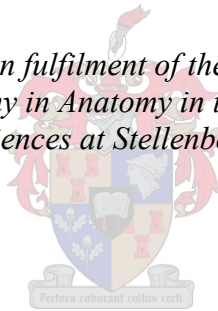


Assessment of health status in a 20th century skeletal collection from the Western Cape

by
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DECLARATION

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ABSTRACT

Studies on the health status of skeletal remains give insight into the standard of living and survival pattern of historic populations. Analysis of trauma and pathological conditions in human skeletal remains are important in biological anthropology, explaining patterns of malnutrition, stress, disease and trauma in a population. However, the difficulty to overcome is the fact that the majority of skeletal pathological conditions are limited in their interpretive significance, since they are nonspecific and a range of stressors can cause the lesions. By analysing multiple conditions within a known population, pathological responses for specific insults can be outlined and in return help in interpretation of the frequencies and distributions within and between populations.

The Kirsten Skeletal Collection, housed at Stellenbosch University, Division of Clinical Anatomy broadly represents individuals from mainly low socio-economic communities of different population groups in the Western Cape, dating throughout the 20th century. The aim of this study was to macroscopically and radiologically examine adult individuals (n=624, nmales=438, nfemales=186) in this collection for skeletal markings that included malnutrition (diet and metabolic deficiencies), osteoarthritic lesions, neoplasms, infective diseases and antemortem trauma lesions to be used as baseline information in further anthropological studies on the people of the Western Cape region. Statistically, the prevalence of specific diseases or trauma were correlated between the sexes, three different age-at-death and population groups as well as three different time periods throughout the 20th century using two-way frequency-tests and correspondence analyses.

During the 20th century, many factors resulted in poverty, and "non white" people, namely the South African Black (SAB) and South African Coloured (SAC) population groups, was especially disadvantaged by the laws introduced by the ruling political party. The influx of people from rural areas during World War II to work in the manufacturing industry resulted in the already overcrowded, unsanitary informal settlements around the Cape Peninsula to be flooded, influencing the disease patterns in the communities.

The results demonstrated that the lowest prevalence of metabolic deficiencies, iron deficiency anaemia (porotic hyperostosis), growth arrest signs (Harris' lines), infections such as tuberculosis, osteomyelitis and non-specific periosteal reactions were observed in the South African White (SAW) population group. This confirms that higher socio economic societies, that escaped the unsanitary conditions associated with poor housing and overcrowding

environments, were more successfully buffering themselves from malnutrition and exposure to pathogens. Better dental health as well as dental fillings were also more associated with the SAW population group that had unrestrained access to dentists and health care facilities. In contrast, the 'non-white' population groups, that were suppressed during the Apartheids regime, demonstrated a high prevalence of malnutrition, metabolic deficiencies, tuberculosis and trauma lesions. The difference between the higher and lower social categories was especially recognised during the late time period when the Apartheid laws of population group segregation among others, started to show results in the 1960 and 1970. Later, during the 1980s and 1990s, political unrest caused by the suppressed majority, and the world due to the Apartheid laws, resulted in sanctions and lower economic opportunities for South Africa. A notable higher frequency of infective markings on bone was observed during the late time period in the study, an indication of the successful use of antibiotics during the last decades of the 20th century, which provided more time for lesions to manifest on bones due to the increased life-span of people.

Studies on skeletal collections rely on the assumption that the remains represent a past community, population group or populations from a specific region and can be used as a valid comparative reference for reconstructing different aspects of skeletal biology of past people that lived in that population. The Kirsten Skeletal Collection represents adult age groups between 18 and 100, three population groups, both sexes, various time periods over the 20th century as well as known cause of death and last known residence. However, this skeletal collection relies on body donations or retention of adult unclaimed or family donated bodies under the statues of the Inspector of Anatomy, and therefore, resulted in a biased sample. This bias is perceived in the fact that the Kirsten Skeletal Collection have an overrepresentation of males, aged individuals and people with lower socio-economic status. Although three major South African population groups are represented, suggesting depiction in population variation, it is highly unequal, especially representing the overrepresented heterogenous mixed population group (SAC) that lived in and around the northern townships of Cape Town. Despite limitations, in general this study of the Kirsten Skeletal Collection may represent many of the traits in the population at that time and may be useful in future studies on honours, masters and PhD level to refine region- and population specific reference data and play a supportive role in research and training of specialists. These data to be collected and interpreted in future studies, include estimation of demographic parameters (age, sex, ancestry origin) as well as human variation, trauma biomechanics and pathological conditions. If the existing predispositions of

the collection are acknowledged and accounted for by the use of suitable methodology, the Kirsten Skeletal Collection holds much potential to become a valuable resource for future research projects in osteology and related fields.

ABSTRAK/OPSOMMING

Studies oor die gesondheidstatus van skeletoorblyfsels gee insig in die lewenstandaarde en oorlewingspatrone van historiese bevolkings. In biologiese antropologie is die ontleding van trauma en patologiese toestande in menslike skelette belangrik, wat weer patrone van wanvoeding, stres, siekte en trauma in 'n bevolking verduidelik. Een struikelblok wat egter oorkom moet word, is dat die meerderheid patologiese toestande in die skelet moeilik is om te interpreteer, aangesien die tekens nie-spesifiek is en die letsels deur 'n verskeidenheid stressors veroorsaak kan word. Deur die ontleding van veelvuldige toestande binne 'n bekende populasie, kan patologiese reaksies vir spesifieke toestande verduidelik word, en dra dit by om die frekwensies en verspreiding daarvan binne en tussen bevolkings te interpreteer.

Die Kirsten Skeletversameling word aan Stellenbosch Universiteit, in die Afdeling Kliniese Anatomie gehuisves, en verteenwoordig individue van verskillende bevolkingsgroepe wat veral uit swak sosio-ekonomiese gemeenskappe in die Wes-Kaap afkomstig is, en uit die 20ste eeu dateer. Die doel van hierdie studie was om die volwasse skelette in die versameling makroskopies en radiologies te ondersoek ($n = 624$, $n_{\text{mans}} = 438$, $n_{\text{vrouens}} = 186$) vir letsels weens wanvoeding (dieet en metaboliese tekorte), osteoartritiese letsels, neoplasmas, infektiewe siektes en antemortem trauma, wat as basislyn inligting gebruik word vir verdere antropologiese studies oor die inwoners van die Wes Kaapse streek. Statistiese analise (frekwensie-toetse) van die resultate is gekorreleer met die voorkoms van spesifieke siektes of trauma tussen geslagte, tussen drie verskillende ouderdoms- en bevolkingsgroepe, asook tussen drie verskillende tydperke wat oor die 20^{ste} eeu strek.

Baie faktore het 'n invloed gehad op armoede gedurende die vroeë 20^{ste} eeu en die 'nie-blanke' mense, naamlik die Suid-Afrikaanse Swart en Suid-Afrikaanse Kleurling groepe wat tydens die Apartheidsregering onderdruk is, was veral benadeel deur wette wat gedurende die 20^{ste} eeu ingestel is. Die instroming van mense vanuit die plattelandse gebiede om in die vervaardigingsbedryf te werk gedurende die Tweede Wêreldoorlog, het daartoe gelei dat die reeds oorvol, onhigiëniese informele nedersettings rondom die Kaapse Skiereiland oorstrom was, en dus die siektepatrone beïnvloed het.

Resultate het getoon dat die Suid-Afrikaanse wit populasiegroep die laagste voorkoms het van toestande soos metaboliese gebreke, ystertekort-anemie (porotiese hiperostose), tekens van groeiversteurings (Harris se lyne), infeksies soos tuberkulose en osteomiëlitis, en nie-spesifieke periosteale reaksies. Dit bevestig dat hoër sosio-ekonomiese groepe, wat uit die onhigiëniese toestande wat verband hou met swak behuising en oorbevolking ontsnap het, hulself meer suksesvol kon buffer teen wanvoeding en blootstelling aan patogene. Beter tandheelkundige gesondheid, sowel as tandheelkundige herstellings was ook meer geassosieer met die Suid-Afrikaanse wit populasiegroep wat onbeperkte toegang tot tandartse en gesondheidsorgfasiliteite gehad het. In teenstelling hiermee, het die 'nie-blanke' bevolkingsgroepe 'n hoër voorkoms van wanvoeding, metaboliese tekorte, tuberkulose en trauma getoon. Die verskil tussen die hoër- en laer sosiale kategorieë het veral na vore gekom in die laat tydperke toe, onder andere, rasse segregasie weens apartheidswette onder meer resultate in die 1960s en 1970s getoon het. Terselfdertyd het politieke onrus, veroorsaak deur die oorweldigende onderdrukte meerderheid en 'n ontevrede wêreld weens die apartheidswette, sanksies en lae ekonomiese geleenthede vir Suid-Afrika tot gevolg gehad. Aan die ander kant is 'n noemenswaardige hoër frekwensie infeksieletsels gedurende die laat tydperk op been waargeneem, wat die gevolg was van die suksesvolle gebruik van antibiotika in die middel tot laat dekades van die 20ste eeu. Die letsels was 'n aanduiding dat die lewensduur van mense verleng is en dat daar dus meer tyd beskikbaar was vir letsels om op die skelet te manifesteer.

Studies oor skeletversamelings maak staat op die veronderstelling dat die oorblyfsels 'n vorige gemeenskap, bevolkingsgroep of spesifieke streek verteenwoordig, en kan gebruik word as 'n geldige vergelykende verwysing vir die rekonstruksie van verskillende aspekte van skeletbiologie van persone wat voorheen in daardie populasie woonagtig was. Die Kirsten Skeletversameling verteenwoordig volwasse ouderdomsgroepe tussen 18 en 100, drie bevolkingsgroepe, beide geslagte, verskillende tydperke oor die 20^{ste} eeu, asook bekende oorsaak van dood en die laaste beskikbare woonadres. Innames steun egter baie op liggaamskenkings, of die inname van volwasse onopgeëide liggame en skenking van liggame deur familie, onder die beheer van die Inspekteur van Anatomie, en kan dus lei tot 'n ongelyke inname tussen populasiegroepe. Hierdie ongelykheid in inname word ook in die Kirsten Skeletversameling waargeneem, ten opsigte van die feit dat die versameling geen kinders bevat nie, en dat daar 'n oorverteening van manlike persone, bejaardes en persone vanaf 'n laer sosio-ekonomiese omgewing is. Alhoewel drie groot Suid-Afrikaanse bevolkingsgroepe verteenwoordig word, wat wel bevolkingsvariasie verteenwoordig, is die getalle baie ongelyk,

en dit verteenwoordig meestal die heterogene gemengde bevolkingsgroep (Kleurlinggroep) wat in en om die noordelike lae-behuisingswoonbuurte van Kaapstad gewoon het. Ons erken dat aansienlike beperkings bestaan, maar die algehele voorstelling van die Kirsten Skeletversameling spreek baie van die eienskappe aan en kan dus in toekomstige studies op honneurs-, meesters- en PhD-vlak gebruik word om streek- en populasie spesifieke verwysingsdata te verfyn en 'n ondersteunende rol in navorsing en opleiding van spesialiste te speel. Data wat ingesamel en geïnterpreteer kan word, sluit in die bepaling van demografiese parameters (ouderdom, geslag, herkoms), sowel as menslike variasie, trauma, biomeganika en patologiese toestande.

Indien die bestaande ongelykhede van die versameling in ag geneem word en deur erkende metodiek gekorrigeer word, toon die Kirsten Skeletversameling die potensiaal om 'n waardevolle bron van genoegsame navorsingsprojekte vir toekomstige navorsings in osteologie en relevante velde te bied.

RESEARCH OUTPUTS

PUBLICATIONS AND PRESENTATIONS BASED ON THIS WORK

Articles presented and published:

- Geldenhuys E, Burger EH, Alblas A, Greyling LM, Kotzé SH. 2016. The association between healed skeletal fractures indicative of interpersonal violence and alcoholic liver disease in a cadaver cohort from the Western Cape, South Africa. *Alcohol* 52: 41-48. Doi:10.1016/j.alcohol.2016.02.003. Oral presented at the 44th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State.
- Alblas A, Greyling LM, Geldenhuys E. 2018. Composition of the Kirsten Collection of Human Skeletal Material at Stellenbosch University, South Africa. *South African Journal of Science* 114(1/2). Doi: 10.17159/sajs.2018/20170198. Poster presented at the 44th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State.

National conference presentations:

- Alblas A, Greyling LM, Bastiaanse, BL. 2013. The prevalence of lumbosacral transitional vertebrae (LSTV) in the Kirsten Skeletal Collection at Stellenbosch University. Poster presented at the 41st Annual Conference of the Anatomical Society of Southern Africa (ASSA), 20-24 April 2013, University of KwaZulu Natal, Durban and Abstract published in *Clinical Anatomy* 26(7):911–917.
- Alblas A, Coetzee N, Greyling LM. 2014. The prevalence of interpersonal violence-related post-cranial skeletal trauma in a sample of a Western Cape population. Poster presented at the 42nd Annual Conference of the Anatomical Society of Southern Africa (ASSA), 13-16 April 2014, Stias Conference Centre, Stellenbosch and abstract published in *Clinical Anatomy* 2015:28(2):399–412. Doi:10.1002/ca.22493. Further presentation and discussion at the 1st South African Homicide Research Colloquium Hosted by the Safety and Violence Initiative, University of Cape Town 3-4 September 2015. www.savi2015.co.za. The River Club, Observatory, Cape Town.
- Alblas A, Greyling LM. 2016. Trauma on bones, a case study in the Kirsten Skeletal Collection. Poster presented at the 44th Annual Conference of the Anatomical Society

of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State. Abstract published in *Clinical Anatomy* (2018) 31(8):E11-24. doi.org/10.1002/ca.23046.

- Walters J, Alblas A, Greyling LM. 2016. Congenital malformations in the Kirsten Skeletal Collection. Oral presented at the 44th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State. Abstract published in *Clinical Anatomy* (2018) 31(8):E11-24. Doi.org/10.1002/ca.23046.
- Geldenhuys E, Burger EH, Alblas A, Greyling LM, Kotzé SH. 2016. Prevalence of Spinal Pathology in Embalmed Cadavers used for Medical Dissection at Stellenbosch University, South Africa. Poster presented at the 44th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State. Abstract published in *Clinical Anatomy* (2018) 31(8):E11-24. Doi.org/10.1002/ca.23046.
- Marais JC, Alblas A, Greyling LM. 2016. Dental pathology and the association with co-morbid factors (poor oral hygiene) and systemic disorders. Oral presented at the 44th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 08-11 May 2016, Bloemfontein, Free State. Abstract published in *Clinical Anatomy* (2018) 31(8):E11-24. Doi.org/10.1002/ca.23046.
- Walters J, Alblas A, Greyling LM. 2017. Neoplasms in the Kirsten Skeletal Collection. Poster presented at the 45th Annual Conference of the Anatomical Society of Southern Africa (ASSA), 23-26 April 2017, Club Mykonos, Langebaan. Abstract published in *Clinical Anatomy* (2018) 31(8):E25-E39. Doi.org/10.1002/ca.23050.

WORKSHOPS AND SHORT COURSES COMPLETED RELEVANT TO THIS STUDY

- Attended a workshop on Interpretation of Bone Trauma and Pseudo-trauma in suspected violent deaths between 27 – 31 August 2012, hosted by the Forensic Anthropology Research Centre, School of Medicine, Faculty of Health Sciences, University of Pretoria. The course was presented by Prof. Steve Symes, PhD, D-ABFA, from the Department of Applied Forensic Sciences, Mercyhurst University in Erie, Pennsylvania, USA and Dr. João Pinheiro, MD, MSc from Forensic Medicine and Pathology at the Instituto Nacional de Medicina Legal Coimbra Portugal.

- Attended a short course in Death Scene Archaeology; to locate, recover and interpret human remains from grave sites to ensure that taphonomic trauma effects that may be present on the remains can be accurately interpreted. It was hosted by the Department of Applied Forensic Sciences at Mercyhurst University Erie, Pennsylvania, USA between 3-7 June 2013.
- Attended a short course in Laboratory Methods in the Identification of Human Skeletal Remains. Focus was on the determination of personal identification through assessment of chronological age, sex, ancestry, stature, and pathology. It was hosted by the Department of Applied Forensic Sciences at Mercyhurst University Erie, Pennsylvania, USA between 10-14 June 2013.
- Attended a Bone Pathology Symposium, presented by Prof George Maat from the Netherlands and Prof Niels Lynnerup from Denmark. It was hosted by the Department of Anatomy School of Medicine, Faculty of Health Sciences, University of Pretoria between 2-15 May 2014.
- Attended a short course in the Application of Advanced Statistical Methods for Improving the Biological Profile in Age and Ancestry Estimation in South Africa. It was hosted by the Forensic Anthropology Research Centre, Department of Anatomy School of Medicine, Faculty of Health Sciences, University of Pretoria between 27-31 July 2015. Presenters included Proff Jasper Boldsen, George Milner, Stephen Ousley as well as drs Kyra Stulls and Michael Kenyherscz.
- Attended a two day FASE advanced workshop in Milan Italy on Anthropology and Migration. 14-16 September 2017.

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CONTENTS

DECLARATION	ii
ABSTRACT	iii
ABSTRAK/OPSOMMING	v
RESEARCH OUTPUTS	viii
PUBLICATIONS AND PRESENTATIONS BASED ON THIS WORK	viii
Articles presented and published:	viii
National conference presentations:	viii
WORKSHOPS AND SHORT COURSES COMPLETED RELEVANT TO THIS STUDY	ix
ACKNOWLEDGEMENTS	xi
CONTENTS	xiii
LIST OF TABLES	xvii
LIST OF FIGURES	xviii
ABBREVIATION LIST	xxiii
CHAPTER 1: INTRODUCTION	1
1.1 BACKGROUND	2
1.2 RESEARCH PROBLEMS	3
1.2.1 Significance of project	3
1.3 HYPOTHESES, AIMS AND OBJECTIVES OF THE STUDY	4
1.3.1 Hypothesis.....	4
1.3.2 Pathology assessment: aim and objectives	4
1.3.3 Trauma assessment: aims and objectives	5
1.4 OUTLINE OF THE STUDY	5
CHAPTER 2: LITERATURE REVIEW	7
2.1 PROFILE OF THE STUDY LOCATION (WESTERN CAPE PROVINCE)	8
2.1.1 The origin of the Western Cape population.....	9
2.1.2 The effect of Apartheid on the Composition of the Western Cape population.....	12
2.2 FACTORS INFLUENCING POPULATION HEALTH IN THE WESTERN CAPE	13
2.2.1 Economic growth	13
2.2.2 Housing	13
2.2.3 Health care access	15
2.2.4 Education.....	16
2.2.5 Substance abuse.....	16
2.2.6 Life expectancy and socio-economic factors.....	18
2.3 PROFILE OF MORBIDITY AND MORTALITY	19
2.3.1 Cause of death and disease patterns of the population during the 20 th century	19
2.3.2 Cause of death as represented in the Kirsten Skeletal Collection.....	21
2.4 OVERVIEW OF POSSIBLE PATHOLOGICAL BONE LESIONS STUDIED	21
2.4.1 Congenital/genetic origin	22
2.4.1.1 Klippel-Feil syndrome (KFS)	22
2.4.1.2 Cleft neural arches/spina bifida occulta	23

2.4.1.3 Lumbosacral transitional vertebrae (LSTV)	24
2.4.2 Infectious diseases.....	26
2.4.2.1 Specific bone infections.....	26
2.4.2.2 Non-specific bone infections	32
2.4.3 Deficiency diseases	35
2.4.3.1 Metabolic and nutritional stress disorders.....	35
2.4.3.2 Haematological disorders.....	44
2.4.4 Degenerative diseases and arthropathies.....	47
2.4.4.1 Osteoarthritis (OA)	49
2.4.4.2 Arthropathies.....	53
2.4.5 Neoplastic diseases.....	55
2.4.5.1 Primary bone tumours.....	55
2.4.5.2 Secondary bone tumours and metastatic skeletal lesions	58
2.4.6 Other diseases/conditions	59
2.4.6.1 Hyperostosis frontalis interna (HFI)	59
2.4.6.2 Paget's disease	60
2.4.6.3 Hypertrophic osteoarthropathy (HOA)	61
2.5 PROFILE OF TRAUMA IN THE WESTERN CAPE POPULATION	62
2.5.1 Evidence of trauma.....	62
2.5.2 Age, sex and population group.....	64
2.5.3 Timing or mechanism of trauma on skeletal material	65
2.5.4 Trauma in the Kirsten Skeletal Collection	65
2.5.5 Fracture sites on bones	66
2.5.5.1 Cranial bones involved.....	66
2.5.5.2 Postcranial bones involved.....	67
CHAPTER 3: MATERIALS AND METHODS	71
3.1 MATERIAL	72
3.1.1 The Kirsten Skeletal Collection	72
3.1.2 The study sample: Kirsten Skeletal Collection.....	73
3.1.3 Ethical consideration	75
3.1.4 Limitations of the study.....	76
3.2 METHODS.....	77
3.2.1 Biological profile.....	77
3.2.1.1 Estimation of sex.....	77
3.2.1.2 Estimation of chronological age:	78
3.2.2 Macroscopic observation.....	78
3.2.2.1 Cranial scoring	79
3.2.2.3 Postcranial scoring	79
3.2.3 Radiological observations	80
3.3 PATHOLOGY AND TRAUMA ANALYSIS.....	81
3.3.1 Congenital/Genetic origin	82
3.3.1.1 Klippel-Feil syndrome (KFS)	82

3.3.1.2 Cleft neural arches/spina bifida occulta (SBO).....	82
3.3.1.3 Lumbosacral transitional vertebrae (LSTV)	83
3.3.2 Infectious disease markings.....	84
3.3.2.1 Specific bone infections	84
3.3.2.2 Non-specific bone infections	88
3.3.3 Deficiency diseases	90
3.3.3.1 Metabolic and nutritional stress disorders.....	90
3.3.3.2 Haematological disorders.....	92
3.3.4 Degenerative joint disease markings	93
3.3.4.1 Degenerative peripheral joint osteoarthritic (pOA) markings	93
3.3.4.2 Vertebral column degenerative markings	94
3.3.4.3 Specific DJD markings on vertebrae.....	97
3.3.4.4 Specific degenerative arthropathies	98
3.3.5 Bone reaction and Neoplasms	99
3.3.5.1 Primary and secondary bone tumours	99
3.3.6 Other bone markings	103
3.3.6.1 Hyperostosis frontalis interna (HFI)	103
3.3.6.2 Paget's disease	103
3.3.6.3 Hypertrophic osteoarthropathy (HOA)	104
3.3.7 Trauma assessment.....	105
3.3.7.1 Cranial scoring	105
3.3.7.2 Nasal trauma scoring.....	107
3.3.7.3 Postcranial trauma scoring	108
3.4 OBSERVER ERROR AND STATISTICAL ANALYSIS.....	111
CHAPTER 4: RESULTS	112
4.1 PATHOLOGICAL LESIONS OBSERVED ON BONE	113
4.1.1 Congenital/genetic origin	113
4.1.1.1 Klippel-Feil syndrome (KFS)	113
4.1.1.2 Cleft neural arches/spina bifida occulta	115
4.1.1.3 Lumbosacral transitional vertebrae (LSTV)	117
4.1.2 Infectious diseases.....	118
4.1.2.1 Specific bone infections	118
4.1.2.2 Non-specific bone infections	122
4.1.3 Deficiency diseases	125
4.1.3.1 Metabolic and nutritional stress disorders.....	126
4.1.3.2 Haematological disorders.....	129
4.1.4 Degenerative joint diseases and arthropathies.....	131
4.1.4.1 Osteoarthritis: Degenerative peripheral joint osteoarthritic (pOA) markings	131
4.1.4.2 Osteoarthritis: Vertebral joint osteoarthritic (vOA) markings	133
4.1.4.3 Osteoarthritis: Vertebral osteophytosis (VO).....	135
4.1.4.4 Arthropathies.....	136
4.1.5 Neoplastic diseases	138

4.1.5.1 Malignant primary neoplasms.....	138
4.1.5.2 Benign primary neoplasms.....	139
4.1.5.3 Cancer-related cause of death.....	140
4.1.5.4 Metastatic skeletal lesions.....	143
4.1.6 Other diseases/conditions.....	146
4.1.6.1 Hyperostosis frontalis interna (HFI).....	146
4.1.6.2 Paget's disease.....	146
4.1.6.3 Hypertrophic osteoarthropathy (HOA).....	148
4.2 CORRESPONDENCE ANALYSES.....	148
4.3 TRAUMA MARKINGS ON BONE.....	153
4.3.1 Cranial trauma.....	153
4.3.2 Post-cranial trauma.....	155
4.3.2.1 General post-cranial lesions.....	155
4.3.2.2 Rib fractures.....	159
4.3.3 TOTAL trauma.....	161
CHAPTER 5: DISCUSSION.....	181
5.1 CONGENITAL/ GENETIC INFLUENCES.....	182
5.2 PATHOLOGICAL LESIONS.....	184
5.2.1 Sex.....	184
5.2.2 Age.....	186
5.2.3 Population groups.....	188
5.2.4 Time periods.....	190
5.3 TRAUMA MARKINGS.....	192
5.4 CONCLUSION.....	196
5.4.1 Congenital anomalies during the 20 th century.....	196
5.4.2 Pathological lesions during the 20 th century.....	197
5.4.2.1 Early-time period.....	197
5.4.2.2 Mid-time period.....	198
5.4.2.3 Late-time period.....	198
5.4.3 Traumatic lesions during the 20 th century.....	199
5.4.4 Relevance of Skeletal Collections to research.....	199
CHAPTER 6: REFERENCE LIST.....	181
CHAPTER 7: APPENDIX.....	229

LIST OF TABLES

Table 3.1. Population group, sex and age composition of the cohort.....	75
Table 3.2. The mean age at death for each sex and population group per age group (in years-of-age).....	75
Table 3.3. Classification of lumbosacral transitional vertebrae after Castellvi et al. (1984)...	83
Table 4.1. Statistical analyses of Klippel-Feil Syndrome in the cohort (n=13).....	113
Table 4.2. Statistical analyses of cleft neural arches of vertebrae in the cohort (n=57).....	116
Table 4.3. Statistical analyses of lumbosacral transitional vertebrae (LSTV) in the cohort (n=83).....	117
Table 4.4. Summary of the statistical analyses for possible tuberculous infections.....	118
Table 4.5. Summary of the statistical analysis for possible cranial infections.	120
Table 4.6. Statistical analyses of signs of poor dental health in the KSC cohort (n=269). ...	122
Table 4.7. Statistical analyses of signs of dental work in the KSC cohort (n=16).	122
Table 4.8. Statistical analyses of non-specific reactions on long bones in the cohort.	123
Table 4.9. Statistical analyses of deficiency diseases in the cohort.....	125
Table 4.10. Statistical analyses of deficiency diseases in the cohort.....	129
Table 4.11. Statistical analyses of peripheral joint osteoarthritis (n=399).	131
Table 4.12. Statistical analyses of vertebral joint osteoarthritis (n=511).	134
Table 4.13. Statistical analyses of vertebral fusion and spondylolysis.....	135
Table 4.14. Statistical analyses of metastatic lesions on individuals in the KSC cohort.....	143
Table 4.15. Statistical analyses of HFI and HOA lesions on individuals in the KSC cohort.	146
Table 4.16. Frequency (n) of antemortem cranial trauma per bone, side and type of trauma.	153
Table 4.17. Statistical analyses of trauma lesions on cranial elements in the KSC cohort. ...	154
Table 4.18. Trauma per bone (%) in different age groups, population groups, eras and sexes.	156
Table 4.19. Trauma per post-cranial bone (%) compared between sexes and among population groups.....	157
Table 4.20. Trauma complication (n) per post-cranial bone.....	159

LIST OF FIGURES

Figures 2.1A-B. A) The four Provinces of the Union of South Africa and later the Republic of South Africa prior to Democracy (1910 to 1994); and B) the nine provinces of SA, showing the much smaller WCP the south-western province in South Africa (1994 to present).	9
Figure 2.2. The average life expectancy for the four population groups in South Africa during selected years.	18
Figures 2.3A-C. Tuberculosis are seen as pulmonary or extra-pulmonary infections A) PTB as periosteal reaction on the visceral surface of ribs, or B) Pott's disease on the vertebral column; or C) manifest on postcranial bones (right radius that fused with the scaphoid).....	28
Figure 2.4. Suppurative complications in otitis media infections.....	31
Figure 2.5. Diagram showing location of dental abscesses.	32
Figures 2.6A-D. Signs of rickets observed on dry bone includes A) bowing of long bones; B) rachitic rosary (beading) at the costo-chondral junctions; C) wrist widening; and D) triradiate configuration of the pelvis.	40
Figure 2.7. A young patient with signs of rickets such as rachitic rosary (beading) at the costo-chondral junctions (block arrows) and the horizontal Harrison's sulcus (line arrows).	41
Figures 2.8A-D. Signs of osteomalacia observed on dry bone of adults include A) Bowing of the long bones; B) pectus carinatum of the sternum (arrow); C) protruding or bossing of the frontal bone (arrow); and D) coxa vara (arrow), compared to a normal femur head on the right side.	42
Figure 2.9A-D. Signs of scurvy on dry bone is evident as marked porosity and woven bone activity on A) the endocranial aspect of the petrous bone; B) the lateral aspect of the greater sphenoid wing; C) the outer mandible; and D) gross new bone formation on shafts of the ulnae.	44
Figure 2.10. Posterolateral view of the ribs and vertebral articulation.....	51
Figure 3.1. Individuals were laid out on a table in anatomical order for macroscopic analysis.	79
Figure 3.2. Cranial bones scored in this study.	79
Figure 3.3. Skeletal diagram showing peripheral joints scored for degenerative lesions.....	80
Figure 3.4. Individuals were laid out on the Lodox [®] Statscan [®] table in anatomical order for digital X-rays.	81
Figures 3.5A-B. Ankylosed C2-C3 cervical vertebrae, indicating Klippel-Feil syndrome on A) dry bone and B) Lodox [®] image.....	82
Figures 3.6A-D. Cleft neural arch in the A) atlas; B) lower thoracic vertebra; C) SBO on S1, but high hiatus on the inferior aspect of the canal; and D) complete SBO of all the sacral vertebrae.....	83
Figure 3.7. Anterior view of sacra showing the types of lumbosacral transitional vertebrae (LSTV).....	84
Figures 3.8A-D. Localised infections A) concha bullosa; B) otitis media; C) ossified spicules (arrow) in the external acoustic meatus; D) petrositis on inner petrous part of the temporal bone.....	86

Figures 3.9A-F. Dental disease and modification. A) dental work (block arrow), also note the cervical carie (arrow); B) high calculus; C) prominent dental caries; D) stains on teeth; E) periodontal abscess; and F) periapical cyst.....	88
Figures 3.10A-C. A) Striated; B) nodular; and C) severe irregular periosteal reaction.	89
Figures 3.11A-B. Osteomyelitis of a long bone on A) on dry bone with sequestrum, (line arrow) and involucrum, (block arrow); B) a cloaca (arrow) for pus draining	89
Figures 3.12. Harris' lines across the diaphysis of the tibia (arrow).	90
Figures 3.13A -B. Examples of A) linear horizontal grooves of the tooth enamel; and B) pits and lines in the tooth crown of archaeological specimens.....	91
Figures 3.14A-B. Signs of anaemia seen as: A) cribra orbitalia B) porotic hyperostosis (arrows).....	93
Figures 3.15A-B. Eburnation as A) smooth polished lesions; and B) smooth polished lesions with grooves.....	94
Figures 3.16A-C. Osteophytes on the peripheral joints: A) lipping with round ridges (mushrooming); B) lipping with sharp ridges (spiculed); and C) a combination of mushrooming and sharp lipping.....	94
Figures 3.17A-B. Markings on the anterior bodies of the vertebrae: A) Inferior and/or superior lipping on the marginal surface of the anterior body; B) kissing osteophytes (or curved spicules) between two adjacent vertebrae.	95
Figures 3.18A-D. Markings on facet joints of the posterior arch (vOA) of the vertebrae: A) laminal spurs (arrow); B) spurs at the zygapophyseal articulation joints; C) spurs on the costotransverse joints (line arrow) and the costovertebral joint (block arrow); and D) VO on the fibro-cartilaginous joint on the vertebral body.	96
Figures 3.19A-D. Types of vertebral compression fractures: A) crushed vertebral body B) anterior wedging; C) posterior wedging; D) lateral wedging.	97
Figures 3.20A-F. Ankylosing spondylitis as: A) a 'bamboo spine'; or B) with anterior fusion causing kyphosis. Diffuse idiopathic skeletal hyperostosis (DISH) on C) dry bone; and D) X-ray image; and spondylolysis on E) dry bone; and F) X-ray image.	98
Figures 3.21A-B. Reactive arthritis (ReA) seen on: A) the thoracic vertebral column with "skip lesions" (arrows); and B) X-ray showing a whisker sign (small arrow heads) and osteophytes in the acetabular rim (large arrow head).	99
Figures 3.22A-C. Difference between porosity and erosion were observed as: A) macroporosity or porous markings; B) microporosity or pitting; and C) erosion.	100
Figures 3.23A-D. A) Large lytic metastatic lesion on calvarium without defined margin; B) lytic lesion on rib with well-defined margins (arrow) and some sclerotic activity within lesion. C) Dry manubrium with multiple punched-out lytic lesions; D) also visible on the Lodox [®] scan.	101
Figures 3.24A-B. Sclerotic response as A) unorganised woven bone, or B) well-organised sclerosis of mature bone.....	102
Figures 3.25A-B. Surface osteosarcomas as: A) parosteal; and B) periosteal varieties visible on X-ray images of the distal femora.....	102

Figures 3.26A-C. Presence of HFI on endocranial surface of the frontal bones with A) low; B) medium; and C) high sclerotic activity.	103
Figures 3.27A-D. Signs of Paget's disease on A) os coxa showing the exposed trabecular bone below damaged cortical bone B) scapula with layered woven bone deposited on cortical bone C) X-ray image of cross-sectioned vault; and D) dry bone showing layered expanded diploe.	104
Figures 3.28A-B. Severe HOA showing sclerotic activity on A) distal tibia and fibula; and B) os coxae.	105
Figures 3.29A-D. Types of fractures on the skull as A) pathological fracture; B) blunt force trauma (BFT); C) healed sharp force trauma (SFT); and D) other (surgical intervention). ..	106
Figures 3.30A-C. Trauma complications on the skulls was regarded as A) an open fracture with a missing part; B) infection with a periosteal response; and C) deformation of the healed fracture.	107
Figures 3.31A-D. Nasal bones with: A) no trauma; B) antemortem fracture with remodelling; C) post mortem damage; and D) antemortem fracture with further post mortem damaged..	108
Figures 3.32A-C. Type of fractures in post-cranial elements: A) blunt force trauma (BFT) B) healed sharp force trauma (SFT); and C) pathological fracture on weakened bone.	109
Figures 3.33A-F. Trauma complication A) fusion of two bony elements after remodelling; B) periosteal response after trauma; C) non-union of two bone elements, D) dislocation, traumatic arthritis or myositis ossificans; E) deformation or misalignment of bone; and F) surgical intervention at a fracture site.	110
Figure 4.1. Distribution and frequency of other disorders associated with Klippel-Feil syndrome in this study.	114
Figures 4.2A-D. Anomalies associated with Klippel-Feil Syndrome A) misshapen and fused ribs; B) fused ribs 1 and 2; C) cleft neural arches of T3 (top) and C1 (bottom); and D) several (C2-C7) fused vertebrae.	114
Figure 4.3. Distribution of Klippel-Feil syndrome per cervical level in the cohort. Note that some individuals have more than one group of fused vertebrae.	115
Figures 4.4A-C. Examples of the cases that presented with A) full spina bifida occulta (SBO); B) most of the spinous processes of the sacral vertebrae open; and C) cleft transverse spines.	115
Figure 4.5. Distribution and frequency (%) of vertebrae with a cleft neural arch.	116
Figure 4.6. Frequency (%) of LSTV classes, using classification criteria of Castellvi et al. (1984).	117
Figures 4.7A-B. Examples of A) sacralisation IIIb and B) lumbarisation Ib. Note the difference in the number of sacral segments.	118
Figure 4.8. Distribution of extra-pulmonary TB in the cohort.	119
Figure 4.9. Total number of maxillae and mandibles in the KSC cohort present.	121
Figure 4.10. Type of periosteal reaction on long bones in the cohort (%).	123
Figure 4.11. Percentage of bone elements or bone combinations with periosteal reaction in the cohort.	124

Figures 4.12A-C. Osteomyelitis on A) proximal end of the right fibula and distal shaft of the left tibia, showing B) the Lodox [®] image and C) the dry bone.....	125
Figure 4.13. Distribution of Harris' lines per bone in the cohort (%).....	126
Figures 4.14A-B. Examples of A) pectus carinatum ("pigeon breast"); and B) pectus excavatum ("funnel breast") in the KSC cohort.	127
Figures 4.15A-B. Example of articulated bowed tibia and fibula A) on dry bone; and B) on Lodox [®] image of the same individual.....	128
Figures 4.16A-B. Examples scurvy lesions on the A) sphenoid wings of the cranium; and B) long bone lesion showing woven bone deposits on the shaft.	129
Figure 4.17. Distribution frequency (%) of OA on peripheral joints.....	132
Figure 4.18. Distribution frequency (%) of eburnation at peripheral joints.	133
Figure 4.19. Distribution frequency (%) of dislocation of peripheral joints.	133
Figures 4.20A-B. Examples of dislocated joints A) shoulder and B) hip.....	133
Figure 4.21. Vertebral levels with spondylolysis in the cohort (%).....	135
Figure 4.22. Vertebral levels involved in fusion in DISH.	136
Figure 4.23. The number and distribution of the fused vertebrae in the KSC cohort.....	137
Figures 4.24A-C. Reactive arthritis differential diagnostic traits, including A) vertebral ligament ossification, B) unilateral OA and eburnation on the knee; and C) bilateral ossification of the SIJ.	138
Figures 4.25A-B. Parosteal surface osteosarcoma with a large sclerotic deposit on the distal end of the left femur A) posterior view B) Lodox [®] scan A-P view.....	139
Figures 4.26A-B. Lymphosarcoma with: A) fine spiculed osteoblastic activity on the ileum of os coxa; or B) woven bone depositing on the scapula (encircled).....	139
Figures 4.27A-B. Enchondroma in the proximal shaft of the left femur seen on the A) Lodox [®] scan; and B) shows expansion of the femur shaft on the dry bone (arrows).	140
Figure 4.28. Percentage of specific cancers indicated on the death certificates of the individuals in the cohort (%).	140
Figure 4.29. Distribution of the type of registered cancers per sex in the cohort (%).	141
Figure 4.30. Distribution of the type of registered cancers per age group in the cohort (%).	142
Figure 4.31. Distribution of the type of registered cancers as COD per population group in the cohort.	142
Figure 4.32. Distribution of the type of registered cancers per time period in the cohort (%).	143
Figure 4.33. Lesions observed on various bone elements in the cohort (%).	144
Figure 4.34. Type of lesions observed per sex in the cohort (%).	144
Figure 4.35. Type of lesions observed per bone element compared to COD reported on the death certificates.	145
Figures 4.36A-F. Signs of Paget's disease on dry bone: A) hyperostosis of calvarium; B) Lodox [®] image; C) disorganised, densely woven bone; D) Lodox [®] image; E) 'cotton wool'	

effect on a radiological image; and F) disorganised woven bone deposits on a radiological image.	147
Figure 4.37. The number of skeletal elements with signs of HOA in this cohort.	148
Figure 4.38. Correspondence analysis of sex (male and female) correlated to broadly categorised pathology groups.	149
Figure 4.39. Correspondence analysis of age groups (young adult, mid-adult and old adult) correlated to broadly categorised pathology groups.	150
Figure 4.40. Correspondence analysis of population groups (SAB, SAC and SAW) correlated to broadly categorised pathology groups.	151
Figure 4.41. Correspondence analysis of 20 th century time periods (Early, Mid and Late) correlated to broadly categorised pathology groups.	152
Figure 4.42. Prevalence (%) of post-cranial trauma observed per bone in KSC cohort.....	155
Figure 4.43. Frequency (%) of post-cranial trauma observed per side, sex and bone in KSC cohort.	158
Figure 4.44. Percentage post-cranial trauma observed per long bone section.....	158
Figure 4.45. The number of ribs present in the cohort.....	160
Figure 4.46. The number of ribs present and the number of healed fractures on the ribs.	160
Figure 4.47. Percentage of healed trauma per cranial and post-cranial bones analysed.....	162
Figure 4.48. Percentage of healed trauma per sex.	163
Figure 4.49. Percentage of healed trauma per age group.....	164
Figure 4.50. Percentage of healed trauma per population group.	165
Figure 4.51. Percentage of healed trauma per time period in the 20 th century.	166

ABBREVIATION LIST

°	degree	HIV	human immunodeficiency virus
%	percentage	HL	Harris' lines
®	registered sign	HOA	hypertrophic osteoarthropathy
A	mid-adult age group	HPOA	hypertrophic pulmonary osteoarthropathy
AIDS	acquired immunodeficiency syndrome	IBD	inflammatory bowel disease
A-P	antero-posterior view	IPV	interpersonal violence
AS	ankylosing spondylitis	KFS	Klippel-Feil Syndrome
BFT	blunt force trauma	KSC	Kirsten skeletal collection
BMI	body mass index	L	lumbar
BO	button osteoma	LC	late 20 th century
C	cervical	LEH	linear enamel hypoplasia
CA	cardiac arrest	LSTV	lumbosacral transitional vertebrae
Ca	cancer	M	males
CE	common era	MA	medial aspect of the ischio-pubic ramus
CDH	dislocation of the hip	MC	mid 20 th century
CPA	cardiopulmonary arrest/failure	MCP	metacarpophalangeal joints
CVA	cerebrovascular accident	MDR	multi drug resistant <i>Mycobacterium</i> -strains
CO	cribra orbitalia	MI	myocardial infarction
COD	cause of death	Mm	millimeter
COPD	chronic obstructive pulmonary disease	MRI	magnetic resonance imaging
CT	computer topography	MTP	metatarsophalangeal
DEH	dental enamel hypoplasia	MVA	motor vehicle accident
DIP	distal interphalangeal	NTDs	neural tube defects
DISH	diffuse idiopathic skeletal hyperostosis	O	old adult age group
dist	distal	OA	osteoarthritis
DJD	degenerative joint disease	PH	porotic hyperostosis
DM	diabetes mellitus	PIP	proximal interphalangeal
DOB	date of birth	pOA	peripheral joint osteoarthritis
EC	early 20 th century	prox	proximal
F	females	PsA	psoriatic arthritis
FAS	Foetal alcohol syndrome	PTB	pulmonary tuberculosis
FMHS	Faculty of Medicine & Health Sciences	PVA	pedestrian vehicle accident
HFI	hyperostosis frontalis interna		

R	right	SIJ	sacroiliac joint
RA	rheumatoid arthritis	SPC	subpubic concavity
ReA	reactive arthritis	T	thoracic
RS	Reiter's syndrome	TB	tuberculosis
SA	South Africa	TMJ	temperomandibular joint
SAB	South African Black	VA	ventral arc
SAC	South African Coloured	VCF	vertebral compression fractures
SAI	South African Indian	vert	vertebra
SAW	South African White	VO	vertebral osteophytosis
SBC	spina bifida cystica or aperta	vOA	vertebral osteoarthritis
SBO	spina bifida occulta	WCP	Western Cape Province
SD	standard deviation	WWII	World War II
SES	socio-economic status	XDR	drug resistant <i>Mycobacterium</i> -strains
SFT	sharp force trauma		

CHAPTER 1: INTRODUCTION

*A skeleton is a human being in its most naked form. A life stripped down to its essence.
Chip Cowell Oct 2017*

1.1 BACKGROUND

The human skeleton offers a wealth of information related to different aspects of the person's life including development, individual variation in sex, age-at-death, genetic influences, population history, environmental influences, disease patterns, as well as health and activity levels (White et al., 2012; İşcan & Steyn, 2013). Identification of unknown people from a specific population can be aided by using inherited skeletal features (e.g. congenital anomalies) and the predisposition of diseases (e.g. diabetes or anaemias). Similarly, nutritional assessments on bone may be used to explain living conditions, population relationships, the history of disease patterns, nutrition as well as the impact of cultural and political change on health (Buikstra & Ubelaker, 1994).

Anthropologists are making use of various worldwide reference skeletal collections (see list at <https://skeletal.highfantastical.com>) available to address a number of research questions in archaeological, as well as forensic context (Albanese, 2003). Unfortunately, skeletal research is often limited by the lack of adequately large, modern skeletal collections with known demographic information. Lately, skeletal collections worldwide tend not to add new material due to funding restrictions as well as repatriation of sensitive remains (Endere, 2002. Kakaliouras, 2012). The number of cadavers received at anatomy schools, which is a source from cadaver donation and therefore skeletal collection, are declining as a number of these schools have opted for digital software programmes or prosections for teaching anatomy (Komar & Grivas, 2008; Gregory et al., 2009). No new material is therefore available to add to skeletal collections, or to start new collections in many countries. Another restriction for not sustaining large skeletal collections is that there is substantial funding needed for processing, cataloguing, storage, and curating of skeletal remains and supporting documentary information (Albanese, 2003). Moreover, established reference collections such as the Terry Collection or the Hamman-Todd Collection in USA, as well as cemetery collections in Europe, have been described as not representative of the specific population in the various countries because of the source of the collections. For example, cadaver donations for anatomy teaching are biased to a over-representation of males, the elderly and individuals of low socio-economic standings (Hunt & Albanese, 2005; L'Abbé et al., 2005; Komar & Grivas, 2008; Dayal et al., 2009). Furthermore, older archaeological collections may no longer be useful for development of forensic identification methods due to the source of the skeletons and the age of the collections. The majority of the collections have individuals who were born before or during the 19th century

and the collections may not reflect some of the major secular changes in the 20th and 21st centuries (Albanese, 2003).

1.2 RESEARCH PROBLEMS

Not many large skeletal collections are available to assess the health of a population during a specific time period (Usher, 2002). The theoretical framework for this research is based on a broad, descriptive review of the historical, political, economical and demographic profile of the Kirsten Skeletal Collection (KSC) contributing to a better understanding of adaptation and regional variability of a Western Cape population (Larsen, 2001; Lambert, 2002). Factors that may influence susceptibility of a population to disease are: sex, ancestry, age-at-death, genetics, occupation, nutrition, socio-economic status (SES), religious and social customs, immunisation and disease history. Therefore, research of the health assessment of a population gives insight into the social complexity and hierarchy, presenting disparity between different SES groups, with lower SES groups undergoing a decline in their health, and higher SES groups buffering themselves more successfully from malnutrition, exposure to pathogens and the unsanitary conditions often associated with urbanisation and settlement density. If the health history and historic context of this known collection is identified, it will be available for researchers as a reference tool when developing research projects such as expanding biological profile techniques in age-at-death, ancestry and sex estimation, development of new regional specific and ancestral standards as well as updating anthropometric data on secular trends.

1.2.1 SIGNIFICANCE OF PROJECT

The significance of this research spans several fields including biological- and forensic anthropology, skeletal biology, anatomy, and anthropometrics. Research is necessary in the field of forensic and biological anthropology because of morphological variation among different population groups (Buikstra & Ubelaker, 1994). The KSC, housed at Stellenbosch University, Faculty of Medicine and Health Sciences (FHMS), Division of Clinical Anatomy, is a contemporary collection representing, in part, a population that lived during the 20th century. The KSC with individuals with known records provides the opportunity to develop a reference tool to investigate human variation with regards to age, sex, ancestry and stature differences in a region specific population. Furthermore, it provides the unique opportunity to test the influence of various demographic, ecological, behavioural and environmental variables on pathology and trauma patterns in a contemporary population.

The KSC is the largest collection for the heterogeneous mixed population group in South Africa, namely the South African Coloured (SAC) population. Very little population- and regional specific reference data are available for this population and there is a need to establish base-line information to use as a tool in unknown victim identification when applied to forensic cases. Reference to ancestry in this thesis context is therefore to establish parameters to apply to forensic identification of the many unknown individuals found in SA on a daily basis.

1.3 HYPOTHESES, AIMS AND OBJECTIVES OF THE STUDY

1.3.1 HYPOTHESIS

Analysis of the life history (health, disease, diet and trauma) of the skeletal material in this collection will give insight into the lifestyle of a Western Cape population during the 20th century. Interpersonal violence and diseases such as tuberculosis are prominent issues in the disadvantaged communities of the Western Cape, where alcoholism, lack of proper nutrition, poor health and trauma are frequently encountered.

The hypothesis tested was that there would be a relationship between the variables of sex, age, time period and population group and the presence of disease and trauma markings on the skeletons. The null hypothesis was therefore, that the demographic data would not be dependent or co-dependent on the time periods and/ or the health status of the cohort analysed.

The focal aim of the study is to report on the pathological, metabolic, nutritional and trauma lesions visible on the individuals of the KSC to be used as baseline information in further anthropological studies on the people of the Western Cape region.

1.3.2 PATHOLOGY ASSESSMENT: AIM AND OBJECTIVES

The aim of the study is to describe and evaluate the disease patterns and skeletal indicators of metabolic disorders and nutritional deficiencies and their distribution as presented on the skeletal material. In order to achieve this aim, the following objectives are proposed:

1. To statistically assess whether there is an association between age, sex and specific disease lesions.
2. To statistically assess the correlation between disease lesions and the timeline through the 20th century to determine if the disease patterns varies between eras.

3. To statistically assess the association between disease lesions and population groups.

1.3.3 TRAUMA ASSESSMENT: AIMS AND OBJECTIVES

A further aim of this study is to describe and evaluate the type and distribution of trauma patterns on the skeletal material. In order to achieve this aim, the following objectives are proposed:

1. To statistically assess whether a correlation exists between the prevalence of trauma on the skeletal material and age, ancestry and sex.
2. To statistically assess whether a correlation exists between the type of trauma (classified as interpersonal violence or other injuries such as motor vehicle accidents, falls, sport injuries, etc) inflicted on the KSC population and the changes that occurred during the 20th century due to social, political, geographical or economical factors.

1.4 OUTLINE OF THE STUDY

Chapter 2 is divided into five major sections. In the first section, the current profile of the study location, namely the Western Cape Province (WCP) of South Africa (SA) is reviewed, followed by a broad, descriptive view of the origin, history and demographic profile of the Western Cape population. The next section refers to the profile of the population health in the Western Cape during the 20th century, including economic growth, housing, health care access, education, substance abuse, life expectancy and the socio-economic circumstances of the Western Cape inhabitants. This is followed in the third section by the profile of disease and mortality where the cause of death and disease patterns of Western Cape communities during the 20th century are discussed and compared to the cause of death as represented in the KSC. The fourth section reviews studies on disease lesions on bone; this includes some congenital anomalies seen on individuals in the KSC, infectious diseases, deficiency diseases, haematological disorders, degenerative diseases and arthropathies, as well as neoplastic diseases. The last section discusses the profile of violence and trauma in the Western Cape population during the 20th century. The mechanism of trauma and fracture sites on bones are reviewed thereafter. Chapter 3 lays out the materials and methods used for this project. This includes an explanation of the study sample from the KSC used, and the ethical consideration.

The methods for macroscopic, as well as radiological observations are explained and the statistical methods used to compare disease and trauma lesions among age-at-death, sex, population groups, and time periods are discussed. Chapter 4 presents the statistical results of the above-mentioned comparisons with graphs and tables. Chapter 5 gives a discussion on the results and presents concluding remarks, with future research possibilities. References and Appendices follows thereafter in Chapter 6 and 7 respectively.

CHAPTER 2: LITERATURE REVIEW

2.1 PROFILE OF THE STUDY LOCATION (WESTERN CAPE PROVINCE)

South Africa (SA), a medium sized high middle-income country at the southern tip of Africa, although many of the people live below the national minimum income line. The inhabitants of SA are a diverse nation with a rich biological history, displaying a wide range of differences in ancestral origin and cultural backgrounds. The heterogeneous population consisting of four socially defined population groups, namely the South African Black (SAB), South African White (SAW), South African Indian (SAI) and a mixed ancestry group called the South African Coloured (SAC) population group (Stats SA Census, 2012).

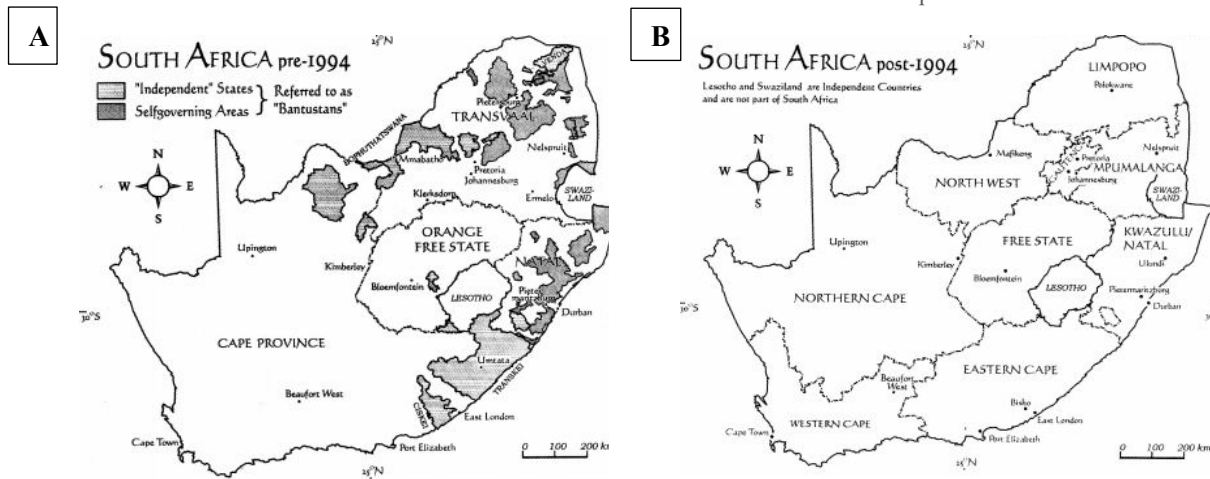
The population numbers of the southernmost province of South Africa are difficult to establish, as the borders have changed over the years. This south-western province was first named the Cape Colony when the Dutch took reign in the mid 1600s. When the Union of South Africa was established in 1910, under British ruling, it was renamed the Cape of Good Hope Province (or the Cape Province). At the time, it was the largest of South Africa's four provinces, encompassing two-thirds of South Africa's land (Fig. 2.1A). The borders included the north-western parts of the country, known today as the Northern Cape Province. Currently, the Republic of South Africa is separated into nine provinces (Fig. 2.1B) with the Western Cape Province much smaller, influencing census numbers when comparing the old Western Cape Province to the democratic new era.

Table 2.1 provides population numbers between 1960 and 1991 (Labuschagné & Matthey, 2000) of the Cape Peninsula, that enclosed the Cape Town City bowl and surrounding areas. Between 1991 and 2011, census data (2011) reflect information of the WCP that included the Boland towns surrounding Cape Town.

Table 2.1. Population composition: Cape Peninsula and Western Cape Province (%)

	Cape Peninsula (%)				Western Cape (%)			
	1960	1970	1980	1991	1991	1996	2001	2011
SAC	51.8	54.7	53.6	53.6	58.3	54.2	53.9	48.8
SAB	9.3	9.8	12.7	19.3	16.4	20.9	26.7	32.8
SAW	37.8	34.5	32.6	26.1	24.5	20.8	18.4	15.7

Source: Labuschagné and Matthey (2000), Stats SA Census (2012).



Source: <http://www.sahistory.org.za/archive/map-south-africa-pre-1994-bantustans>.

Source: <http://www.sahistory.org.za/archive/map-south-africa-post-1994>.

Figures 2.1A-B. A) The four Provinces of the Union of South Africa and later the Republic of South Africa prior to Democracy (1910 to 1994); and B) the nine provinces of SA, showing the much smaller WCP the south-western province in South Africa (1994 to present).

Currently, the WCP houses approximately 5.8 million people. This constitutes roughly 11.2% of the total South African population (Stats SA Census, 2012) of 53 million people. The regional population composition of the province differs considerably from that of other provinces in SA. According to the most recent census (Stats SA Census, 2011), the SAB population group has the highest representation in the country, making up over 70.0% of the population in all provinces with the exception of the Northern Cape (32.9%) and Western Cape (32.8%). In contrast, the SAC population group count is the highest in the Northern Cape (40.3%) and Western Cape (48.8%). However, the percentage of the SAC population group in the Western Cape decreased from 58.3% in 1991 (Labuschagné & Mathey, 2000) to 48.8% in 2011 (Stats SA Census, 2012).

According to Census 2011 figures (Stats SA Census, 2012), the City of Cape Town's total population escalated from 2.9 million in 2001 to 3.7 million in 2011. Currently, the composition of the Cape Town metropole population is: Coloured 42.4%, Black African 38.6%, White European 15.7%, Indian / Asian 1.4% and other 1.9%.

2.1.1 THE ORIGIN OF THE WESTERN CAPE POPULATION

The most ancient ancestral people of southern Africa are the original San (hunter-gatherers) and the Khoikhoi (or Khoena) (pastoralists) populations (Chen et al., 2000; Schuster et al., 2010; Schlebusch et al., 2012). According to Mountain (2003), the San inhabitants carry the oldest genetic stock of modern humanity and the presence of the San in South Africa can

be traced back over thousands of years. KhoiKhoi herder immigrants arrived in the Western Cape region more than 2000 years ago to co-exist with the San. The KhoiKhoi and San are jointly referred to the Khoisan or the Khoe-San (Schlebusch et al., 2012) and are not found elsewhere in Africa (Byrnes, 1996). The Khoe-San sustained an isolated prehistoric lifestyle for centuries, living in small kinship-based villages (Byrnes, 1996). These people, with their characteristic physical traits remained genetically secluded from other population groups; however, the genetic profiles of some tribes (such as the Khwe-groups) show high proportions of Bantu-speaking admixture (Schlebusch et al., 2012; Schuster et al., 2010).

The SAB population group originated from the Great lakes region of sub-equatorial Central/East Africa and migrated as part of the greater Bantu-group to occupy much of the East and Southern parts of Africa. The Nguni group is a subfamily of this Bantu-family which can be subdivided into the northern- (Xhosa, Zulu, Swati and Ndebele tribes) and southern (also called the Sotho-Tswana) groups (Southern Sotho, Northern Sotho, Tswana, Venda and Tsonga tribes) (Plaza et al., 2004; Coelho et al., 2009; Tishkoff et al., 2009; De Filippo et al., 2011; Thompson, 2013). These tribes migrated towards the south over many centuries and settled in different regions in SA. For example, Ndebele stayed in the north, the Swazi in the north east, the Zulu towards the east and the Xhosa in the south (Thompson, 2013). These migration patterns started around 2000 years ago, followed by larger migration impressions around 1400 CE (Coelho et al., 2009; De Filippo et al., 2011).

The European community settled in the Dutch-controlled Cape Colony after 1652. Cape Town became a busy refreshment station where European traders and sailors, either stopped over to obtain fresh supplies or settled at the harbour-town at the foot of Table Mountain. British settlers colonised the Cape Colony during the mid-1800s (Van der Ross, 2005; Patterson et al., 2010). The Asian population group originated from Indian and Chinese immigrants, as well as Capetonian slave-forefathers from countries in the East (Van der Ross, 2005).

The first slaves imported in the mid 1600s were a few hundred Bantu-speaking slaves from the West Coast of Africa, at the time seen as the chief slave market (Schoeman, 2007). In the following years, slaves were rather imported from East Africa or the East. East African slaves were the Bantu-speaking Mozambiquen slaves (known then as “Masbiekers”) or Malagasy-speaking slaves from Madagascar, with a genetic mix between Black and Indonesian blood (Marais, 1939). The slaves imported from the East through the Indian Ocean Slave Trade

included people from the mainland of India, Batavia (now Jakarta) in Indonesia, Ceylon (now Sri Lanka), Siam (now Thailand) the Malayan Archipelago, and southern China (Marais, 1939; Boeseken, 1977).

In the early days of the settlement, it was common for European settlers to have irregular union with slave women. Marais (1939) reported that during the first 20 years of white settlement in the Cape, 75% of children born to slave mothers had white fathers.

During the 18th century, the Khoisan tribes increasingly lost their land, and their flocks and herds to the white farmers, especially after the Europeans introduced smallpox into the settlement in 1713, and again in 1755 and in 1767. This devastated these small tribes, and left them broken-spirited fragments of communities without hope (Marais, 1939). They then entered the service of the white farmers. Sexual intercourse among the European farmers and the Khoi women was a common occurrence, and the term “Bastards” was in general use to signify the cross between European and other population groups, especially favouring the European-Khoi mix (Marais, 1939). However, with time, European farmers lost standing among their own people if they married Khoi women. By the second half of the 18th century, the “Bastards” became people apart from the white farmers and Khoi people, tending to intermarry only among themselves. As they generally had large families, this heterogenic group was rapidly increasing in numbers by the beginning of the 19th century. This newly formed population group was named the Cape Coloureds (Van der Ross, 2005; Patterson et al., 2010). In the period extending between 1807-1821, the importation of slaves gradually stopped. With the emancipation of slavery between 1834-1938, under the British Act 73 of 1833, there were approximately 30 000 slaves in the Cape Colony (Marais, 1939). In the time period of 180 years of slavery in the Cape, the total number of new imported slaves sold were documented at 78 539. Their children and descendants all led lives of enslavement (Giliomee & Mbenga, 2007). There were more slaves than Europeans in the Cape Colony in the 18th century; by 1769, the locally born slaves had reached 50.0% of the entire slave population (Shell, 1997) and a census taken in 1798 showed that the slaves (25 754) outnumbered the Europeans (20 000) (Giliomee & Mbenga, 2007).

When the representative government, under British rule, was established as the Parliament of the Cape of Good Hope in 1853, equal political rights were granted to all men, regardless of colour (Pfeiffer et al., 2016). The draft constitution that had been drawn up prohibited population group or class discrimination. During this time, marriages among the white

European males and free Black populations or Khoisan were common (Jacobson et al., 2004; Patterson et al., 2010). Colonisation and migration of immigrants from many parts of the world became the foundation of modern variation in South African groups (Stull et al., 2014), thereby producing a diverse new population group, with distinctive physical and biological traits, as well as genetic variations (Friedling, 2007). Genetic studies (Quintana-Murci et al., 2010) illustrated that the genetic enhancement of the SAC population group of the Cape is more diverse in admixture than any other population group in the world. The genetic distribution of the Coloured population group in the Cape Town metropole has been characterised as foremost Khoisan, followed by Black, White and Indian/Asian (De Wit et al., 2010; Daya et al., 2013). A gradual decrease in cross cultural relationships between the late 1800s (Sherman & Steyn, 2009) and the mid 1900s, was established by apartheid laws which resulted in a decrease in cross population gene flow (Stull et al., 2014).

2.1.2 THE EFFECT OF APARTHEID ON THE COMPOSITION OF THE WESTERN CAPE POPULATION

Apartheid is a political system of racial separation that was enforced by the white minority governing party that ruled between 1948 and 1994 (Erasmus, 2012), and which brought about unequal rights and privileges to population groups in the country. Although SA was already segregated prior to apartheid, these apartheid laws made segregation more systematic and discrimination more absolute (Bundy, 2016). Under this ruling African people, who were not Khoisan and were a product of the Bantu expansion, were grouped as “Black” people (Jacobson et al., 2004) while people with an admixture of mixed origin, including Khoisan, were classified as “Coloured” people, persons from European descent grouped as “White” and from Indian descent as “Indian” people. The population Registration Act of 1950 was used to categorise all South Africans as Black, White, Indian or Coloured (Jacobson et al., 2004). This discriminatory way of living was deep-rooted into the ‘white’ or ‘non-white’ groups, where the ‘white’ category included the people from European descent only, and the ‘non-white’ groups, included all the other groups. The lives of the non-white majority were tightly controlled and regulated, for example occupation, education, and wages for ‘non-whites’ were kept much lower than those for ‘whites’.

Although South Africa has changed to a democratic political dispensation since 1994, residential segregation among population-classified groups still persists (Christopher, 2001) in addition to the continued differences in the socio-economic status (SES) among groups.

2.2 FACTORS INFLUENCING POPULATION HEALTH IN THE WESTERN CAPE

The environment, in which people live, has an impact on their bones in terms of development, maturation, and commencement of degradation (Inwood & Masakure, 2013; Pfeiffer et al., 2016). By studying past populations, the correct identification of these environmental stressors and the impact thereof on the skeletons as well as the history of some diseases becomes evident (Cox & Mays, 2000). Population health in the Western Cape was influenced by various factors during the 20th century.

2.2.1 ECONOMIC GROWTH

It was easy and inexpensive for South Africa to import many products during the 19th century. During World War I it became more difficult to import many goods and some even impossible. South Africa was left dependent on its own resources, encouraging development of industrial assets. The trade structure of the WCP was dominated by three industries during the 20th century, namely clothing and textiles, processing and preparation of foods and beverages and printing. Between 1920 and 1960 the labour services in these industries have increased by 900%, 238% and 335% respectively (Whittingdale, 1973), although the effect of the Great Depression, between 1929 and the mid 1930s, rendered a decline in the rate of economic development. In the following period, 1934-35 to 1938-39, there was an upsurge (Whittingdale, 1973). During World War II (1939-1945), a rapid increase in industrial output was observed and the SA industry was forced to adjust swiftly to a greatly expanded market, and become as self-sufficient as possible. The WCP industries doubled during this time and by the early 1950s large manufacturing estates were in demand and large open areas around Bellville and Parow were set out to build industrial facilities. These included for example, Kasselsvlei Road in 1954 and Sacks Circle in 1957. At the same time 'Coloured' townships (Belhar, Mimosa, Delft, Ravensmead) were established around these industrial parks together with direct rail and road links to supply labourers for these industries (Whittingdale, 1973; Friedling, 2004).

2.2.2 HOUSING

During the industrial revolution time (1939-1945), the number of people in the manufacturing industry doubled and many rural inhabitants moved to the cities in search of a better life (Byrnes, 1996). Most of the 'non-white' community of Cape Town were living in

squatter camps on the outskirts of Cape Town. Compared to the rest of the country, where the SAB population groups mainly represented the poor and destitute, these areas had a far larger number of people classified as SAC. In 1941, Batson published a “Social Survey of Poverty” report, reporting that 53.0% of Cape Town’s ‘non-white’ households (predominantly SAC) were living below the poverty line, compared to 5.0% of the SAW population. By the end of 1946, thirty squatter camps around Cape Town housed an estimated 25 000 men and 5000 families, and conditions in these camps were dire (Bundy, 2016).

In 1950, the Group Areas Act was passed, giving the apartheid government powers to displace ‘non-whites’ living in racially mixed areas in the cities to ‘non-prime’ areas on the outskirts of the city, it also restricted ownership and occupation of prime land to the SAW group. In 1952, the Government started to clear the unhygienic slums in and around Cape Town by building scheme houses in the low-lying Cape Flats areas between the False Bay and Table Bay areas (Friedling, 2004). These plain, uniformly built houses in orderly grids were to relieve the dire conditions of the overcrowded informal slums that was without proper sanitation and water supply.

Many of the SAC people were forced to relocate to the “Coloured” Bellville-Parow townships close to the large industrial estates. This is also where most of the Tygerberg cadaver donations were received from (Whittingdale, 1973; Friedling, 2004). Although conditions improved, these townships did not have the resources, complexity or variety that the ‘White’ cities had. Huge numbers of residents were concentrated in these townships without basic civic services, poor access to utilities, public space, work opportunities, schooling, or transport (Bundy, 2016).

Although adequate housing for the population of the Cape metropole was estimated at only 31.0% in the early 1990s (Pfeiffer et al., 2016), presently, households living in informal housing add up to about 12.2% (Stats SA Census, 2012). Residents of the Cape Flats still constitute one of the most vulnerable groups in need of social and economic support, living in inadequate housing, with poor living conditions, low SES, high gang-related activities, and high levels of interpersonal violence that go hand in hand with alcohol and drug abuse (Stats SA Census, 2012; Inwood & Masakure, 2013).

2.2.3 HEALTH CARE ACCESS

During the 20th century, an overview of the health system in SA revealed a close relationship and interdependence between health care and the level of society, with distinct differences among population groups. There was, and still is, a coexistence of two types of health care systems in SA. On the one hand, there is highly organised, Western health care (allopathic medicine), available to all population groups, based on a constantly improving scientific approach and successful medical technology. South Africa's health care facilities include hospitals, day hospitals, community health care centers, and clinics (Byrnes, 1996). In contrast to this, there is a traditional alternative health system, followed mainly by the SAB population group. Traditional tribal medicine (ethnomedicine) emphasises the supernatural nature centered around a traditional healer (Van Rensburg & Mans, 1982).

The western health care system was (and still is) subsidised by the government of SA and until the 1990's apartheid was practiced in most facilities to varying degrees. The distribution of health services among population groups was not reconciled with the actual demand or needs of the different groups. Some admitted patients of one racial group only, while in others, designated operating rooms and special care facilities for patients of certain racial groups existed (Van Rensburg & Mans, 1982). The government employed trained, specialised health care personnel, although there was an unequal allocation of services and health personnel favouring the privileged SAW population. During 1976, for example, the doctor:population ratio in the Cape Peninsula was 1:500, while in the Cape Province it was 1:1200, with a 1:300 ratio for 'whites' and 1:900 for 'non-whites' (Van Rensburg & Mans, 1982). The number of dentists in SA increased from a total of 818 in 1946 to 2 155 in 1976, improving the dentist:population ratio from 1:13958 in 1946 to 1:12328. This ratio of dentist to the total population was misleading since most dental practices were situated in 'white' urban areas and did not service the largest portion of the SA population (Van Rensburg & Mans, 1982). At the end of the 20th century, SA had a well-established health care system, but the quality of health care services across the country was still uneven, and regarded a legacy of the apartheid regime.

The unequal access to health care facilities and poor standard of living conditions had a negative influence on the health status of the inhabitants of the Cape Flats and rural residences of the Cape Province (Bundy, 2016).

2.2.4 EDUCATION

Education in SA during the 20th century was compulsory for all racial groups, but at different ages, and the law was enforced differently. The SAW population group were required to attend school between the ages of seven and sixteen years-of-age and for the SAC and SAI population groups, education was compulsory between the ages of seven and fifteen years-of-age. Children from the SAB group; however, were required to attend school from the age of seven until the equivalent of the seventh grade, or at the age of sixteen, was attained, but this law was poorly enforced. In addition, a number of the ‘black’ areas had no schools available for children to attend (Byrnes, 1996). The Bantu Education Act (No. 47) of 1953 widened the gaps in educational opportunities for different population groups. Black education was kept inferior to the other population groups to ensure that they were not employed above the level of certain forms of labour (Bundy, 2016). ‘Non-white’ education was not supposed to drain government resources away from ‘white’ education.

The spending gap of government funds on education between different population groups slowly began to taper from the late 1980s. When the apartheid laws were lifted in the 1990s, new policies and frameworks were developed to restructure equal education for all population groups (Byrnes, 1996). There is a connection between educational achievement, employment and income and the low literacy levels of the vulnerable residents of the Cape Flats was directly linked to their unemployment levels and income (Schneider et al., 2007; Inwood & Masakure, 2013).

2.2.5 SUBSTANCE ABUSE

Southern Africa has a major problem with substantial alcohol consumption and it is especially prevalent in the SAC communities in Cape Town (Parry et al., 2005; Pluddemann et al., 2004; Schneider et al., 2007; Richard et al., 2010; Ward et al., 2012; Corrigan & Matzopoulos, 2013; Schuurman et al., 2015). The high levels of alcohol abuse among the SAC population group could be regarded as a legacy from even before the apartheid era. Vines were planted in the early days of the Dutch settlements and the wine produced was plentiful and cheap. Wine was regularly supplied to the slaves and Khoi labourers in the agricultural districts to help keep them obedient and dependent on the farm owners (Marais, 1939; Pfeiffer et al., 2016). After slave emancipation in 1834, many farmers found it necessary to provide more alcohol to their labourers during harvest time, mainly to keep them on the farm, than was regarded as the general custom (Marais, 1939). Even during apartheid, SAC labourers on the

farms were largely paid in alcohol, as part of the so-called “dop-stelsel” (London, 1999; Parry et al., 2005; Schneider et al., 2007; Peltzer & Ramlagan, 2009; Gossage et al., 2014). The drinking habit was therefore passed from generation to generation (Marais, 1939). Furthermore, it was (and still is) tradition for the SAB community to brew their own fermented iron-rich beer, called umgombothi, from maize malt, sorghum malt, yeast and water (Odhav & Naicker, 2002). A recent study found that up to 40% of the sorghum and maize used in fermenting the beer, are contaminated with mycotoxin-producing fungi (Odhav & Naicker, 2002) and previous research by the Medical Research Council found a very high incidence of oesophageal cancer in the Eastern Cape that may be caused by toxin-producing fungi (Isaacson et al., 1985).

Studies noted a significant association between alcohol abuse and violence (Silvennoinen et al., 1992; Chrcanovic et al., 2004; Norman et al., 2007). As alcohol and drug abuse is fairly common amongst young adults in the SAC social setting, increased incidence of drug related crimes and interpersonal violence (IPV) have been reported (Lee et al., 2007). In a survey to investigate alcohol intake trends in SA, in general, males were shown to consume, on average, more alcohol compared to females (Peltzer & Ramlagan, 2009).

Tobacco products, cultivated in the Cape Colony since 1882 (Mantzaris, 1995) have been widely used during the 20th century. Males tend to use more tobacco products than females (Sieminska & Jassem, 2014) and this bias is related to cultural and behavioural factors (Cosgrove et al., 2014). Many research projects on tobacco-related diseases and cancers have been done over the years and proof of tobacco chemicals causing adverse effects in the human body are numerous (Doll et al., 1994; Ozlü & Bülbül, 2005; Jha, 2009; Swerdlow et al., 2010; Banks et al., 2015). Smoking increases the risk of at least 15 types of cancers such as lung, mouth, pharynx, larynx, oesophagus, liver, pancreas, stomach, kidney, intestines, ovary, bladder, cervix, and some types of leukaemia. Scientists proved a positive correlation between the number of cigarettes smoked daily and the risk of cancer (Swerdlow et al., 2010), furthermore, they found the number of years a person spend smoking affects the cancer risk even stronger (Doll et al., 1994; Ozlü & Bülbül, 2005). Specifically, the association between cigarettes and lung cancer has been reported to be the main cause of 90.0% of male and 79.0% of female lung cancers and 90.0% of deaths from lung cancer are estimated to be due to smoking (Ozlü & Bülbül, 2005).

It was a tradition in SA, since the 15th century, for the native SAB population group to cultivate and use cannabis, known as ‘dagga’ in SA (Rubin, 1975). Use expanded during the 18th and 19th centuries. The use of dagga was largely limited to the SAB population group and gained popularity only in the SAC and SAW population groups over the last few decades (ODCCP, 1999). The first drug laws with regards to the use of dagga was introduced by the Government of SA in 1928 (Wright, 1991). Use of Methaqualone (Mandrax), as a psychoactive depressant substance, was widespread after World War II (ODCCP, 1999). The SA government subsequently identified the abuse potential of Mandrax and removed it from the legal market and classified it as illegal in the SA narcotics law (Act 41 of 1971). Today, many variants of different illegal substances such as crack-cocaine, heroin and Mandrax are drugs of choice (ODCCP, 1999). High unemployment, low SES, low literacy levels and the weakening of the family and social structures in the Cape Flats townships, strengthened the pattern of drug abuse over the last few decades (Atkins, 1997; ODCCP, 1999). Furthermore, the easy accessibility of drugs, underground laboratories manufacturing drugs and the Cape Flats gang wars, related to the illegal drug trade, have a hugely negative effect on the communities in these areas, causing the death of numerous young men and innocent bystanders, including children (Samara, 2011).

2.2.6 LIFE EXPECTANCY AND SOCIO-ECONOMIC FACTORS

As a result of the vastly dissimilar life styles among the inhabitants of SA, lower and higher socio-economic classes showed different patterns of disease and life expectancy. During the 20th century, there was a considerable discrepancy in life expectancy among the different population groups, with the privileged SAW with the highest and the SAC and SAB with the lowest life expectancy (see Fig. 2.2 for numbers for life expectancy between different population groups in selected years between 1945 to 1971).

YEAR	WHITES		ASIANS		COLOUREDS		BLACKS*	
	M	F	M	F	M	F	M	F
1945-1947	63,8	68,3	50,7	49,8	41,7	44,0	39,1	39,2
1959-1961	64,7	71,7	57,7	59,6	49,6	54,3	47,9	53,7
1969-1971	64,7	72,3	59,2	63,2	48,9	55,8	49,0	55,6

* Figures for Blacks are estimates.

Source: Van Rensburg & Mans, 1982.

Figure 2.2. The average life expectancy for the four population groups in South Africa during selected years.

During the 1990s the life expectancy figures still varied substantially between population groups. For SAW these figures were as high as 62.7 years-of-age for SAW males, and 68.3 years-of-age for SAW females, placing South Africa just below the global median; life expectancy for the other population groups were persistently lower. For instance, the life expectancy figures for the SAB males was about nine years less than that for SAW males (Byrnes, 1996). The decrease in age were mostly attributed to the effects of HIV/AIDS and infectious diseases such as tuberculosis (Andrews et al., 2007; Den Boon et al., 2007).

2.3 PROFILE OF MORBIDITY AND MORTALITY

Data on morbidity and mortality are indicators of the standard of health in a population. An assessment of disease patterns can provide insight into the interrelationship between disease, diet, ecology, social- or political structure, immunological resistance and psychological stress (Kunitz & Euler, 1972; Krogman & İşcan, 1986; Mann & Murphy, 1990; Aufderheide & Rodríguez-Martín, 2011) of a particular population group. Signs on skeletal remains indicating diseases of excess or diseases of deficiency, are indicators of the status of the health and wealth of a community and can be useful in explaining local patterns of disease and mortality. The differences in the mortality profile of South African population groups is clearly associated with their divergent socio-economic circumstances, with the SAW group in an advantageous position compared to the SAC and SAB population groups (Van Rensburg & Mans, 1982). However, it should be taken into account that morbidity and mortality indicators are not exact in a population, especially in a largely chronic disease profile.

2.3.1 CAUSE OF DEATH AND DISEASE PATTERNS OF THE POPULATION DURING THE 20TH CENTURY

Before and during World War II (until 1945) large scale migration and urbanisation, resulting from the industrial revolution and discovery of gold and diamonds lead to a challenge in dealing with urban sanitation. This was further complicated by several infectious disease epidemics, such as smallpox (1883 epidemic), bubonic plague (1901 epidemic), Spanish influenza (1918 pandemic), cholera (several outbreaks during the 20th century), tuberculosis, malaria, yellow fever, typhus, and venereal diseases (Van Rensburg & Mans, 1982) .

World War I (1914-1918), the various disease epidemics and the Great Depression (1929 to mid 1930s) brought about considerable economical devastation and political change during the early 20th century, influencing the poverty levels of all population groups in SA (Byrnes,

1996). In 1932, the Carnegie commission specifically made recommendations to uplift the poor White people (Byrnes, 1996), which brought about the first steps of the ruling political party to uplift the White population group to economic independence (or the onset of apartheid ideas).

Post World War II (after 1949), advancement in vaccines and the development of antibiotics world-wide greatly improved the quality of life and outcome of infective conditions. Medical progress, with enhanced understanding of microorganisms and the diseases caused by them, helped to eradicate (smallpox, bubonic plague) and control (yellow fever, cholera) many of these diseases (Van Rensburg & Mans, 1982). Very little of these advances; however, extended to the 'non-white' population groups in SA during the apartheid era (1948-1994) as poverty-related diseases persisted in these population groups, while, in contrast, non-communicable diseases increased in the SAW population during this period (Coovadia et al., 2009). The statistical figures from the Union Census and Statistics Office in the mid to late 1900s for cause of death (COD) in SA showed that the SAB group died mainly from gastrointestinal diseases. However, it should be considered that life expectancy includes individuals of all ages. As infants are particularly prone to die of gastrointestinal disease, and described by Van Rensburg & Mans (1982) to be up to 25%, the cause of death for the adults may include other diseases. The SAC population group were more inclined to contract pulmonary infections and parasitic diseases. In contrast, the SAW group, in general, showed a low profile for these diseases, but a high profile for ischaemic heart diseases, neoplasms and ulcers (Cluver, 1959; Van Rensburg & Mans, 1982) in line with developed Western countries.

When comparing COD between the sexes of the various population groups, it did not follow the overall pattern of each particular population group (Van Rensburg & Mans, 1982). For example, in 1976, the highest cause of death in males of the SAC population was homicide or willful injury by others, followed by pneumonia-related deaths. In contrast, the SAC females showed the highest COD due to pneumonia, closely followed by enteritis and diarrhoeal diseases (Van Rensburg & Mans, 1982).

In post-apartheid democracy (1994 ongoing), diseases of poverty, non-communicable diseases and HIV/AIDS are the main contributors to the high burden of disease. The public health system serves all the population groups, although it still remains underfunded, unskilled and understaffed (Coovadia et al., 2009). The most affluent 20.0% of the population use the private system and are far better off (Coovadia et al., 2009).

2.3.2 CAUSE OF DEATH AS REPRESENTED IN THE KIRSTEN SKELETAL COLLECTION

The foremost COD of the cadaver intake at the Anatomy department (later division) as recorded by Labuschagné & Mathey (2000) for years between 1956 and 1996, was recorded as respiratory-associated diseases such as pulmonary tuberculosis (PTB), bronchiectasis, pneumonia and asthma (24.1%). These diseases were with and without co-morbid conditions such as cardiopulmonary failure, renal failure, or diseases of the liver and gastro-intestinal tract in general (Alblas et al., 2018). After respiratory-associated diseases, cardiovascular disease, (15.1%) and cancer of various origins (14.3%), followed. When the main cause of death among population groups in the KSC are compared, heart disease appeared to be the main cause of death among the SAW; while various cancers was the highest among the SAB and SAC groups (Labuschagné & Mathey, 2000).

2.4 OVERVIEW OF POSSIBLE PATHOLOGICAL BONE LESIONS STUDIED

Bone can react in only three ways; therefore, the origin of the pathology can be classified as resorption (bone destruction), deposition (bone addition) or both, each depending upon interpretation of its pattern for diagnosis (Ortner, 2003). If bone addition is present, the added bone will be characterised as either poorly organised (this typically means rapid growth) or well organised (usually slow growth). If destruction or osteoclastic activity is present, the lesions can be characterised with well-defined margins with evidence of well-organised bony repair (circumscribed and generally less aggressive), or with poorly defined margins (permeative and generally more aggressive) (Ortner, 2003; White et al., 2012). A single resorptive (lytic) lesion of a vertebral body could represent infection (e.g. tuberculosis), a primary cancer (plasmacytoma), or a benign lesion (Schmorl's node). If a single lytic margin is surrounded by a reactive growth of bone, the process is probably a chronic infection (e.g. tuberculosis). Aggressive lysis with no reaction favours malignant disease, while a smooth regular margin is probably both benign and inactive.

Deposition of bone is most frequently due to inflammation of the periosteum. This is most readily seen in the vigorous callus formation after a fracture, but is also seen in more subtle injuries such as muscular injuries next to the bone retracted the periosteum and blood collected underneath. The haematoma is organised into scar tissue, which can be converted into bone. Chronic infections (for example, osteomyelitis) of bone rarely heal spontaneously, producing

years of inflammation resulting in very large deposits of bone, often with a central sinus which continually drained pus (Aufderheide & Rodríguez-Martín, 2011).

2.4.1 CONGENITAL/GENETIC ORIGIN

Pathological changes occurring during embryological development and birth are called congenital malformations (Aufderheide & Rodríguez-Martín, 2011) and can be diagnosed prenatally, at birth, during childhood, or in non-related cases during adulthood. These congenital conditions can be due to an environmental insult, infection, maternal malnutrition during developmental stages, or have a genetic influence (Ortner, 2003; White et al., 2012). Any type of tissue or organ of the body can be affected by these malformations, with skeletal manifestation representing about 40.0% of all congenital malformations (Aufderheide & Rodríguez-Martín, 2011), especially seen in the vertebral column due to its multifaceted embryological development (Masniková & Beňuš, 2003).

The genetic composition of an individual or population can influence the structure of bone and play a role in possible diseases or disorders a person or population can acquire, for example, osteoporosis (Inwood & Masakure, 2013). Several authors found that the SAC population have a lower bone mass and density than global average (Daya et al., 2013; Pfeiffer et al., 2016; Beresheim et al., 2018); while others found that the SAC population have a smaller stature and are affected by skeletal TB (Goldblatt & Cremin, 1978). Although genetics could have played a role in these factors, environmental and social stressors in this population group should not be excluded in the diagnosis (Silvennoinen et al., 1992; Chrcanovic et al., 2004; Schneider et al., 2007).

During vertebral embryological development, it is relatively common to observe skeletal defects such as developmental delays of vertebral elements, for example, NTD's; spondylolysis, cranial-caudal border shifts, and failure of segmentation of the mesodermal sclerotome during cervical development, causing Klippel-Feil syndrome (Masniková & Beňuš, 2003). It must be considered that, although many congenital conditions do exist, this literature research is based on conditions visible on the KSC cohort only.

2.4.1.1 Klippel-Feil syndrome (KFS)

This is a rare congenital defect in the embryological formation of the cervical vertebrae or somites between the third and eighth weeks of development (Fernandes & Costa, 2007; Aufderheide & Rodríguez-Martín, 2011), resulting in the congenital synostosis of any two or

more of the cervical vertebrae. The underlying cause of this condition is not fully understood, although there is evidence that genetic mutations on specific GDF6 and GDF3 genes can cause this disease (Tracy et al., 2004; Tassabehji et al., 2008). Other studies have suggested an association between KFS and Foetal alcohol syndrome (FAS) (Lowry, 1977; Neidengard & Carter, 1978; Schilgen & Loeser, 1994).

Several clinically based studies by retrospective plain radiographic assessment of the vertebral column of patients have described the presence of KFS, usually associated with a classic triade of symptoms (abnormally short neck, restricted movement of the head and neck, and a low hairline at the back of the head). However, not that many studies on skeletal archaeological and contemporary collection material (Ortner & Putschar, 1985; Legge, 2004, Fernandes & Costa, 2007) are described as diagnosis is more difficult without clinical symptoms and ankylosed vertebrae may have other origins as well. Other skeletal abnormalities that can be present include occipitalisation, cranial-caudal border shifting, hemivertebrae, cervical ribs, supernumerary vertebrae, rib anomalies, spina bifida, scoliosis, cleft palate, dental problems, or Sprengel's deformity, a congenital structural abnormality of the shoulder girdle (Ortner & Putschar, 1985; Barnes, 1994; Fernandes & Costa, 2007; Paradowska et al., 2007; Samartzis et al., 2006). Different classification systems for this syndrome have been suggested (Samartzis et al., 2006). In a study of 462 patients with KFS, Gray et al. (1964) found that the highest level of fusion occurred from the occiput to C3. A study by Somartzis and colleagues in 2006, found that 74.1% of the ankylosed segments in the cervical vertebrae were at level C2-C3, also noted by other authors (Bonola, 1956; Gray et al., 1964; Baird et al., 1967). The prevalence of this disease in populations is still unknown, and several studies reported that females are more affected (up to 70.0%) than males (Gray et al., 1964; Helmi & Pruzansky, 1980; Pizzutillo et al., 1994, Thomsen et al., 1997; Larson et al., 2001; Fernandes & Costa, 2007; Paradowska et al., 2007; Samartzis et al., 2007) and it occurs one in every 40 000 or 42 000 people (Thomsen et al., 1997; Paradowska et al., 2007).

2.4.1.2 Cleft neural arches/spina bifida occulta

Failure of the neuropores of the neural tube to close during embryological development can cause neural tube defects (NTD's) such as spina bifida, which involves midline openings in the posterior segment of the vertebral column. It is observed on skeletal material as a cleft between the two halves of the unfused neural arches. Spina bifida can be divided into two basic types, namely, the more serious spina bifida cystica or aperta (SBC), and spina bifida occulta

(SBO). In SBC, the meningeal and/or neural tissue protrude through the defect resulting in a severe type of spina bifida, which can be fatal or cause severe abnormalities (Aufderheide & Rodríguez-Martín, 2011). The meningeal and/or neural tissue do not protrude through the opening in cases of SBO, and can therefore go unnoticed (Ortner, 2003). This congenital defect can be caused by a combination of genetic and environmental factors, such as a genetic predisposition, a lack of maternal dietary supplementation (folic acid), or environmental factors such as teratogenic drugs, exposure, social drugs, smoking, glucose metabolism and alcohol (Groza et al., 2012). The cleft neural arch of spina bifida can occur in any vertebra, although it has been mentioned in several studies that it is predominantly present in the lumbosacral region of the vertebral column with the sacrum affected the most (Boone et al., 1985; Botto et al., 1999; Shin et al., 2008; Groza et al., 2012). In a study by Groza et al. (2012) spina bifida was reported to be the most common congenital malformation of vertebrae and several studies found a higher prevalence in males than females (Lorber & Levick, 1967; Vannier et al., 1981; Fidas et al., 1987). In a USA study by Shin et al. (2008), it was reported that the White population study group showed a higher prevalence of spina bifida than their Black counterparts. The same results were reported in a study by Eubanks & Cheruvu (2009). A large variation in open sacral segments occurs in studies done on different population, as reviewed by Albrecht and colleagues (2007), ranging between 1-30%. This wide range among population groups can be due to the use of different classification methods of open arches rather than population variation. Spina bifida of the sacrum was scored in some studies if S1 was involved, regardless if any of the other segments had cleft arches (Saluja, 1988; Avrahami et al., 1994; Henneberg & Henneberg, 1999), while other studies included sacral involvement of the second and third segments as well (Boone et al., 1985; Fidas et al., 1987; Schweitzer et al., 1993).

2.4.1.3 Lumbosacral transitional vertebrae (LSTV)

Transitional vertebrae result from overlapping developmental fields due to a cranial or caudal shift of somite during embryological development, being unilateral or bilateral. The direction of this cranial-caudal shift can also result in an increase or decrease of the total number of vertebrae (Savage, 2005; Thawait et al., 2012). These abnormal transitional vertebrae take on the features of adjacent vertebrae and can be present throughout the vertebral column; however, it occurs most often in the lumbosacral region (Konin & Walz, 2010; Aufderheide & Rodríguez-Martín, 2011). The neural arches of vertebrae are more susceptible to change than the vertebral bodies. Carlson (2014) suggested that a clear association exists

between boundary shifts and mutations in certain Hox genes. Although no external factors such as teratogenic drugs, smoking or environmental conditions are linked to this anomaly, it has been included in this study as the prevalence in the KSC cohort is substantial and should be compared to other population groups.

Congenital spinal anomalies of the lumbosacral region, known as lumbosacral transitional vertebrae, (LSTV) were first observed by Bertolotti in 1917 (Bertolotti, 1917; Delport et al., 2006; Kubavat et al., 2012). In this region, the anomalies can either occur in the form of sacralisation, where the fifth lumbar vertebra (L5) acquires characteristics of the sacral vertebrae, or lumbarisation, where elements of the first sacral segment (S1) acquire characteristics of the lumbar vertebrae (Konin & Walz, 2010; Aufderheide & Rodríguez-Martín, 2011; Sharma et al., 2011). A border shift towards the cranium result in sacralisation of the last lumbar vertebra, while a caudal shift will result in lumbarisation of the first sacral segment (Barnes, 1994). Lumbosacral transitional vertebrae have been reported in various forms or degrees. Variation occurring in sacralisation can range from incomplete, where L5 vertebrae have thickened elongated transverse processes (unilateral or bilateral) to complete, where the L5 vertebrae is completely fused to the sacrum, resulting in only four lumbar vertebrae and six sacral vertebrae. Varying degrees of lumbarisation also exist. These include total separation of S1 from the sacrum (resulting in six lumbar vertebrae), various degrees of well-formed lumbar-type facet joints, articulation rather than fusion between S1 and S2, and the formation of a full-sized disk between S1 and S2, rather than the typical smaller-sized disk usually present (Sharma et al., 2011). Several studies have determined the prevalence of LSTV in a number of population groups. The prevalence of LSTV has shown to be as low as 3.6% in an American population group (Moore & Illinois, 1925) and as high as 38.0% in an Indian population group (Kalyan et al., 2013). A review of studies previously conducted on the prevalence of LSTV of at least 19 different population groups showed an average of 16.5%. Relating to sexual differences, authors mainly found that males were more commonly affected than females (Dharati et al., 2012); however, Aufderheide & Rodríguez-Martín (2011) and Mahato (2010) found that females were more commonly affected than males.

Clinically, the correct identification of LSTV is important as failing to recognise LSTV prior to spinal procedures may result in the incorrect numbering of vertebrae. This could lead to problems with the administration of epi/intradural anaesthetics as well as the possibility of spinal surgery occurring at an unintended level (Malanga & Cook, 2004). A further clinical

implication of LSTV is its association with lower back pain, commonly referred to as Bertolotti syndrome. The authenticity of this association has been widely discussed and it is currently believed that low back pain associated with LSTV is due to various aetiologies arising from numerous locations (Konin & Walz, 2010) such as intervertebral disk herniation or degeneration, facet joint arthrosis and foraminal stenosis (Bron et al., 2007), all of which may result in low back pain.

2.4.2 INFECTIOUS DISEASES

In the earliest times, infectious diseases was responsible for many premature deaths. Microbe epidemics were developing after the establishment of settlements and later with civilisation and crowded cities, killing thousands within a short time (Ôsz et al., 2009). It was particularly the immune compromised children and the elderly mostly affected. The invention and use of antibiotics since the mid-20th century controlled many of the bacteria that caused the devastating diseases, granting that bacterial epidemics have remained a serious problem especially in developing countries (Ôsz et al., 2009). Signs of an infectious disease, as observed on skeletal elements, are complex and challenging, although it discloses much about human adaptation to their environment (Friedling, 2007). The interaction of many variables must be considered, such as host resistance (e.g. Black Africans with sickle-cell anaemia do not get malaria), pathogen virulence (antibiotic resistant bacteria), ecological settings, cultural practices (alcoholism), malnutrition and overcrowding (low SES). The type of infections towards which the body developed sufficient immunity to, will co-exist in the body, causing a chronic infection evident on the skeleton, although these chronic infections most probably have a lesser demographic impact on populations (Ortner, 2003; İşcan & Steyn, 2013). In bone, bacteria are the main causative agent, as viruses causing diseases such as the plague and smallpox have a tendency to kill or is cured very quickly and therefore, does not affect the skeleton easily (Kumar et al., 2007; Ôsz et al., 2009).

Signs of infectious diseases on skeletal material from past populations can provide therefore information about the life history of a population or an individual, helping to understand how humans evolved and adapted to their environment (Cox & Mays, 2000).

2.4.2.1 Specific bone infections

The most common specific bone infections (diagnosed by their specific distribution pattern or focus on a particular part of the body), described in paleopathology literature are caused by

bacteria, such as *Staphylococcus aureus*, *Treponema pallidum* (syphilis), *Mycobacterium* (tuberculosis, leprosy) and *Brucella* (brucellosis) are recognised in human remains (Cox & Mays, 2000; Ortner, 2003; Chhem & Brothwell, 2008).

2.4.2.1.1 Tuberculosis (TB, Pott's disease)

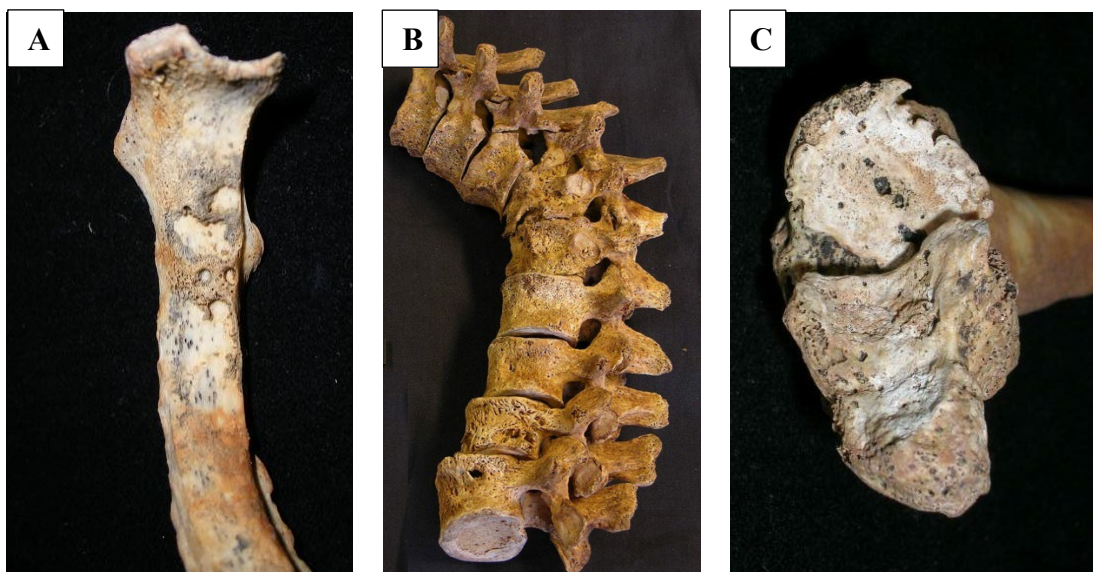
Tuberculosis (TB) is a chronic infection resulting from *Mycobacterium tuberculosis* bacteria, that can express itself in two phases, namely a primary and a secondary phase. The secondary phase can occur years later when a patient can become infected again by new tubercle bacilli (reinfection), or an old primary lesion can break down to release dormant tubercle bacilli contained within it (reactivation). Reactivation of the disease can occur when treatment is discontinued before completion (Steyn et al., 2013; O'Gradaigh & Conway, 2017). This is a common problem in SA and other third world countries as there is limited access to healthcare and individuals begin to feel better after about three months of treatment. However, the disease is not eradicated from the body until six to 18 months of treatment has been completed (Steyn et al., 2013). In many cases, this incomplete use of antibiotics causes drug resistant (XDR) and multi drug resistant (MDR) *Mycobacterium*-strains; (Steyn et al., 2013; Geldenhuys, 2014).

Although TB mainly affects the lungs, it is known to that can spread haematogenously to other organs as well, and is then classified as pulmonary (PTB) and extra-pulmonary TB (Mason et al., 2010; Steyn et al., 2013).

The prevalence of pulmonary TB on skeletal material can not accurately be determined without soft tissue analyses, although several authors (Roberts et al., 1994; 1998; Bennike, 1999; Souza, 2002; Steyn et al., 2013; Geldenhuys, 2014) indicated periosteal reaction on the visceral surface of ribs (Fig. 2.3A) are indicative of PTB on skeletal material, as the lungs are generally first to be affected at inhaling of the infected airborne droplets. However; these lesions is not exclusively TB lesions, it may be caused by other types of infections as well as respiratory system neoplasms (Matos & Santos, 2006; İşcan & Steyn, 2013; Geldenhuys, 2014). Worldwide males tend to have a higher chance to develop TB than females (Caracta, 2003; Austin et al., 2004; Balasubramanian et al., 2004, Neyrolles & Quintana-Murci, 2010). Collectively with the poor housing, overcrowding and low sanitation, an association between high alcohol consumption and the risk of PTB has been observed (Addolorato et al., 1998; Rehm et al., 2010). In the cadaver study by Geldenhuys (2014) and Geldenhuys et al. (2016) a

statistical noteworthy association was observed between the prevalence of TB and liver disease associated with heavy intake of alcohol.

Deaths due to PTB were recorded throughout the 20th century with the highest mortality rate among the SAC population group (Cluver, 1959; Van Rensburg & Mans, 1982; Coovadia, 2009; WHO, 2011). Although, recent studies suggested that polymorphisms in the human *NRAMP1* gene resulted in increased susceptibility to the *Mycobacterium*-complex infection, mainly in the West African population (Stead et al., 1990; Bellamy et al., 1998; Thwaites et al., 2000). This devastating disease is life-threatening and in 1993, it was estimated that half the individuals who acquired TB died within six to 24 months (Steyn et al., 2013). In a study done on a Western Cape cadaver cohort (n=127), mainly from the SAC population group, dissected by Stellenbosch University's medical students, Geldenhuys (2014) found that pulmonary tuberculosis (PTB) was present in 76.4% individuals, with 22.8% presenting with extra-pulmonary TB. The average age of the individuals affected with rib lesions in the Geldenhuys (2014) study was 47.3 years with the young adult age group (ages between 20 to 39 years) statistically significantly more affected.



Source: Museum of London. St. Bride's lower churchyard photographs.
<https://www.museumoflondon.org.uk/collections>

Figures 2.3A-C. Tuberculosis are seen as pulmonary or extra-pulmonary infections A) PTB as periosteal reaction on the visceral surface of ribs, or B) Pott's disease on the vertebral column; or C) manifest on postcranial bones (right radius that fused with the scaphoid).

Extra-pulmonary TB can cause Pott's disease in the vertebral column (Fig 2.3B) or tuberculous arthritis on the peripheral joints (Fig 2.3C). Tuberculous arthritis occurs when

Mycobacterium bacilli are spread from the primary infection site, usually the chest, to lodge in the vertebral column or weight-bearing joints (El Titi et al., 1987; Pigrau-Serrallach & Rodríguez-Pardo, 2013). Extra-pulmonary TB of the musculoskeletal system is usually chronic with the progression of the infection being slow (Matos & Santos, 2006; Mason et al., 2010; Steyn et al., 2013; O'Gradaigh & Conway, 2017). Only 3-5% of patients with TB will show osteoarticular dissemination (Kumar et al., 2007; Steyn et al., 2013). Pott's disease presents as lytic activity of the anterior vertebral body, causing collapse, creating a wedge-shaped vertebra that will result in angular kyphosis in the advanced stages (Aufderheide & Rodríguez-Martín, 2011) or it can present as a bony ankylosis of several vertebral bodies. The vertebral attack by tuberculous-bacteria is rarely on the posterior elements of the vertebrae, as the bacteria are deposited particularly in areas of trabecular bone (as seen anteriorly), which has a high circulatory rate (Ôsz et al., 2009). Authors report on vertebral lesions are localised mostly in the thoracolumbar region and involve the vertebral bodies of one to three, maybe four, vertebrae at the most (Ortner, 2003).

Other joints include monoarticular involvement in mainly the weight-bearing joints such as the knee and hip joints due to good blood supply which provide an optimal environment for tuberculosis-bacteria (Ôsz et al., 2009). It causes fibrous ankylosis of the joint with obliteration of the joint space as well as the sacroiliac joint (up to 15.0%) and less than 10.0% in the other joints such as the ribs, shoulder, ankle, elbow, and wrist in order of prevalence (La Fond, 1958). It is difficult to detect, especially in skeletal remains as examination and culture of the synovial fluids is mainly used for positive diagnosis.

2.4.2.1.2 Localised cranial infections

a) Concha bullosa

This condition, also known as pneumatized turbinate and nasal turbinate hypertrophy, is a common anatomical variant and presents as enlarged nasal conchae that appear swollen and rounded. Clinical differential diagnosis of hypertrophic nasal conchae have included conditions such as fibrous dysplasia, rare neoplasms or concha bullosa (Mays et al., 2011). Although still under debate, concha bullosa has been suggested by various authors to be a result of weather or temperature changes, stress, fatigue, medication, sinusitis, chronic allergies and hormone changes from thyroid disorders (Campillo, 2005; Hatipoğlu et al., 2005; Kwiatkowska et al., 2011; Mays et al., 2011; Cukurova et al., 2012; Mays, 2012; Mays et al., 2012). The presence of concha bullosa in clinical publications ranged from 9% (Lothrop, 1903) to 56% (Ünlü et al.,

1994) and even up to 72.2% (Keleş et al., 2010). It is unclear if this large variation is due to population variation or methodological differences. Several clinical reviews, studying CT scans from patients, found a strong association between the presence of a unilateral concha bullosa and deviation of the nasal septum (Lebowitz et al., 1995; Aktas et al., 2003; Stallman et al., 2004) which itself has been linked to sinus diseases (Calhoun et al., 1991). Some authors found no increased incidence of paranasal sinus disease (sinusitis) in patients with concha bullosa (Calhoun et al., 1991; Stallman et al., 2004); however, other studies have suggested a relationship between a concha bullosa and sinus disease, as these enlarged concha can cause a blockage which can prevent sinuses from draining properly, resulting in frequent sinus infections (Lam et al., 1996).

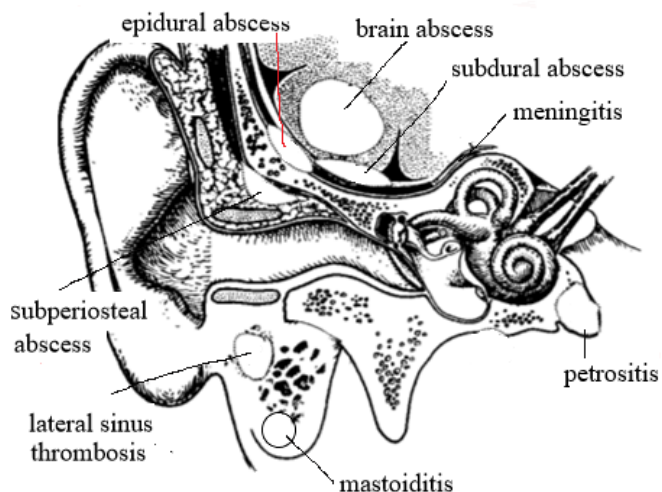
b) Otitis media, petrositis and mastoiditis

Anatomically, the middle ear is attached to the nasopharynx by the Eustachian tube and the mastoid pneumatic cells lies posteriorly. The air pressure in the middle ear, compared to the outside environment, is slightly negative and is relieved during yawning and chewing. If the Eustachian tubes are obstructed due to congestion after an upper respiratory tract infection (pneumonia, influenza, tuberculosis, exanthemata), it can prevent the air in the middle ear to be relieved. The negative pressure increases, causing an increase in the microbe-loaded secretion in the middle ear, resulting in symptomatic otitis media (Rovers et al., 2006; Winther et al., 2007). Chronic otitis media result in osteitis that destroys the bone and helps the infection to penetrate deeper. A complication of otitis media is consequently further spread of these bacteria from the middle ear leading to inflammation of the petrosal and mastoidal air cells (petrositis and mastoiditis) (Fig. 2.4). Therefore, chronic allergies of infections result in osseous changes in the internal acoustic meatus (otitis media) the petrous part of the temporal bone (petrositis), and mastoid process (mastoiditis) (Flohr & Schultz, 2009). Bony layers can be deposited, forming plate-like proliferations with spicules in the external acoustic meatus and on the tympanic plate while complete sclerosis of the mastoid air cells due to pressure in the cells caused by the exudate (pus) non draining, can appear when mastoiditis is suspected (Flohr & Schulz, 2009). Dalby (1994) attempted to determine standard non-destructive criteria for diagnosis.

Risk factors for otitis media include exposure to tobacco smoke, air pollution (Greenberg et al., 2006), asthma (Eldeirawi & Persky, 2004) lack of medical care access, family history, genetics and population groups (Casselbrant et al., 1995). Clinical studies demonstrated that

susceptibility varies by age and population group (Mann & Murphy, 1990). Otitis media is common in the first period of life (birth to 28 days), declining after the first birthday. A clinical study by Teele et al., (1984) on children in Boston, USA, found that 9.0% experienced at least one episode of otitis media by age three months, and 65.0% by 24 months of age. Pestalozza (1984) reported that 21.1% of infants in a neonatal pathology ward, were diagnosed with otitis media.

Risk factors suggested for further complications to occur include immune compromised adults, elderly patients with low resistance as well as communities with poor SES where over crowding, poor health education, poor personal hygiene and limited access to health facilities (Vasquez et al., 2003). Authors reported a high incidence of otitis media in Indians (Jaffe, 1969; Gregg et al., 1965), Eskimos (Kaplan et al., 1973), and Australian aborigines (Morris, 1998), while, American Whites (Casselbrant et al., 1995) and Blacks (Griffith, 1979) experience low frequencies of these infections.



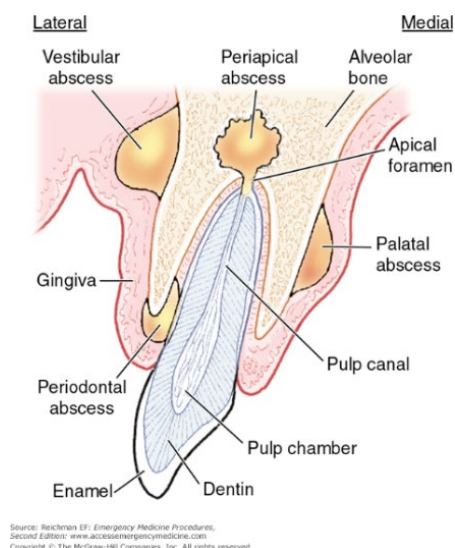
Source: Vasquez et al., 2003.

Figure 2.4. Suppurative complications in otitis media infections.

c) Periodontal disease and oral health

Globally, societies are affected by dental caries, irrespective of sex, age or SES (Peckmann, 2002). The type of food ingested, and the manner in which it is prepared strongly influences the caries rates in human populations (Peckmann, 2002). Maize, in particular, has been historically shown to have a high sucrose content and can cause high caries rates as sucrose is rapidly metabolised by oral bacteria (Larsen et al., 1991).

Dental plaque or calculus builds up on teeth due to precipitation of minerals from saliva and gingival crevicular fluid on teeth. This may cause the health of the gingiva to deteriorate. No reports on calculus rates for population groups are available. A periodontal or parietal abscess occurs alongside a tooth (Fig. 2.5) and is a localised acute, destructive bacterial infection with a collection of pus within the tissue of the periodontium, but not arising from the tooth pulp (Avelar et al., 2009). It has been reported to be the third most frequent dental emergency demonstrating 7-14.0% of all dental emergencies, and often develops as a result of gingival disease. Conversely, a periapical or radicular abscess is caused by a bacterial spread arising from the tooth pulp that endured pulpal necrosis at the apex. A granuloma or cyst has an epithelial lining and develops over a period of time (chronic) where the granulation tissue is chronically inflamed. The lesion may penetrate the mandible or maxilla. The prevalence of these abscesses ranged from 7.8% (Ledesma-Montes et al., 2000; Avelar et al., 2009) to 55.0% (Ramachandran Nair, 2003). It was reported that periapical cysts are more prevalent on the maxilla than the mandible (Ramachandran Nair, 2003) and are more common in the 30 to 39 age group, with a male predilection (Avelar et al., 2009). On dry bone, the healed abscess will show pitting due to the periosteal reaction around the sinus that formed and the margins will be smooth, rounded, and of similar texture as the surrounding bone (Avelar et al., 2009).



Source: Reichman, 2013 p1142

Figure 2.5. Diagram showing location of dental abscesses.

2.4.2.2 Non-specific bone infections

The non-specific infections most commonly reported in literature include infections affecting the periosteum (periostitis), cortex (osteitis) and medullary cavity (osteomyelitis) of

bone (Cox & Mays, 2000). The presence of non-specific periosteal bone lesions increase as adverse living conditions increase and are important in the assessment of health in a community (Larsen, 1997) such as the low SES communities in and around Cape Town.

2.4.2.2.1 Periosteal reaction

A periosteal reaction, mainly (incorrectly) called periostitis in literature, is the most common non-specific inflammatory reaction of the periosteum (the connective tissue layer surrounding the bone) resulting from pathological changes of the bone beneath it (Weston, 2012). The reaction can occur on any bone of the skeleton and has no sex bias (Ôsz et al., 2009). A systemic inflammatory response will generally be observed as a bilateral reaction on the bones, while an inflammatory response after a traumatic event will be evident as an unilateral focal reaction (Aufderheide & Rodríguez-Martín, 2011).

The lesions manifesting on certain bones or areas are specific for the area, for example, a systemic periosteal reaction of new bone production, is general seen on the long bones of an individual, particularly the tibiae (Weston, 2012). Furthermore, lesions on the maxillary sinuses (evidence for sinusitis), the endocranial surfaces of the skull (referring to meningitis) and the visceral surface of ribs (suggesting a lung infection) suggest specific periosteal reactions (Schultz, 2001). In paleopathological literature, periosteal reactions have been described in specific infectious diseases including TB (Roberts et al., 1994, Steyn et al., 2013), syphilis (Hackett, 1975; Rothchild & Heathcote, 1993), leprosy (Lewis et al., 1995), HOA (Fennell & Trinkaus, 1997) and scurvy (Ortner et al., 2001). Specific diagnosis for these lesions is however difficult, since mechanical stress on the periosteum, for example trauma or subperiosteal bleeding as it is seen in scurvy (Ortner et al., 2001; Schultz, 2001), can produce very similar changes (Ôsz et al., 2009). Therefore, most authors mention a periosteal reaction caused by a non-specific infection (Larsen, 1997; Ortner, 2003; Larsen et al., 2007), meaning that an infection was present, but the cause was unknown. The periosteal reaction may; however, have various aetiologies, and can be caused by trauma, dislocation of joints, localised ulcerations, malnutrition, neoplasms or venous insufficiency due to varicose veins (Mann & Murphy, 1990; Larsen, 1997; Ortner, 2003) and to assume that the inflammatory response is due to an infection is a precarious assumption (Weston, 2012).

2.4.2.2.2 Osteitis

Osteitis, also known as ostitis, refers to inflammation of the bone substance that can be caused by a primary bone infection, trauma or degeneration of the bone due to age-related

erosion (Tiemann & Hofmann, 2009). This condition can lead to destruction of the bone, as well as the surrounding soft tissue. Hofmann and colleagues (1997) classified osteitis as: a) postoperative osteitis where, within the first eight weeks of trauma or after an operation, a bacterial infection of the bone and surrounding soft tissue occurs; and b) chronic osteitis, where the infection occurs more than eight weeks after a surgical intervention or injury.

Schmidt et al. (1997) classified osteomyelitis and osteitis according to the predisposing factors involved, namely: a) endogenous factors: age over 65 years, obesity, nicotine and alcohol abuse, diabetes, vascular diseases, immunosuppressive therapy, cancer or general debility which can lead to suppression of cellular and humoral resistance to infection; and b) exogenous factors: bacterial invasion and soft tissue damage due to trauma or surgical manipulation, which are the main contributing factors for the development of post-traumatic bone infection.

The main difference between osteitis and osteomyelitis is the way in which the infection affects the bone (Tiemann & Hofmann, 2009), although in the later stages, it is difficult to differentiate between chronic haematogenous osteomyelitis and exogenous (post-traumatic or postoperative) osteitis. Osteitis and osteomyelitis are among the most severe diseases affecting bone and the surrounding soft tissue in terms of duration of the treatment and complications (Tiemann & Hofmann, 2009). The mortality rate for patients with open fractures was up to 60.0% as recently as the late 20th century, but due to modern targeted therapy strategies, it is presently as low as 2.0% in first world countries (Schwameis et al., 1996).

2.4.2.2.3 Osteomyelitis

Osteomyelitis is an infection of the bone (osteitis) and bone marrow (myelitis) caused by pus-producing bacteria. It is a highly variable disease in patients with regards to its clinical presentation and management, as well as its pathophysiology (Berbari et al., 2010). Unique characteristics of osteomyelitis include the presence of a sequestrum (necrotic bone), an involucrum (highly vascular, enveloping new bone) and cloacal openings (for pus to escape). Without all three features present, one cannot diagnose osteomyelitis with confidence and other disease possibilities should be considered. Osteomyelitis of the vertebrae can be confused with Pott's disease (TB) when destruction of several adjacent vertebrae are involved. In diagnosis, TB rarely shows destruction of the vertebral arches, joints and transverse processes, but in osteomyelitis it can occur (Ortner, 2003). This condition affects males and females equally (Lew & Waldvogel, 2004) and is usually more common in children and adults of 50 years and

older (Underwood, 2004). It may be present in any portion of any bone (Lew & Waldvogel, 2004). Risk factors include a removed spleen, recent trauma, diabetes, hemodialysis and intravenous drug abuse (Lazzarini et al., 2004). The most common bones involved in osteomyelitis are long bones such as the femur, tibia, humerus and radius (Agaja & Ayorinde, 2008; Beckles et al., 2010; Aufderheide & Rodriguez-Martín, 2011).

2.4.3 DEFICIENCY DISEASES

The study of deficiency diseases plays an important role in improving the understanding of aspects of life in both past and present communities. These aspects include living and environmental conditions, social and cultural practices as well as the impact and effects of the aging process (Brickley & Ives, 2008).

2.4.3.1 Metabolic and nutritional stress disorders

A variety of diseases or disorders are caused by malnutrition, that can include both undernourishment (not having enough to eat) and malnourishment (not having the right balance of nutrients), or insufficient absorption of ingested nutrients (Ortner, 2003). Specific metabolic disorders, nutritional deficiencies and hormonal disturbances can be difficult to determine because malnutrition usually comprises of deficiencies of more than one specific nutrient (Goodman & Armelagos, 1988; Huss-Ashmore et al., 1982).

Bone is easily affected by nutrition and hormones, and evidence of nutritional disorders can be observed in bones, causing, for example, inadequate bone mineralisation, resulting in excessive deossification of bone as seen in vitamin D deficiency diseases (osteomalacia), or insufficient osteoid production resulting in a reduction in bone mass as seen in vitamin C deficiency or scurvy (Ortner & Putschar, 1985; White et al., 2012). Malnutrition and endocrine disorders are linked to hormonal influences as bones serve as a storage organ to maintain balanced calcium levels in the blood. When there is too little calcium in the blood (hypocalcaemia), the parathyroid hormone activates osteoclasts to resorb bone to release calcium; if the lack of calcium continues, osteoblasts cannot lay down the same amount of bone that is being resorbed and the bones become osteoporotic (Cox & Mays, 2000).

2.4.3.1.1 Harris' lines (HL)

Harris' lines are thin, radiopaque, sclerotic, transverse lines which develop in the metaphyses of long bones, and are visible on anteroposterior radiographs (Harris, 1933). Several authors have suggested that these lines form during the recovery phase after episodes

of childhood diseases, when longitudinal growth in bone was temporary arrested (Park & Richter, 1953; Platt et al., 1963; Park, 1964; Byers, 1991; Larsen, 1997; Aufderheide & Rodriguez-Martín, 2011). Although it is still under debate whether HL form as a reaction to short or long-term factors delaying an individual's growth, Nowak & Piontek (2002) found that the morphology and growth of the long bones showing HL, are not affected. Most studies conducted in the past found that the stature of individuals were not affected by the presence of HL (Gindhart, 1969; Wells, 1967; Papageorgopoulou et al., 2011). However, a study on Guatemalan children showed children with HL were shorter when compared to children with no HL (Acheson et al., 1974).

Authors have, in addition, suggested that childhood episodes resulting in disturbance in growth, can include malnutrition (Platt et al., 1963; Manzi et al., 1989), infection (Gindhart, 1969), trauma (Resnick, 1995), psychological stress (Sontag & Comstock, 1938), or dietary imbalances, specifically resulting from protein or vitamin deficiencies (Dreizen et al., 1964; Gray, 1967; Clark, 1978; Goodman & Clark, 1981), and may not necessarily be related to a specific disease (Peckmann, 2002). These lines are therefore used to determine the stress history of an individual, and along with periodontal diseases, dental enamel hypoplasia (DEH) and metabolic deficiency indicators, such as cribra orbitalia, the health status of a population can be estimated. Papageorgopoulou and colleagues (2011), however, suggested that HL should rather be considered a result of normal growth and growth spurts during childhood, rather than a pure outcome of nutritional or pathological stress. Although their findings are valid and should be explored, this current study did not include estimation of age-at-formation of HL and can therefore not comment on this finding.

Harris' lines form when the growth plates of an individual are most active and before the epiphyseal plates close. It was observed that these lines decrease in frequency with age (Harris, 1933). As an individual grows, the lines can undergo resorption and appear broken and thinner, or even disappear with age (Caffey, 1978; Garn et al., 1968). Therefore, the radiographic appearance of HL will decrease with age due to remodelling of bone (Marshall, 1968). It can therefore be deduced that the older an individual is, and the more remodelling bone has undergone, the greater the chances that the HL will no longer be visible.

Nowak & Piontek (2002) identified that, regardless of sex, the periods of the most concentrated influence of stress factors responsible for the formation of HL were between the ages of 3 and 6, and 6 and 12 years-of-age. Ameen et al. (2005) compared the incidence of HL

in tibiae of children of two medieval populations (n=112) from Switzerland with those of a modern population (n=138) living in the same geographical area. While no difference in incidence were found between the sexes, 80.0% of the medieval skeletons showed HL on the tibiae, while only 20.0% of the modern individuals showed HL on their tibiae. This discrepancy can be ascribed to hardships experienced during the medieval period, such as the lack of medical treatment and poor nutrition.

According to various authors, Harris' lines are most commonly seen on both ends of the tibia, followed by the femur and the distal radius (Park, 1964; Wells, 1967; Garn et al., 1968, Clarke, 1982; Maat, 1984; Goodman & Armelagos, 1988; Hughes & Saifuddin, 2006). Other bone involvement includes the anterior ends of the ribs (Park, 1964) and the metacarpals (Garn et al., 1968). No lines have been observed in the ulna or proximal radius as yet. Although males and females show no difference in frequency of HL on long bones (Huss-Ashmore et al., 1982; Webb, 1984), age (Piontek et al., 2001) and stress factors in the environment (Hatch et al., 1983; Ameen et al., 2005) can have an influence on its presence on bones.

2.4.3.1.2 Dental enamel hypoplasia (DEH)

Dental enamel forms during juvenile growth and, in contrast to bone, never remodels after this formation. Dental enamel hypoplasia (DEH), also called dental hypoplasia, hypoplastic lines, linear enamel hypoplasia (LEH), or enamel dysplasia is a nonspecific quantitative enamel defect, presenting as three main forms of defects on teeth, as classified by Lukacs (1989): a) linear horizontal grooves (LEH), b) linear/non linear pits, and c) linear grooves and pits in the tooth crown enamel as a result of defects in ameloblastic activity. This dental defect represents a short period when growth slowed down or stopped during the formation of the tooth crown (the foetal period) until the age of eruption of the last tooth (Goodman & Rose, 1990; Steckel & Rose, 2002). These lines or pits are excellent measures of childhood nutritional and morbidity stress, indicating the individual's health or/and nutritional stress during the growth stages (Huss-Ashmore et al., 1982; Goodman & Armelagos, 1988). The exact aetiology of hypoplasia cannot be specified potential contributing factors include periapical inflammation or trauma to a deciduous tooth, fever, disease, nutritional deficiencies, endocrine dysfunction, and generalised infection during odontogenesis (Robinson & Miller, 1983). Although genetic predisposition should not be excluded, differences in living conditions, environmental influences and underlying diseases, such as hypoparathyroidism and hypocalcaemia (Goepferd & Flaitz, 1981) should also be taken in consideration.

Dental enamel defects are more favoured for assessing growth disruptions when compared to HL. Firstly, this is due to the process of bone remodelling throughout an individual's life leading to the resorption of HL and, secondly, due to the fact that dental development is much less affected by environmental stress than bone growth. According to research, minor stresses episodes in a person's life may result in the formation of HL, but not to DEH (Mays, 1995; Papageorgopoulou et al., 2011).

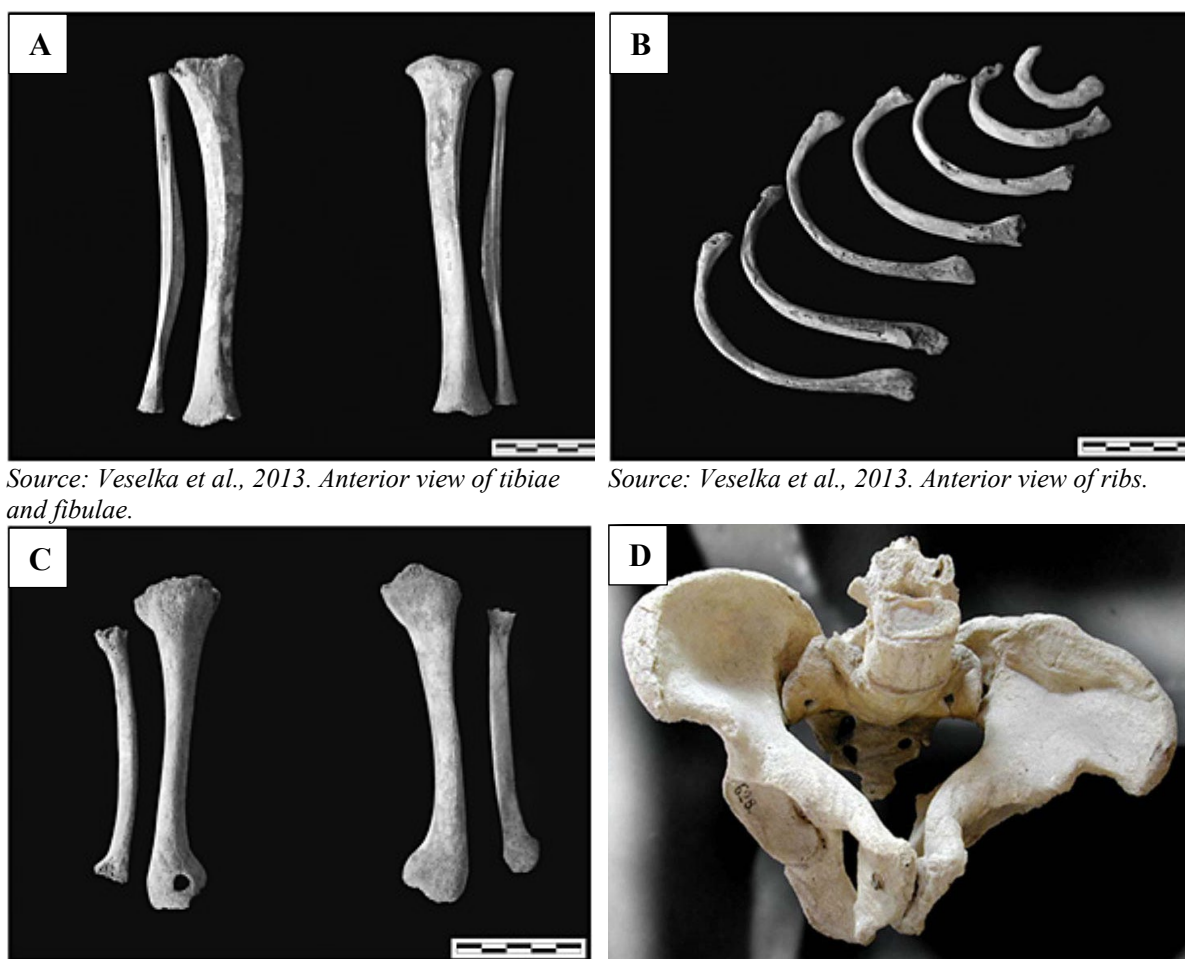
In population-specific studies, the prevalence of DEH in different populations vary between 3.0% to 99.0%. Genetic factors, as well as exposure to similar environmental stresses can have an influence on the susceptibility of DEH (Hart et al., 2002; Brook, 2009), for example, a study on young Australian Aboriginal children from Bathurst Island (Pascoe & Kim Seow, 1994), showed a prevalence of 99.0% DEH, with nearly all the patients presenting with a full range of medical problems from similar environmental stressors. A similar study on Guatemalan children, however, showed a significantly higher prevalence of DEH among siblings, while the general prevalence of DEH in the population fell between 18.0% and 24.0% (Infante & Gillespie, 1974), and in this instance, suggesting a genetic influence. A Polish study by Kozak & Krenz-Niedbala (2002) showed a total of 22.4% of individuals with prevalence of DEH. Children, from mainly the White population group, from an Australian hospital in Brisbane showed a prevalence of only 3.0% DEH on their teeth (Seow et al., 1987). Most studies comparing sexes showed no difference in the prevalence of DEH/LEH (Infante & Gillespie, 1974; Kozak & Krenz-Niedbala, 2002), although a study conducted by Li et al. (1995) showed a significantly higher prevalence noted in males compared to females. The maxillary incisors and the mandibular canines are the preferred teeth to be examined for DEH, according to Goodman and his colleagues (1980).

2.4.3.1.3 Rickets / Osteomalacia

Vitamin D is found in some food, including fatty fish, liver and eggs, but is also synthesised by the skin from sunlight. The body needs Vitamin D to absorb calcium in the intestinal canal. Calcium is needed to mineralise growing bone and for normal bone turnover and repair, even in adults. Vitamin D-related deficiencies from chronic lack of sunlight, a diet poor in animal fats and calcium, or the body's inability to break down and use Vitamin D (Bringhurst et al., 2008) leads to poor mineralisation and high flexibility of bone (Vigorita, 1999). Vitamin D deficiency can be due to risk factors such as darker skin pigmentation, pollution, rainy and overcast winters, and covering skin for religious or cultural reasons (Elder

& Bishop, 2014; Kumar et al., 2007). Studies on children from several sub-Saharan African countries have found that rickets is caused by chronic dietary calcium deficiency and can be cured nutritionally by calcium supplementation alone (Thacher et al., 1999; Braithwaite et al., 2016). Thacher et al. (2013) recorded an increase in rickets and osteomalacia in more industrialised nations due to the increase in infrastructure, taller buildings blocking out sunlight, and city dwellers remaining mostly indoors. Recent dietary and biochemical studies in Kenya reported a significant burden of rickets amongst children living in informal urban settlements where rickets was associated with acute malnutrition and developmental delay. The researchers provided evidence that deficiencies in both calcium and vitamin D are playing a role in this metabolic disease (Edwards et al., 2014; Jones et al., 2017).

Vitamin D deficiency in children is called rickets and in adults it is called osteomalacia, and can be observed in bone (Ortner, 2003). In children, before epiphyseal closure, rickets can cause a variety of skeletal abnormalities varying in relation to age and developmental stage, including bowing of the tibia and fibula (Fig. 2.6A), rachitic rosary (beading) at the costochondral junctions (Fig. 2.6B), pectus carinatum (pigeon breast), thoracic kyphosis, lumbar lordosis-scoliosis, wrist widening (Fig. 2.6C) and triradiate configuration (Fig. 2.6D) of the pelvis due to a loss of structural rigidity in developing bone (Kumar et al., 2007; Aufderheide & Rodriguez-Martín, 2011; Veselka et al., 2013; Elder & Bishop, 2014).



Source: Veselka et al., 2013. Anterior view of tibiae and fibulae.

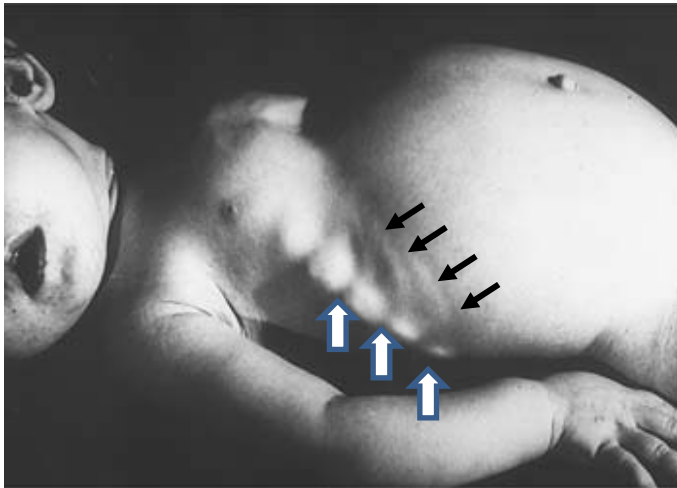
Source: Veselka et al., 2013. Anterior view of ribs.

Source: Veselka et al., 2013. Anterior view of humeri and radii.

Source: Brickley, 2008. Anterior view of pelvis

Figures 2.6A-D. Signs of rickets observed on dry bone includes A) bowing of long bones; B) rachitic rosary (beading) at the costo-chondral junctions; C) wrist widening; and D) triradiate configuration of the pelvis.

Clinically, the rachitic rosary at the rib ends is radiologically seen as round ossified knobs and as the bones of the ribcage are weakened, the diaphragm pulls the softened lower rib bones inward inferiorly and forms a horizontal Harrison's groove or sulcus (Fig. 2.7) along the lower border of the intact thorax (Brickley & Ives, 2008). Young infants can present with symptomatic hypocalcaemia (Ladhani et al., 2004; Hogler, 2015), and softening of the skull (craniotabes) with frontal bossing and delayed fontanelle closure. Impaired linear growth and developmental delays are common.



<http://gabeents.com/Data/Patologia/Pathologic/nutk1/record0006.html>

Figure 2.7. A young patient with signs of rickets such as rachitic rosary (beading) at the costo-chondral junctions (block arrows) and the horizontal Harrison's sulcus (line arrows).

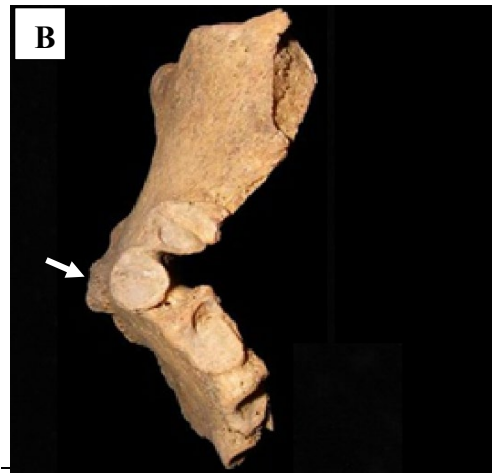
A child can recover from this disease, although the curvature of the long bones (Fig. 2.8A) and the pigeon breast (Fig. 2.8B) are often retained throughout life. Rickets during childhood can cause dolichocephaly or premature closure of the sagittal suture that may persist to adulthood, presenting as bossing or protruding of the frontal bone (Fig. 2.8C). Osteomalacia in adults is less severe as the period of bone growth has passed and is defined by defective mineralisation of osteoid in mature compact and spongy bone, occurring during normal bone turnover. It may not be the result of a defect in vitamin D metabolism, although it usually is (Brickley & Ives, 2008). The main abnormality in osteomalacia is the presence of high amounts of inadequately mineralised osteoid laid down by osteoblasts, which deranges normal bone remodelling that occurs throughout life. (Ortner, 2003; Kumar et al., 2007; Brickley & Ives, 2008; Aufderheide & Rodriguez-Martín, 2011). Although the bone contours are not affected, the bone is weak and exposed to both macro or microfractures. These fractures do not repair properly due to the deficiency in available calcium. The area of healing is relatively weak, and in some cases will fracture persistently. Eventually, a mass of poorly mineralised bone will develop around the fracture site. This weakened bones can cause bilateral coxa vara, a deformity of the femoral head and proximal shaft whereby the angle between the head and the shaft is less than 120° (Fig. 2.8D).

Radiographically, these pseudo or stress fractures can be seen as Looser's zones, often symmetrical in distribution, and are mainly seen in the pubic rami and femoral necks, scapulae, ribs, long bones, and metatarsals. Looser's zones are focal accumulations of osteoid in compact

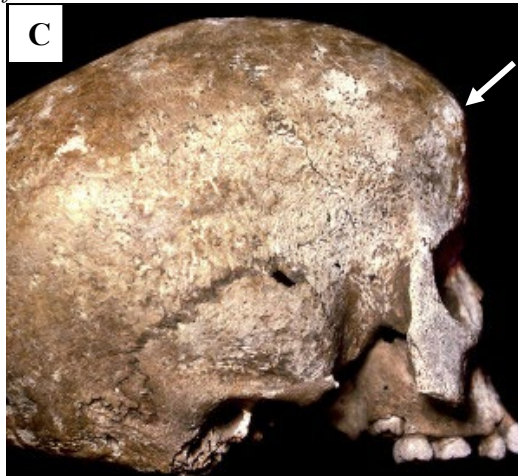
bone perpendicular to the long axis of the bone (Brickley & Ives, 2008). Other radiological findings include intracortical bone resorption or cortical tunnelling of the long bones (Favus et al., 1999), thinning and loss of secondary trabeculae, causing the trabecular pattern to appear coarsened, and hyperplasia of the metaphyseal cartilage, presenting as widening of the joints as seen in children.



Veselka et al., 2017. Anterior view of tibiae and fibulae



Source: Armit et al., 2015. Lateral view of manubri sternum.



Source: Tilley, 2015. Right lateral view of skull.



<https://www.theptdc.com/2015/02/why-people-must-have-different-squat-stance/>
Anterior view of femora.

Figures 2.8A-D. Signs of osteomalacia observed on dry bone of adults include A) Bowing of the long bones; B) pectus carinatum of the sternum (arrow); C) protruding or bossing of the frontal bone (arrow); and D) coxa vara (arrow), compared to a normal femur head on the right side.

2.4.3.1.4 Scurvy

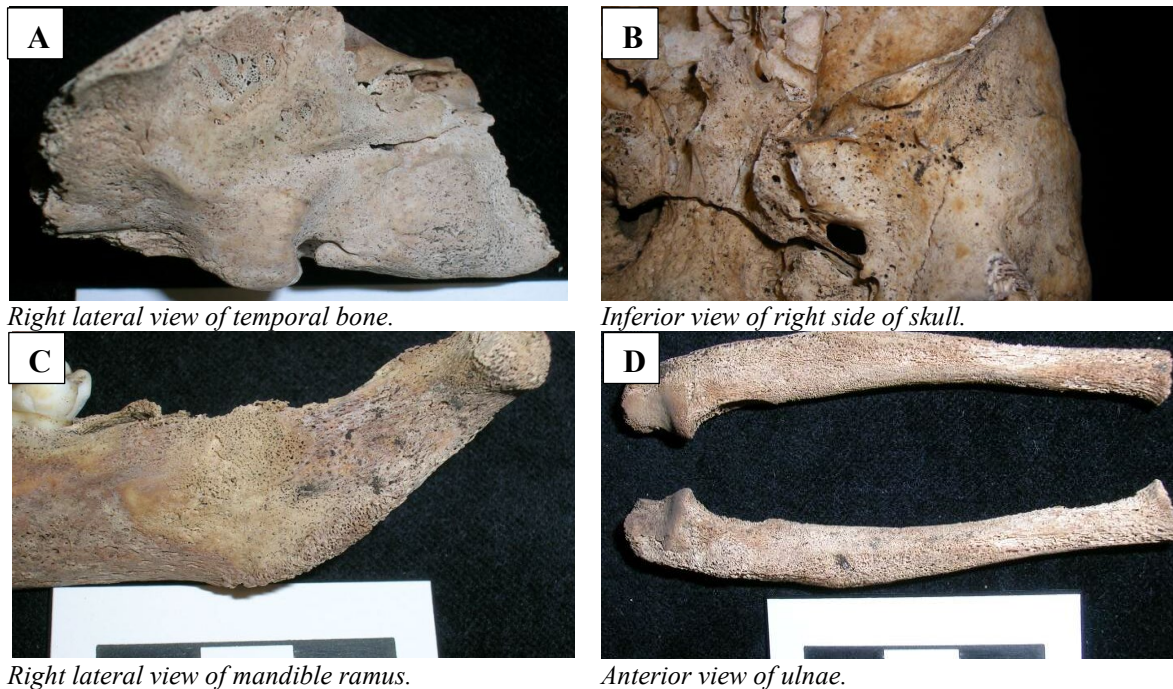
Vitamin C is needed by the body for the formation of collagen, which is found in tendons, ligaments, skin, bone, cartilage and blood vessels. Scurvy is produced by a deficiency in ascorbic acid (vitamin C), resulting in a reduction in the formation of osteoid in bone and general weakness in the connective tissue in the body (İşcan & Kennedy, 1989; Aufderheide

& Rodríguez-Martín, 2011). Although the human body is not able to produce vitamin C, it does store it (Waldron, 2009). Therefore it can take months for an adult on a vitamin-deficient diet to show symptoms of scurvy as seen in men at war or at sea. In bioarchaeological studies it is commonly accepted that a lack of fresh fruit and vegetables is the cause of this deficiency (Fain, 2005), however, there are a multitude of other non-dietary variables that can lead to, or play a role in the development of scurvy (Delanghe et al., 2007). Causes of scurvy can be classified into four groups (Halcrow et al., 2014): 1) reduced intake of vitamin C, 2) increased requirements for vitamin C, 3) malabsorption of vitamin C, and 4) genetic predisposition to lowered vitamin C levels (Delanghe et al., 2007).

Scurvy is primarily seen as haemorrhages, which occur from ruptured blood vessels, which have a weakened structure from impaired collagen formation (Brickley & Ives, 2008). The reaction of this proliferation of blood vessels can cause an increased porosity in the cortical bone (Ortner & Ericksen, 1997; Ortner, 1999; Ortner et al., 2001). Scorvic individuals show signs of scurvy at locations where mechanical stress is experienced and where the arteries lie between the muscle and the bone. Muscle contraction can trigger a haemorrhage, and chronic bleeding from small blood vessels near the skin's surface or areas of muscle activity, can induce an inflammatory response followed by vascular proliferation (Ortner & Ericksen, 1997; Brickley & Ives, 2006; Halcrow et al., 2014). On the skull, for example, the temporalis muscle is an ideal site for signs of scurvy. Porosity and woven bone deposits are observed in areas associated with the temporalis muscle such as the infratemporal (posterior) surface of the maxillae, internal surface of the zygomatic bone and temporal fossa (Fig. 2.9A), which includes the greater wing of the sphenoid (Fig. 2.9B), that has been suggested to be a predominantly diagnostic symptom (Ortner & Ericksen, 1997). Furthermore, signs of haemorrhage are particularly visible on the lateral roofs of the eye orbits, the result of minor eye movements, which are enough to rupture the weakened ocular vessels of individuals with scurvy (Ortner, 1999; Ortner et al., 2001). Other evidence of scurvy in dry bone is based on the presence of antemortem tooth loss due to gingival bleeding (Fain, 2005; Popovich et al., 2009) with necrosis and reactive new bone formation (Ortner et al., 2001), alveolar osteitis or resorption of alveolar bone (Maat, 2004) (Fig. 2.9C). According to Brickley & Ives (2006) cranial lesions of scurvy will only appear with accompanying postcranial lesions, therefore, regardless of the distinctive anatomical patterning of cranial lesions, a diagnosis of scurvy should not be made based on the presence of only orbital and cranial lesions. Postcranially, the skeletal elements with muscles with vascular supplies adjacent, include the scapulae and the long bones (Fig.

2.9D). On the tibiae and fibulae symmetrical ossified subperiosteal haematomas can be evident (Maat, 2004) and on dry bone, the osseous lesions are visible as symmetrical layers of porous woven bone on the bone surfaces, and periostitis of the long bone diaphysis. However, once the person recovers from scurvy, the capillaries stop bleeding and the bone heals; hence, scurvy is only seen in people who died either when the disease was active or shortly after they recovered.

Numerous literature on scurvy is presented on individuals from the Western world (Maat, 2004; Ortner & Ericksen, 1997; Ortner, 1999; Ortner et al., 2001; Ferreira, 2002; Brickley & Ives, 2006; Mays, 2008; Brown & Ortner, 2011; Geber & Murphy, 2012), a few studies on individuals with scurvy in tropical environments (Ortner et al., 2001; White et al., 2012) and almost no reports of scurvy from Asia (Halcrow et al., 2014).



Source: *St. Bride's lower churchyard photographs*. Source: <https://www.museumoflondon.org.uk/collections>
 Figure 2.9A-D. Signs of scurvy on dry bone is evident as marked porosity and woven bone activity on A) the endocranial aspect of the petrous bone; B) the lateral aspect of the greater sphenoid wing; C) the outer mandible; and D) gross new bone formation on shafts of the ulnae.

2.4.3.2 Haematological disorders

The survival and function of most metabolising body cells are dependent upon a continuous and sufficient supply of oxygen. A lack of oxygen in the blood can cause premature red blood cell death and increased erythropoiesis (or red blood cell production). Lack of oxygen in the blood can be caused by iron-deficiency-, hereditary-, megaloblastic and haemolytic anaemias (Ôsz et al., 2009) as a result of insufficient iron in the diet or an inability to absorb

sufficient amounts of iron in the gastrointestinal tract (Cohen & Armelagos, 1984). This group of anaemic conditions are, however, multifaceted (Brickley, 2018). In literature, various risk factors for megaloblastic, haemolytic or iron deficiency anaemia have been suggested. Risk factors suggested as a cause of megaloblastic anaemias are, for example, vitamin deficiencies, in particular Vitamin B₁₂ and folic acid, caused by either alcoholism, drugs, malabsorption due to toxins, or congenital factors (Walker et al., 2009). Haemolytic anaemias can have a congenital (autoimmune, Hodgkin's lymphoma, rheumatoid arthritis) or acquired (drug, malaria-infection) origin (Walker et al., 2009), while iron deficiency anaemia have been associated with age, lower income, diabetes, vascular diseases, and hypertension (Zakai et al., 2008).

Skeletal indicators of haematological disorders can be seen in anaemias, lymphomas and multiple myelomas. Various signs on bones are diagnostic to the anaemic disorders, such as radiologically visible metaphyseal widening and cortical thinning of long bones, as well as widening of diploe, seen as the "hair-on-end" appearance on flat bones, caused by coarsening of the trabeculae, or the multiple "punched-out" lytic lesions of multiple myeloma lesions (Angel, 1966; Resnick & Niwayama, 1995). In clinical literature notable skeletal lesions due to acquired anaemia are rarely reported, and only severe, chronic cases of anaemia will show lesions on bone. Brickley (2018) suggested that another mechanism for PH and CO should be found to explain the frequency thereof in skeletal material.

In particular, signs of iron deficiency anaemia in dry bone, include hyperostosis of the cranial bones, expansion of the diploe, porous foramina or pitting on the outer vault named porotic hyperostosis (PH), and pitting in the outer layer of the anterolateral roofs of the orbits, named cribra orbitalia (CO) (Mann & Murphy 1990, Ortner, 2003; Walker et al., 2009; Aufderheide & Rodríguez-Martín, 2011). Authors do not all agree as to whether the development of PH and CO are related to anaemia and an association exists between the two types of lesions (Brickley, 2018). Brickley (2018) suggested that another mechanism for PH and CO should be found to explain the frequency thereof in skeletal material. It has been suggested, in previous studies, that CO is an earlier expression of bone marrow expansion than the porous lesions seen in the cranial vault (Stuart-Macadam, 1985; Blom et al., 2005; Brickley, 2018). However, Rivera and colleagues (2017) suggested that the form of anaemia that manifests as CO and PH do not originate from the same type of underlying anaemic conditions.

They suggest that PH is associated with bone marrow hypercellularity and hyperplasia, where CO is associated to anemias that lead to diploic bone hypocellularity and hypoplasia.

Some studies found a male predilection for anaemia (Facchini et al., 2004), however, it has been suggested that females are more prone to anaemia during menstruation, pregnancy and lactation (Bharati & Basu, 1990; Brabin & Brabin, 1992; Scholl & Hediger, 1994) and that developing countries have an anaemic bias towards young females due to young pregnancies, malnutrition and poor access to health care facilities (Rosso & Lederman, 1982). Children are particularly susceptible to anaemias because of their rapid growth and associated high iron requirements (Facchini et al., 2004) and active iron deficiency anaemia can easily present on children's bone as PH or CO. Body growth slows down in late in adolescence, at which point the iron status of boys appears to improve. Adult men, therefore, typically have larger iron stores than women (Cohen & Armelagos, 1984).

Several risk factors for iron deficiency anaemias have been suggested, such as malnutrition (El-Najjar, 1976; Holland & O'Brien, 1997) by a maize dependent diet due to the poor absorption of iron by maize, as seen in prehistoric Mexican Mayans and southwestern North American Anasazi populations. Anaemias may have a hereditary origin such as thalassaemia found in skulls from Greece and the early Neolithic village of Catal Hiiyiik, Turkey (Miles, 1975), or sickle cell anaemia in Africa (Steinbock, 1976). The interaction between diet and bacterial pathogens (for example *Mycobacterium tuberculosis*) or parasites (helminthiasis or malaria) (Mensforth et al., 1978; Cohen & Armelagos, 1984) have been emphasised to play a role in iron uptake (Mensforth et al., 1978). Anaemia can also be the result of abnormal blood loss through bleeding from a variety of causes, including chronic gastrointestinal bleeding (Blom et al., 2005), extensive diarrhoea and menstruation (Ortner, 2003). Iron deficiency anaemias may also be related to vitamin and mineral deficiencies, particularly of magnesium ions, chloride ions, iron, Vitamin B₁₂ and folic acids (McKern & Stewart, 1957; El-Najjar, 1976; Holland & O'Brien, 1997). These deficiencies may be caused by malnutrition, alcoholism, malabsorption due to toxins, drugs or congenital factors (Walker et al., 2009).

Zakai et al. (2008) found anaemias to be three times more common in American Blacks than American Whites by using World Health Organisation criteria among 19 836 Blacks and Whites even after adjusting for demographic variables, socioeconomic factors, and comorbid conditions.

2.4.3.2.1 *Cribra orbitalia* (CO)

Cribriform orbitalia (CO) is represented by visible pitting (small holes) in the outer layer of bone in the roof of the orbits, mainly suggested as a nutritional deficiency suffered in childhood (Steinbock, 1976). In children with active iron deficiency, the bone can be thickened with large foramina, while in adults only remnants of the holes (frequently only pits) remain. Lesions seen in adults result from bone changes occurring in the growth period that have not undergone complete remodelling (Stuart-MacAdam, 1985; Kosak, 2002; Van der Merwe, 2007). Paleopathological findings reported that CO is more frequent than PH (Walker et al., 2009) and studies have suggested CO has a more complicated aetiology than PH. The cause of CO lesions have been suggested to be subperiosteal bleeding associated with nutritional deficiencies including scurvy, gastrointestinal disorders, diarrhoea, and parasitic infections (Mensforth et al., 1978; Steckel & Rose, 2002; Walker et al., 2009).

2.4.3.2 Porotic hyperostosis (PH)

Porotic hyperostosis (also called symmetric osteoporosis) is classified as lesions that present as areas of sieve-like holes, symmetrically on the outer table and diploe of the frontal and parietal bones (Ortner, 2003), and the occipital bone (Van der Merwe, 2007). Porous lesions on the cranial vault should be evaluated carefully to ensure that they were caused by underlying marrow expansion before classifying it as iron deficiency anaemia (Brickley, 2018). It should only be classified as porotic hyperostosis if the porosities are accompanied by increased vault thickness. If scattered fine porotic pitting of the parietal and occipital bones are observed giving it an "orange-peel" texture, and is not accompanied by thickened bone, it would rather be scored as non-specified ectocranial porosis (Angel, 1966; Ortner, 2003), caused by non-specific diseases or processing artefacts, and not porotic hyperostosis associated with iron deficiency anaemia.

One of the key difficulties in evaluating skeletal material is the need to establish if observed porous lesions are caused by underlying marrow expansion (Wapler et al., 2004).

2.4.4 DEGENERATIVE DISEASES AND ARTHROPATHIES

Degenerative diseases are the most commonly diagnosed disease on dry bone (Weiss & Jurmain, 2007; Waldron, 2009). This progressive pathological condition is a result of loss of the articular surfaces (cartilage) at joints, causing direct contact between the bones in joints (Aufderheide & Rodriguez-Martín, 2011).

Degenerative disease is commonly subclassified as a) primary (idiopathic), in which the cause is unknown, but can result from a combination of factors e.g. age, sex, hormones, mechanical stress, and genetic predisposition, and b) secondary, in which the joint has been changed by some other disease or event such as wear and tear (physical), trauma, metabolic diseases (e.g. rickets) or vascular, invasion of the joint by bacteria (infectious), or other arthritic events for example obesity, occupational stress, congenital deformities or limb asymmetry (Steinbock, 1976; Hall, 2003; Aufderheide & Rodriguez-Martín, 2011). Arthritis is a term commonly used for any disease affecting the joints and joint spaces, and can be specific or non-specific.

The mechanism of degenerative diseases can be categorised into two, namely:

a) Lesions of abnormal bone formation

Osteophytes are also called bone spurs or marginal lipping, and naturally form as a person ages as a sign of degeneration in joints and the vertebral column. It forms as a result of the increase in a damaged joint's surface area due to aging, degeneration, mechanical instability or disease, and is most commonly seen from the onset of arthritis. These spurs can also appear on other joints such as hands, ankles, knees, elbows and shoulders (Aufderheide & Rodriguez-Martín, 2011). Bone spurs usually limit joint movement and typically cause pain. Often osteophytes form in osteoarthritic joints due to damage and wear from inflammation. Calcification and new bone formation can also occur in response to mechanical damage in joints, or at the attachment points for ligaments and tendons. Lipping can be visible as round (mushrooming) or sharp (spiculed) osteophytes on joints or syndesmophytes on vertebral bodies (Wilczak & Jones, 2012).

b) Lesions of bone erosion

Eroding signs resulting from degenerative diseases are visible on bone, as either porosity, or erosion and eburnation with long-standing irritation of the joint.

Eburnation, or polishing of the bone, is typically indicative of degenerative diseases and is caused by the breakdown and eventual loss of the hyaline cartilage covering the joints. This loss eventually leads to bone-to-bone contact, producing the polished effect on the joint surfaces. Osteoblasts then respond by depositing subchondral bone (osteophytes) around the edges of the joints (Waldron, 2009). The surface can appear smooth and shiny or can have striations. Eburnation can be associated with load bearing joints, which suggests strenuous physical activity by the individual, particularly through the area/joint where the

eburnation is visible. Common sites involved is the small joints of the hands, elbows, knees and wrists, with the ankles are usually the least involved (Rogers et al., 2004). Eburnation on joint surfaces is not always indicative of physical activity, it is multifactorial but the ageing process is an important factor (Ortner, 2003). Rogers et al. (2004) found a clear relationship between age over 45 years-of-age and eburnation. Post-menopausal women are especially vulnerable to the condition due to hormonal changes, especially at the knee (Rogers et al., 2004).

2.4.4.1 Osteoarthritis (OA)

This is the most common form of noninflammatory arthritis, present in approximately 80.0% of a population older than 50 years-of-age (Mann & Murphy, 1990). The cause is wear and tear on the joints; causing degeneration of the articular cartilage of load bearing joints such as the vertebral joints, knees and hips resulting in osteophytes and eburnation. When the small joints of the fingers are involved, OA is visible in the proximal (PIP) and distal (DIP) interphalangeal joints. Radiographic features include asymmetric narrowing of the joint space, subchondral sclerosis, subchondral cyst (geode) formation, formation of osteophytes, and a lack of osteoporosis. Factors influencing the likelihood of having OA is largely age, although strenuous activity, especially from an early age, trauma, and infection can also lead to biomechanical changes in joint function, increasing the risk for OA in any age (Mann & Murphy, 1990). Osteoarthritis of the knee, hip and also the lower back and feet have been associated with obesity in modern populations due to the additional strain on the main weight bearing joints. On the other hand, OA of the vertebral column is common in archaeological populations, and is directly related to age (Ortner, 2003).

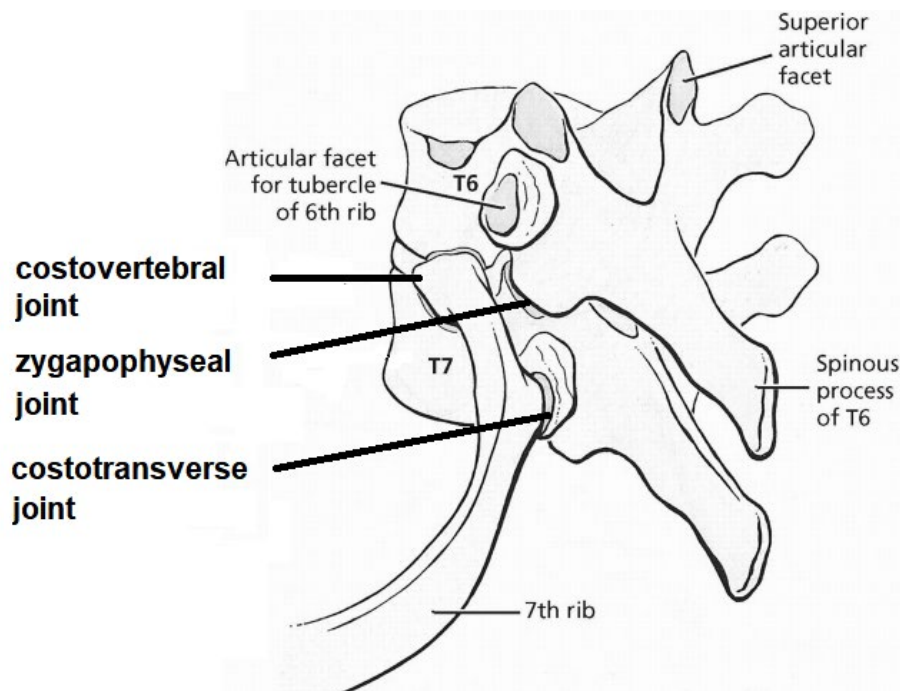
A number of authors have compared the incidence of OA in various traditionally male occupations, for example, increased frequencies of OA on various synovial joints were found in persons doing heavy lifting or farming (Hall, 2003), bus drivers and cotton pickers (Lawrence, 1961, 1969), workers casting metal (Mintz & Fraga, 1973), coal miners (Kellgren & Lawrence, 1957) and pneumatic hammer operators (Burke & Fear, 1977). Specific synovial joint OA have also been associated with specific activities, for example, OA on hand joints of print setters, from brushing letters across the tray with their thumbs (Dieppe et al., 1986), and long distance runners (Puramen et al., 1975) should expect increased frequencies of OA on their knee and ankle joints.

2.4.4.1.1 Peripheral joint osteoarthritis (pOA)

This presents as degeneration of the non-vertebral synovial joint cartilage, for example the elbow, shoulder, wrist, knee and temporomandibular joint (TMJ). Pitting of and osteophyte response in the bone tissue follows a synovial inflammation (Steinbock, 1976). The elbow, hip and knee joints are the most commonly involved, and lesions are often seen bilaterally in dry bones (Steinbock 1976, Roberts & Manchester, 2005; Aufderheide & Rodriguez-Martín, 2011). Shoulder OA is not as common as OA of the hip or knee, but it is estimated that nearly one in three people over the age of 60 have shoulder OA to some degree. Females bear a higher burden of osteoarthritic joints than males do, especially when considering the hips and knees, (Perruccio et al., 2017). Additionally, females experienced a higher affinity for symptomatic osteoarthritic changes in the knee than males; there was also a correlation found between their endogenous hormone levels and this pathological presentation (Jin et al., 2017).

2.4.4.1.2 Vertebral joint OA (vOA)

Vertebral osteoarthritis (vOA), also called facet joint osteoarthritis, results from the breakdown of cartilage between posteriorly located facet joints of the vertebral column, including facet joints of zygapophyseal, costovertebral and costotransverse joints (Fig. 2.10). Costovertebral joints are where the ribs articulate with the facets on the posterior aspects of the vertebral bodies. Costotransverse joints are where the ribs articulate with the transverse processes of the vertebrae, while articulation in the zygapophyseal joints are between the superior and inferior articular facets of the posterior arches of two adjacent vertebrae. It causes degeneration of synovial joint cartilage with inflammation of the synovial joints, with a bone tissue response to it. The vertebral column is a common area for stress injuries and pain to occur, mainly due to human bipedalism. Data collected worldwide, indicates the highest prevalence of vOA in the lumbar section of the vertebral column followed by the cervical section (Peckmann, 2002). Target joints for the formation of marginal osteophytes include facet joints of the vertebral arches and the costovertebral joints. Vertebral osteoarthritis generally occurs in all persons older than 40 years and is a common form of pathology observed in the vertebrae (Steinbock, 1976).



Adapted from: Agur & Lee, 1999

Figure 2.10. Posterolateral view of the ribs and vertebral articulation.

a) *Spondylolysis*

Spondylolysis is described as the presence of a fracture in the *pars interarticularis* of the vertebral neural arch; the weakest portion of the neural arch. The severity can range from unilateral hairline fractures to complete separation of the bone (Masniková & Beňuš, 2003) and the prevalence in studies ranged from 3.5% to 7.0% (Eisenstein, 1978; Soler & Calderón, 2000; Kalichman et al., 2009). Although some pars defects can be congenital, the aetiology is known to be caused by mechanical stress, for example repeated axial loading of the lumbar vertebral column while it is hyperextended (Frederickson et al., 1984; Standaert & Herring, 2000; Hall, 2003). Spondylolysis is mostly present in the lumbar vertebrae, in particular L5, but can, although very rarely, be present in cervical vertebrae as well (Ortner, 2003). Spondylolysis is often asymptomatic and is clinically diagnosed coincidentally during radiographic evaluation (Attiah et al., 2014). Males are more affected than females (Kalichman et al., 2009; Aufderheide and Rodríguez-Martín, 2011) and when comparing population groups, White population groups are more affected than the Black population groups (Aufderheide & Rodríguez-Martín, 2011).

2.4.4.1.3 *Vertebral osteophytosis (VO)*

Vertebral osteophytosis (VO) is degeneration of a non-synovial, solid, fibro-cartilaginous joint and are present on the vertebral column as ossification on the vertebral endplates, causing

marginal osteophytes, that may end in fusion of adjacent vertebrae. It is usually present at the most commonly flexed sites such as C5-C6, T8-T9, L4-L5 (Aufderheide & Rodríguez-Martín, 2011). The pathogenesis of VO has been described as wear and tear, aging and hereditary factors. Vertebral osteophytosis are seen in individuals over 34 years-of-age (Jurmain & Kilgore, 1995).

a) Diffuse Idiopathic Skeletal Hyperostosis (DISH)

Diffuse idiopathic skeletal hyperostosis (DISH) is also known as Forestier's disease, which is a non-inflammatory, degenerative disease of the axial skeleton, causing ossification of the anterior longitudinal spinal ligament. This fusion occurs typically on the right side of the vertebral column and not left as the aortic pulsation is high on the left side, preventing ossification. It has the characteristic "dripping candle wax" appearance (Aufderheide & Rodríguez-Martín, 2011). The aetiology of DISH is still under discussion, although trauma, occupational stress, endocrine abnormalities and genetic factors have been suggested (Weinfeld et al., 1997; Kim et al., 2004; Sarzi-Puttini & Atzeni, 2004). In addition, risk factors such as a protein rich diet, gout, substantial drinking, obesity and late-onset diabetes type II have been suggested to influence the prevalence of DISH (Resnick et al., 1978; Rogers & Waldron, 2001; Mader, 2002; Hannallah et al., 2007). This ankylosed condition has been widely described in clinical studies.

Criteria to diagnose DISH include fusion along the anterolateral aspect of at least four adjacent vertebrae (with or without osteophytes), no fusion of the facet joints, the intervertebral disc spaces remain intact and an absence of sacroiliac erosion, sclerosis or bony fusions of the sacroiliac joint (SIJ) (Ortner, 2003; Aufderheide and Rodríguez-Martín, 2011). Clinically, the patient initially suffers from pain in the mid back, and over time experiences increasing stiffness as the bones of the vertebral column fuse together. In severe cases, the ribs can fuse to the vertebral column. Other non-vertebral skeletal involvement may be ossification of the iliolumbar ligaments, thickening of short bones of the hand, hyperostosis of the distal clavicle, olecranon and calcaneal spurs, enthesophytes on the ischial tuberosity and iliac wing, and periarticular osteophytes (Hannallah et al., 2007). This condition is mostly observed in vertebrae of older individuals after their 5th decade of life and a predisposition for males exists (Cammisa et al., 1998; Hannallah et al., 2007; Aufderheide & Rodríguez-Martín, 2011). Previous studies found the European population group have a higher prevalence for DISH than

Asian, Black, and Native American groups (Weinfeld et al., 1997; Rogers & Waldron, 2001; Aufderheide & Rodríguez-Martín, 2011; Kim et al., 2012).

2.4.4.2 Arthropathies

Arthropathies are either sero-positive (positive rheumatoid factor) or sero-negative. In general, sero-positive patients have more severe joint deformities and inflammation outside the joints.

2.4.4.2.1 Rheumatoid arthritis (RA)

Rheumatoid arthritis (RA) is a sero-positive arthropathy. Unlike osteoarthritic markers on the skeleton, recognised as markers of conditions the body has endured, RA is rather linked to a genetic origin. The exact cause is unknown, but evidence exists that it varies with genetic background (Van der Merwe, 2007) or occasionally caused by an outside vector such as a parasite or fungus. Other causes include infection of the synovial joint, or nonsuppurative proliferative secondary synovitis, after a *Streptococcus* throat infection that can progress to a cyst-like invasive destruction of the articular cartilage and bone, with formation of a joint ankylosis (Cawley & Paine, 2015).

Rheumatoid arthritis presents as chronic inflammation of joints throughout the body. It is the most painful form of arthritis (Hall, 2003). It has an auto-immune origin in which the body's immune system attacks its own cartilage, causing inflammation and thickening of the synovial membranes and breakdown of the articular cartilage. This results in limitation of movement and eventually, ossification of the articulating bones. Other symptoms include fatigue, anaemia, muscular weakening, and osteoporosis (Hall, 2003). It especially targets the hands and feet (bilaterally) where symmetric pitting of the small joints such as the metacarpophalangeal (MCP), metatarsophalangeal (MTP), and PIP joints of hands and feet, occur. Although the wrist, elbow, and the knee can be targeted, less often and less destruction is seen on large joints (Brothwell, 1968). It typically does not affect the vertebral column or SIJ (Cawley & Paine, 2015). This form of arthritis is present in 1.0% of adults, of which with up to 75.0% are women (Langton & Njeh, 2004). The peak ages for RA are between 10 and 29 years-of-age. Although RA is high in menopausal or middle-aged women, it is, in general unrelated to age (Kilgore, 1989).

2.4.4.2.2 Ankylosing spondylitis (AS)

Ankylosing spondylitis, previously known as Von Bechterew's disease and Marie-Strümpell disease, is a seronegative chronic inflammatory spondyloarthropathy of the axial

skeleton, with non-typically, variable involvement and fusion of peripheral joints and nonarticular structures. This disease causes abnormal immobility of the intervertebral synovial joints of the vertebral column, resulting from pathological changes in the joint. Although the aetiology is not known, it is not related to normal aging of the body and it has been suggested that genetic, immunologic, and/or environmental factors may play some role (Braun & Sieper, 2007; Jordana et al., 2009). It causes progressive ossification of the associated connective tissue ligaments (e.g. interspinous ligaments), progressing up the vertebral column from the pelvis, resulting in increased immobility as the joints are fused (Braun & Sieper, 2007). On the lumbar vertebrae, only the lateral edges of the vertebral bodies are joined, but the spinous processes can become a solid fused mass, giving a ‘bamboo spine’ appearance, while the costovertebral joints of the thoracic vertebrae can fuse. Kyphosis can be present (Ortner, 2003). Other axial skeletal elements that can be targeted include fusion of the manubriosternal joint and symmetric sacro-iliitis (bilateral ossification of sacro-iliac joint) (Braun & Sieper, 2007; Aufderheide & Rodríguez-Martín, 2011).

Although it is an ancient disease of the vertebral column, it can be difficult to distinguish and differentiate between osteophytes of spondylosis, diffuse idiopathic skeletal hyperostosis (DISH) and AS (Rogers et al., 1987). Characteristics that can be used for differential diagnosis of AS includes bilateral fusion of the SIJ (Jordana et al., 2009) and ‘bamboo spine’, where smooth sheets of bone are ossified (Braun & Sieper, 2007).

2.4.4.2 Reactive arthritis (Reiter’s syndrome)

Reactive arthritis (ReA), formerly known as Reiter’s syndrome (RS) is a sterile inflammatory seronegative arthritis (or synovitis) typically develops one month after a systemic infection, most commonly, genitourinary/venereal acquired infections. The most common pathogenetic organisms associated with this condition are *Chlamydia trachomatis* and gram-negative enterobacteria, for example, *Shigella*, *Salmonella*, *Yersinia* or *Campylobacter* spp. (Cawley & Paine, 2015). It is less destructive or erosive than other seronegative arthropathies. Clinically this condition results in a triad of conjunctivitis, urethritis, & arthritis (commonly memed as "can't see, can't pee, can't climb a tree" among clinicians) (Kumar et al., 2007).

On dry bone it causes patches of asymmetrical ossification within the ligamentous structures (syndesmophytes) along the vertebral column (known as “skip lesions”), although asymmetric (unilateral) ossification of the sacro-iliac joint (SIJ) may also be observed (Ortner, 2003). Unilateral purulent infective arthritis of one or two large joints of the lower limbs may

be involved, usually the knee or ankle. It appears in young patients from adolescence, before 40 years-of-age, mainly between 15 and 35 years-of-age, and it features more in males than females, in some cases a ratio of 20:1 has been reported (Khan, 2002; Aufderheide & Rodríguez-Martín, 2011). Genetical studies showed a HLA-B27 positive result in approximately 80% of White patients, less in Coloured and even less in Black patients (Khan, 2002).

2.4.5 NEOPLASTIC DISEASES

The term neoplasia refers to any abnormal growth, both malignant and benign. On bone, it can present as areas of resorption, with or without reactive deposition to produce a sclerotic margin (Ortner, 2003; White et al., 2012). Cancers is a term used for malignant neoplastic diseases. Bone tumours, both benign and malignant, can be classified according to tissue origin: a) Primary bone tumours, cancers that develop initially in bone, causing the bone itself to grow tumours; b) Secondary bone tumours: cancer in the soft tissue adjacent to a bone which lead to pressure, causing the tissues to react and metastasise to the bone, leaving visible lesions or holes in the bones; or c) Soft tissue lesions prompting a bone response without metastases such as hyperostosis secondary to cranial meningioma (Cox & Mays, 2000; Aufderheide & Rodríguez-Martín, 2011; White et al., 2012). Diagnosis of neoplasms in skeletal remains is difficult, especially when lytic lesions that differentiated from other pathology, are observed, as many cancers manifest only in soft tissues (Brothwell, 1991). Many factors can have an influence on the development of cancers in population groups, including geographic location, social status, environmental or occupation stressors, sexual activity, hereditary factors and diet and vitamin intake (Cox & Mays, 2000).

2.4.5.1 Primary bone tumours

Generally, primary tumours seen in bone are benign lesions since malignant bone tumours are rare. However, most bone tumours, whether they are benign or malignant, can negatively affect human health and leave skeletal changes. The nature of tumorous lesions can be detected microscopically for differential diagnosis (Ortner, 2003).

2.4.5.1.1 Malignant primary bone tumours

Primary bone tumours are usually present in actively growing young people, with some suggestions that tall rapidly growing individuals are more prone to primary bone neoplasms. These tumours generally produce wildly aberrant patterns of new bone admixed with areas of

aggressive lysis (Ortner, 2003). Malignant tumours are rare in British archaeological material with infrequent reports of osteosarcoma of a knee joint (Brothwell, 1968), or osteochondroma of the limb bones (Cox & Mays, 2000) and primary bone lymphoma. In the KSC cohort, primary malignant bone tumours are rare; therefore only three types are discussed.

a) Osteosarcoma

This malignant bone tumour is the second (40-60%) most common primary malignant neoplasm of the skeleton and comprises 19.0% of all tumours on bone (Hogendoorn, 2010). It is present in growing metaphysis of long bones in children (15–19 years-of-age) and in young males in their 20s (Picci, 2007; Hogendoorn, 2010). Risk factors include radiation therapy, medications and genetics (Capasso, 2005). The typical distribution of an osteosarcoma is in the distal femur metaphysis, followed at a lesser frequency the hip, and shoulder joints (Dorfman & Czerniak, 1998; Hogendoorn, 2010).

Conventional osteosarcoma, a high-grade malignancy, accounts for 80%–90% of all osteosarcomas (Hogendoorn, 2010) and subtypes that most frequently occur include osteoblastic, chondroblastic and fibroblastic neoplasms. Surface osteosarcoma is a rare form of osteosarcomas observed in approximately 3-6% of all osteosarcomas. Three major groups of surface osteosarcomas are low grade parosteal, intermediate grade periosteal, and the high grade surface osteosarcomas. Of these, the parosteal osteosarcoma is the most common. Radiologically, the parosteal osteosarcoma will show an intact underlying cortex (Kumar et al., 2014), while the periosteal osteosarcoma will show a lifting of the periosteum (Unni et al., 1976; Kundu, 2014), although both originate from the surface of the bone (juxtacortical). Generally, the site where these are observed most, is the lower end of the femur followed by the upper end of the tibia, and upper end of humerus, in that order (Kumar et al., 2007). Differentially, the parosteal osteosarcoma is generally seen as a lobulated, broad based mass on the posterior aspect of the distal femur, while the periosteal osteosarcoma affects the tibial shaft more commonly (Kundu, 2014). Studies showed a female predilection for parosteal osteosarcoma (Song et al., 2011). Surface osteosarcomas occur at a later age, compared to other osteosarcomas (Bertoni et al., 2005; Song et al., 2011; Kumar et al., 2007). In clinical studies the age of patients ranged between 8-64 years-of-age (Okada et al., 1994), although Bertoni et al. (2005) reported and average age of 36 years (range 15-85 years-of-age).

b) Primary bone lymphoma (lymphosarcoma)

Lymphoma of bone is a malignant neoplasm arising from one site in bone; in the early 20th century, it was included in the same category as Ewing's sarcoma (Boston et al., 1974), but was later classified separately. Lymphosarcoma is defined as a lymphoma occurring primarily (between 44-71%) in long bones (Limb et al., 1994; Dubey et al., 1997; Stein et al., 2003), forming about 7.0% of all primary bone malignancies (Pettit et al., 1990), with non-Hodgkins lymphoma the most common. Several studies indicated that the diaphysis of the femur is the most common site for this malignancy (Heyning et al., 1999; Mulligan et al., 1999) and it is more frequent in ages between 50 and 60 years-of-age (Mulligan et al., 1999); however, Non-Hodgkin's, in particular, is rather seen in ages between 20-25 years. Generally, there is a bias towards males (Heyning et al., 1999; Zinzani et al., 2003; Barbieri et al., 2004). Reticulum cell lymphosarcoma (of late known as histiocytic lymphoma of bone) is an extremely rare malignant lymphosarcoma that may be present either as an unifocal disease (Vos et al., 2005) or a metastatic multi-systemic disease (Kayikcioglu et al., 2017). Studies showed this malignancy as lytic and/or sclerotic lesions, that affects the pelvis, femur, humerus, ribs and tibia in that order (Potdar, 1970; Boston et al., 1973). Radiological features are generally non-specific and can be easily confused as metastatic lesions. Although difficult without using various image modalities, histopathological biopsies as well as knowledge of clinical features, diagnosis is attainable. In most cases, the presentation was found to be extra-nodal, involving the skin, spleen and the gastrointestinal tract (Vos et al., 2005) although skeletal lesions have been reported (Kayikcioglu et al., 2017). It is predominantly seen in males (Potdar, 1970; Boston et al., 1974) between 50-60 years-of-age (Vos et al., 2005; Yoshida & Takeuchi, 2008; Kayikcioglu et al., 2017) in clinical studies.

2.4.5.1.2 Benign primary bone tumours

Benign tumours are slow growing, well-differentiated, small osteoblastic lesions that do not destroy surrounding tissue or metastasise to other areas of the body (Ortner, 2003; Aufderheide & Rodríguez-Martín, 2011; White et al., 2012). Circular, slow growing osteomata (button osteoma and osteoid osteoma) as well as enostosis (bony islands), are seen as hamartomatous lesions rather than true neoplasms with no clinical significance and are therefore omitted in this study. Hamarta is described as islands of mature cortical bone surrounded by spongy bone. Enchondromas are small, benign intermedullary cartilage lesions that are remnants of hyaline cartilage at the epiphyseal growth plate, and that failed to undergo enchondral ossification. It is radiographically seen as minor cortical scalloping, with a clear

margin surrounding a mineralised matrix (Kendell et al., 2004). It is mainly present on hands, however, it can occur as larger lesions in long bones (Ortner, 2003; Walters, 2014). It makes up about 10% of benign bone tumours and is common in young adults, although it may be seen at any age (Kendell et al., 2004).

2.4.5.2 Secondary bone tumours and metastatic skeletal lesions

The spread of neoplastic cells in the skeleton is mostly from organ cancers (Resnick, 1995) and present as either osteolytic lesions, sclerotic lesions or a combination of these in the form of single lesions or multiple foci. Haematogenous routes are mainly used for spreading these cells throughout the skeleton, although lymphatic routes or direct extension from adjacent soft tissue or via the lymphatics or cerebro-spinal fluid (Binder et al., 2014). Therefore, metastatic response to secondary bone tumours favours areas of rich red marrow found in the axial skeleton and long bones (Kim et al., 1987; Coleman, 2006). In order of decreasing frequency, the most common affected elements include the vertebrae, pelvis, ribs, sternum, humeral and femoral heads, skull, clavicles and scapulae (Miller et al., 1992). Organ cancers that in particular develop bone metastases in 60-80% of patients include breast, lung, or prostate cancer (Huben, 1992; Sone & Yano, 2007; Zhang et al., 2009; Hofbauer et al., 2014),

Occupational stressors have an influence on the type of cancers as suggested by researchers on older material, for example, Raven (1990) found that in the 1700s, soot was implicated in scrotal cancers (called chimney sweepers cancer), whereas coal miners were found to be more susceptible for gastric carcinomas (Raffle et al., 1978). It has been suggested that blood groups have an influence in the susceptibility of specific cancers, for example, people with blood group A, have a weakened immune system, in which the body fails to destroy cancer cells such as gastric and prostatic cancers (Raffle et al., 1978; Glucksmann, 1981). Mourant (1985) suggested that people with blood group O, have an overactive immune system, causing a tendency to develop autoimmune diseases. Testosterone hormones were directly linked to development of prostate cancers (Vesey & Grey, 1985) while other studies suggested that the amount of sexual intercourse, the number of partners and the age at first intercourse can all play a role (Raffle et al., 1978). Breast cancers have directly linked to genetic hereditary (Mould, 1983) and the higher social classes in urban context (Glucksmann, 1981). Colon and rectal cancers were closely related to the civilised diets of the Western world, namely the lack of fibre with excess fat and animal protein (Glucksmann, 1981; Raven, 1990). People with low SES are rather at risk of carcinoma of the liver and oral cavity (Mould, 1983).

2.4.6 OTHER DISEASES/CONDITIONS

2.4.6.1 Hyperostosis frontalis interna (HFI)

Hyperostosis frontalis interna is also known as hyperostosis cranii (Moore, 1955) is a unique, benign condition, limited to lesions on the endocranium and no other pathology. The irregular, undulating bony patterns located on the endocranial lamina of the frontal bone (Raikos et al., 2011), are usually bilateral. The external surface and the inner midline area around the *crista galli* are usually not affected (She & Szakacs, 2004).

The aetiology is highly debated and includes aging, dietary factors, obesity (Cocheton et al., 1974; Verdy et al., 1978) genetic predisposition and epigenetics (Rosati, 1972; Glab et al., 2006), arterial hypertension (Mayer, 1962; Devriendt et al., 2005), diabetes mellitus (Bartelheimer, 1939; Boulet & Mirouze, 1954) as well as endocrine disturbances (Mayer, 1962; Perou, 1964; Chaljub et al., 1999; Hershkovitz et al., 1999; Ortner, 2003; Belcastro et al. 2006; May et al., 2010). She and Szakacs (2004) found hormonal influences on bone growth in the endocranium is involved and believed that prolonged estrogen stimulation is linked to HFI, which explain the increased prevalence in postmenopausal females.

Clinically, it has been associated with Morgagni-Stewart-Morel Syndrome, which is a hormonal disorder in females presenting with obesity, psychiatric disorders, masculinity and facial hair growth (Eldridge & Holm, 1940). Other clinical disorders associated with HFI included behavioural disturbances, visual disorders or chronic alcoholism (Devriendt et al., 2005), and in severe cases memory loss and chronic apathy (Chaljub et al., 1999).

A review of population group prevalence by Barber et al. (1997) reported that it is an uncommon finding in archaeological populations ranging from 1.0% (Stroud, 1993) to 10.0% (Lazer, 1994) of people affected; however, it is common in post-menopausal females in modern societies, up to 62% (Gershon-Cohen, 1955). Most studies were done on the general population of people from various countries and not specific population groups within countries. Henschen et al. (1949) and Gershon-Cohen (1955) both studied elderly citizens in the United States, while Verdy et al. (1978) studied Canadian nuns and Devriendt et al. (2005) studied individuals in the Hospital de la Timone, Marseille, France. In 1962 Salmi and colleagues studied normal citizens aged 15-70+ years in Finland (Salmi et al., 1962).

The estimated male:female ratio is 1:9 in literature and the mean age in modern patients include the 40-60 years age group (Salmi et al., 1962). Although most literature found post-menopausal women of mature age to have HFI (Barber et al., 1997; Hershkovitz, et al., 1999;

She & Szakacs, 2004; Devriendt et al., 2005; Raikos et al., 2011), Marlet (1974) found in a total of 300 women, two young individuals with HFI aged 15 and 19. The youngest patient in a study by Grollman & Rousseau (1944) was 20 years old, while Cocheton et al. (1974) reported the youngest in their study was a 21 year old male.

A study by May and colleagues (2010) of males over 60 years old, found that the patients, treated with hormones for prostate cancer, are at a higher risk of developing HFI compared to healthy males, although benign prostatic hypertrophy does not support HFI manifestation.

2.4.6.2 Paget's disease

Osteitis deformans, also known as Paget's disease, is an uncommon chronic inflammatory condition that results in proliferation and softening of bone that can affect any or all bones in the skeleton, although the bones most frequently affected are the vertebral column, skull, pelvis and lower limbs (Barker, 1981). In the early stages of this disease the lesions are typically lytic (resorptive or osteoclastic), originating in one focus of bone, and spread until the entire bone is affected (Mirra, 1987). Late phases result in grossly enlarged dense reformed (osteoblastic) woven bones. This newly deposited bone is dense, but fragile and therefore be structurally compromised. This results in an increased likelihood of spontaneous fractures at sites of Paget's involvement (Barker, 1981; Mirra, 1987; Kanis, 1998).

The exact cause of Paget's disease is unknown and in most cases, no specific cause can be identified. Genetic, as well as environmental factors have been suggested. A family history of Paget's have been identified in 15-30.0% of cases (Reid et al., 2005). Genes associated with Paget's disease include sequestosome 1 gene, the TNFRSF11A gene, and the VCP gene. Other researchers suggested that Paget's disease might be caused by a "slow virus" infection of bone, a condition that is present for many years before symptoms appear. This hereditary factor may be the reason that family members are susceptible to the suspected virus (Mirra, 1987).

Clinically, most individuals with this disease are asymptomatic or only develop mild symptoms (Singer & Krane, 1998). Skeletal signs include enlargement and bowing of the femora and tibiae, hyperostosis of the anterior vault, proliferation of one or more of the bones in the skeleton, and rarely, hydrocephalus and spinal stenosis. Osteogenic sarcoma is a rare complication (Reid et al., 2005). On the inner vault, pits along the meningeal grooves can be seen. Radiological diagnosis includes localised enlargement of bone (due to rapid bone formation), marked cortical thickening, increased bone density (seen as decreased radiolucency in radiographs) enhancement of the trabecular pattern, sclerotic changes (disorganised newly

formed bone) and osteolytic areas. Signs of Paget's disease on the pelvis X-rays shows thickening of the iliopectineal line ("brim sign") and *protrusio acetabuli* (Murray & Jacobson, 1979). The disease seldom appears before the age of 40 years, but may occur in up to 80.0% of the population over the age of 60 years-of-age (Ortner & Putschar, 1985). Both male and females are affected with a slight male predominance (Singer & Krane, 1998). Paget's disease affects individuals of any population group, although findings shows that it affects individuals of Asian descent less frequently. Studies of Europeans, including a 1.0% prevalence among 1778 Italians (Gennari et al., 2005), 0.3% among 6935 Europeans (Poór et al., 2006), and 1.0% prevalence among 4528 Spaniards (Guañabens et al., 2008) illustrated that Paget's disease of bone is, although a rare occurrence, commonly found in the White population group.

2.4.6.3 Hypertrophic osteoarthropathy (HOA)

Hypertrophic osteoarthropathy is also known as Marie-Bamberger syndrome or the previously called hypertrophic pulmonary osteoarthropathy (HPOA). Several clinical studies have been described, mainly case studies reporting on clinical symptoms. The hallmarks of this condition are systemic periostitis on multiple sites, digital clubbing and osteoarthritis. Skeletal involvement of HOA includes mainly the appendicular skeleton (Ali et al., 1980; Vigorita, 1999; Aufderheide & Rodríguez-Martín, 2011). Studies have suggested that the long bones distal to the elbow and knee joints are more commonly involved than the bones proximal to these joints, and in particular, nodular periostitis of the tibiae and fibulae are characteristic of HOA (Ali et al., 1980). Involvement of the clavicles and scapulae as well as tubular bones of the hands and feet, are indicative of severe HOA (Vigorita, 1999). Pelvic involvement has been reported by Ali et al. (1980), but cranial involvement in HOA is extremely rare (Ortner, 2003; Halcrow et al., 2014). The cause of HOA is still under debate (Ali et al., 1980; Bhat et al., 1989); however, it has been associated with malignancies, primarily bronchial carcinoma, PTB, bronchiectasis, congenital cyanotic heart disease, liver cirrhosis, pulmonary fibrosis and empyema, amongst others (Stenseth et al., 1967; Carroll & Doyle, 1974; Vigorita, 1999; Ali et al., 1980; Bhat et al., 1989; Dickinson, 1993; Armstrong et al., 2007; Geldenhuys, 2014). The prevalence of HOA in association with an underlying pulmonary or pleural pathology (Carroll & Doyle, 1974; Geldenhuys, 2014) and inflammatory disorders such as PTB (Ali et al., 1980; Resnick & Niwayama, 1995; Vigorita, 1999; Armstrong et al., 2007) have been reported in a substantial number of patients (95,0%). Non-discriminatory characteristics that can contribute to accurate diagnosis includes age and sex (Segal & Mackenzie, 1982). This disease affects

adults, mostly in their 5th to 7th decade and occurs less commonly in children (Hammarstan & O’Leary, 1957; Ali et al., 1980; Segal & Mackenzie, 1982; Geldenhuys, 2014; Halcrow et al., 2014). It distinctly shows a bias towards males compared to females (Hammarstan & O’Leary, 1957; Segal & Mackenzie, 1982; Morgan et al., 1996; Ito et al., 2010; Geldenhuys, 2014).

2.5 PROFILE OF TRAUMA IN THE WESTERN CAPE POPULATION

Skeletal trauma is indicated by the presence of osseous changes, such as callus formation of the bone, bone remodelling, fractures and joint dislocation (Walker, 2001). Fractures on bone occur when there is more force applied to the bone than the bone can absorb and result from a single traumatic incident, repetitive stress, or abnormal weakening of the bone (pathological fracture). Fracture healing takes place in four stages, namely: the inflammatory phase; reparative phase; callus formation and lastly, remodelling phase and is observed on bone as a thickened callus at the site of the fracture (Ortner, 2003)

2.5.1 EVIDENCE OF TRAUMA

Evidence of fracture patterns on human skeletal remains may provide insight into the socio-economic, environmental (e.g. availability of medical care or environmental stresses) and cultural behaviour that cause people to resort to violence (Steinbock, 1976; Ortner, 2003; Van der Merwe, 2007). Skeletal trauma is observed by the presence of osseous changes, such as healing of the bone, callus formation, fractures, burnt bone or joint dislocation (Walker 2001). Due to the limited number of available collections with known records, research based in the behavioural mechanism of trauma in skeletons are relatively scarce. The KSC gives the unique opportunity to test the influence of various demographic ecological and environmental variables on individual trauma in a contemporary population.

Trauma refers to the injury of living tissue by an external force and the epidemiology of trauma is mainly dependant on the population studied (Chrcanovic et al., 2004). Causes of traumatic injuries are generally classified as violence-related injuries, sport injuries, or accidents (e.g. falls or motor vehicle accidents) (Lee et al., 2007). Worldwide, mortality due to trauma is comparable to the total deaths caused by HIV, malaria and tuberculosis (TB) combined (Schuurman et al., 2015).

Violence-related injury- and mortality rates in SA suggest that this country is one of the most violent countries in the world (Norman et al., 2007) exceeding global averages, resulting

in a critical burdening of the health care system and great financial implications to the South African public (Alvi et al., 2003; Norman et al., 2007; Lee et al., 2007). Violence became the accepted approach for resolving conflict in this country (Norman et al., 2007), making SA one of the very few countries in the world with a higher rate of intended injury than unintended injury (including falls and MVAs) (Norman et al., 2007; Schuurman et al., 2015). The murder rate in SA exceeds the rate of the USA over five times (Brooks & Barker, 2003). Factors that had an influence on the high violence levels in this country, include the social injustice and political violence during apartheid with the state promoting violence against certain communities (Norman et al., 2007). Other factors include the predisposition of communities with a low SES and underprivileged circumstances to have a higher incidence rate of violence, especially IPV, as unemployment and lower levels of education are major risk factors for IPV (Kyriacou et al., 1999; Brickley, 2006; Brickley & Smith, 2006; Lee, 2009).

Prinsloo (2007) reported that 39.0% of the trauma-related deaths in SA in 2005 were caused by interpersonal violence (IPV) which is defined as the intentional use of physical force against another person or a group of people, resulting in injury. Generally, assault or battered victims suffer from blunt force trauma (BFT), which results from punching, kicking, shoving or striking an individual with a blunt object (Brink et al., 1998; Kyriacou et al., 1999; Abrahams et al., 2006). Not only do more than 1,5 million people die worldwide due to violence, many more are rendered permanently disabled (Schuurman et al., 2015).

According to the global trend, IPV is the major cause of death in developed countries, while motor vehicle accidents (MVAs) are the major cause of death in developing countries (Adi et al., 1990; Oji, 1999; Krug et al., 2002, Peden et al., 2002; Alvi et al., 2003; Adeyemo et al., 2005; Hofman et al., 2005; Lee et al., 2007). However, in SA, a country classified as a developing country, IPV is seen as the leading cause of death with a rate of nine times higher for men compared to any other country in the world (Norman et al., 2007) while MVAs caused twice as many deaths as the global rate (Hofman et al., 2005). In the Western Cape Province (WCP) of SA, IPV together with MVAs, are the leading causes of injury (Brooks et al., 1999; Goosen et al., 2003) and a primary cause of death among young males (Lee et al., 2007; Groenewald et al., 2014).

Studies noted a significant association between alcohol abuse, violence and road traffic accidents (Silvennoinen et al., 1992; Van der Spuy, 2000; Chrcanovic et al., 2004). It is acknowledged that the WCP of SA has a major problem with excessive alcohol consumption

(Schneider et al., 2007; Bowman et al., 2010; Ward et al., 2012; Corrigan & Matzopoulos, 2013; Schuurman et al., 2015), also among women, as seen in the high prevalence of Fetal Alcohol Syndrome (FAS) among paediatric patients (Schneider et al., 2007). Alcohol abuse is common amongst young adults in the social setting, leading to increased incidence of IPV and MVA (Lee et al., 2007). In a study by van der Spuy (2000) in SA, 7.0% of drivers with blood alcohol levels over the limit caused 30.0% of non-fatal and 47.0% fatal driver deaths. Mabunda et al. (2008) established, using surveillance data of SA's four largest cities (Cape Town, Durban, Pretoria and Johannesburg), that more than half of the pedestrian deaths between 2001 and 2005 occurred when the drivers of vehicles were under the influence of alcohol. Deaths and injuries due to pedestrian-vehicle accidents (PVA) are particularly high in SA. Sukhai and van Niekerk (2002) reported that PVAs accounted for more than half of all SA road-traffic accidents in 2001. A study in pedestrian-vehicle related accidents, implicated alcohol and other substances in 72.0% of the deaths of intoxicated pedestrians (van der Spuy, 2000).

2.5.2 AGE, SEX AND POPULATION GROUP

Worldwide, statistical significance was found between the age of patients and the occurrence of trauma (Ugboko et al., 1998; Ansari, 2004). Multiple studies assessing the incidence of trauma between male and female victims in hospitals or *via* case studies or surveys, concluded that the majority of trauma and death as a result of IPV or MVA occurs in males between the ages of 24-35 years, with an average age of 28 years-of-age (Krug, 2002; Lee, 2009; Groenewald et al., 2014). Surveillance data of SA's cities reported that pedestrian victims in PVA were between the ages of 20 and 44 years (Mabunda et al., 2008), globally McCoy et al. (1988) found injuries due to PVA occur mostly among school-aged children, old-aged persons and the intoxicated. The prevalence of trauma is higher in males compared to females in most studies, although most findings show a non-significant correlation (Butchart & Brown, 1991; Judd, 2008; Murphy et al., 2010). The reasoning behind these high numbers is that younger people, especially males, are more likely to participate in more dangerous activities than the rest of the population (Chrcanovic et al., 2004).

Studies done in SA and New Mexico, USA showed that one in every two women was killed by an assailant known to them, such as an intimate partner, husband or family member (Norman et al., 2007; Groenewald et al., 2014). Buckhart & Brown (1991) came to a similar conclusion in their study assessing 1592 victims in Soweto in Johannesburg. Compared to

global statistics females in Africa and India have the highest incidence of IPV, MVA and suicide rates than any other country in the world (Norman et al., 2007).

2.5.3 TIMING OR MECHANISM OF TRAUMA ON SKELETAL MATERIAL

Skeletal trauma can be classified into blunt force, sharp force, ballistic and heat related or burnt trauma (Symes et al., 2012). Blunt and sharp-force trauma can inflict different types of injuries and fractures (by analysing tool marks). The type of trauma observed on bones can therefore be valuable to forensic scientists by assisting in estimating the cause, time and manner of death (İşcan & Steyn, 2013). Blunt force trauma (BFT) result from a blunt object and will generally cause depression of bone surface that will either crack or break the bone if this slow-loading force exceeds the natural elasticity of the bone (İşcan & Steyn, 2013). When bone is hit by a direct blow, the bone will fracture at the point of impact, for example, a parry fracture on the distal shaft of the ulna when fending off a blow. In contrast, during an indirect blow, the bone may fracture far from the point of impact, for example a spiral fracture that may be caused away from the point of impact. Major external forces may be applied in MVAs where the forces (direct or indirect) may be severe and cause crushing injuries (İşcan & Steyn, 2013). Sharp force trauma (SFT); however, results from contact with a pointed or sharp-edged object (knife, saw, axe) and will generally cause a linear, open fracture (Symes et al., 2012).

2.5.4 TRAUMA IN THE KIRSTEN SKELETAL COLLECTION

The cadaver-derived KSC is mostly donations or unclaimed individuals and therefore no unnatural deaths that underwent forensic autopsies are included (Alblas et al., 2018). As a result thereof, skeletal injuries on the skeletal elements from the KSC, were classified as blunt force- and sharp force trauma, no ballistic or burn trauma was observed. Further classification was antemortem- (healed) and post mortem- (damaged) trauma and no to very little perimortem injuries should be visible in this collection. Postmortem damage to skeletal elements in this collection is common because of processing artefacts and rough handling by students over a period of more than 50 years. The KSC does not represent the full Western Cape population, although the last residential address of most of the individuals in the collection indicates low SES areas in the Cape Flats region (Alblas et al., 2018).

2.5.5 FRACTURE SITES ON BONES

Although signs of antemortem trauma are preserved on human bones after death, it is estimated that only 16.0% of trauma-related assaults are visible on skeletal material (Kyriacou et al., 1999). The cause of trauma visible on bone will be dependent on the population studied due cultural and economic influences; however, the location of fractures on bone may give an indication as to what type of trauma was involved. The location of fractures should therefore be carefully assessed as they may aid in the determination of causality (Kyriacou et al., 1999). Hussain et al., (1994) verified in 950 patients that soft-tissue injuries were more commonly observed in accidental falls, while IPV recurrently resulted in craniofacial fractures. Injury as a result of the high-speed impact observed during MVA tends to result in multiple and more complex fractures that include all the maxillofacial bones as well as long bones, ribs and pelvic bones (Petridou et al., 2002; Lee et al., 2007).

2.5.5.1 Cranial bones involved

The cranium, divided into the neurocranium and viscerocranium, are commonly involved in traumatic injuries, as it is the most exposed part of the body (Adeyemo et al., 2005; Lee et al., 2007). The cranial bones (neurocranium) where signs of IPV are commonly observed include the two parietal and mid-cranially frontal bones (Gassner et al., 2003; Eggenesperger et al., 2007; Lee, 2009; O'Meara et al., 2012). The facial skeleton (viscerocranium) is divided into three areas when it comes to describing fractures: the upper face (orbits, and frontal sinuses), midface (maxillary, zygomatic, and nasal bones) and mandible. The temporal bones on both sides of the skull, and the occipital, sphenoid and ethmoidal bones mid-cranially are usually more involved in MVA cases (Lee et al., 2007). Trauma, more likely to be due to MVA, in the upper face, usually includes the superior orbits and the sinuses. Authors agree that fractures related to incidents of IPV are high in the head and neck area as it is commonly the most exposed site of the body (Oji, 1999; Adeyemo et al., 2005; Lee et al., 2007; Geldenhuys et al., 2016) with the most frequent site of fractures in the mandible, nasal and zygomatic bone areas (Lee, 2009). A direct blow to the midface region usually includes in-bending of the centre part of the zygomatic arches (İşcan & Steyn, 2013) with the two fractures to the sides of it. Brink et al. (1998) suggested that emergency personnel in hospitals should be suspicious that craniofacial fractures may be due to domestic violence and should be followed up. The midface and mandible are commonly involved on both IPV and MVA cases. Various skull injuries may result from a PVA where the pedestrian struck by a vehicle is knocked to the ground (McCoy et al., 1988).

2.5.5.2 Postcranial bones involved

Individuals which are frequently assaulted, will show evidence of multiple trauma sites at various stages of healing; therefore, individuals which displayed various skeletal lesions, especially of the upper extremities, including the scapula, radius, ulna, phalanges and ribs (Kyriacou et al., 1999; Judd, 2004; Fibiger et al., 2013), may have been involved in incidences of ongoing IPV (Brink et al., 1998; Fox, 2011). Conversely, tibial fractures in combination with fibular fractures are generally caused by PVAs, MVAs or falls from heights (Lovell, 1997).

Spinal column injuries

Injury to the spinal column or individual vertebrae is rare during IPV. It is more common during frontal or rear impact collisions in the course of MVAs, when the unstrained occupant of a car move into the steering column and windscreen after abrupt stopping of the vehicle, or PVAs at ground impact after being hit. (McCoy et al., 1988). An acute overload event that causes microfractures, or more likely, chronic trauma with repeated stress, may cause spondylolysis, it is known as a 'fatigue fracture' among palaeopathologists. Clinically, it is observed among labourers or athletes whose activities include lumbar hyperextension and lumbar flexion). The differences in prevalence between male and female were reported as activity related (Lovell, 1997).

Pelvis

Pelvic fractures is uncommon and is usually present on the superior and/or inferior ischio-pubic ramus and the wing of the ilium (Lovell, 1997). Although soft tissue damage will generally be involved in lateral impact during a frontal collision MVA, possible compressive pelvic injuries may occur (McCoy et al., 1988). Trauma to the pelvis are uncommon during IPV.

Shoulder

Clavicle injuries are commonly associated with MVA, falls onto the shoulder or an outstretched hand, and sport- or work-related injuries (Nordqvist & Petersson, 1995; Lovell, 1997; Postacchini et al., 2002). A study by Throckmorton & Kuhn (2007) found that only two of the 55 study subjects presented with clavicle fractures as a result of IPV. Clavicular fractures are commonly occur at the junction of the middle and lateral third of the shaft (Lovell, 1997). Scapula fractures are rare due to the complex system of surrounding muscle protecting it (Vigorita, 1999). A scapula will therefore only fracture if it is subjected to a considerable

amount of force or high energy, blunt injuries experienced during a high speed MVA, falls from heights or sharp kick to the shoulder blade (Weideman et al., 2000). Fractures to the scapula are often accompanied by other injuries to the shoulder, clavicle and ribs (Livingston & Hauser, 2003).

Upper limb

Forearm fractures predominantly result from a direct blow, which may be caused by MVAs, fall and physical conflict (Lovell, 1997; Judd, 2004). Although injuries to the middle and distal thirds of the humerus are largely caused by falls or due to MVA (Lind et al., 1989; Lovell, 1997), a direct blow from an assailant may similarly result in an injury in these regions (Lovell, 1997). However, a torsion fracture to the humerus may be caused during a sports injury (e.g. competitors in throwing events) (Lind et al., 1989; Cummings & Nevit, 1994; Lovell, 1997).

The prevalence of ulnar fractures was reported the highest of all long bones in studies by Judd (2008) (13.8%), Kilgore et al. (1997) (13.1%), and Alvrus (1999) (8.1%). The most common radial fractures are Colles' fracture on the distal shaft that happens during a fall on an outstretched hand (Lovell, 1997). Colles' fractures are common in females over 40 years-of-age (Lovell, 1997). Fractures in different regions of the ulna may indicate different causes of trauma. Fractures in the distal shaft of the ulna are called Parry fractures or defence fractures ascribed to shielding off an attacker with an upraised arm, resulting in a direct blow to the ulna (Lovell, 1997; Kilgore et al., 1997; Alvrus, 1999; Hertel & Rothenfluh, 2010). In most studies, Parry fractures have a higher occurrence in females (Ali, 2003; Judd, 2008). Conversely, proximal ulnar fractures near the olecranon are commonly a result of a fall onto the elbow (Lovell, 1997).

Fractures of the distal radius near the wrist joint are called Smith's fractures, causing displacement anteriorly and is caused by falling (or a direct blow) on to the back of the hand while Colles' fractures are fractures of the distal radius, causing displacement posteriorly and is caused by falling onto an outstretched hand. In the elbow region, a radial head fracture is the most common part broken and result from a fall onto an outstretched hand. Therefore, distal radial shaft fractures such as Colles', Smith's, and radial head fractures are associated with falls (Judd, 2004) rather than sport injuries, because a much larger force is needed to result in injury (Hertel & Rothenfluh, 2010). However, Solgaard & Peterson (1985) found that distal radial fractures were caused by a blow from a fist or blunt object in 15.0% of cases. In cases

where both the radius and ulna are involved on the same side, an accidental fall may be the cause (Judd, 2006). However, Hertel & Rothenfluh (2010) state that a primary mechanism of injury may be a direct blow, therefore listing IPV as well as MVA as a potential cause.

Ribs

Rib fractures may be caused by a variety of factors, such as accidental falls, contact sports, MVA- and IPV-related trauma and progressive spasmodic spells of coughing, or pathological fractures due to osteoporosis (Cummings & Nevit, 1994; Simarli et al., 2003; Brickly, 2006). However, isolated rib fractures, without accompanying long bone injuries may be indicative of IPV, as MVAs and accidental falls generally result in injury to multiple skeletal elements. Therefore, individuals that display rib fractures without any long bone injuries are strongly suspected to be involved in incidences of IPV. Brickley (2006) describes rib fractures as consistently being the most frequently fractured skeletal element in both historical and modern populations, occurring mostly in males. In addition, the prevalence of rib fractures increases with age. In living populations, rib fractures are described as occurring in 60.0% - 70.0% of all individuals admitted to hospital for blunt thoracic trauma (Lovell, 1997). In MVAs, unbuckled drivers may fracture several ribs against the steering column and windscreen (McCoy et al., 1988).

Lower limbs

The femora are the sturdiest bones in the body; therefore, fractures will only happen by severe direct or indirect high-energy trauma, such as MVAs, PVAs or falls from a height (Kolmert & Wulff, 1982; Lovell, 1997). In general, when an occupant in a car is unrestrained and the vehicle is involved in a frontal collision, the first impact point is usually the lower limbs as the body still moves forward after the vehicle comes to a sudden stop. This results in fractures of the ankles or femora, or dislocation of the knee or hip (McCoy et al., 1988). Isolated tibial shaft fractures, predominantly the distal third of the shaft, are commonly caused by falls, PVAs, MVAs or direct force as a result of sport related injuries; IPV are involved in less than 5% of tibial injuries (Lovell, 1997; Alvrus, 1999). Tibial fractures may occur in conjunction with fibular fractures, especially in a PVA as the vehicle bumper will hit an upright adult at the level of the lower limbs (McCoy et al., 1988) and team sport games.

In summary, South Africa is a high middle-income country, however majority of its people are classified in a low socio-economic category. This has the potential to influence health and disease in the country. The population of South Africa is made up of genetically diverse groups, contributing to a diverse genepool. The Apartheid regime in South Africa was a large determinant of health in the 20th century, favouring the SAW population group to the SAB and SAC population groups. This showed in other determinants of health such as housing, education, and substance abuse. This study looks specifically at a Western Cape Province population, using individuals in the Kirsten Skeletal collection, during the 20th century time period. The health of these individuals was evaluated based on pathology seen on skeletal material. This pathology included infectious diseases, congenital diseases, deficiency diseases, degenerative diseases, neoplastic diseases, amongst others. In addition, health was also reviewed based on traumatic injuries on bone. This also alluded to social constructs within the population groups. Violence was categorised according to the nature, such as intrapersonal violence or motor vehicle accidents. Groups such as age, sex and population groups were assessed separately for comparison purposes. Thus, the overall health of a Western Cape population was assessed in multiple facets of the lifestyles of the individuals examined.

CHAPTER 3: MATERIALS AND METHODS

3.1 MATERIAL

3.1.1 THE KIRSTEN SKELETAL COLLECTION

The Kirsten Skeletal Collection (KSC), which was started in the early 1960's (Labuschagné & Mathey, 2000), is housed at Stellenbosch University's Medical campus at the Division of Clinical Anatomy, Department of Biomedical Sciences, Faculty of Medicine and Health Science. The skeletal specimens (n=1161) were obtained mainly from cadavers used for the training of medical students at the University (Labuschagné & Mathey, 2000; Alblas et al., 2018).

Under the Human Tissue Act (65 of 1983), the more recent National Health Act (61 of 2003), and the protection of the regional Inspector of Anatomy, Stellenbosch University is allowed to receive cadavers for both teaching and research purposes. These cadavers are comprised of consented donations and unclaimed bodies of persons who died from natural causes in the Western Cape region.

While most of the cadaver intake is skeletonised, according to the protocol of the Division of Clinical Anatomy, to use as teaching aids for undergraduate and postgraduate students at the University, some of the processed skeletal material is received, registered and curated at the KSC Ossuary. All pertinent information of each individual skeleton is entered into a database, contributing to a full skeletal inventory. The KSC is not considered representative of the current population of the WCP, mainly due to the unequal distribution in terms of age, population group and sex in the donated cadavers. It has been established (Hunt & Albanese, 2005; L'Abbé et al., 2005; Komar & Grivas, 2008; Dayal et al., 2009) that cadaver donations for anatomy teaching include an over-representation of males, the elderly, and individuals of low socio-economic standing. However, the data derived from the KSC remain useful for examining trends within a specific community.

The KSC consists of individuals from three main population groups, namely South African Coloured (SAC, 59.7%), South African Black (SAB, 16.5%) and South African White (SAW, 12.2%) groups. These cadavers were acquired mostly from teaching hospitals such as Tygerberg (32%) and Karl Bremer (10.1%), both situated in Bellville, a Northern suburb of Cape Town. Townships close to these hospitals were established in the mid-20th century to supply the large industrial parks in the Bellville area with labourers. These townships housed mainly families from the SAC population group. Therefore, the major over-representation of

the SAC population group in this study can be attributed to the ‘catchment’ areas of the hospitals (Whittingdale, 1973, Friedling, 2004). Furthermore, the regional population composition of the Western Cape Province differs considerably from that of other provinces in South Africa. Census figures from the mid-1900s show that at least 50.0% of the Western Cape inhabitants at the time were from the SAC population group (Labuschagné & Mathey, 2000), and not the SAB population group, as was the case in the rest of SA. Therefore, the unequal population group distribution in the KSC, derived mainly from Western Cape individuals, is to be expected.

The KSC represents a population who lived between the mid- and late 20th century, as most of the individuals were born between 1920 and 1949 (42%) and died between 1970 and 1989 (54%) (Pfeiffer, et al., 2016; Alblas et al., 2018). The sample comprises approximately 60% male skeletons and 40% female skeletons (Alblas et al., 2018), which does not reflect the male to female ratio in the SA population group as, according to the most recent census, the national population is predominantly female (Stats SA Census, 2012). The average age at death in the collection was between 40 and 60 years (Alblas et al., 2018). Ages ranged from 10 to 103 years, with a mean age of 51 years (Alblas et al., 2018). When comparing the age categories in which the most individuals died among population groups, it becomes apparent that the highest number of individuals in the SAB and SAC population groups died in the mid-adult age category (40-60 years old) (males in their 50s, and females in their 40s); however, in the SAW group more individuals belonged to the old-adult category (older than 60 years), with males mostly being in their early 60s and females in their late 60s at the time of death. During the mid-1900s, members of the SAW population had a life expectancy of 60-69 years, while for the SAC group it was 40-49 years, and even lower for the SAB population (van Rensburg & Mans, 1982).

3.1.2 THE STUDY SAMPLE: KIRSTEN SKELETAL COLLECTION

The sample used in this study included cadaver-derived adult individuals from the KSC, born in the 20th century and with known cadaver records. Where disparity existed between collection identification number and cadaver number, individuals were excluded from the study. Known records of COD and demographics of individuals were blinded. A total of 624 individuals from the 1161 included in the KSC met the aforementioned criteria and were included in the current study. Twenty of the unclaimed individuals in the cohort had limited records available, and information such as age and sex were unknown. Established age- and

sex-estimation methods were used to complete the missing data. Of the individuals included in the study, 414 (66.3%) had all skeletal elements present, while the rest of the skeletons (33.7%) were incomplete, missing some skeletal elements as a result of losses due to loans to students or other departments/institutions in the past (Alblas et al., 2018). Males made up 70.2% (n=438) of the sample, and females 29.8% (n=186). A male bias is common in many collections originating from cadaver donations (Hunt & Albanese, 2005, L'Abbé et al., 2005; Komar & Grivas, 2008, Dayal et al., 2009, Alblas et al., 2018).

The age at death of skeletons in the study sample ranged from 20 to 90 years. Three age categories were established according to age at death (Table 3.1), namely: 1) a young adult age group (Y) with an age at death of between 20 and 39 years (n=146); 2) a mid-adult age group (A) of between 40 and 59 years old at the time of death (n=320); and 3) an old age group (O) with individuals of between 60 and 90 years old at the time of death (n=158). Age classification was limited to three categories in order to allow adequate numbers for statistical analyses per category. The mean age at death of the study population was 49.1 years, with males having a mean age at death of 49.8 years, and females a mean age of 46.2 years. The mean age at death for each sex and population group in the cohort, is indicated in Table 3.2.

A median was calculated for the time period during which each individual lived. For example, an individual born in 1935 and who died in 1990 lived for 55 years during the 20th century with the median of this life span being 1962. The 20th century period was subsequently subdivided into three time periods, according to these medians, namely 1) early 20th century (n=67), including individuals with a median of between 1928 and 1940 (coinciding with economic restraints due to the Great Depression, and the pre-antibiotic period); 2) mid-20th century (n=283), comprising individuals with a median between 1941 and 1956 (a period with many socio-economic challenges, such as WWII and the start of apartheid, but also the introduction of antibiotics); and 3) the late 20th century and beginning of the 21st century (n=274), including individuals with a median between 1957 and 1995 (representing the height of apartheid, the start of HIV infections, political unrest, and the establishment of democracy). The under-representation of the early era can be ascribed to the fact that the collection was only started when the medical school at Stellenbosch University opened in the late 20th century (Alblas et al., 2018).

The available last-residence records for the individuals of the KSC indicate mostly low SES areas such as the Cape Flats, implying poor employment, health care, and housing (Pfeiffer

et al., 2016), even after the end of apartheid in 1994. Full personal medical histories of all individuals are unknown.

Table 3.1. Population group, sex and age composition of the cohort.

	N	SAB (n=102)		SAC (n=439)		SAW (n=83)	
		Male	Female	Male	Female	Male	Female
Young adult (20-39yrs)	146	24	7	61	51	3	0
Mid adult (40-59yrs)	319	45	9	174	59	23	9
Old adult (>60yrs)	159	15	2	68	26	25	23
Total:	624	84	18	303	136	51	32

SAB=South African Black; SAC=South African Coloured; SAW=South African White

Table 3.2. The mean age at death for each sex and population group per age group (in years-of-age).

	SAB (avg=48 yrs)		SAC (avg=48 yrs)		SAW (avg=54 yrs)	
	Male	Female	Male	Female	Male	Female
Young adult (20-39yrs)	33	31	32	31	36	0
Mid adult (40-59yrs)	50	46	49	48	53	46
Old adult (>60yrs)	64	63	65	63	67	69

SAB=South African Black; SAC=South African Coloured; SAW=South African White

3.1.3 ETHICAL CONSIDERATION

The Human Tissue Act (65 of 1983), and the more recent National Health Act (61 of 2003), make provision for the use of human tissue or skeletal material in medical research and education, provided that some form of consent has been documented. In circumstances where original (donor) consent or next-of-kin consent could not be obtained, as is the case of some individuals in this study, legislation allows for the Inspector of Anatomy, as the representative of the Director General, to provide consent by proxy, provided that he/she is satisfied that all attempts have been made to contact next of kin for consent. In the KSC, the exact number of donated versus unclaimed bodies between 1957 and 1996 were not recorded, therefore the ratio of donated to unclaimed bodies in this study could not be accurately determined. This study was ethically approved by the Health Research and Ethics Committee (HREC) of Stellenbosch University (S13/05/100) and conforms to the principles of the Declaration of Helsinki (1964).

3.1.4 LIMITATIONS OF THE STUDY

Several limitations were taken into account during this study. The KSC cadavers were mainly males, older persons, and individuals of low socio-economic background, causing an unequal distribution of individuals available to study. Furthermore, no access to medical histories or personal information of these donated or unclaimed individuals are known, therefore, disease and trauma lesions on skeletal material in the KSC cannot be referenced to social context of the society and their usefulness as indicators of a healthy or diseased society are therefore not exact. This study was considered in the context of the osteological paradox limitations first described by Wood and colleagues in 1992. According to the authors, where such limitations are present, a study should allow for the underlying vulnerabilities in the skeletal population, including heterogeneity in disease risk, selective mortality, and demographic variability (Wood et al., 1992).

Some of the cadavers were unclaimed and unknown individuals, without written records of age and sex. In these cases, biological profiles were estimated, possibly resulting in incorrect information. However, the low number of cases of unknown age and sex in the cohort (3.2%) should not have a significant impact on the overall results.

In some cases, skeletal remains were incomplete due to loans to students and other departments or institutions, influencing the number of bones available for analysis. The handling of bones by students over the course of almost 50 years caused damage and erosion to some of the bones, while processing artefacts also played a role. The impact of both these influences on the skeletal material were taken into account during analysis of the skeletal elements. Handling and processing damage were seen as postmortem signs of erosion, discolouration and exposure of spongy bones of skeletal elements. Some bones of older individuals showed more damage due to osteoporotic influences.

Additionally, the dissection methods used in anatomy training resulted in damaged skeletal elements, including horizontally sectioned calvaria for removal of brains, a mid-cut of the mandibulae, as well as sectioning of ribs 3-7 to open the thoracic cavity. Most of these ribs were cremated by previous curators before the bones were acquired by the KSC Ossuary. Many individuals did not have a sternum present, as it is often destroyed in the process of opening the chest during dissection. The hand and feet bones were articulated by technical staff prior to storage in the Ossuary. More recent intakes are still awaiting articulation and were not available for examination.

3.2 METHODS

Age and sex were taken into account when diagnoses were made, as many diseases may be more prevalent in a specific sex or age group (İşcan & Steyn, 2013). Comparisons of findings between the sexes and among age groups were made; therefore, a biological profile estimation was completed for individuals in the KSC of unknown age and sex. Death certificates (which should indicate at least sex and ancestry), were available for all individuals included in the study; however, three individuals needed a sex estimation due to incomplete records, while 20 individuals (3.2% of study cohort) had no age recorded on the available documents and demographic estimations were done.

3.2.1 BIOLOGICAL PROFILE

Various non-metric scoring methods of both cranial and postcranial landmarks were used to estimate biological profile information, including chronological age and sex of individual skeletons with unknown demographic records.

3.2.1.1 Estimation of sex

The skulls and/or the pelvis bones were used for sex estimation in cases of unknown sex. A combination of three methods were used, depending on which bones were available:

- a. A scoring system based on five aspects of skull morphology by Acsadi and Nemeskéri (1970), and modified by Walker (2008), was used to estimate sex based on cranial features.
- b. The Phenice method (Phenice, 1969), modified by Klales et al. (2012), also known as the KVO method, was used to estimate adult sex. The subpubic concavity (SPC) on the ventral pubic ramus, the medial aspect of the ischio-pubic ramus (MA), and the ventral arc (VA) on the os coxae of unknown individuals were macroscopically studied. When the SPC is viewed from the dorsal aspect, the inferior border of the ischio-pubic ramus is seen as a relatively straight line from the pubic symphysis in males, while it is more concave in females. The MA is broader and flatter in males, and narrow or ridged in females. If an elevated ridge of bone was visible on the ventral surface of the pubis, lateral to the pubic symphysis, it indicated female sex. When absent, male sex was assumed.

- c. A scoring system for the greater sciatic notch on the os coxa was used, where a score of “1” is allocated for the broad greater sciatic notch (typically present in females), and a score of “5” for a narrow greater sciatic notch (typically seen in males) (Buikstra & Ubelaker, 1994).

3.2.1.2 Estimation of chronological age:

- a. The Transition Analysis Method (Boldsen et al., 2002) was generally used in the estimation of age in this study. It involves scoring of the changing morphological characteristics of the pubic symphysis (Brooks & Suchey, 1990), the auricular surface morphology of the ilium (Lovejoy et al., 1985; Buckberry & Chamberlain, 2002), and degree of closure of the cranial sutures (Meindl & Lovejoy, 1985; Todd & Lyon, 1924, 1925).
- b. In addition, scoring of the changing morphological characteristics of the sternal ends of the 3rd, 4th and 5th ribs was used (İşcan et al., 1985; Krogman & İşcan, 1986; Oettlé & Steyn, 2000) if these ribs were available.
- c. Signs for young age (e.g. non closure of the medial end of the sternum) and old age (osteophytic changes in joints) were additional used for age-at-death estimation.

3.2.2 MACROSCOPIC OBSERVATION

The skeletal elements were placed in anatomical order (Fig. 3.1) and each skeletal element was macroscopically analysed for signs of pathology or trauma, using a standard fluorescent lamp with a magnifying glass at 3x magnification. All skeletal elements present or absent were recorded. Individuals with one rib or vertebra were reported as “ribs/vertebra absent”. All observations were recorded on scoring sheets in *Excel* (MS Windows 10, Microsoft Cooperation, USA), using drop down menus. The position of each lesion was scored, including the side, aspect, and section of the bone element.

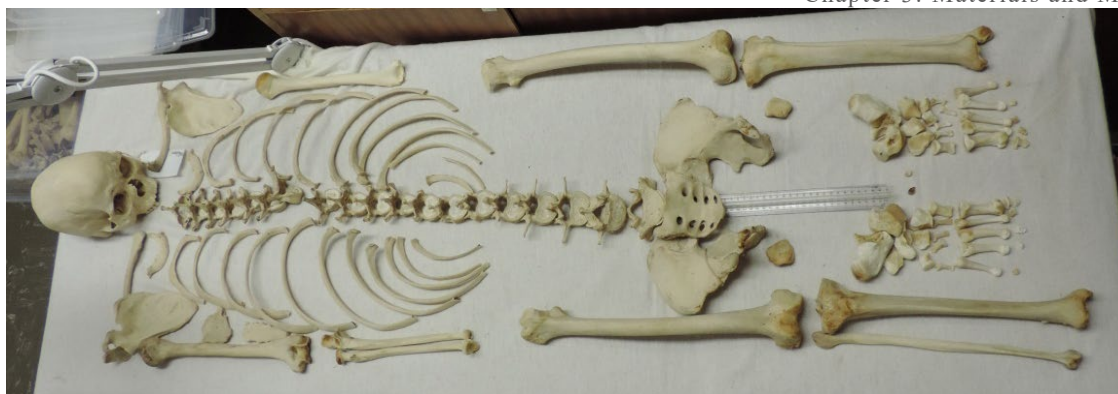


Photo by: A Alblas

Figure 3.1. Individuals were laid out on a table in anatomical order for macroscopic analysis.

3.2.2.1 Cranial scoring

The cranial elements scored for signs of pathology and trauma included the frontal bone, both parietal bones, both temporal bones, both zygomatic bones, the occipital bone, the sphenoid bone, both sides of the maxillary bones, both nasal bones, and the mandible (Fig. 3.2). As most individuals analysed showed extensive post-mortem damage to the fragile ethmoidal plate, vomer, and palatine bones (due to extensive handling of the skulls over a 50-year period), data derived from these bones were excluded.

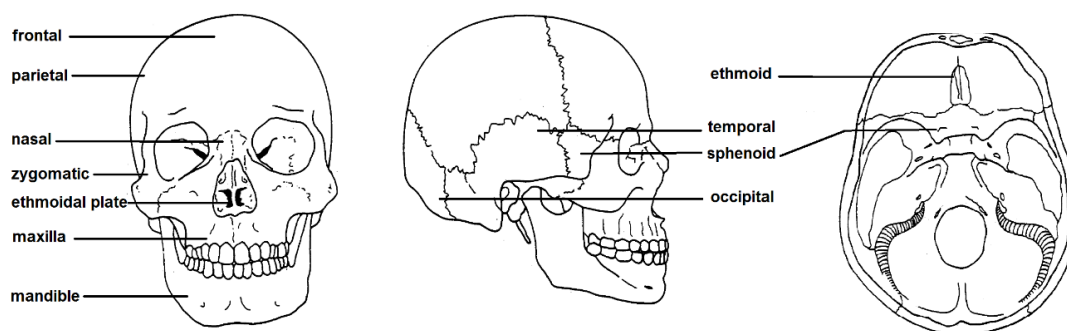


Figure 3.2. Cranial bones scored in this study.

3.2.2.3 Postcranial scoring

Long bones (humeri, ulnae, radii, femora, tibiae, fibulae, clavicaulae), scapulae, manubriosternum, ribs, vertebrae, sacra, ossa coxae, and hands and feet present were scored for signs of pathology or trauma. The large joints examined for signs of DJDs included the shoulders, elbows, wrists, hips, knees, and ankles (Fig. 3.3). The unit of analysis for each skeletal element is explained later in the methods.

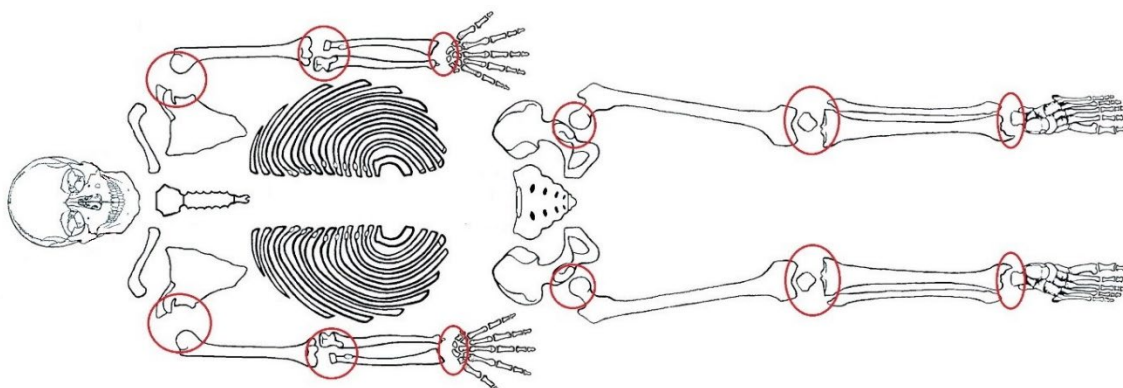


Figure 3.3. Skeletal diagram showing peripheral joints scored for degenerative lesions.

3.2.3 RADIOLOGICAL OBSERVATIONS

All complete individuals in the KSC were imaged in anatomical order with a cost-effective Lodox[®] Statscan digital low dose X-ray scanning system (Lodox[®] Systems Pty., Sandton, South Africa) to visualise the skeletal elements of each individual (Fig. 3.4). Bones with lesions from incomplete individuals were scanned separately. The DVS[®] vs 2.8 software package was used to view these digital x-rays, and observations were done in DICOM[®] image format, viewed by ImageJ[®] 1.4r freely available software. Additionally, lucid[®] software was used to enhance the bone on images (Kotzé et al., 2012). Skeletal elements with evidence of pathological lesions, surgical interventions, and trauma were given specific attention. Lesions observed on the digital scans were confirmed by a qualified radiologist (Dr Carl Holdt) from the Division of Radiodiagnosis (Department of Medical Imaging and Clinical Oncology, FMHS, Stellenbosch University).



Photo by: A Alblas

Figure 3.4. Individuals were laid out on the Lodox[®] Statscan[®] table in anatomical order for digital X-rays.

3.3 PATHOLOGY AND TRAUMA ANALYSIS

Bone has limited ways in which it will react to pathological stimuli; hence, pathology visible on dry bone, lacking organic material, is difficult to diagnose. Even when dry bone is examined by radiological or histological methods, any changes, which may have occurred in, for instance, the marrow, are unknown. These can include changes in the vascularity, hyperplasia of the haemopoietic elements, and the presence of chondroid and osteoid matrices (Ortner, 2003; Waldron, 2009; Aufderheide & Rodríguez-Martín, 2011). For this study, the lesion pattern observed on the skeletal material of each individual was described by location, gross (macroscopic), and radiographic (digital X-ray) appearance. Pathology, metabolic and nutritional disorders, trauma, and some congenital variations in individual skeletons were analysed and diagnosed as per criteria set out by Ortner (2003), Waldron (2009), and Aufderheide & Rodríguez-Martín (2011).

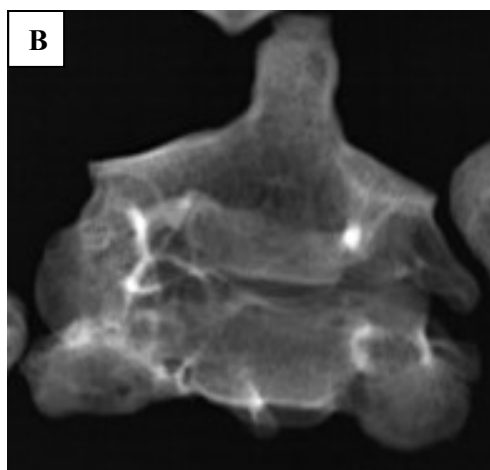
3.3.1 CONGENITAL/GENETIC ORIGIN

3.3.1.1 Klippel-Feil syndrome (KFS)

The presence of two or more adjacent fused cervical vertebrae (Fig. 3.5A&B) was analysed to determine whether the fusion could be the result of a congenital disorder (KFS) or of a traumatic event or degenerative disease. If any other trauma was visible on the skeleton, indicating that the ankylosed vertebrae may have been due to a traumatic event, it was scored as trauma. Otherwise, without supporting evidence of KFS, it was scored as degeneration. Abnormalities known to be associated with KFS, such as rib anomalies, spina bifida, scoliosis, cranial-caudal border shifts, cleft palate, or dental problems (Hensinger et al., 1974; Barnes, 1994; Fernandes & Costa, 2007) were taken into account when diagnosing KFS.



Photo by: A Alblas (AN 637). Anterior view.



Lodox® image: J Walters (AN 732). A-P view.

Figures 3.5A-B. Ankylosed C2-C3 cervical vertebrae, indicating Klippel-Feil syndrome on A) dry bone and B) Lodox® image.

3.3.1.2 Cleft neural arches/spina bifida occulta (SBO)

Non-fusion between the two halves of the neural arches of any of the vertebrae (Fig. 3.6A&B) was scored as presence of a cleft neural arch (Barnes, 1994; Ortner, 2003). Each vertebra was scored, including sacral elements. A cleft neural arch on the sacrum was scored present if at least one or more of the neural arches of S1, S2, and S3 were open (Fig. 3.6C, line arrow), while open S4 and S5 segments were regarded as normal variations of a high sacral hiatus on the inferior aspect of the canal (Fig. 3.6C, block arrow) (Barnes, 1994). If the spinal arches of all the sacral vertebrae were unfused (Fig. 3.6D), it was regarded as a complete SBO.

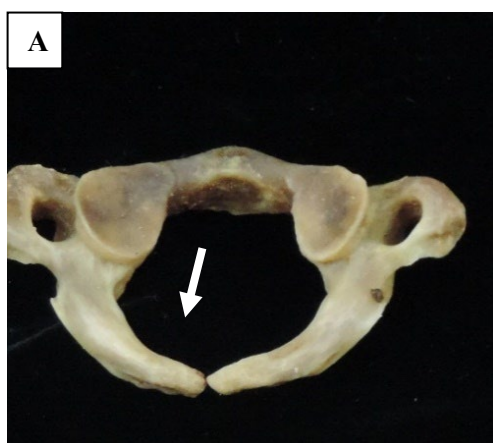


Photo by: A Alblas (AN 1198),
superio-posterior view.

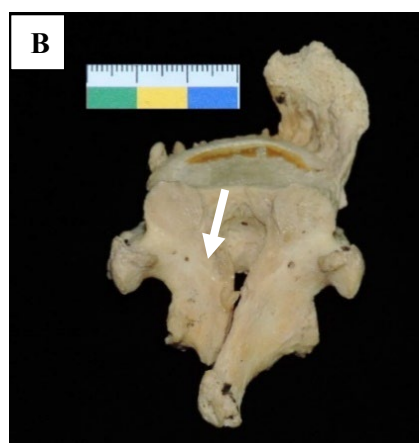


Photo by: A Alblas (AN 1198),
posterior view.

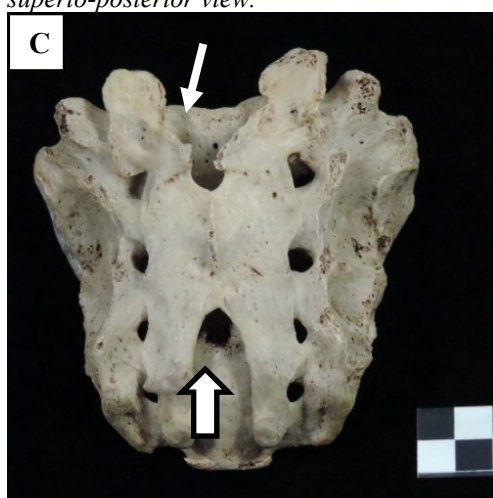


Photo by: A Alblas (AN 126), posterior view.

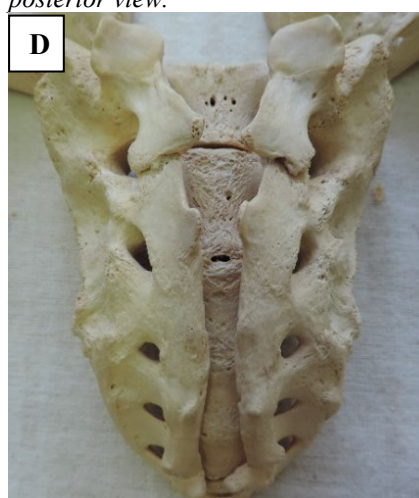


Photo by: A Alblas (AN 849), posterior view.

Figures 3.6A-D. Cleft neural arch in the A) atlas; B) lower thoracic vertebra; C) SBO on S1, but high hiatus on the inferior aspect of the canal; and D) complete SBO of all the sacral vertebrae.

3.3.1.3 Lumbosacral transitional vertebrae (LSTV)

Lumbar vertebrae (where present) were examined to determine if a 6th sacral vertebra resulted from fusion of either a lumbar or a coccygeal vertebra to the sacrum. Supernumerary vertebrae were also taken into account if a 6th lumbar vertebra was present. The presence and type of LSTV were indicated by examining all vertebrae between L5 and S1. Classification was done according to Castellvi et al. (1984) (Table 3.3, Fig 3.7).

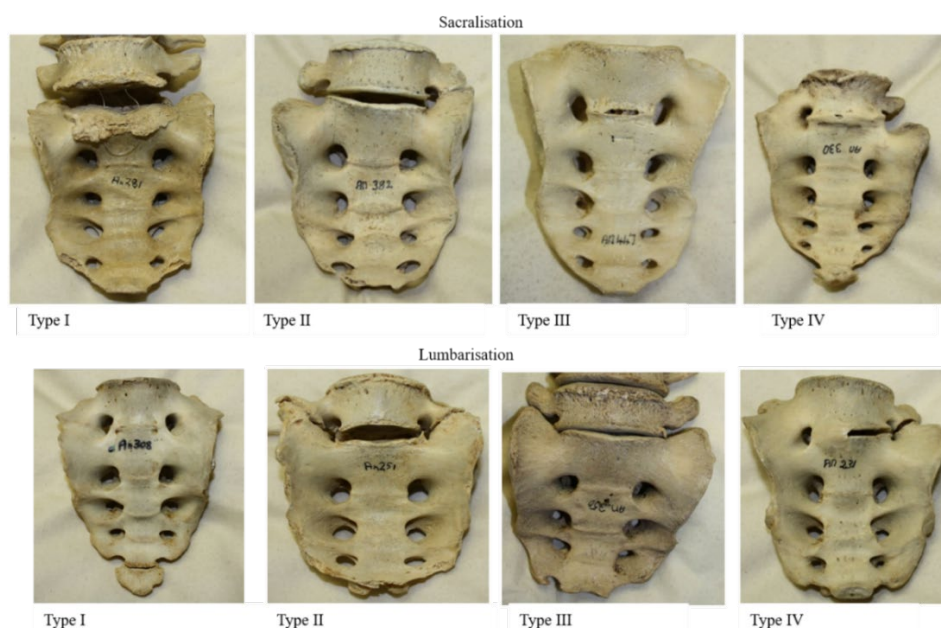
Table 3.3. Classification of lumbosacral transitional vertebrae after Castellvi et al. (1984)

	Grade	Description
Sacralisation	Type I ^a	Dysplastic transverse process of L5 with measurement of >19mm.
	Type II ^a	Incomplete sacralisation (diarthrodial joint between the transverse process and sacrum).

	Type III	Complete sacralisation with complete fusion of L5 with the neighbouring sacral basis, resulting in 4 lumbar and 6 sacral vertebrae.
	Type IV	Mix of Type II on the one side and Type III on the other side.
Lumbarisation	Type I ^a	Well-formed lumbar-type of facet joints on S1 viewed posteriorly.
	Type II ^a	Anomalous articulation of a full sized disk rather than fusion between S1 & S2.
	Type III	Complete lumbarisation with complete separation of S1 from the sacrum, resulting in 6 lumbar and 4 sacral vertebrae.
	Type IV	Mix of Type II on the one side and Type III on the other side.

^aType I and Type II lumbosacral transitional vertebrae may be further classified as (a), where fusion between the transverse process and the sacrum is unilateral, or (b), where the fusion is bilateral.

Source: Konin & Walz (2010)



Photos by: A Alblas. Anterior view.

Figure 3.7. Anterior view of sacra showing the types of lumbosacral transitional vertebrae (LSTV).

3.3.2 INFECTIOUS DISEASE MARKINGS

Specific chronic bacterial infections were diagnosed according to characteristic distribution patterns.

3.3.2.1 Specific bone infections

3.3.2.1.1 Tuberculosis (TB, Pott's disease)

Macroscopic and radiographic analyses were carried out on all bones present. In an effort to avoid destructive analysis, no bone samples were taken for histological confirmation. If only one rib was present in an individual, ribs were scored as 'absent'. Signs of pulmonary tuberculosis (PTB) was seen as a periosteal reaction on the visceral surfaces of any of the ribs present, resulting from inflammation of the contiguous pleura, as described by several authors (Roberts et al., 1998; Bennike, 1999; Souza, 2002; Roberts & Manchester, 2005; Steyn et al.,

2013; Geldenhuys, 2014). However, it was taken into account that such periosteal reactions may also have been caused by pulmonary cancer. Each rib number and side with lesions were noted, although many ribs between rib 3 and 7 were cremated by previous curators of the KSC. All skeletal elements showing signs of extra-pulmonary tuberculosis, as described by Ortner (2003), were noted. These skeletal signs include periosteal reactions, woven bone deposits, and lytic lesions on the bone surfaces (Ortner, 2003). The vertebral column was divided into three groups, namely the cervical, thoracic and lumbar regions. The vertebrae present for each of the groups were scored for Pott's disease lesions or TB on the vertebral column. Pott's disease was scored in the presence of two or more of the following traits described by Ortner (2003): 1) cavitation on the vertebrae due to destruction of the trabecular bone; 2) collapse of the affected vertebral body; 3) loss of intervertebral disc space; 4) fusion of the adjacent infected vertebrae; and 5) kyphosis. All the vertebrae affected were scored in each region. If one or more vertebrae were absent for each group, the rest of the vertebrae in the group were scored. If one or less vertebrae were present, the individual was reported as 'vertebrae absent' on the scoring sheet.

3.3.2.1.2 Localised infections

There are no standard methods to determine the presence of concha bullosa, otitis media, petrositis, or mastoiditis. Possible infection in the cranial pneumatised cells and sinuses are seen as bone remodelling after resorption and proliferation (Flohr & Schultz, 2009). However, gross and radiological observations cannot explain the cause, pathogenesis, or type of disease involved in the osseous changes that took place in localised cranial areas (Rathbun & Mallin, 1977). All observations were therefore scored with caution, as normal variation or other diseases may have caused bone remodelling. Histological studies or opening of the pneumatised cells were not attempted, and Computer Topography methods were unavailable. Concha bullosa (Fig. 3.8A) were scored as present if, in a unilateral anterior and posterior view, the middle or inferior concha visible in the nasal aperture were enlarged when compared to the other conchae of the individual. An association with a marked shift of the nasal septum was also noted.

Osseous changes in the temporal bones were observed macroscopically with the aid of a magnifying ring light. Scoring followed the criteria set out by Rathbun and Mallin (1977). A sclerotic, thickened, or layered tympanic cavity or plate (Fig. 3.8B), external pitting or porosity on the suprameatal triangle on the temporal bone, and ossified spicules (Fig. 3.8C) in the

external acoustic meatus were scored as otitis media, while porosity or pitting and extensive sclerotic activity on the superior and/or the inferior petrous part of the temporal bone (Fig. 3.8D) were scored as petrositis or infection of the internal acoustic meatus.

Osseous changes in the pneumatized cells of the mastoid bones were observed macroscopically, as well as on Lodox[®] X-ray images. Signs of bone remodelling, such as sclerotic and thickened walls, massively enlarged air cells, pitting and proliferation, were taken as possible infectious disease markings in the mastoids.

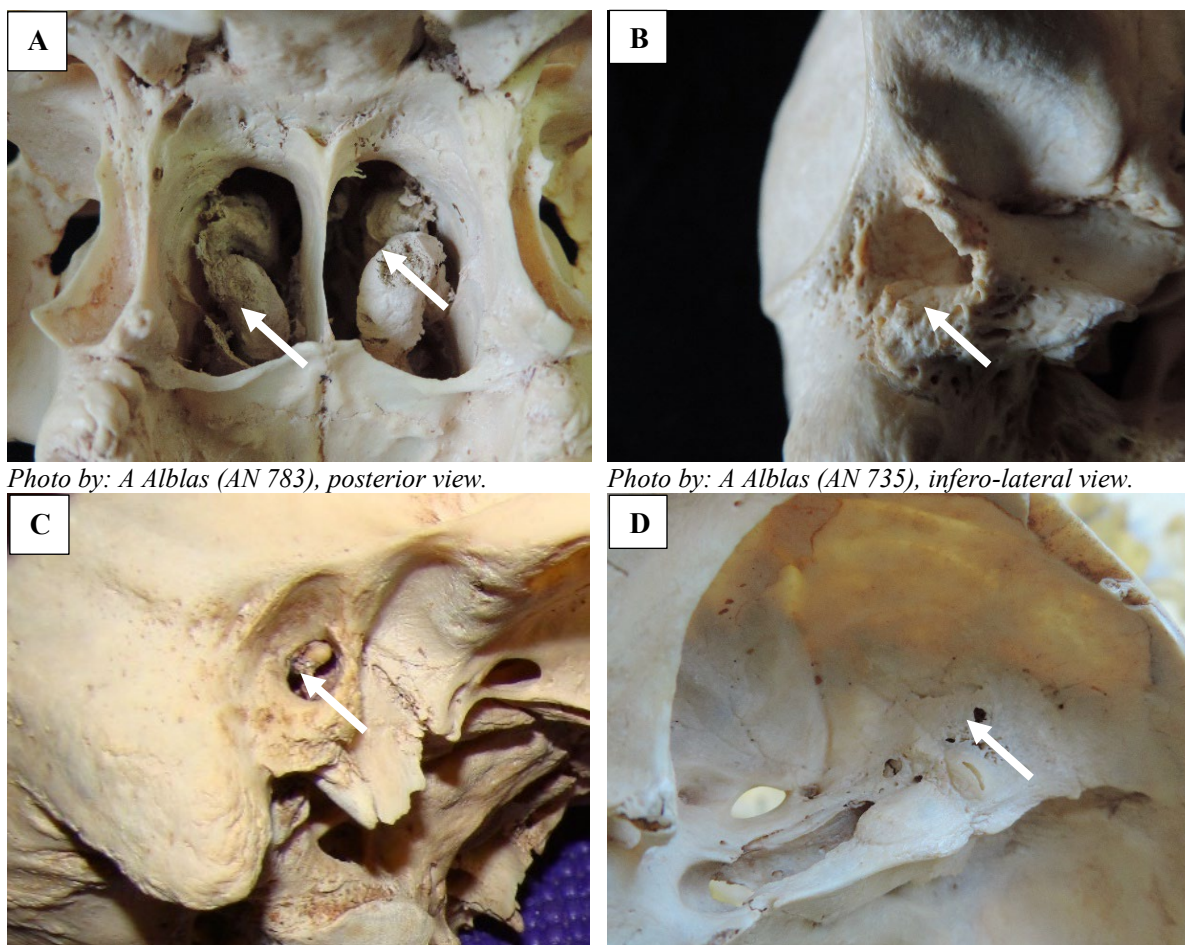


Photo by: A Alblas (AN 783), posterior view.

Photo by: A Alblas (AN 735), infero-lateral view.

Photo by: A Alblas (AN 380,) lateral view.

Photo by: A Alblas (AN 810,) inner-cranial view.

Figures 3.8A-D. Localised infections A) concha bullosa; B) otitis media; C) ossified spicules (arrow) in the external acoustic meatus; D) petrositis on inner petrous part of the temporal bone.

3.3.2.1.3 Periodontal diseases or infections and dental health

Modification and extraction of partial or full dentition was normal for inhabitants of the Western Cape during the 20th century. The SAC community, for example, commonly extracted their incisors as part of a cultural practice (Friedling & Morris, 2007). It was also more economical and easier to extract infected teeth than to visit a dentist or treat the infection with

antibiotics, as dentists and antibiotics were not readily available to all individuals during the apartheid regime. Many individuals in the KSC consequently had few or no teeth to examine for dental health; therefore, dental health findings may not be a true representation of the dental health status of the study sample.

All available teeth on the maxillae and mandibles were examined for signs of calculi, caries, abscesses and stains. The total dentition was taken as the unit of analysis. If any of the teeth showed signs of one of these traits, the trait was scored as present, otherwise it was scored as absent (Fig. 3.9A-F). Any dental work on the teeth was also indicated. The assistance of a qualified dentist, Dr André Uys from the Faculty of Health Sciences, School of Dentistry, Department of Oral Pathology and Oral Biology at the University of Pretoria, was obtained to help with diagnosis of dental diseases and anomalies seen on the teeth of individuals in the KSC.

Dental caries (Fig. 3.9A&C) were scored as present if the enamel and dentin layer underneath were eroded due to demineralisation (Southam & Soames, 1993). Although dental staining and calculi are not regarded as dental diseases, both are indications of poor oral hygiene and health (Silk, 2014).

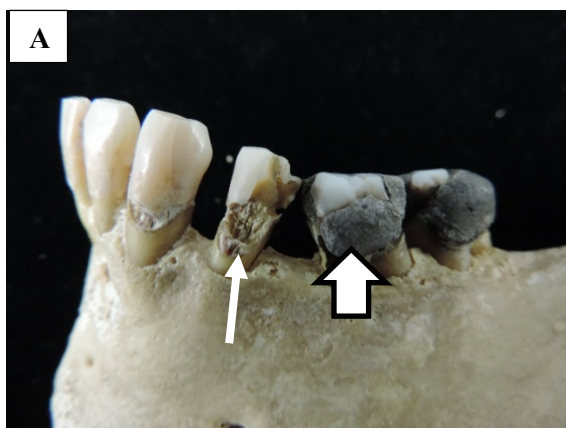


Photo by: JC Marais (AN 691) lateral view of mandible.



Photo by: JC Marais (AN 818), lateral view of maxilla.



Photo by: JC Marais (AN 853), superior view of mandible.



Photo by: JC Marais (AN 853), lateral view of mandible.

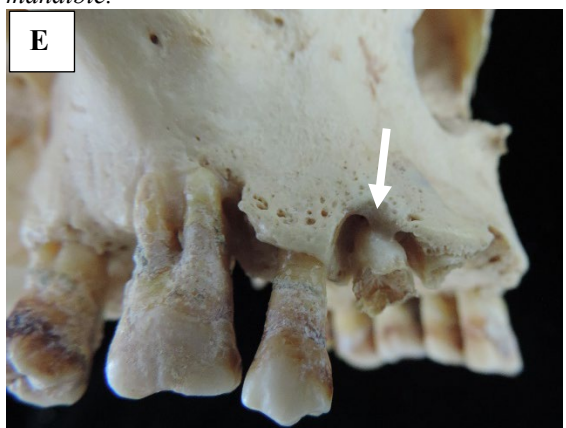


Photo by: JC Marais (AN 806), lateral view of maxilla.



Photo by: JC Marais (AN 619), lateral view of maxilla.

Figures 3.9A-F. Dental disease and modification. A) dental work (block arrow), also note the cervical carie (arrow); B) high calculus; C) prominent dental caries; D) stains on teeth; E) periodontal abscess; and F) periapical cyst.

3.3.2.2 Non-specific bone infections

3.3.2.2.1 Periosteal reaction

Non-specific periosteal responses in long bones are associated with the healing phases of periostitis and was scored based on surface appearance. This appearance was scored according

to the classification system of Wilczak and Jones (2012) as: 1) striated (where deposits of woven bone resulted in a ridged appearance; 2) nodular (if a sclerotic proliferation was visible); or 3) irregular (where the sclerotic activity may appear undulated and elevated in an irregular pattern) (Fig. 3.10A-C).

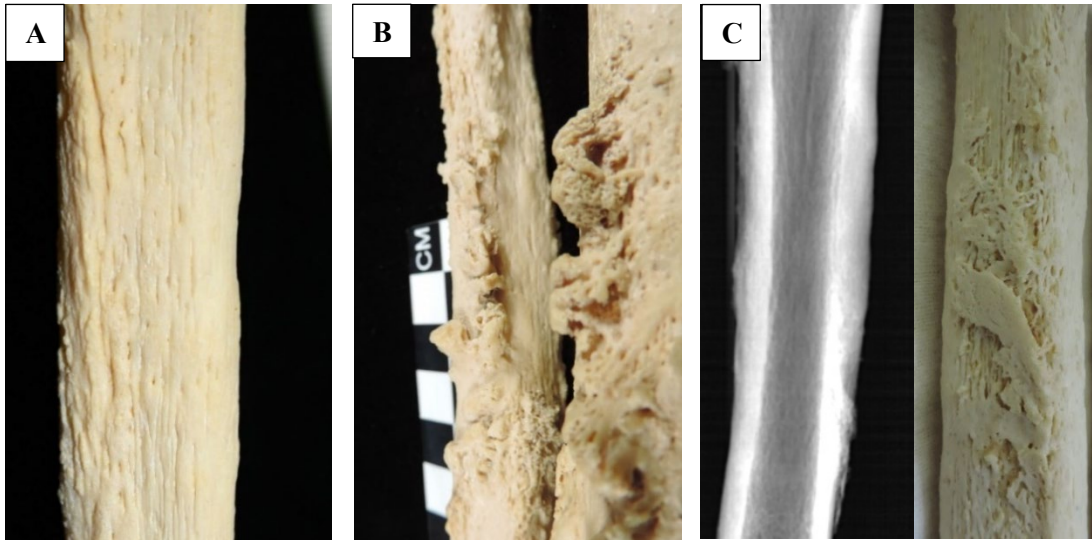


Photo by: A Alblas (AN 828). Photo by: A Alblas (AN 476). Photo by: A Alblas (AN 240); Lodox[®] image by: JC Marais.

Figures 3.10A-C. A) Striated; B) nodular; and C) severe irregular periosteal reaction.

3.3.2.2.2 Osteomyelitis

Osteomyelitis was scored as present in cases with visible sequestrum, involucrum, and/or a cloaca (Fig. 3.11A&B), as suggested by Wilczak & Jones (2012). The affected skeletal element and the location of the osteomyelitic response on the bone were also noted.



Photo by: A Alblas (AN 107), anterior view of ulnar shaft.

Photo by: A Alblas (AN 436) anterior view of tibial shaft.

Figures 3.11A-B. Osteomyelitis of a long bone on A) on dry bone with sequestrum, (line arrow) and involucrum, (block arrow); B) a cloaca (arrow) for pus draining.

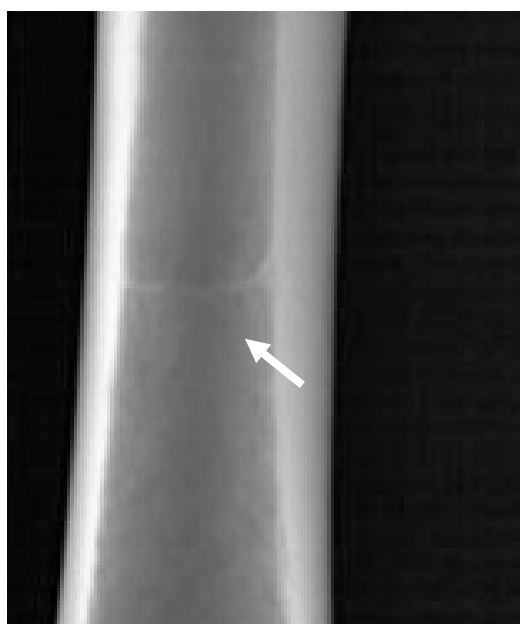
3.3.3 DEFICIENCY DISEASES

The bones of all individuals were macroscopically and radiologically assessed and scored for skeletal indicators of metabolic disorders, nutritional deficiencies, and hormonal disturbances

3.3.3.1 Metabolic and nutritional stress disorders

3.3.3.1.1 Harris' lines (HL)

Radiologically, growth arrest lines, or Harris' lines (HL), are visible as wide transverse lucencies across long bones (Fig. 3.12). The criterium used to identify HL in this study was the presence of one or more lines, observable on enlarged digital Lodox[®] scan images in an anteroposterior position. These lines extended across the endosteal border towards the diaphysis of the humeri, ulnae, radii, femura, tibiae and fibulae, as described by Clark (1978). The lines were only scored as present if they were symmetrical on both long bones. The number of lines per long bone was not considered in this study, only the presence of HL and the long bone involved.



Lodox[®] image by: J Walters, A-P view.

Figures 3.12. Harris' lines across the diaphysis of the tibia (arrow).

3.3.3.1.2 Dental enamel hypoplasia lines (DEH)

Teeth were macroscopically analysed with a magnifying glass and a light for signs of DEH, while a dental probe was run across the tooth surface to detect surface unevenness, such as horizontal grooves or pitting (Fig. 3.13A&B). Although the scoring system of Lukacs (1989) was used as guide, any sign of growth-arrest lines or pits in the crown enamel of a tooth as a

result of defects in ameloblastic activity was positively scored. No attempt was made to record the position of the lesion on the surface of the crown, and the number of lines per tooth were not counted. According to Goodman et al. (1988), the two maxillary incisors and the two mandibular canines are to be used for scoring the presence of DEH on the teeth. In this study, all teeth were analysed for signs of DEH, as many individuals did not have incisors to analyse. Therefore, the true number of individuals with DEH on specific teeth may be inaccurate.



Source:

<http://forums.canadiancontent.net/history/137396-tragedy-civil-wars-child-soldiers.html>



Source:

https://sites.google.com/site/stressedoutconference/_/rsrc/1475850351826/config/

Figures 3.13A -B. Examples of A) linear horizontal grooves of the tooth enamel; and B) pits and lines in the tooth crown of archaeological specimens.

3.3.3.1.3 Osteomalacia

Vitamin D-related deficiencies result in poor mineralization and high flexibility of bone (Vigorita, 1999), making bones weak and prone to chronic macro- or micro-fractures. Specific bone elements were macroscopically analysed for signs of persistent rickets, such as bowing (anterolateral bending) of the long bones, thoracic kyphosis, lumbar lordosis-scoliosis, pectus carinatum, and bossing of the frontal bone (Kumar et al., 2007; Aufderheide & Rodríguez-Martin, 2011; Elder & Bishop, 2014). Each of these signs were scored as present or absent. Osteomalacia was not confirmed histologically, as no destructive methods were used on the bones of the KSC. The presence of pectus excavatum was also noted, but not regarded as a sign of osteomalacia.

3.3.3.1.4 Scurvy

Vitamin C-related deficiencies reduce the formation of osteoid in bone, causing a general weakness in the body's connective tissue (Aufderheide & Rodríguez-Martin, 2011). As scurvy is primarily seen as haemorrhage, especially in areas where superficial bloodvessels, lying between muscles and bones, can proliferate due to an inflammatory reaction (Halcrow et al., 2014). In dry bone, the vascular proliferation can manifest as porous foramina or pitting in the cortical bone (Ortner et al., 1999, 2001; Ortner & Ericksen, 1997) and new bone proliferation is visible on the bones as woven bone (Mays, 2008) at these sites. Pitting with woven bone on specific cranial bones, including the ectocranium, the roof and lateral walls of the orbits, the infratemporal (posterior) surface of the maxilla, the internal surface of the zygomatic bone, and the external surfaces of the greater wings of the sphenoid bone, were noted (Ortner & Ericksen, 1997; Ortner et al., 1999, 2001; Brickley & Ives, 2006). Antemortem tooth loss and bleeding gums are common clinical symptoms of scurvy and display on dry bone as porotic lesions at the alveolar margins of the maxilla and mandible (Fain, 2005; Popovich et al., 2009). Pitting on the alveolar margins with reactive bone formation (Ortner et al., 2001) was scored as possible presence of scurvy. Postcranially, symmetrical layers of porous woven bone on the surfaces of the tibiae and fibulae, and periostitis of these long-bone diaphysis were scored as positive for signs of possible scurvy (Maat, 2004). Scurvy-like lesions were only scored as present if both cranial and post cranial elements were affected.

3.3.3.2 Haematological disorders

Iron deficiency anaemia was indicated by pitting observed macroscopically in the roof of the orbits (cribra orbitalia) (Fig. 3.14A) and on the outer table of the calvarium (porotic hyperostosis (PH)) (Fig. 3.14B). PH was only scored as present if thickening of the cranial vault was present (Stuart-MacAdam, 1985), and an expanded diploe was also considered. Storage erosion and processing of artefacts were taken in account when lesions were analysed, using a magnifying ring light. All grades of PH and cribra orbitalia (CO) indicated in the grading system of Buikstra and Ubelaker (1994), were scored as present in the individual. The orbits were graded separately for CO presence. The side involved was reported as well. Any sign of pitting with accompanying vault thickness on the frontal, parietal and/or occipital surfaces was scored positively for each individual.

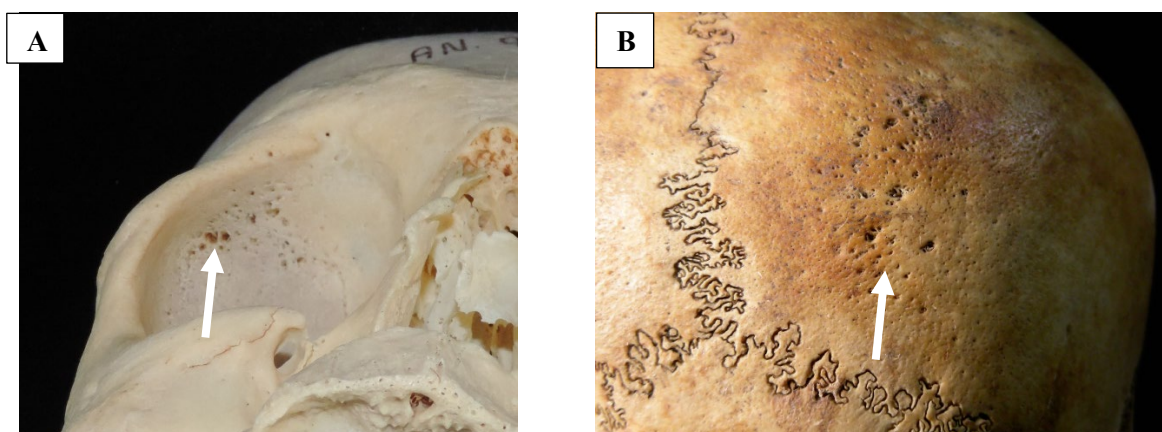


Photo by: A Alblas (AN 901). Inferior view of skull. Source: Wilczak & Jones, 2012. Superior view of skull. Figures 3.14A-B. Signs of anaemia seen as: A) cribra orbitalia B) porotic hyperostosis (arrows).

3.3.4 DEGENERATIVE JOINT DISEASE MARKINGS

Macroscopic signs of DJD on the skeletal elements were recorded for each individual, including side, section, and joint involved. Criteria scored included presence of osteoarthritic surface porosity, osteoporotic erosion, eburnation, and formation of osteophytes/sclerotic lipping at joints (Figures 3.15-21). The joints of the appendicular skeleton of each individual were scored separately from the vertebral column. Processing of artefacts and student handling of the bones, especially the vertebral column, were considered before scoring. Processing and handling were taken as highly eroded and brittle bones.

3.3.4.1 Degenerative peripheral joint osteoarthritic (pOA) markings

If signs of pOA, as described by Wilczak and Jones (2012), were seen on the appendicular skeleton, this condition was scored as present per individual. In this study, surface porosity was regarded as porous foramina or pitting with a smooth margin in the cortical bone. Erosion was seen as sharp-edged pitting in the trabeculae of the exposed spongy bone below the cortical bone. Eburnation was regarded present when a polished surface, resembling ivory, was visible under a light source, with or without porous lesions (Fig. 3.15A) or grooves (Fig. 3.15B), as described by Wilczak and Jones (2012). Specific sclerotic activity at joints was observed as lipping or osteophyte formation that was either round with a mushroom effect (Fig. 3.16A), spiculed with sharp ridges (Fig. 3.16B), or a combination of both (Fig. 3.16C).

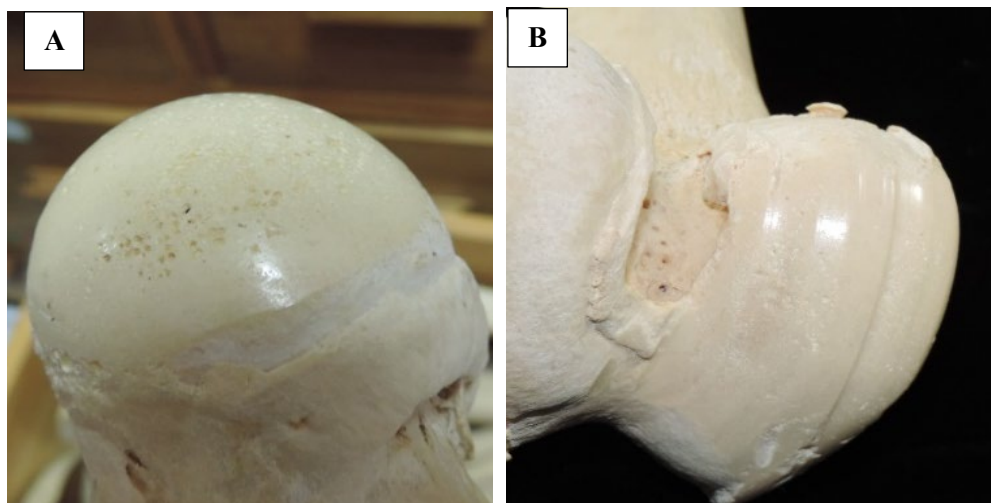


Photo by: A Alblas (AN 624), head of femur.

Photo by: A Alblas (AN 249), posterior view of distal femur.

Figures 3.15A-B. Eburnation as A) smooth polished lesions; and B) smooth polished lesions with grooves.

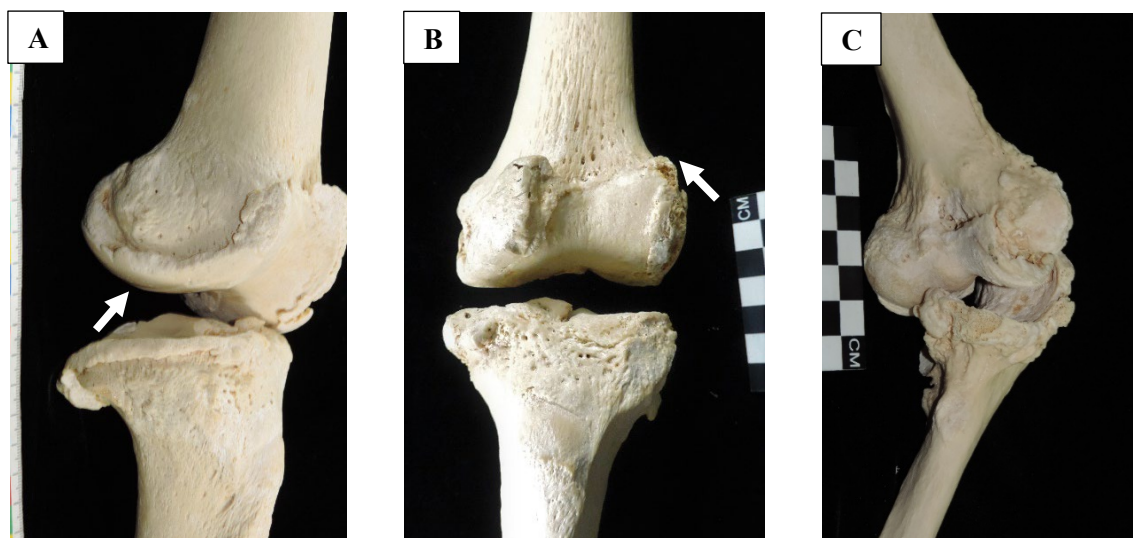


Photo by: A Alblas (AN 856), right-lateral view of knee.

Photo by: A Alblas (AN 414), anterior view of knee.

Photo by: A Alblas (AN 288), lateral view of elbow.

Figures 3.16A-C. Osteophytes on the peripheral joints: A) lipping with round ridges (mushrooming); B) lipping with sharp ridges (spiculed); and C) a combination of mushrooming and sharp lipping.

3.3.4.2 Vertebral column degenerative markings

The vertebral column was grouped into three regions, namely cervical, thoracic and lumbar, and an accumulative score for degenerative markings was allocated per region per individual. If one or more vertebrae were absent for each group, the rest of the vertebrae in the group were scored. If one or less vertebrae were present, the individual were indicated as 'no vertebrae present'.

Criteria for a positive score included the presence of peripheral osteophytes on the anterior vertebral bodies. These were scored as inferior and/or superior round or sharp lipping on the marginal surface of the vertebral endplates (Fig. 3.17A), or kissing osteophytes (or curved spicules) between two adjacent vertebrae, forming the characteristic “parrot beak” appearance (Fig. 3.17B).

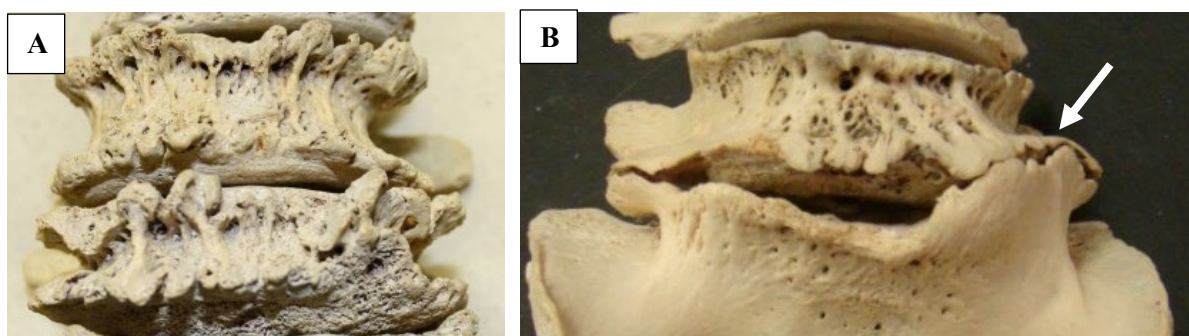


Photo by: A Alblas (AN 435), anterior view. Photo by: A Alblas (AN 604), anterior view.

Figures 3.17A-B. Markings on the anterior bodies of the vertebrae: A) Inferior and/or superior lipping on the marginal surface of the anterior body; B) kissing osteophytes (or curved spicules) between two adjacent vertebrae.

Vertebral osteoarthritis (vOA) (also called facet joint osteoarthritis) on the posterior arch of the vertebrae was scored separately for each group of vertebrae per individual. This included laminar spurs on the posterior laminar surface (indicative of ossification of the ligamentum flavum) (Brickley & Ives, 2008) (Fig. 3.18A), the zygapophyseal facet joints (Fig. 3.18B), the costovertebral and costotransverse joints (Fig. 3.18C). The presence of vertebral osteophytosis (VO) in the fibro-cartilaginous joint on the bodies of the vertebrae was also indicated (Fig. 3.18D).



Photo by: A du Plessis (AN 607), posterior view.

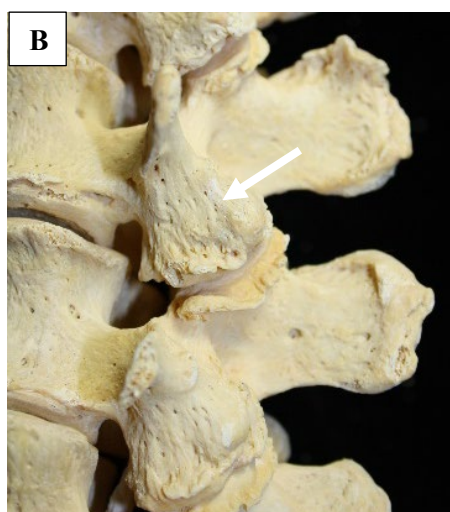


Photo by: A Alblas (AN 838), lateral view.

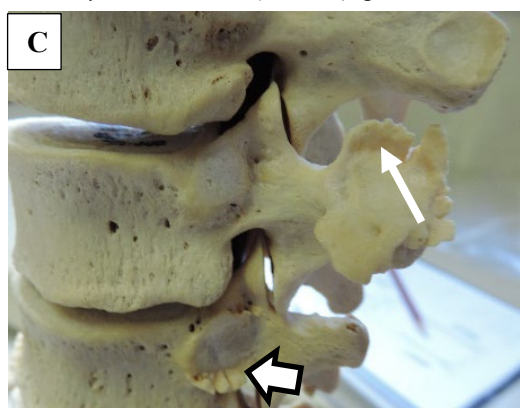


Photo by: A Alblas (AN 668), lateral view.

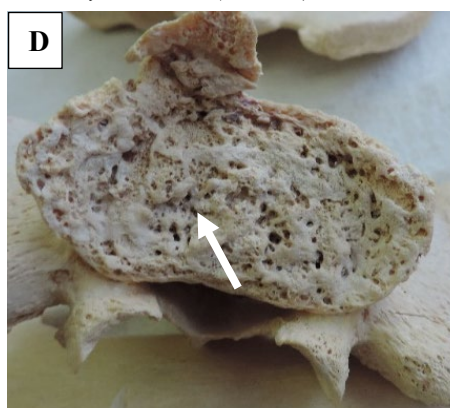
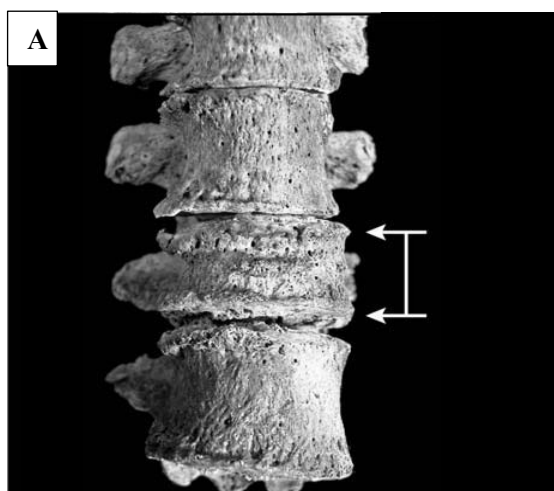


Photo by: A Alblas (AN 623), superior view.

Figures 3.18A-D. Markings on facet joints of the posterior arch (vOA) of the vertebrae: A) laminal spurs (arrow); B) spurs at the zygapophyseal articulation joints; C) spurs on the costotransverse joints (line arrow) and the costovertebral joint (block arrow); and D) VO on the fibro-cartilaginous joint on the vertebral body.

Vertebral compression fractures (VCFs) include a range of vertebral deformations of both the spongy and cortical bone (Fig. 3.19). The scoring system used for VCF included a crushed vertebral body where the anterior or posterior height, or in advanced cases, almost the entire height, was lost (known as plana fractures; Fig. 3.19A). Single end-plate depression with anterior wedging occurs when only the anterior part of the body is collapsed (Fig. 3.19B), while posterior wedging occurs when only the posterior part of the body is collapsed (Fig. 3.19C). Lateral wedging occurs when either the left or right side of the anterior body is collapsed (Fig. 3.19D). The scoring method was adapted from the scoring system used in the Osteoware Manual (Wilczak & Jones, 2012). The type of vertebral collapse was used to determine kyphosis and/or scoliosis of the vertebral column. Each of these traits were scored separately per individual. Processing and student handling erosion were taken into account during scoring.



Source: Brickley, 2008, anterior view.

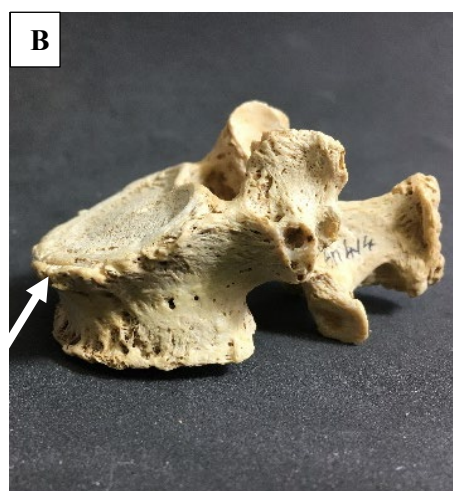


Photo by: A Naudé (AN 414) lateral view.



Photo by: A Naudé, lateral view.

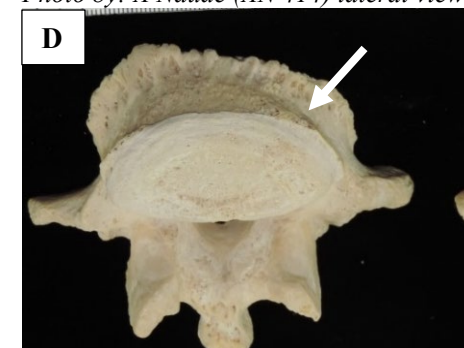


Photo by: A Alblas (AN 833) inferior view.

Figures 3.19A-D. Types of vertebral compression fractures: A) crushed vertebral body B) anterior wedging; C) posterior wedging; D) lateral wedging.

3.3.4.3 Specific DJD markings on vertebrae

In addition, specific degenerative anomalies manifesting as vertebral degeneration were scored as present or absent for each individual. Ankylosing spondylitis (AS) was regarded to be present in the individual, if smooth asymmetrical ossification of two or more adjacent vertebrae was present, forming a “bamboo”-like ossification (Fig. 3.20A), as described by Braun and Sieper (2007) with no sacro-iliac joint fusion. However, in severe cases SIJ and costovertebral joints fusion may be present (Olivieri et al., 2009). Anteriorly-fused vertebral bodies can be present and can result in kyphosis (Fig. 3.20B). Diffuse idiopathic skeletal hyperostosis (DISH) was scored according to criteria described by Resnick and colleagues (1978), when ossification of the anterior longitudinal spinal ligament was seen on the right side (Fig. 3.20C), extending across three or more vertebrae without loss of intervertebral space (Fig. 3.20D). Spondylolysis or stress fractures on the pars interarticularis of a vertebral arch (Fig. 3.20E&F) were regarded as present if the pars interarticularis showed a complete or partial fracture bilaterally or unilaterally on any of the vertebrae.



Photo by: A Alblas (AN 838), right-lateral view.

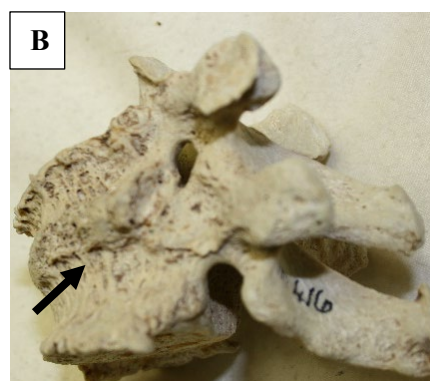
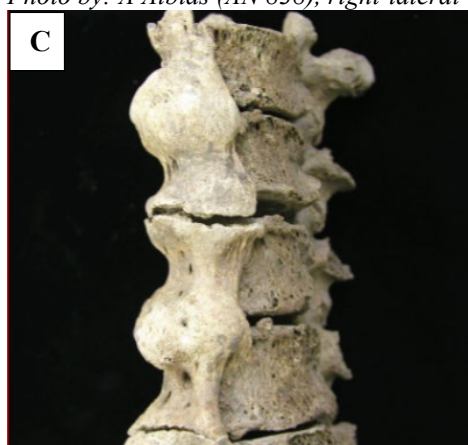


Photo by: A Alblas (AN 416,) left-lateral view.



Source: Ósz et al., 2009. Dry bone image (anterior view) and X-ray image (A-P view).

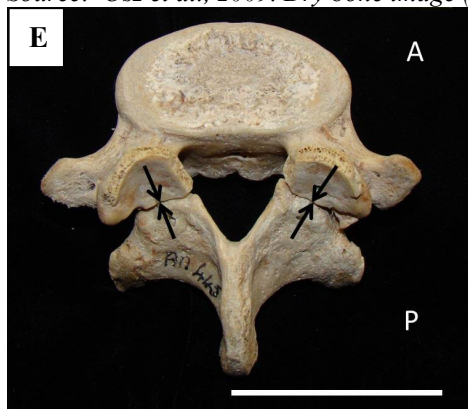
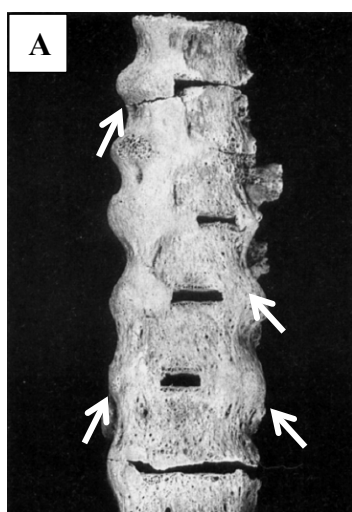


Photo by: J Walters (postero-superior view) and Lodox[®] image by: J Walters (AN 463) superior-inferior view. Figures 3.20A-F. Ankylosing spondylitis as: A) a 'bamboo spine'; or B) with anterior fusion causing kyphosis. Diffuse idiopathic skeletal hyperostosis (DISH) on C) dry bone; and D) X-ray image; and spondylolysis on E) dry bone; and F) X-ray image.

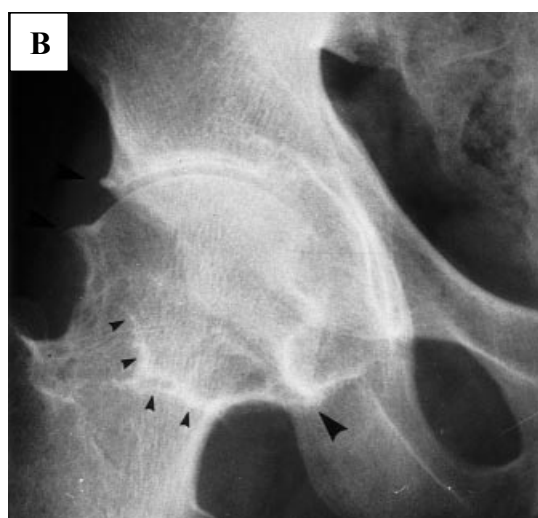
3.3.4.4 Specific degenerative arthropathies

Specific degenerative arthropathies were diagnosed using combinations of scoring criteria for peripheral and vertebral degenerative disease markings. Rheumatoid arthritis (RA) was scored as present if destructive erosion was seen bilaterally on the peripheral synovial joints, and fusion of hand (rather than feet) joints was observed. It was also taken into account that fusion of the vertebral column and sacroiliac fusion is generally absent in cases of RA. Reactive

Arthritis (ReA) was diagnosed by the presence of asymmetrical patches (“skip lesions”) of ligament ossification along the vertebral column (Fig. 3.21A), asymmetrical sacroiliitis, unilaterally degenerative changes in a large joint (e.g. knee), and fusion of feet (rather than hand) joints. On X-ray images, ReA were seen as a whisker sign (Fig. 3.21B, small arrow heads) and osteophytes in the acetabular rim (Fig. 3.21B, large arrow head). If two or more of these characteristics were present, a diagnosis of possible ReA was made (Ortner, 2003; Aufderheide & Rodriguez-Martin, 2011; Waldron, 2009).



Source: Rogers et al., 1987, anterior view.



Source: Kim et al., 1999, A-P view.

Figures 3.21A-B. Reactive arthritis (ReA) seen on: A) the thoracic vertebral column with “skip lesions” (arrows); and B) X-ray showing a whisker sign (small arrow heads) and osteophytes in the acetabular rim (large arrow head).

3.3.5 BONE REACTION AND NEOPLASMS

Living bone has a repetitive balance of resorption (bone destruction) and construction (bone addition) (Mann & Murphy, 1990; Ortner, 2003; Ragsdale et al., 2018). When stimulated, this balance is upset, and the bone will react in only one of three ways, namely, 1) destruction, 2) addition, or 3) both, to meet changes in the stress load (Ortner, 2003). The pattern of lesions and the diagnostic criteria used to identify dry bone lesions were recorded for each bone with lesions. In addition, the side, aspect, and section involved were recorded for each affected skeletal element.

3.3.5.1 Primary and secondary bone tumours

Age of the person, distribution of the lesions, and type of activity were taken into consideration when scoring bone tumours, as these characteristics help with the diagnosis of specific primary bone neoplasms and secondary neoplasms (Ragsdale et al., 2018). For

example, primary bone tumours are seen in actively growing young people, while secondary or metastatic lesions may be visible at any age. Also considered, was that primary bone lesions have specific medullary and/or cortical bone activity visible on radiological images, for example, medullary and/or cortical distribution with lytic and/or sclerotic activity (Ortner, 2003). Abnormal bone loss results from an increased osteoclastic activity and/or a reduced osteoblastic activity; therefore, the pattern of bone loss within the skeleton was identified to narrow down the range of possible causative factors. An osteolytic response was observed as porous, or pitted surface markings (Fig. 3.22A&B respectively) with smooth margins, as described by Ragsdale (1993). Erosion markings (Fig. 3.22C) on bone elements showing exposed trabeculae of spongy bone and sharp margins around cortical bone margins, were considered as processing or post mortem handling damage of fragile or osteoporotic skeletal elements.



Photo by: A Alblas (AN 400)



Photo by: A Alblas (AN 249)

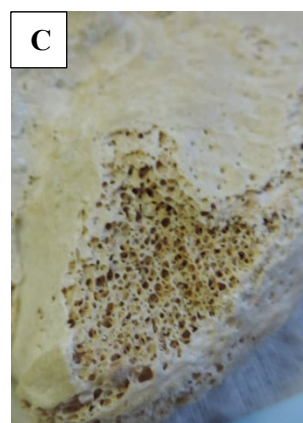


Photo by: A Alblas (AN 664)

Figures 3.22A-C. Difference between porosity and erosion were observed as: A) macroporosity or porous markings; B) microporosity or pitting; and C) erosion.

Lesions can present as a single lytic lesion without (Fig. 3.23A) or with (Fig. 3.23B) defined margins or as multiple foci (Fig. 3.23C,D). Lytic, “punched-out” foci with a poorly defined reactive margin was regarded as local aggressive lytic lesions. These lesions form at a high speed, resulting from an infiltrative process, and was seen as a malignant neoplasm such as multiple myeloma (Ragsdale et al., 2018).



Photo by: A Alblas (AN 716), superior view of skull.



Photo of the visceral view of rib.
Source: Nicklisch et al., 2012

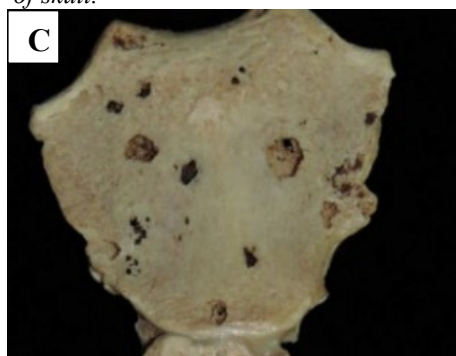
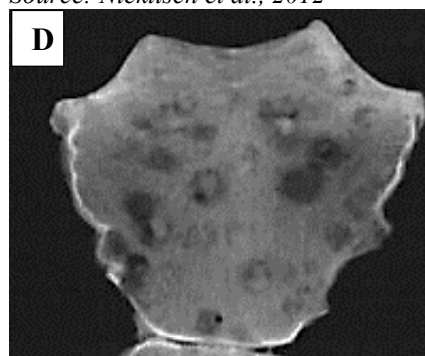


Photo by: A Alblas (AN 372), anterior view.

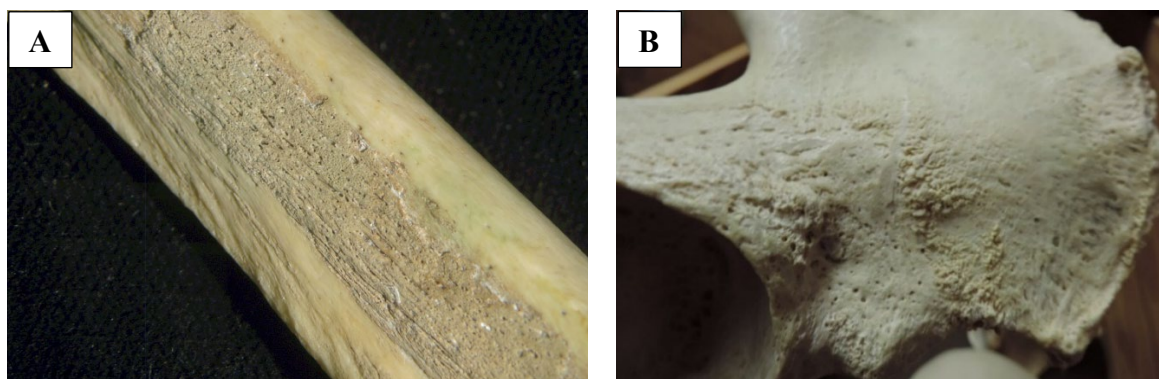


Lodox[®] by: JC Marais (AN 372), A-P view.

Figures 3.23A-D. A) Large lytic metastatic lesion on calvarium without defined margin; B) lytic lesion on rib with well-defined margins (arrow) and some sclerotic activity within lesion. C) Dry manubrium with multiple punched-out lytic lesions; D) also visible on the Lodox[®] scan.

An osteoblastic or sclerotic response was diagnosed when bone deposits were seen, either as disorganised woven bone (Fig. 3.24A) or well-organised sclerosis of mature bone (Fig. 3.24B). If both bone destruction (lytic) and bone deposits (sclerotic) were seen on the surface of a skeletal element, it was scored as such.

The radiological diagnostic trait for conventional osteosarcomas is a Codman's triangle on the long bones, while higher-grade osteosarcomas or Ewing's sarcoma are seen as an "onion-peel" or "sunburst" appearance (Ragsdale et al., 2018).

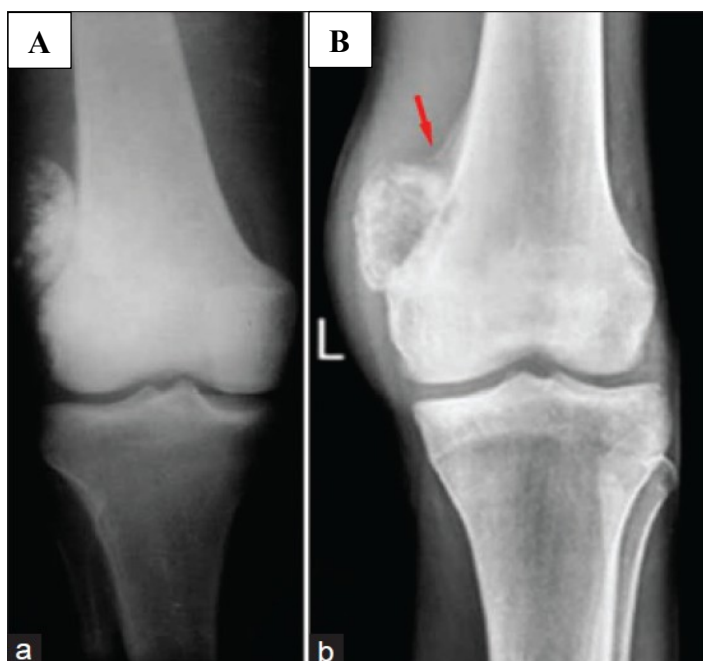


Source: Wilczak & Jones, 2012.

Photo by: A Alblas (AN 522).

Figures 3.24A-B. Sclerotic response as A) unorganised woven bone, or B) well-organised sclerosis of mature bone.

A surface osteosarcoma differs in radiological appearance from a low-grade parosteal and intermediate-grade periosteal osteosarcoma. The parosteal osteosarcoma was scored as present if an intact underlying cortex was observed (Fig. 3.25A), with the lesion arising from the surface of the bone, and medullary involvement in less than 25% of the medullary cavity (Kumar et al., 2007). On the other hand, the periosteal osteosarcoma was observed when lifting of the periosteum was present and the lesions were lobulated islands of malignant cartilage with little tendency to invade skeletal muscles (Fig. 3.25B).



Source: Kundu, 2014, A-P view.

Figures 3.25A-B. Surface osteosarcomas as: A) parosteal; and B) periosteal varieties visible on X-ray images of the distal femora.

3.3.6 OTHER BONE MARKINGS

Other bone markings observed were scored as present or absent.

3.3.6.1 Hyperostosis frontalis interna (HFI)

All the available dry calvaria and crania were examined for areas of localised thickening of the endocranial table of the frontal bone. Striated, irregular, or nodular activity were scored as present or absent, but the degree of hyperostosis was not quantified. Low, medium, and high involvement were all scored as present (Fig. 3.26A-C).

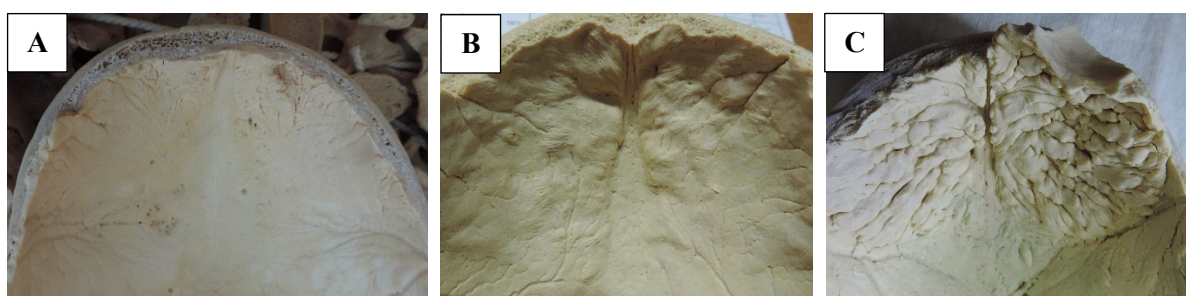


Photo by: A Alblas (AN 801).

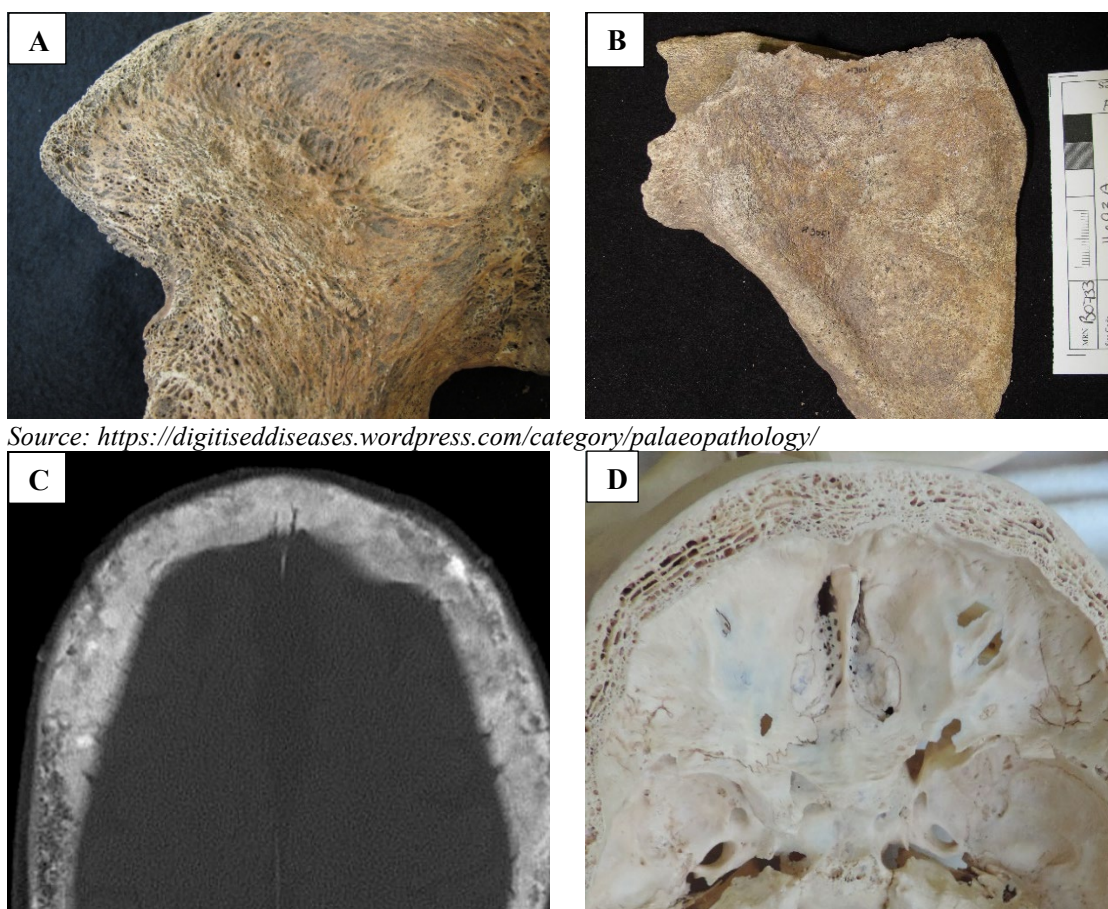
Photo by: A Alblas (AN 455).

Photo by: A Alblas (AN 899).

Figures 3.26A-C. Presence of HFI on endocranial surface of the frontal bones with A) low; B) medium; and C) high sclerotic activity.

3.3.6.2 Paget's disease

Skeletal signs evaluated include enlargement and bowing of the femora and tibiae, hyperostosis of the anterior vault, deposition of thickened disorganised porous new bone on one or more of the bones in the skeleton (for example, the os coxa and scapula, Fig 3.27A,B), and lytic lesions with irregular sharp margins. No invasive procedures, including histological methods, were used in the study, therefore Paget's disease could not be confirmed histologically. Radiological diagnosis of Paget's disease was made based on standardised criteria observed, including localised enlargement of bone (due to rapid bone formation), marked cortical thickening, increased bone density (seen as decreased radiolucency in radiograph) (Fig. 3.27C&D), enhancement of the trabecular pattern, sclerotic changes (disorganised newly formed bone), and osteolytic areas (Ortner, 2003). Special attention was given to the pelvis, sacrum, femoral heads and lumbar vertebrae. The pelvis was evaluated for thickening of the iliopectineal line ("brim sign") and protrusio acetabuli (Murray & Jacobson, 1977).



Source: <https://digitiseddiseases.wordpress.com/category/palaeopathology/>

Source: Bhargava & Maki, 2010, superior-inferior view.

Photo by: A Alblas, inner cranial view.

Figures 3.27A-D. Signs of Paget's disease on A) os coxa showing the exposed trabecular bone below damaged cortical bone B) scapula with layered woven bone deposited on cortical bone C) X-ray image of cross-sectioned vault; and D) dry bone showing layered expanded diploe.

3.3.6.3 Hypertrophic osteoarthropathy (HOA)

Hypertrophic osteoarthropathy is clinically characterised by a triad of digital clubbing, bilateral periostitis of long bones (particularly distal to the elbow and knee joints), and arthritis with accompanying arthralgia (Ali et al., 1980; Vigorita, 1999; Armstrong et al., 2007; Yao et al., 2009). In dry bone, clinical symptoms such as digital clubbing and arthralgia cannot be diagnosed. Therefore, HOA was scored present if symmetrical nodular periostitis of the diaphyseal long bones were present, together with OA on any of the peripheral joints, as described by Aufderheide and Rodríguez-Martin (2011) (Fig. 3.28A&B).

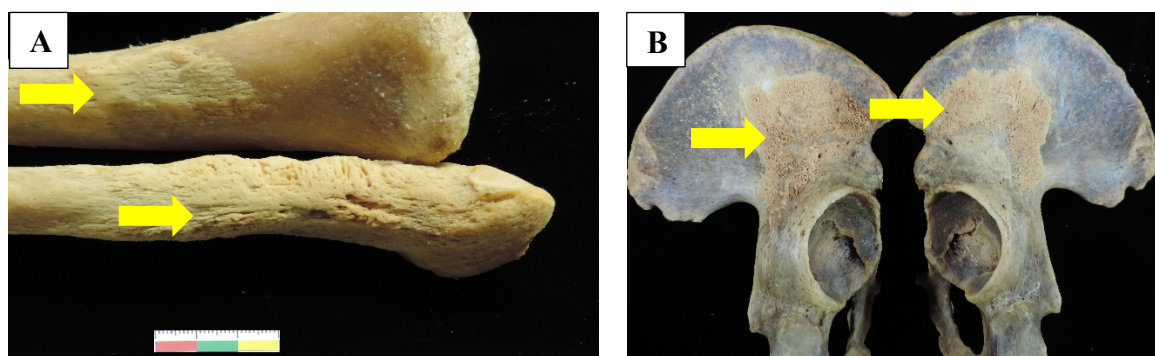


Photo by: A Alblas (AN 1197), anterior view.

Photo by: A Alblas (AN 1197), posterior-lateral view.

Figures 3.28A-B. Severe HOA showing sclerotic activity on A) distal tibia and fibula; and B) os coxae.

3.3.7 TRAUMA ASSESSMENT

Skeletal trauma was evaluated by the presence of osseous changes, such as callus formation, bone healing, fractures, and joint dislocations (Walker, 2001). None of the individuals in the cohort died of unnatural causes resulting in forensic autopsies. Due to processing methods and many years of handling by students, some of the skeletal elements, however, showed signs of post-mortem damage, therefore careful consideration was given to antemortem trauma versus post-mortem damage during trauma assessment. Post-mortem damage was considered if the trauma site did not show signs of remodelling and visible discolouration at the damaged site could be observed.

All skeletons (n=624) were analysed macroscopically with the aid of a ring light and magnifying glass for signs of antemortem fractures or remodelling. A total of 346 individuals were examined on full-body Lodox[®] Statscan[®] images to verify healed fractures.

In each individual, trauma to a specific bone was scored as present or absent. Evidence of healed or partially healed fractures was reported in a customised spreadsheet, including the side involved, the position of the trauma on the skeletal element (for example, in long bones, the proximal, middle, or distal third), and trauma complications. The results were compared between sexes, and among different race and age groups and different time periods during the 20th century by using Pearson's chi-square statistical tests.

3.3.7.1 Cranial scoring

The cranial elements scored included the frontal bone, both parietal bones, both temporal bones, both zygomatic bones, the occipital bone, both sides of the maxilla, both nasal bones, and the mandible. The side and aspect involved in each of the cranial bones were indicated.

For the crania, no antemortem trauma could be established on the ethmoidal perpendicular plate and vomer, due to their fragility. Therefore, these bones were excluded from the trauma analysis. Types of fractures were scored as pathological fractures, blunt force trauma, sharp force trauma, and other. Pathological fractures were regarded as present if the bone structure was weakened by a pathological process (Fig. 3.29A). The typical appearance of a blunt or depressed skull fracture is a concave defect in the outer vault, with or without radiating fractures (Fig. 3.29B), while a sharp or edged injury is usually caused by a penetrating force applied with a sharp tool, such as a knife, sword, or axe (Fig. 3.29C). All other traumas observed were recorded as other/unknown (Fig. 3.29D). These included projectile entry, embedded objects, surgical interventions, and traumatic deformities.



Photo by: A Alblas (AN 788), anterior view.



Photo by: A Alblas (AN 955), posterior-lateral view.



Photo by: A Alblas (AN 622), posterior-lateral view.



Photo by: A Alblas (An 1279), lateral view.

Figures 3.29A-D. Types of fractures on the skull as A) pathological fracture; B) blunt force trauma (BFT); C) healed sharp force trauma (SFT); and D) other (surgical intervention).

Trauma complications on the skull were categorised as open, non-union, or missing/damaged bone (where the fracture did not heal and the two bones remained separate or where a part is missing; Fig. 3.30A). Infection was scored as present if a periosteal response was seen on the bone (Fig. 3.30B) and deformation was scored positively if a fracture did not align properly after healing (Fig. 3.30C). Surgical procedures observed in cranial elements were scored as a trauma complication.



Photo by: L Greyling, lateral view of calvarium.



Photo by: A Alblas, superior view of skull.

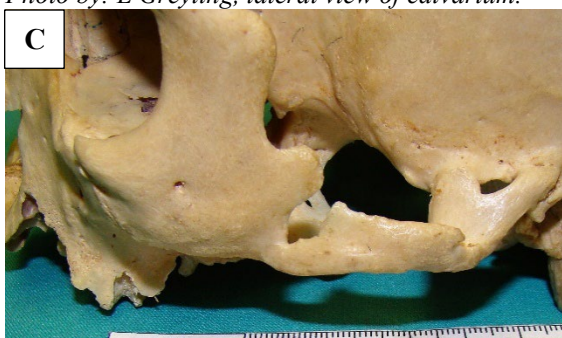


Photo by L de Beer (AN 597), left lateral view of skull.

Figures 3.30A-C. Trauma complications on the skulls was regarded as A) an open fracture with a missing part; B) infection with a periosteal response; and C) deformation of the healed fracture.

3.3.7.2 Nasal trauma scoring

A cautious approach of under-estimation of fractures of the nasal bones was followed, as post-mortem damage can easily influence diagnosis of antemortem fractures (Fig. 3.31A-D). Due to the difficulty in distinguishing antemortem trauma from post-mortem trauma on the nasal bones, each individual was scored using a magnifying glass. Nasal trauma was scored as antemortem when remodelled smooth edges were visible on the inferior ends of the nasal bones, and obvious remodelling has taken place. The adjacent frontal process of the maxillary bone was considered in the scoring of healed trauma. If the nasal bones were rough, sharp, or discoloured on their inferior edges, it was regarded as post-mortem damage.



Photo by: A Alblas (AN 948), anterior view.



Photo by: A Alblas (AN 516), anterior view.



Photo by: A Alblas (AN 510), anterior view.



Photo by: A Alblas (AN 1202), anterior view.

Figures 3.31A-D. Nasal bones with: A) no trauma; B) antemortem fracture with remodelling; C) post mortem damage; and D) antemortem fracture with further post mortem damaged.

3.3.7.3 Postcranial trauma scoring

Long bones (humeri, ulnae, radii, femora, tibiae, fibulae, clavicaulae), scapulae, ribs, and ossa coxae were scored for signs of antemortem trauma lesions. Each bone of each individual was documented as absent, present but normal, or present with antemortem trauma. The type of fracture, namely blunt force trauma (Fig. 3.32A), sharp force trauma (Fig. 3.32B), or pathological fracture (Fig. 3.32C), was indicated for each trauma lesion observed. The side (left, right or both), and section (proximal third, middle third, or distal third) affected in each of the long bones were indicated.

Due to dissection methods previously used, the ribs present in the KSC were often fragmented, with only the costovertebral parts available for examination. The number of ribs with trauma lesions was expressed as a percentage of the total number of ribs present per individual to calculate rib involvement. For example, if only seven ribs or rib fragments were present and three of them had evidence of callus formation, evidence of trauma was seen in 12.5% of 24 ribs, but in 42.9% of the ribs present. Seven individuals in the cohort had only one

rib present, while five individuals had two ribs present these individuals were excluded from the rib analysis, as the number of ribs would not allow accurate representation of the individuals' pathology and trauma. Hands and feet were excluded for trauma analyses, and each vertebra was only assessed for stress fractures and spondylolysis (Steyn et al., 2010).

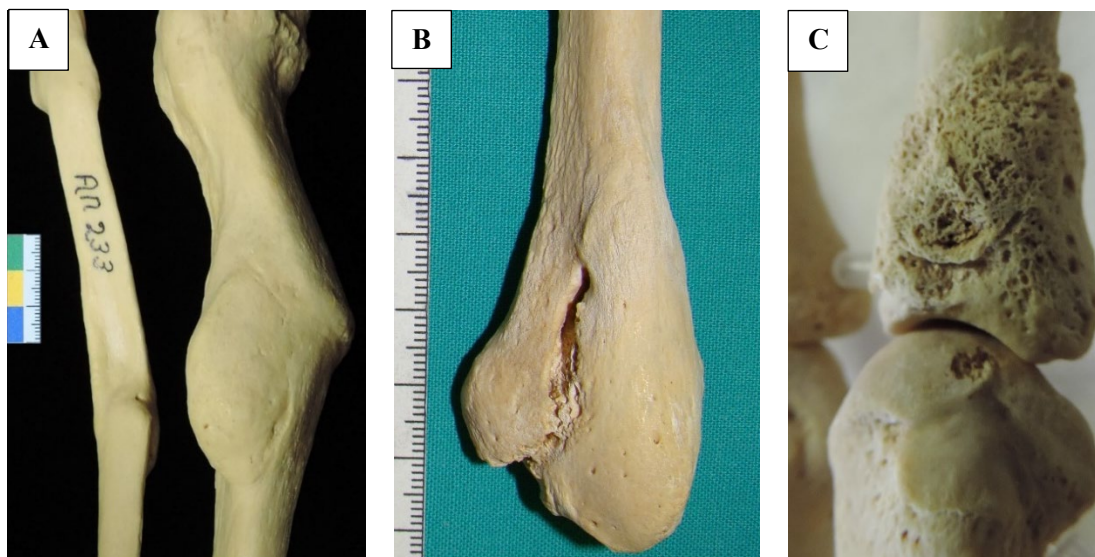


Photo by: A Alblas, midshaft of right tibia and fibula.

Photo by: N Coetzee (AN 697), distal end of right fibula.

Photo: A Alblas, DIP joint of phalanges.

Figures 3.32A-C. Type of fractures in post-cranial elements: A) blunt force trauma (BFT) B) healed sharp force trauma (SFT); and C) pathological fracture on weakened bone.

Pathological fractures were scored positively if the bone structure was weakened by a pathological process. Traits scored as trauma complication included: 1) fusion of two bone elements after excessive remodelling of a fracture (Fig. 3.33A); 2) periostitic response after trauma, where distinct signs of infection was visible at the trauma site (Fig. 3.33B); 3) non-union, or missing/damaged bone, where the fracture failed to heal, including formation of pseudoarthrosis (Fig. 3.33C); d) dislocation, traumatic arthritis or myositis ossificans at joints where a fracture was remodelled (Fig. 3.33D); e) a deformed bone element where the bone was not aligned properly after the fracture (Fig. 3.33E); and f) surgical intervention at a fracture site, such as orthopaedic wires, screws, plates or implants (Fig. 3.33F). Although surgical procedures observed in postcranial skeletal elements were scored as a trauma complication, it was not always possible to determine if the prostheses resulted from severe joint degeneration (for example a hip replacement) or a traumatic event.

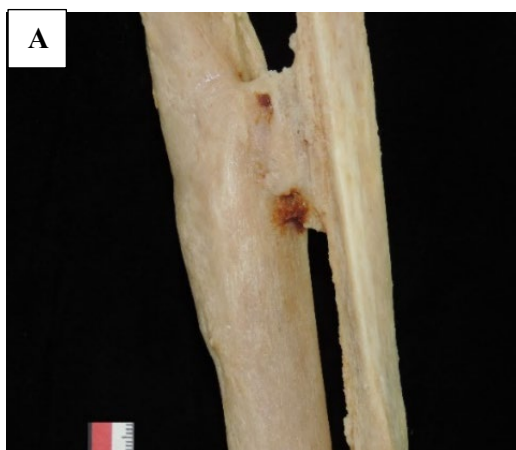


Photo by: A Alblas (AN 1406).

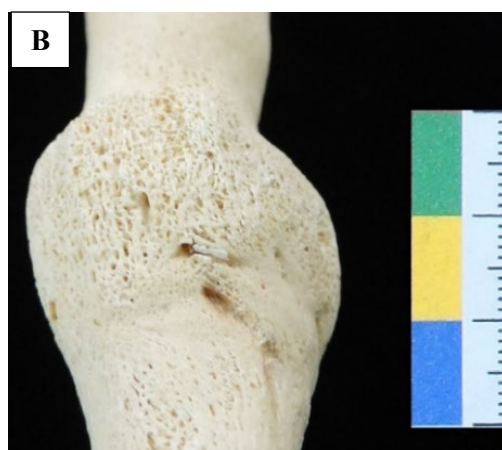


Photo by: A Alblas (AN 815).



Source: Wilczak & Jones, 2012.

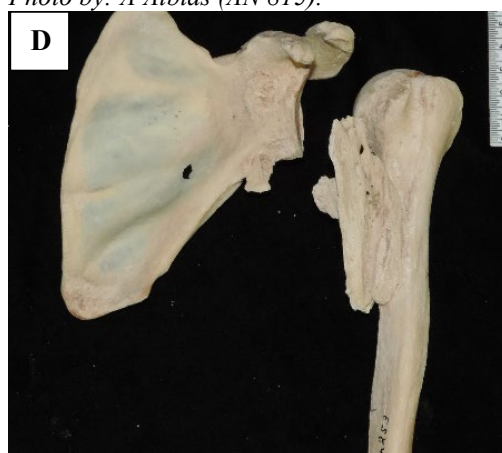


Photo by: A Alblas (AN 253).



Photo (anterior view) by: A Alblas; Lodox® (A-P view) by: J Walters (AN 791).



Photo (anterior view) by: A Alblas; Lodox® (A-P view) by: J Walters (AN 785).

Figures 3.33A-F. Trauma complication A) fusion of two bony elements after remodelling; B) periosteal response after trauma; C) non-union of two bone elements, D) dislocation, traumatic arthritis or myositis ossificans; E) deformation or misalignment of bone; and F) surgical intervention at a fracture site.

3.4 OBSERVER ERROR AND STATISTICAL ANALYSIS

Although not statistically confirmed, radiological and dental interpretation made by the candidate were confirmed by qualified researchers in the relevant fields of research. Intra-observer error were evaluated by the candidate over several years after various short courses, workshops, discussions with experts and conference attendance. Radiological confirmation of lesions on the Lodox[®] Statscan[®] images was done by Dr Carl Holdt from the Division of Radiodiagnosis (Department of Medical Imaging and Clinical Oncology, Faculty of Medicine and Health Sciences, Stellenbosch University). Interpretation of trauma on bones was mentored by Prof Steven Symes, currently at the Mississippi State Medical Examiner's Office, Jackson Mississippi, and Prof Ericka L'Abbé, Department of Anatomy, University of Pretoria. Dr André Uys from the School of Dentistry, Department of Oral Pathology and Oral Biology at the University of Pretoria, helped with identification of dental diseases in the cohort via photographs.

Data were analysed using Statistica statistical software version 13.3 (Tibco[®] Software) and Microsoft Office Excel[®]. Descriptive statistics included correlational analyses between dependant and independent variables. Pearson's Chi-square frequency tests were used to determine if the observed cell frequencies of each pathology tested, differed significantly from the expected frequencies for sex, age, ancestry and, time period. Fisher's exact test was used when sample sizes were too small ($n < 5$) for accurate analysis by the Chi-square test. For all analyses, the probability of 0.05 or less ($p \leq 0.05$) was considered to be significant, although 10% and 1% levels were also considered as the sample sizes of skeletal elements present varied and the prevalence of some of the variables were very low. The null hypothesis was formulated as "no difference", for example, no difference between trauma values for males and females. Correspondence analyses were plotted on contingency tables to summarise the association of qualitative variables in coordinates. Prof Martin Kidd, Director of the Centre for Statistical Consultation at Stellenbosch University, completed statistical analyses on the data generated, while Dr Marie Dussault aided with the interpretation of some of the trauma data.

CHAPTER 4: RESULTS

4.1 PATHOLOGICAL LESIONS OBSERVED ON BONE

4.1.1 CONGENITAL/GENETIC ORIGIN

4.1.1.1 Klippel-Feil syndrome (KFS)

The vertebral column was complete, or near complete, in 511/624 individuals (81.9% of the cohort). Ankylosed cervical vertebrae were found in 19 individuals, of whom 2.5% (Table 4.1) were confirmed to have KFS, a congenital condition associated with various other concurring anomalies (Table 7.1, Appendix 1). The other 1.2% (n=6) of cases with ankylosed cervical vertebrae were ascribed to trauma or pathology of the cervical vertebrae (Table 7.2, Appendix 1). The mean age of study subjects with KFS was 52.8 years (range: 35-66 years, SD:11.1).

Table 4.1. Statistical analyses of Klippel-Feil Syndrome in the cohort (n=13).

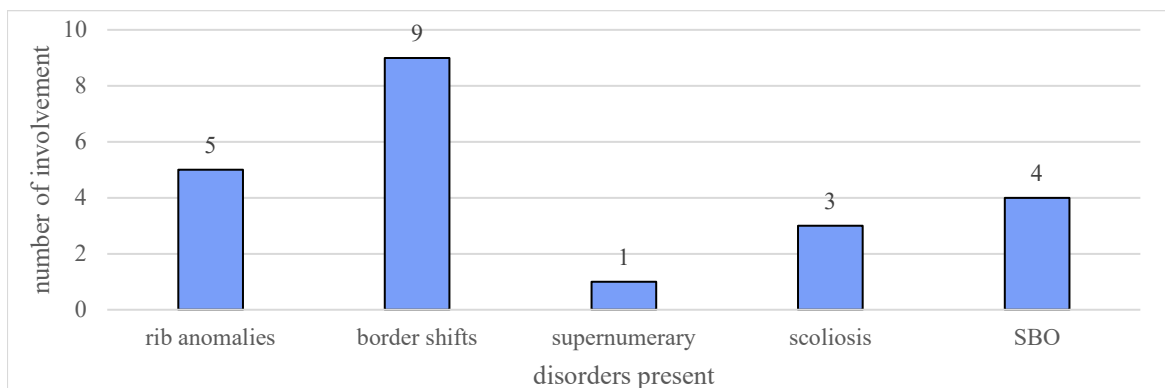
Trait	Age Group (%)			<i>P</i>	Population Group (%)			<i>P</i>	Time Period (%)			<i>P</i>	Sex (%)		<i>P</i>
	Y (n=1)	A (n=9)	O (n=3)		SAB (n=1)	SAC (n=4)	SAW (n=8)		EC (n=1)	MC (n=7)	LC (n=5)		F (n=4)	M (n=9)	
KFS	0.0	1.5	0.8	0.52	0.0	0.3	5.9	†<0.01	0.0	2.2	0.6	0.14	0.7	1.1	0.64

KFS=Klippel-Feil Syndrome

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

†p<0.01

The SAW population group showed a significantly higher prevalence of KFS than the SAC population group (5.9% vs. 0.3%; p<0.01), while one individual from the SAB population group was affected by the anomaly. Males had a slightly higher prevalence rate of KFS than females, while the mid-adult age group displayed a higher rate than the young- and old-adult age groups, respectively. However, the disparity in sample sizes of the different groups should be noted. A difference for KFS prevalence in the three time periods was observed, with all KFS cases belonging to the mid and late time periods, and no KFS representation in the early period. Anomalies (Fig. 4.1) associated with KFS in this study were comprised mainly of cranial-caudal border shifting (69.2%), followed by rib anomalies and fusion (38.0%; Fig. 4.2A,B), cleft neural arches or spina bifida of vertebrae (30.0%; Fig. 4.2C), supernumerary vertebrae, and scoliosis. The C2-C3 segment was the most commonly fused level in the cohort, occurring in six skeletons (46.1%; Fig. 4.3). Two individuals had more than one group of fused vertebrae: in one C2-C3 and C6-C7 were ankylosed, while the other showed fusion in C3-C4 and C5-C6. One individual had a group of 6 vertebrae (C2-C7) fused (Fig. 4.2D).



SBO=*spina bifida occulta*

Figure 4.1. Distribution and frequency of other disorders associated with Klippel-Feil syndrome in this study.



Photo by: A Alblas (AN 408), visceral view.



Photo by: A Alblas (AN 691), posterior view.

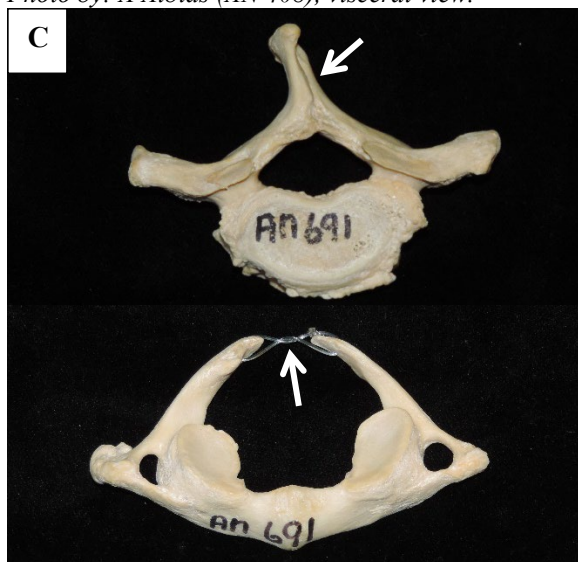
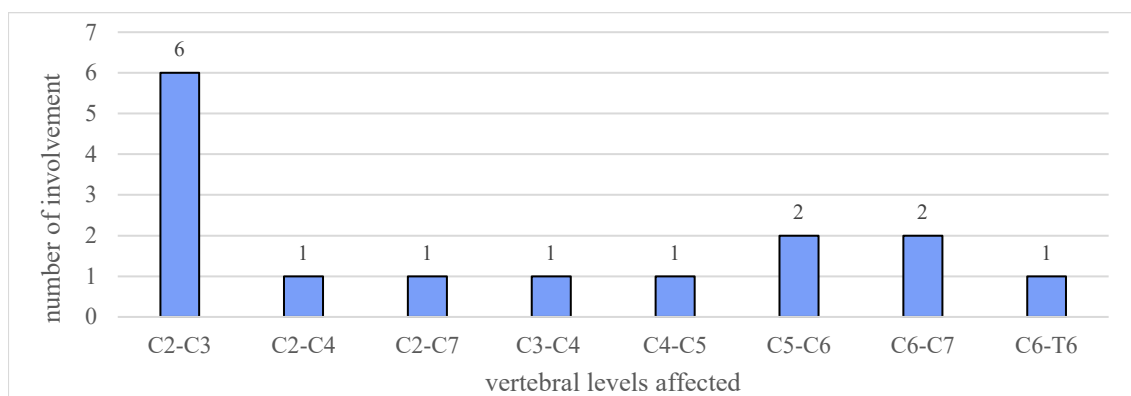


Photo by: A Alblas (AN 691), superior view.



Photo by: A Alblas (AN 691), posterior view.

Figures 4.2A-D. Anomalies associated with Klippel-Feil Syndrome A) misshapen and fused ribs; B) fused ribs 1 and 2; C) cleft neural arches of T3 (top) and C1 (bottom); and D) several (C2-C7) fused vertebrae



C= cervical vertebra

Figure 4.3. Distribution of Klippel-Feil syndrome per cervical level in the cohort. Note that some individuals have more than one group of fused vertebrae.

4.1.1.2 Cleft neural arches/spina bifida occulta

Further analysis of the vertebral columns revealed that 56/511 (11.0%) of cases presented with cleft neural arches of one or more spinous process (Table 7.3; Appendix 1). Only two cases presented with full spina bifida occulta (SBO) of all the sacral vertebrae (Fig. 4.4A), while another two cases presented with open spinous processes of all the sacral vertebrae, except S3 (Fig. 4.4B). One individual (not included in the total) presented with a cleft transverse spine (AN 307; Fig. 4.4C).



Photo by: A Alblas (AN 849), sacrum posterior view.



Photo by: A Alblas (AN 930), sacrum posterior view.

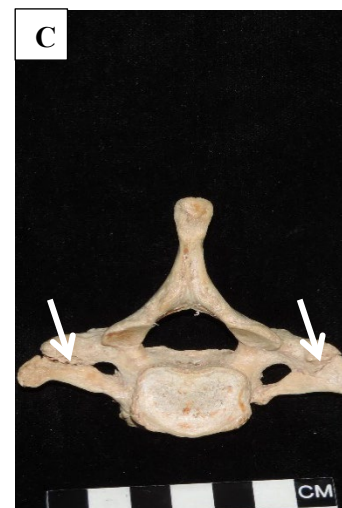


Photo by: A Alblas (AN 307), C7 vertebra inferior view.

Figures 4.4A-C. Examples of the cases that presented with A) full spina bifida occulta (SBO); B) most of the spinous processes of the sacral vertebrae open; and C) cleft transverse spines.

Table 4.2. Statistical analyses of cleft neural arches of vertebrae in the cohort (n=57).

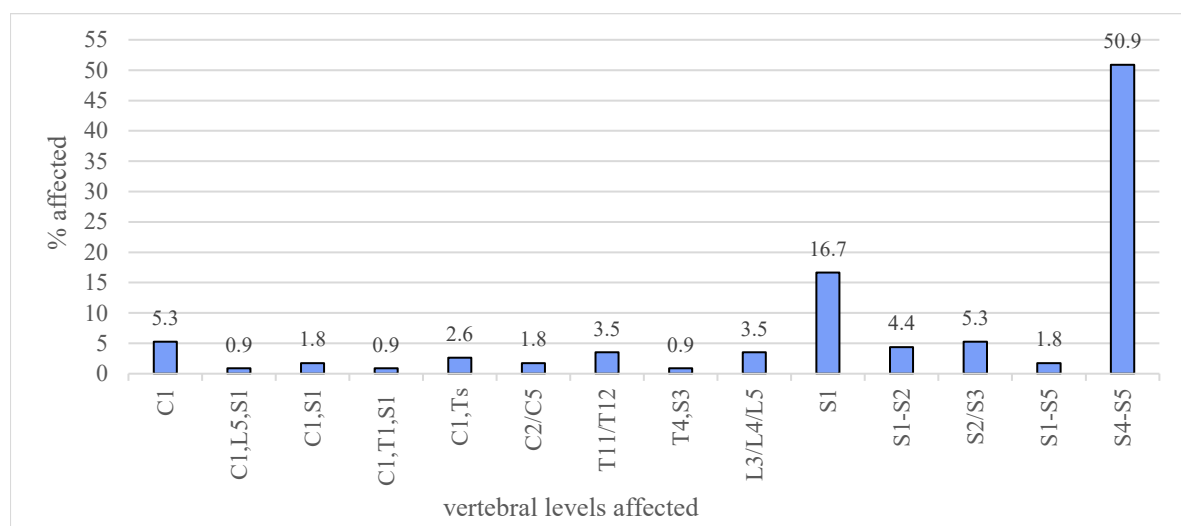
Vertebral Fusion Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)		
	Y (n=14)	A (n=30)	O (n=13)	<i>P</i>	SAB (n=9)	SAC (n=34)	SAW (n=14)	<i>P</i>	EC (n=1)	MC (n=27)	LC (n=29)	<i>P</i>	F (n=18)	M (n=39)	<i>P</i>
SBO	11.4	11.4	9.2	0.78	9.6	9.7	19.4	0.09	12.2	10.9	4.4	0.32	11.4	10.8	0.83

SBO=spina bifida occulta

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

^op<0.1

The mean age of individuals with cleft neural arches in their vertebral columns was 47 years (range: 20-81 years, SD:11.4). No statistically significant difference in the occurrence of this neural tube defect (NTD) was observed between sexes, among age groups or time periods (Table 4.2). However, the SAW population group showed a weakly significantly ($p=0.09$) higher prevalence of the disorder, followed by almost equal occurrence in the SAC and SAB population groups (Table 4.2). The first sacral vertebra revealed the highest number of cleft neural arches (16.7%) in this cohort, followed by C1 and a combination of S2 and S3 (5.3% each; Fig. 4.5). Although a high hiatus of the inferior canal was not scored as a cleft neural arch of the sacral vertebrae, 50.9% of individuals had open arches at the level of S4 and S5 (Fig. 4.5).



C= Cervical vertebra; T=Thoracic vertebra; L=Lumbar vertebra; S=Sacral vertebra

Figure 4.5. Distribution and frequency (%) of vertebrae with a cleft neural arch.

4.1.1.3 Lumbosacral transitional vertebrae (LSTV)

The prevalence of LSTV in the sample was determined from 506/624 cases (81.1% of cohort) for which the lumbar vertebrae and sacra were available for analysis. Of these, 16.3% (n=83) presented with LSTV (Table 7.4; Appendix 1). The age at death of individuals with LSTV ranged from 22 to 75 years (SD:12.9), with an average age at death of 48.8 years. No statistical significance was observed for any of the comparison groups, except for the early time period that showed a weak significance of LSTV prevalence compared to the mid and late eras ($p=0.06$; Table 4.3).

Table 4.3. Statistical analyses of lumbosacral transitional vertebrae (LSTV) in the cohort (n=83).

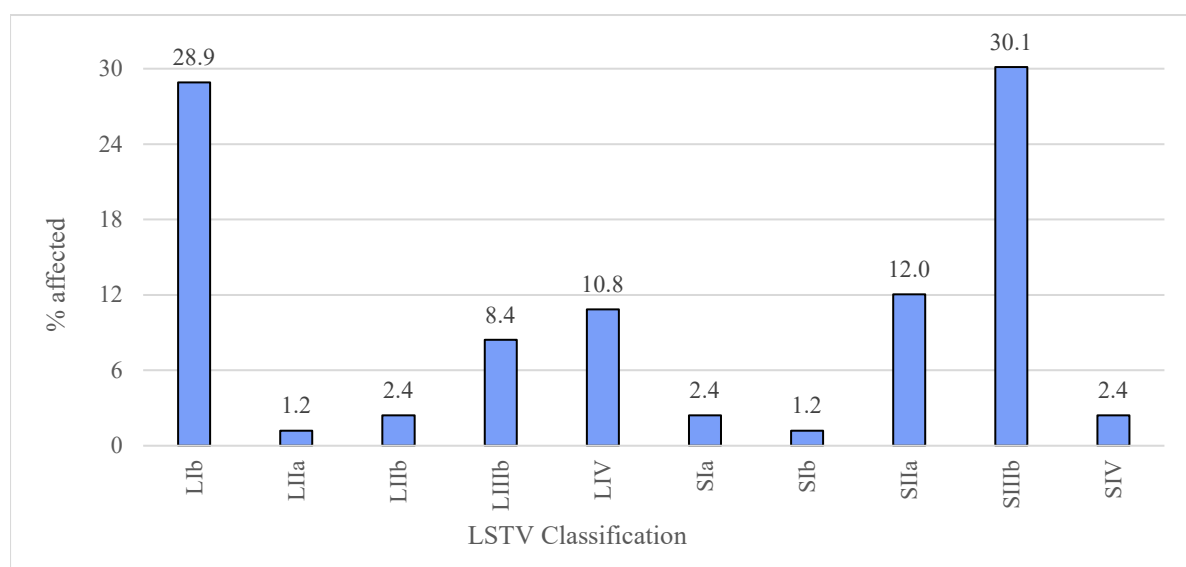
Trait	Age Group (%)				Population Group (%)				Time Period (%)			Sex (%)			
	Y (n=21)	A (n=44)	O (n=18)	P	SAB (n=16)	SAC (n=60)	SAW (n=7)	P	EC (n=13)	MC (n=40)	LC (n=30)	P	F (n=25)	M (n=58)	P
LSTV	17.1	17.1	14.4	0.78	19.5	16.5	11.7	0.44	27.1	17.5	13.1	0.06	16.6	16.3	0.95

LSTV=Lumbosacral transition vertebra

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

$^{\circ}p<0.1$

Each LSTV case was classified according to the criteria set out by Castellvi et al. (1984) (Fig. 4.6). Sacralisation IIIb, or complete sacralisation with fusion of the fifth lumbar vertebra to the first sacral vertebra, resulting in four lumbar and six sacral vertebrae, was the most prevalent (30.1%), followed by lumbarisation Ib (28.9%), where the first sacral vertebra has well-formed lumbar-type of facet joints when viewed posteriorly, although the number of segments remain normal (Fig. 4.7A,B).



L=Lumbarisation; S=Sacralisation; a=only one side involved; b= both sides affected

Figure 4.6. Frequency (%) of LSTV classes, using classification criteria of Castellvi et al. (1984).

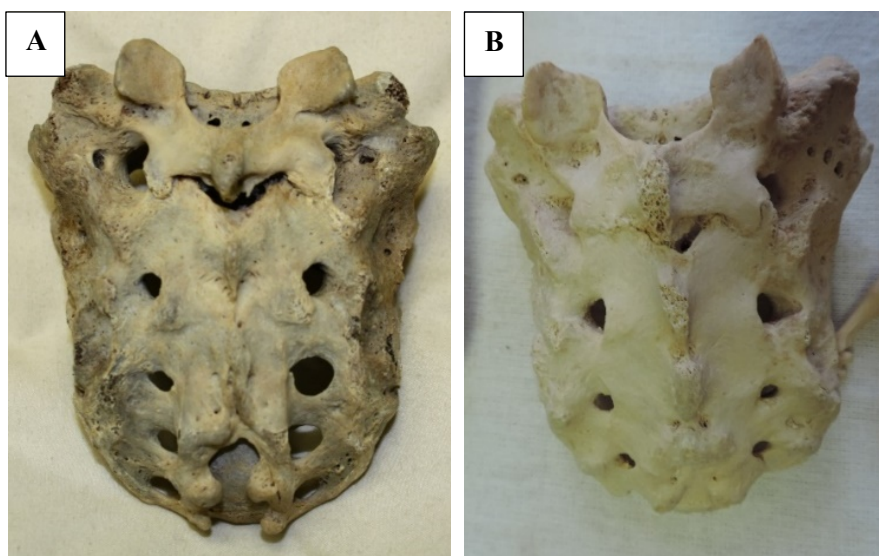


Photo by: A Alblas, posterior view. Photo by: A Alblas, posterior view.
 Figures 4.7A-B. Examples of A) sacralisation IIIb and B) lumbarisation Ib. Note the difference in the number of sacral segments.

4.1.2 INFECTIOUS DISEASES

The following tables (Table 4.4-4.8) give an overview of the frequency and statistical significance of all infectious conditions present in this KSC study. Specific and non-specific conditions were analysed and compared among age, population and time groups, as well as sexes.

4.1.2.1 Specific bone infections

Specific bone infections, in particular, signs of periosteal reactions on the visceral side of the ribs that may represent infectious diseases such as TB (Steyn et al, 2013; Geldenhuys, 2014) on bone elements, as well as disseminated TB infections, are presented in Table 4.4.

Table 4.4. Summary of the statistical analyses for possible tuberculous infections.

TB Infection Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total Number
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p	
Ribs lesions	(n=22) 18.9	(n=29) 9.8	(n=20) 8.1	$\ddagger < 0.01$	(n=9) 10.1	(n=55) 14.2	(n=7) 10.4	0.44	(n=5) 10.3	(n=20) 15.7	(n=46) 16.5	0.12	(n=25) 15.6	(n=46) 12.0	0.27	n=71
Potts/TB	(n=4) 1.8	(n=2) 0.0	(n=3) 1.4	0.89	(n=0) 0.0	(n=8) 1.8	(n=1) 1.2	0.50	(n=0) 0.6	(n=4) 1.9	(n=5) 2.7	0.14	(n=5) 2.7	(n=4) 0.9	0.11	n= 9

TB=tuberculosis

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

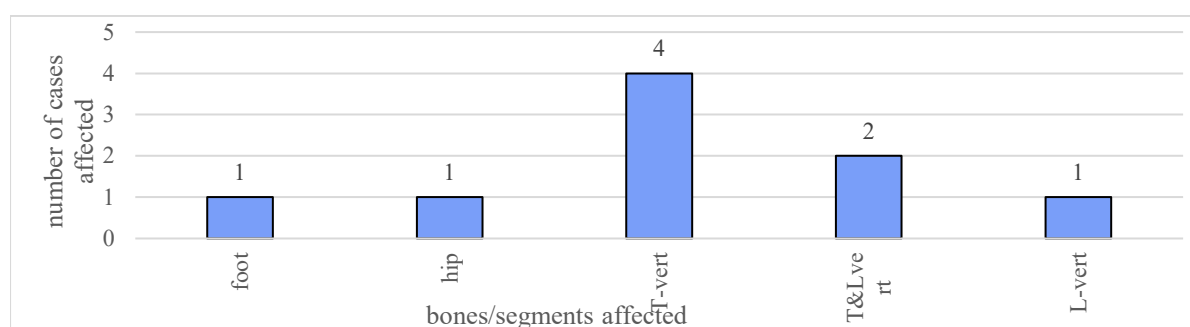
$\ddagger p < 0.01$

4.1.2.1.1 Visceral rib periosteal reaction

A total of 554/624 individuals (88.8% of the cohort) had more than one rib present. Periosteal lesions on the visceral side of the ribs were present in 13.1% of cases (Table 7.5; Appendix 1). The side and exact number of ribs with visceral periostitis on the ribs proved difficult to determine in this study as many ribs between rib 3 and 7 were cremated by previous curators of the KSC. It is possible that, in some cases, visceral rib lesions may have been present only on the missing ribs. Therefore, the figures calculated for this study may be an under-representation of the total number of ribs involved. The young-adult age group showed a significantly higher incidence rate of periosteal lesions on the visceral surface of their ribs compared to the mid-adult and the old-adult age groups, respectively (18.9% (young group) vs 9.8% (mid-adult group) and 8.1% (old adult group); $p < 0.01$; Table 4.4). However, none of the other comparison groups showed significant differences in terms of the presence of rib periosteal lesions.

4.1.2.1.2 Extra-pulmonary tuberculosis (ETB)

All bones of each individual were examined for signs of disseminated TB, including examination of the vertebrae for Pott's disease (TB manifestation in the vertebral column). Only 1.4% of individuals showed signs of extra-pulmonary TB (Table 7.6; Appendix 1). More females than males (2.7% vs. 0.9%) had extra-pulmonary TB lesions, but it is important to keep in mind the unequal sample, biased towards males. None of the comparison groups showed statistically significant differences in terms of presence of TB lesions (Table 4.4). Of the nine cases that presented with extra-pulmonary TB, two were noted outside the vertebral column and seven as Pott's disease. Where vertebral involvement was noted, it was classified as thoracic (T-vert), thoracic with lumbar (T and L vert), or lumbar (L vert) manifestations. The thoracic vertebrae were most affected by Pott's disease in this cohort (44.4% of manifestations; Fig. 4.8).



T=Thoracic; L=Lumbar; vert=vertebrae

Figure 4.8. Distribution of extra-pulmonary TB in the cohort.

4.1.2.1.3 Localised cranial infections

Only 495/624 (79.3%) of cases in the KSC sample had crania available for analysis. Of these, 39.4% (n=195) revealed a localised infection on the cranial bones, including concha bullosa, petrositis and otitis media (Table 7.7; Appendix 1). Petrositis occurred most commonly (n=139; 71.3%), followed by concha bullosa (n=61; 31.3%) and otitis media (n=55; 28.2%). Four individuals (2.0%) showed a combination of all three infections, while 18.0% showed a combination of at least two of the three infections. Due to diagnostic difficulty, data collected for mastoiditis was omitted. The ages involved ranged from 20-90 years (SD:14), including the youngest and the oldest individuals in the cohort. The mean age of affected individuals was 49 years. Females had a higher incidence rate of localised infections than males (34.6% vs. 30.0%). In terms of time periods, the late era had the highest prevalence of localised cranial infections (36.2%), followed by 28.1% in the mid-era and 24.2% in the early era. The SAW population group revealed significantly higher rates of petrositis and concha bullosa than the other two population groups; however, all three population groups were similarly affected by otitis media (Table. 4.5).

Table 4.5. Summary of the statistical analysis for possible cranial infections.

Infection Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)		Total Number	
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M		p
Otitis media	(n=11) 9.0	(n=31) 10.6	(n=13) 8.3	0.84	(n=8) 7.8	(n=39) 8.9	(n=8) 9.6	0.91	(n=7) 9.7	(n=23) 8.3	(n=25) 7.5	0.70	(n=14) 7.6	(n=41) 9.4	0.47	n= 55
Petrositis	(n=28) 28.6	(n=72) 40.0	(n=39) 25.6	0.22	(n=24) 29.6	(n=85) 24.8	(n=30) 42.3	†0.01	(n=14) 28.0	(n=55) 31.0	(n=70) 25.0	0.59	(n=45) 29.2	(n=94) 27.6	0.71	n=139
Concha bullosa	(n=18) 15.9	(n=28) 2.9	(n=15) 9.8	*0.03	(n=10) 12.3	(n=34) 9.9	(n=17) 23.9	†0.01	(n=1) 10.9	(n=21) 11.9	(n=39) 16.1	0.39	(n=23) 14.9	(n=38) 11.1	0.24	n= 61
Local infection	(n=46) 32.0	(n=97) 30.1	(n=52) 33.1	0.78	(n=31) 30.4	(n=121) 27.6	(n=43) 51.8	†0.01	(n=16) 24.2	(n=78) 28.1	(n=101) 36.2	‡0.05	(n=64) 34.6	(n=131) 29.9	0.25	n=195

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

*p<0.05; ; †p<0.01

Thirty individuals showed a deviated nasal septum, with a high incidence rate among old SAW females from the mid-period. A total of 32.8% individuals with unilateral concha bullosa showed an associated deviated nasal septum (p=0.0). However, 2.3% of individuals had a deviated septum not associated with concha bullosa. Of all the CODs indicated on death certificates as upper oral cavity, upper airway or oesophagus carcinomas (n=40; 6.4%), 30.0% showed bone markings on one of these areas.

4.1.2.1.4 Periodontal disease and oral health

Edentulous individuals, where both or either the maxilla and mandible were completely without teeth due to antemortem or post-mortem tooth loss, were excluded from dental analyses. In 552/624 individuals (88.5% of cohort), maxillae and/or mandibles were available for analysis of which 362 had both the maxilla and mandible to form the complete facial structure. The total maxillae and mandibulae with *in situ* teeth to analyse are seen in Fig. 4.9; however, many of the maxillae and mandibles that had teeth, also showed some tooth loss.

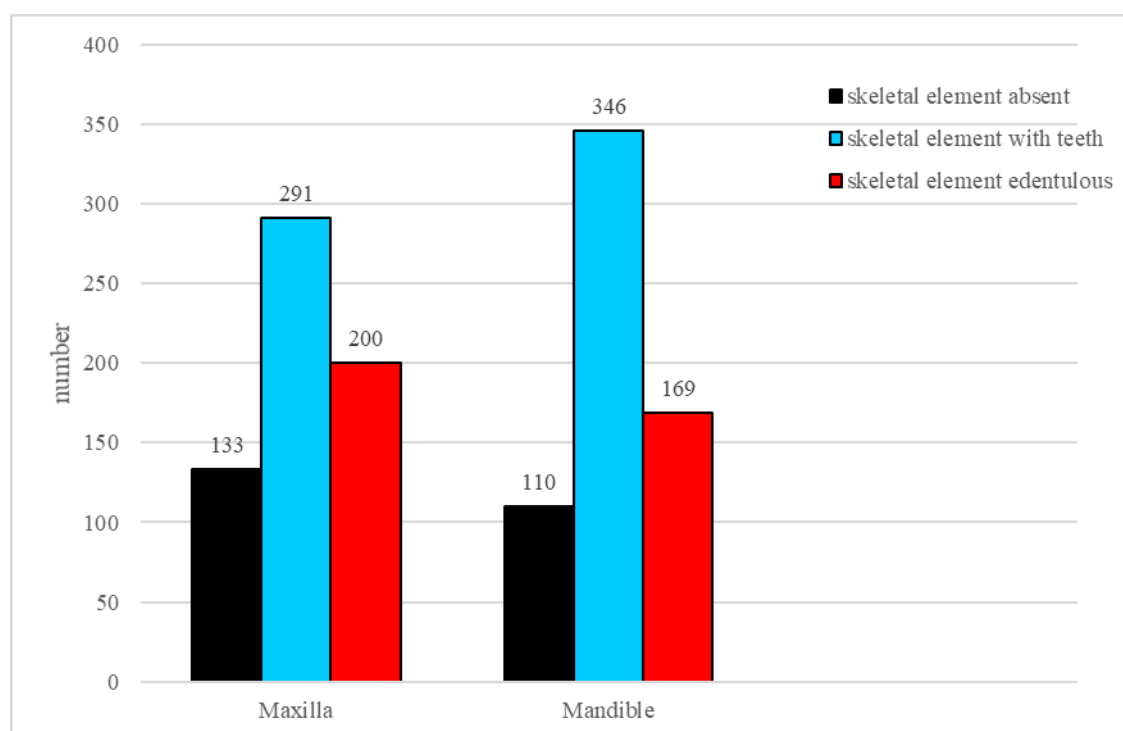


Figure 4.9. Total number of maxillae and mandibles in the KSC cohort present with and without teeth.

Four types of markings on the teeth were considered as indications of poor dental health: calculus and stains (indicating poor oral hygiene), and abscesses and caries (indicating dental diseases) (Table 4.6). The majority of individuals (60.8%, n=269) displayed affected teeth (Table 7.8; Appendix 1). The mean age of these individuals was 46 years (range: 20-81, SD:11.8). A total of 59.7% individuals had at least two of the four traits of poor dental health taken into account in this study. Calculus (35.5%) and staining (32.8%) were most common, followed by caries (23.1%) and periapical abscesses and/or cysts (17.0%). Statistically significant differences in poor dental health were noted among different population groups and time periods.

Table 4.6. Statistical analyses of signs of poor dental health in the KSC cohort.

Dental Health Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total number
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p	
Stains	(n=44) 20.8	(n=79) 35.3	(n=22) 39.3	†0.01	(n=27) 32.5	(n=113) 36.0	(n=5) 11.1	†<0.01	(n=7) 30.5	(n=23) 30.5	(n=25) 37.3	†0.01	(n=33) 25.3	(n=112) 36.1	*0.02	n=145
Calculus	(n=44) 39.3	(n=83) 37.1	(n=30) 28.3	0.18	(n=38) 45.8	(n=108) 34.4	(n=11) 24.4	*0.04	(n=7) 12.5	(n=23) 28.3	(n=25) 44.2	†<0.01	(n=36) 27.3	(n=121) 39.0	*0.02	n=157
Abscess	(n=25) 19.8	(n=42) 14.5	(n=28) 19.3	0.28	(n=14) 15.6	(n=74) 18.9	(n=7) 8.9	0.06	(n=7) 13.5	(n=23) 17.3	(n=25) 17.4	0.07	(n=30) 17.8	(n=65) 16.2	0.74	n= 95
Caries	(n=39) 34.5	(n=64) 28.6	(n=25) 23.6	0.20	(n=30) 36.1	(n=91) 28.9	(n=7) 15.6	*0.04	(n=7) 24.7	(n=23) 24.7	(n=25) 35.1	†<0.01	(n=40) 30.0	(n=88) 28.4	0.72	n=128
Overall health	(n=76) 59.8	(n=137) 47.4	(n=56) 38.6	†<0.01	(n=60) 66.7	(n=194) 49.5	(n=15) 19.0	†0.00	(n=13) 25.0	(n=101) 41.4	(n=155) 58.5	†<0.01	(n=74) 43.5	(n=195) 49.9	0.17	n=269

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

°p<0.1; *p<0.05; †p<0.01

Only 3.5% (n=16) of individuals with teeth displayed dental fillings or other dental repair work (Table 7.9; Appendix 1). A statistically significant difference in dental repair work was seen among population groups, with the SAW group having the highest prevalence (Table 4.7). No difference between sexes was observed. The late time period showed a slight increase in dental work done. The old-adult age group displayed the highest prevalence of dental work. The mean age at death of individuals with signs of dental work was 55.2 years (range: 28-76 years, SD:14.0).

Table 4.7. Statistical analyses of signs of dental work in the KSC cohort (n=16).

Dental work Trait	Age Group (%)				Population Group (%)				Time Period				Sex (%)		
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p
Dental fillings	(n=2) 1.8	(n=8) 3.5	(n=6) 5.5	0.31	(n=0) 0.0	(n=6) 1.9	(n=10) 20.0	†<0.01	(n=1) 3.1	(n=5) 2.7	(n=10) 4.2	0.72	(n=5) 3.8	(n=11) 3.4	0.87

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

†p<0.01

4.1.2.2 Non-specific bone infections

Signs of non-specific bone infections (such as a periosteal reaction or osteomyelitis on skeletal elements) in the cohort are presented in Table 4.7. Due to difficulty in correctly diagnosing osteitis without surrounding soft tissue and its resemblance to periosteal reaction on dry bone, osteitis was excluded from the analysis.

Table 4.8. Statistical analyses of non-specific reactions on long bones in the cohort.

Infections Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total Number
	Y	A	O	<i>p</i>	SAB	SAC	SAW	<i>p</i>	EC	MC	LC	<i>p</i>	F	M	<i>p</i>	
Periostitis	(n=36)	(n=104)	(n=45)		(n=31)	(n=140)	(n=14)		(n=16)	(n=89)	(n=80)		(n=61)	(n=124)		n=185
	23.8	32.9	28.5	0.12	30.4	31.9	16.9	*0.02	24.2	31.9	28.7	0.42	32.8	28.3	0.26	
Osteomyelitis	(n=2)	(n=8)	(n=3)		(n=1)	(n=12)	(n=0)		(n=1)	(n=8)	(n=4)		(n=3)	(n=10)		n= 13
	1.4	2.6	2.0	0.82	1.0	2.8	0.0	0.32	1.6	2.9	1.5	0.56	1.7	2.4	0.62	

Y=young adult; *A*=mid adult; *O*=old adult; *SAB*=South African Black; *SAC*=South African Coloured; *SAW*=South African White; *EC*=Early era; *MC*=Mid era; *LC*=Late era; *F*=Female; *M*=Male.

**p*<0.05

4.1.2.2.1 Periosteal reactions on long bones

A total of 185/624 individuals (29.6% of cohort) showed a periosteal reaction on the long bones (Table 7.10; Appendix 1). A statistical correlation was seen when comparing the presence of periosteal reactions among population groups (Table 4.7): the SAC and SAB population groups presented with a significantly higher incidence rate of periosteal reaction on their long bones compared to the SAW population group (*p*=0.02). Periosteal reactions were classified as: striated, nodular, irregular, or a combination of striated and nodular. The striated surface appearance occurred most frequently (Fig. 4.10). Of all the bones examined, the tibia-and-fibula combination showed the highest rate of a periosteal reaction (Fig. 4.11), with a much lower frequency on all other bones.

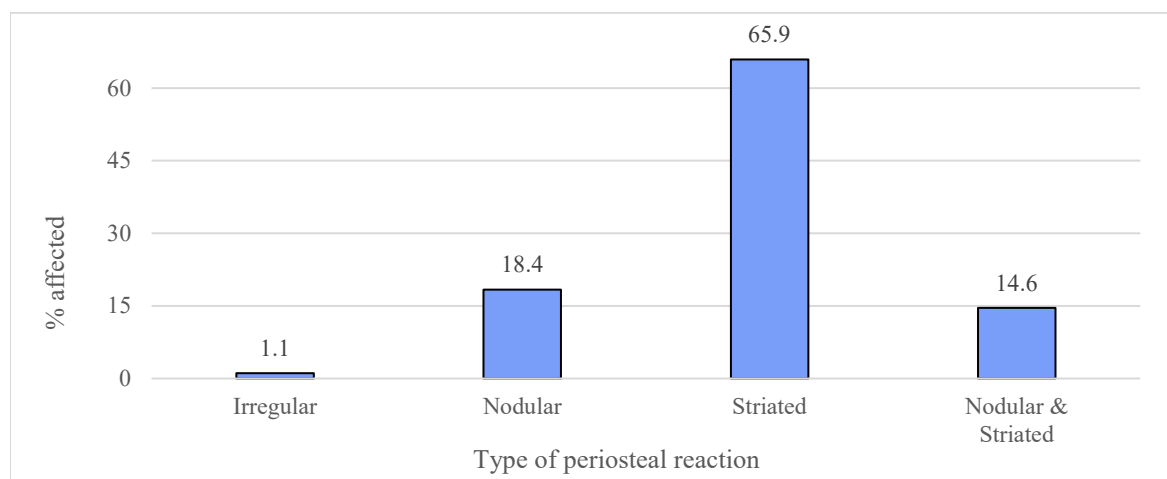


Figure 4.10. Type of periosteal reaction on long bones in the cohort (%).

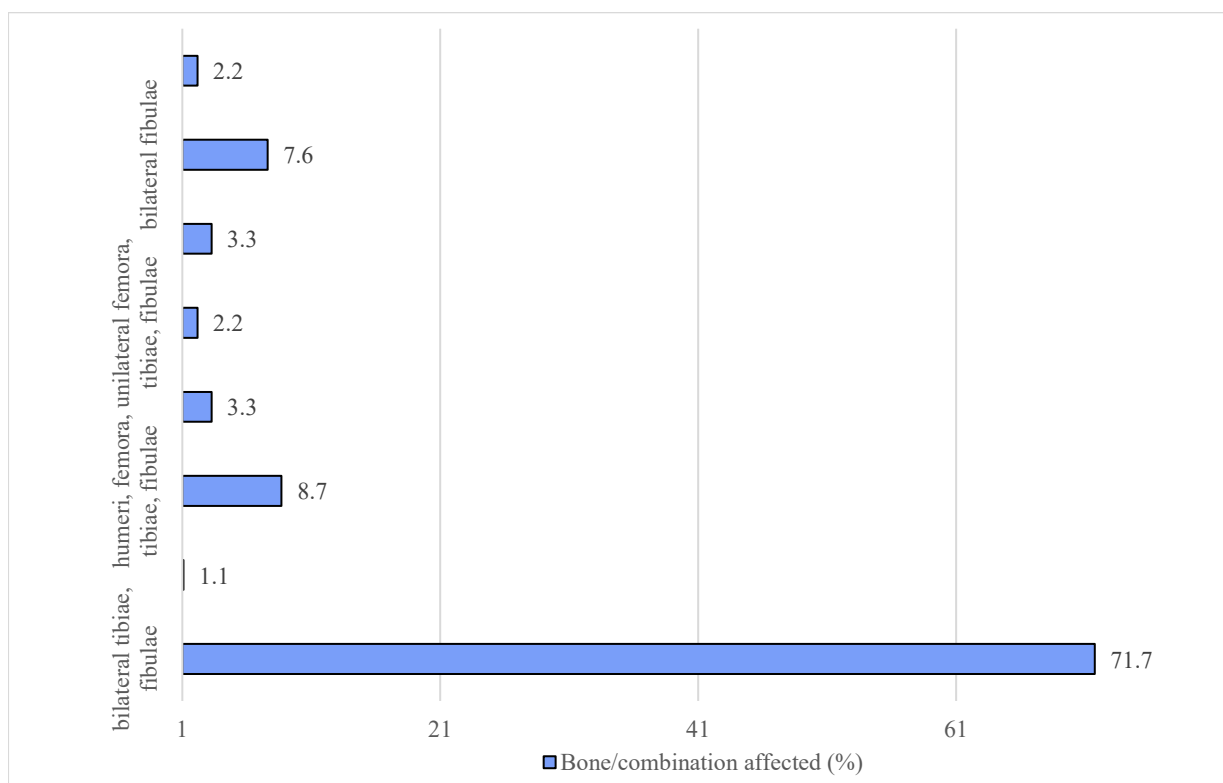


Figure 4.11. Percentage of bone elements or bone combinations with periosteal reaction in the cohort.

4.1.2.2.2 Osteomyelitis

Skeletal elements of 596/624 individuals (95.5% of cohort) were examined for osteomyelitis. The presence of at least two of the three diagnostic criteria for osteomyelitis diagnosis, namely involucrum, sequestrum, or a cloaca (Fig. 4.12A-B), was regarded as a positive indication of osteomyelitis. Only 2.2% (n=13) of individuals showed signs of osteomyelitis according to these criteria (Table 7.11; Appendix 1). No statistical difference was seen in any of the comparison groups. The sample size bias towards males, the mid-adult age group and SAC population group should be taken into account when interpreting the higher values seen in Table 4.8 for these groups. The mean age of affected individuals was 49.4 years (range: 29-67 years, SD:11.3). In this cohort, the tibia and radius displayed the highest incidence rates (30.8% each), followed by the fibula (23.1%). One 59-year-old SAC male showed osteomyelitis on the ischial ramus. Possible causes of pelvic osteomyelitis include pelvic trauma or a haematogenous infection. The most common source of pelvic infection is dissemination from a contiguous focus of infection from either chronic pressure ulcers (bedsores) or surgery (Bodavula et al., 2015; Schmitt, 2017). The individual concerned died of

an unspecified neoplasm with metastatic spread, which may have resulted in long-term bed confinement.

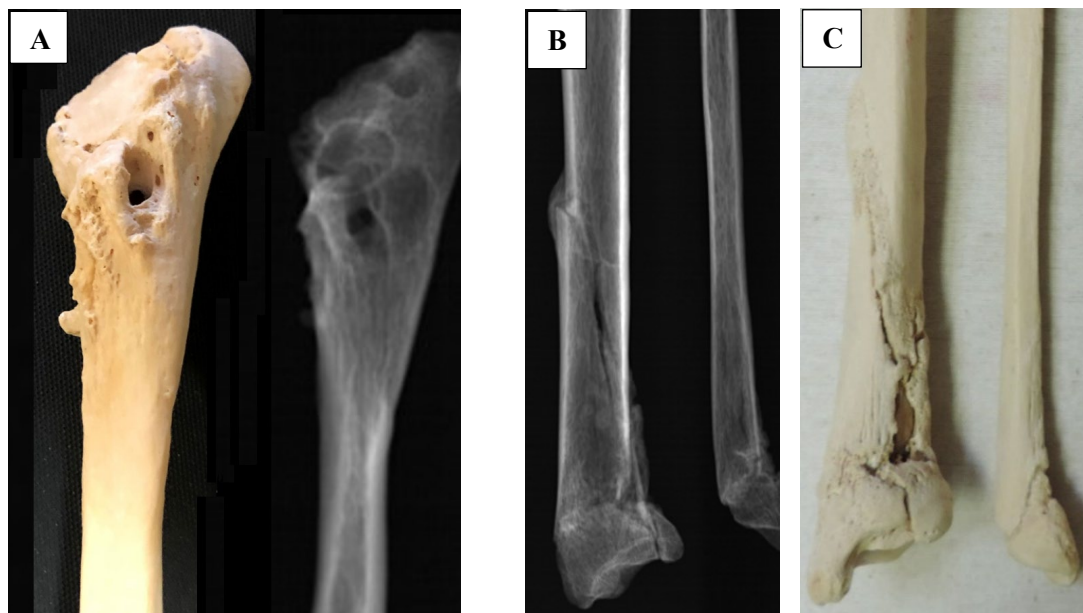


Photo: A Alblas, Lodox®: J Walters (AN 772) Lodox® (A-P view) J Walters, Photo: A Alblas (AN 718).
 Figures 4.12A-C. Osteomyelitis on A) proximal end of the right fibula and distal shaft of the left tibia, showing B) the Lodox® image and C) the dry bone.

4.1.3 DEFICIENCY DISEASES

Skeletal lesions displaying metabolic and nutritional diseases, as well as haematological disorders observed on the bones are shown in Table 4.9. Each of the traits are discussed in further detail under the appropriate headings.

Table 4.9. Statistical analyses of deficiency diseases in the cohort.

Deficiency Traits	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)		Total Number	
	Y	A	O	<i>p</i>	SAB	SAC	SAW	<i>p</i>	EC	MC	LC	<i>p</i>	F	M		<i>p</i>
HL	(n=43) 52.4	(n=189) 47.6	(n=78) 55.1	0.49	(n=30) 55.6	(n=127) 50.8	(n=19) 42.2	0.77	(n=24) 55.8	(n=80) 52.0	(n=72) 47.4	0.54	(n=56) 54.4	(n=120) 48.8	0.34	n=176
DEH	(n=19) 18.1	(n=14) 6.8	(n=5) 5.4	†<0.01	(n=8) 10.3	(n=26) 9.2	(n=4) 9.8	0.89	(n=1) 4.0	(n=8) 5.0	(n=29) 13.3	*0.02	(n=12) 10.0	(n=26) 9.2	0.80	n= 38
pectus carinatum	(n=1) 0.9	(n=0) 0.0	(n=2) 1.8	°0.07	(n=0) 0.0	(n=2) 0.6	(n=1) 1.9	0.36	(n=0) 0.0	(n=2) 1.0	(n=1) 0.5	0.71	(n=1) 0.7	(n=2) 0.6	0.85	n= 3
bowed limbs	(n=2) 1.5	(n=7) 2.4	(n=2) 1.4	0.78	(n=0) 0.0	(n=9) 2.3	(n=2) 2.8	0.26	(n=0) 0.0	(n=5) 1.9	(n=6) 2.4	0.67	(n=5) 3.0	(n=6) 1.5	0.26	n= 11

HL= Harris' lines; DEH=dental enamel hypoplasia

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured;
 SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

°p<0.1; *p<0.05; †p<0.01

4.1.3.1 Metabolic and nutritional stress disorders

4.1.3.1.1 Harris lines (HL)

All available digital Lodox[®] images 349/624 (55.9%) were examined for HL. All long bones on these images were scrutinised by zooming in on each bone. HL were present on the humerus, radius, ulna, femur, tibia and/or fibula of 176 (50.4%) of the individuals (Table 7.12; Appendix 1). No significant difference in the presence of HL was found in any of the comparison groups (Table 4.8). The average age at death of the individuals with HL in this study was 48.9 years (range: 22-82 years, SD: 13.4). The tibia was the bone most commonly affected by HL (65.6%), followed by the fibula (15.8%) (Fig. 4.13).

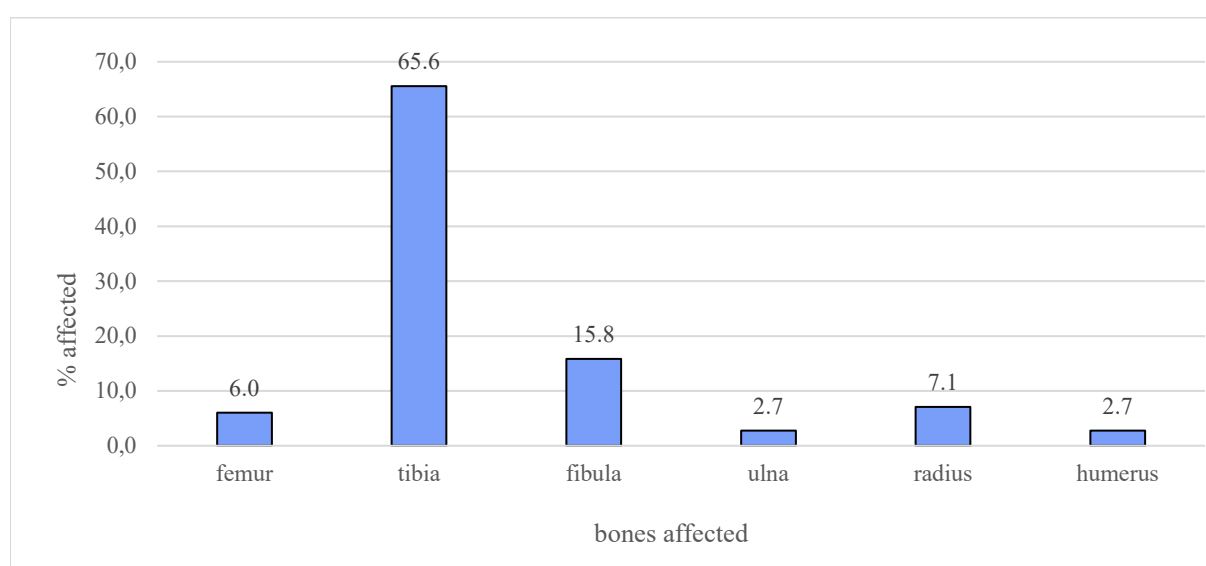


Figure 4.13. Distribution of Harris' lines per bone in the cohort (%).

4.1.3.1.2 Dental enamel hypoplasia (DEH)

Each individual with teeth (442/624) was examined for both forms of DEH, namely pitting and linear lines. DEH was identified in 38 (8.6%) of individuals (Table 7.13; Appendix 1). One must keep in mind that many of the individuals that was classified with teeth, showed antemortem tooth loss of the four incisors of the maxilla and/or the mandible due to cultural modification. Furthermore, many of the teeth present had calculus deposits that obscured possible lines to examine. Therefore this scoring results are not a true representation of the signs of DEH of the individuals in the KSC. The young-adult age group demonstrated a markedly higher frequency of DEH than the other two age groups (18.1% (young-adult group) vs. 6.8% (mid-adult group) and 5.4% (old-adult group); $p < 0.01$). The late time period showed a slightly significant increase in the frequency of DEH when compared to the other two time periods ($p = 0.07$) (Table 4.9).

4.1.3.1.3 Rickets / Osteomalacia

As the KSC sample had no child skeletons for comparison, remnants of chest changes were scored in the adult sample for signs of pectus carinatum (“pigeon breast”) of the anterior thoracic wall (regarded as an important trait for rickets diagnosis). Pectus carinatum is usually less frequently observed than pectus excavatum (“funnel breast”) (Goretsky et al., 2004; Lamrani et al., 2010). The sternum was present in 488/624 individuals (78.2% of cohort), with three of these three (0.6%) displaying pectus carinatum (Fig. 4.14A), and six (1.2%) pectus excavatum (Fig. 4.14B) (Table 7.14; Appendix 1). Only two individuals displayed signs of coxa vara, and no statistical analysis was attempted on this low number. Although a notable difference in the presence of pectus carinatum was seen among age groups (Table 4.9), the small number of individuals presenting with this trait does not allow for any meaningful comparisons. A total of 2.4% (n=15) individuals in the cohort showed traits of osteomalacia, including one or more of the following: pectus carinatum, coxa vara, and bowing of the tibiae, fibulae (Fig. 4.15A&B), femora, ulnae and/or radii. Details of individuals and bones involved are given in Table 7.15, Appendix 1.



Photo by: A Alblas (AN 1216), lateral view



Photo by: A Alblas (AN 197), lateral view.

Figures 4.14A-B. Examples of A) pectus carinatum (“pigeon breast”); and B) pectus excavatum (“funnel breast”) in the KSC cohort.

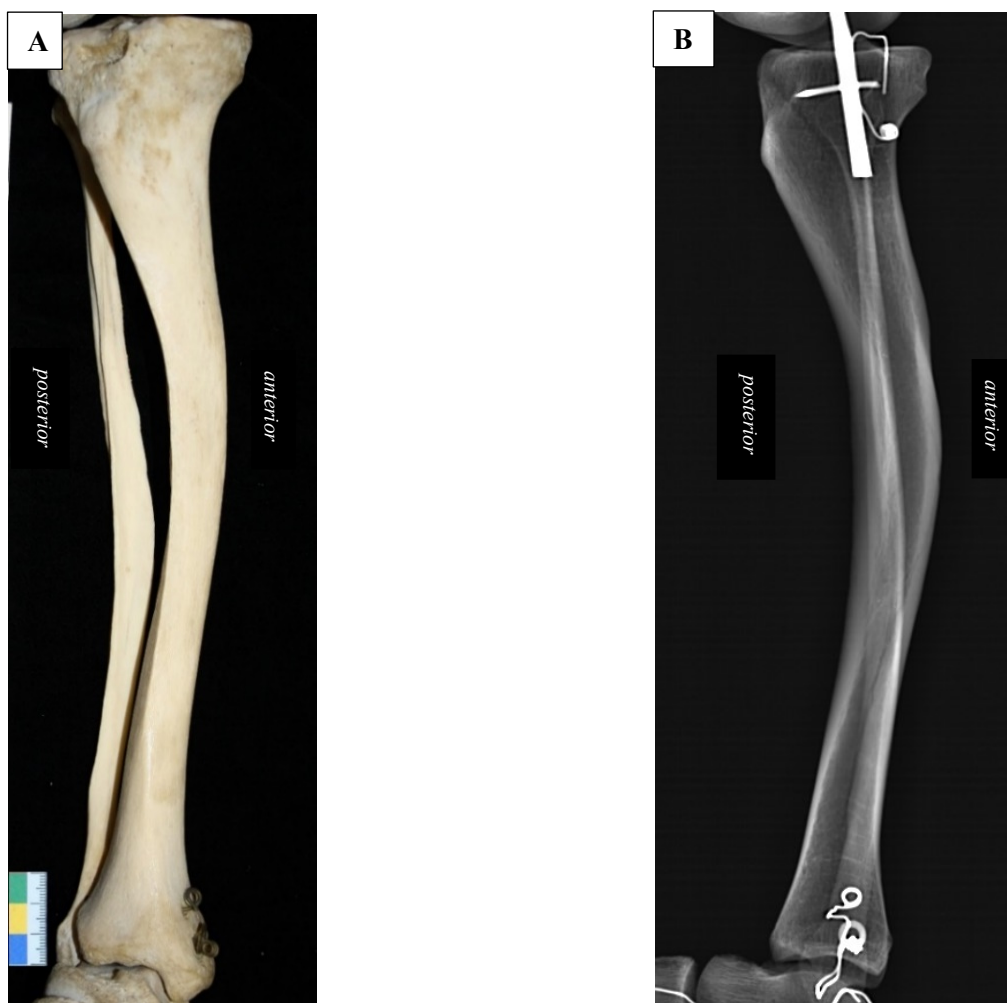


Photo by A Alblas (AN 1), antero-lateral view

Lodox® by: JC Marais, lateral view.

Figures 4.15A-B. Example of articulated bowed tibia and fibula A) on dry bone; and B) on Lodox® image of the same individual.

4.1.3.1.4 Scurvy-related lesions on bones

Skeletal lesions associated with scorbutic deficiency, including ossified haematomas, pitting and woven bone on specific cranial elements, such as the wings of the sphenoid bone (Fig. 4.16A) or the inferior occipital bone, and subperiosteal proliferation of woven bone on post-cranial bones (Fig. 4.16B), were listed in Table 7.16, Appendix 1. Although 240/624 individuals (38.5% of cohort) were observed with these lesions, this is not a true representation of scurvy in the sample, as differential diagnosis was difficult due to the fact that many of these traits overlap with signs of various other diseases. Signs of periosteal reactions on long bones were included in the study as non-specific periostitis (Table 4.8). A summary of the appearance and distribution of these post cranial periosteal lesions can be seen in Table 7.10 (Appendix 1).

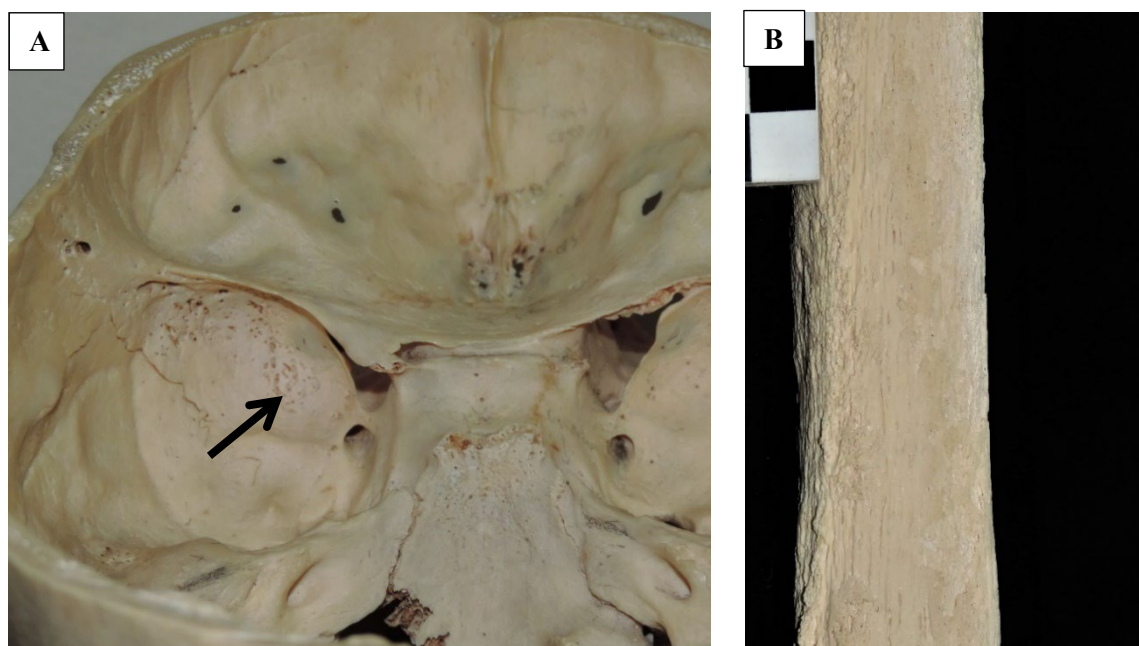


Photo of inner cranium: A Naudé (AN 831), inner cranial view. Photo: A. Alblas (AN 792), anterior view. Figures 4.16A-B. Examples scurvy lesions on the A) sphenoid wings of the cranium; and B) long bone lesion showing woven bone deposits on the shaft.

4.1.3.2 Haematological disorders

Haematological disorders, in particular iron deficiency anaemia, seen as cribra orbitalia (CO) or porotic hyperostosis (PH), associated with hyperostosis of the vault and expanded vault diploe, were seen in a total of 244/544 crania in the KSC cohort (44.9%) (Table 4.10). When all these traits were considered in combination, females showed a significantly higher incidence rate of anaemia than males ($p < 0.01$), although CO on its own showed a male predilection. No significant difference in frequency of any of the other traits of iron deficiency anaemia was observed in any of the comparison groups.

Table 4.10. Statistical analyses of deficiency diseases in the cohort.

Anaemia Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total Number
	Y	A	O	<i>p</i>	SAB	SAC	SAW	<i>p</i>	EC	MC	LC	<i>p</i>	F	M	<i>p</i>	
	(n=20)	(n=42)	(n=15)		(n=13)	(n=51)	(n=13)		(n=2)	(n=33)	(n=42)		(n=22)	(n=55)		
CO	17.9	16.3	11.9	0.38	16.0	14.7	18.3	0.77	5.7	15.4	17.1	0.22	14.3	16.1	0.60	n= 77
	(n=29)	(n=40)	(n=15)		(n=15)	(n=62)	(n=7)		(n=8)	(n=32)	(n=44)		(n=30)	(n=54)		
PH	40.7	35.2	29.2	0.15	17.4	17.0	9.5	0.21	17.8	14.1	17.5	0.57	18.9	14.8	0.24	n= 84

CO=cribra orbitalia; PH=porotic hyperostosis

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; E=Early era; M=Mid era; L=Late era; F=Female; M=Male.

^o $p < 0.1$; ^{*} $p < 0.05$; [†] $p < 0.01$

4.1.3.2.1 Cribra orbitalia (CO)

Of the crania present for analyses of cribra orbitalia, 15.5% (n=77) showed the presence of pitting or porosity in the roof of the orbits (Table 7.17; Appendix 1). The average age of the individuals with CO was 46.5 years (range: 20-70 years, SD:12.0). In 94.8% of cases both orbits were involved. Three individuals (3.9%) showed CO unilaterally in the left orbit and only one (1.3%) in the right orbit alone. No significant differences in the presence of CO were noted for any of the comparison groups (Table 4.10).

4.1.3.2.2 Porotic hyperostosis (PH)

Of the calvaria available for analyses, 16.0% (n=84) showed pitting or porosity on the parietal and/or occipital bones (Table 7.18; Appendix 1), with accompanying vault hyperostosis. If hyperostosis of the vault was not present, pitting was regarded as ectocranial porosis and not PH. Storage and handling erosion were taken into account, along with artefacts resulting from processing. No statistically significant difference in the presence of PH was seen in any of the comparison groups (Table 4.8). The mean age of individuals with PH was 44.8 years (range: 20-72 years, SD:12.9).

4.1.4 DEGENERATIVE JOINT DISEASES AND ARTHROPATHIES

4.1.4.1 Osteoarthritis: Degenerative peripheral joint osteoarthritic (pOA) markings

Table 4.11. Statistical analyses of peripheral joint osteoarthritis (n=399).

pOA	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total
Trait	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p	number
Shoulder L	(n=18) 14.5	(n=65) 25.3	(n=71) 60.2	†<0.01	(n=25) 29.1	(n=100) 28.4	(n=29) 47.5	†<0.01	(n=13) 27.7	(n=102) 42.4	(n=39) 18.0	†<0.01	(n=40) 27.8	(n=114) 32.1	0.34	n= 154
Shoulder R	(n=16) 12.0	(n=76) 27.1	(n=81) 59.6	†<0.01	(n=30) 32.0	(n=111) 28.5	(n=32) 47.8	†<0.01	(n=17) 34.7	(n=108) 42.4	(n=48) 19.5	†<0.01	(n=47) 29.2	(n=126) 32.4	0.46	n= 173
Elbow L	(n=36) 28.1	(n=112) 42.1	(n=66) 55.0	†<0.01	(n=34) 39.1	(n=148) 40.6	(n=32) 51.6	†<0.01	(n=22) 40.7	(n=121) 50.4	(n=71) 32.3	†<0.01	(n=59) 40.1	(n=155) 42.2	0.66	n= 214
Elbow R	(n=37) 26.6	(n=120) 41.5	(n=71) 50.1	†<0.01	(n=36) 37.9	(n=157) 38.9	(n=35) 50.0	0.23	(n=21) 37.5	(n=124) 47.2	(n=83) 33.2	†<0.01	(n=63) 38.0	(n=165) 41.0	0.51	n= 228
Wrist L	(n=9) 6.4	(n=35) 11.7	(n=25) 17.6	†<0.01	(n=11) 11.6	(n=49) 11.9	(n=9) 12.7	0.20	(n=6) 10.2	(n=41) 15.5	(n=22) 8.7	‡0.05	(n=17) 10.1	(n=52) 12.7	0.38	n= 69
Wrist R	(n=9) 6.5	(n=31) 11.5	(n=24) 16.9	0.20	(n=9) 9.5	(n=46) 11.2	(n=9) 12.7	0.98	(n=3) 5.1	(n=39) 14.7	(n=22) 8.7	*0.03	(n=17) 10.2	(n=47) 11.5	0.65	n= 64
Hip L	(n=29) 22.1	(n=82) 29.6	(n=56) 44.1	†<0.01	(n=28) 29.8	(n=104) 27.8	(n=35) 52.2	0.80	(n=22) 40.7	(n=84) 34.4	(n=61) 25.7	*0.03	(n=48) 30.8	(n=119) 31.4	0.89	n= 167
Hip R	(n=29) 31.5	(n=79) 27.5	(n=57) 40.4	†<0.01	(n=27) 28.1	(n=103) 25.9	(n=35) 50.7	†<0.01	(n=20) 34.5	(n=85) 32.8	(n=60) 24.4	‡0.07	(n=45) 27.2	(n=120) 30.2	0.49	n= 165
Knee L	(n=12) 9.2	(n=40) 16.8	(n=44) 36.2	†<0.01	(n=18) 21.3	(n=59) 17.5	(n=19) 31.3	*0.04	(n=11) 24.5	(n=51) 23.6	(n=34) 15.0	*0.04	(n=30) 19.0	(n=66) 20.3	0.73	n= 106
Knee R	(n=11) 8.7	(n=20) 13.8	(n=16) 31.6	†<0.01	(n=6) 18.6	(n=35) 14.8	(n=6) 27.1	‡0.05	(n=3) 19.6	(n=14) 19.8	(n=30) 13.4	0.13	(n=6) 18.1	(n=41) 16.5	0.65	n= 96
Ankle L	(n=11) 7.8	(n=19) 6.6	(n=16) 11.2	0.28	(n=6) 6.1	(n=34) 8.4	(n=6) 8.5	0.71	(n=3) 5.1	(n=14) 11.2	(n=29) 5.4	*0.04	(n=6) 3.5	(n=40) 9.9	†<0.01	n= 47
Ankle R	(n=11) 7.8	(n=19) 6.3	(n=16) 11.2	0.22	(n=6) 6.1	(n=34) 8.2	(n=6) 8.5	0.75	(n=3) 5.1	(n=14) 10.9	(n=29) 5.4	‡0.05	(n=6) 3.5	(n=40) 9.7	†<0.01	n= 46

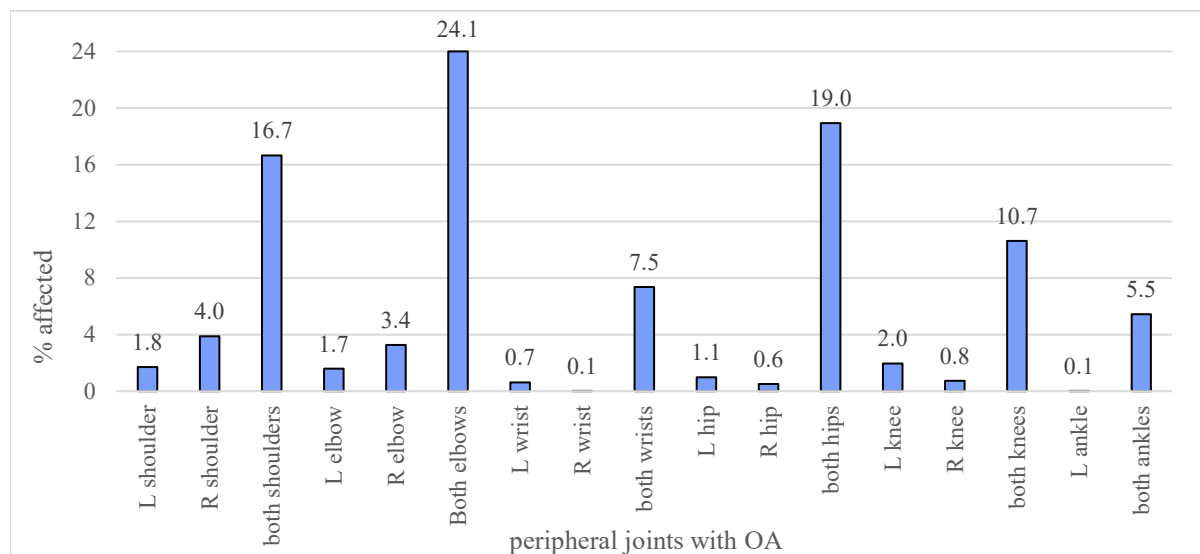
L=left; R=right

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

‡p<0.1; *p<0.05; †p<0.01

A total of 95.5% of the cohort (596/624 individuals) had post-cranial skeletal elements available for examination for signs of OA on the peripheral joints. Of these, 66.9% (n=399) displayed osteoarthritic lesions on the joints (Table 7.19; Appendix 1). Males showed the highest incidence rate for all the joints except the right knee (Table 4.11). A significant difference between the sexes (p=0.01) was observed for both ankles. The old age group demonstrated the highest frequency of OA on all joints. In terms of time periods, the mid-period had the highest incidence rate of OA on all joints except the hips and knees (where, in both cases, OA markings were more frequently observed in the early group). The SAW population group showed a higher frequency of OA markings than the other race groups – a finding that is to be expected, as SAW individuals were, on average, much older than the other

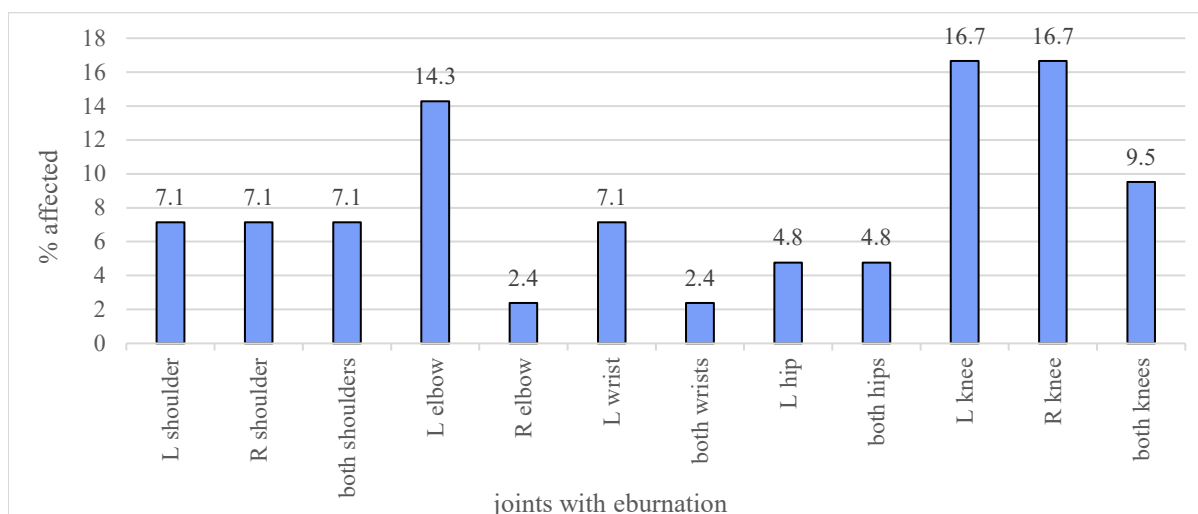
groups. The elbows, followed by the hips and shoulders (all bilaterally), demonstrated the highest rate of OA markings (Fig 4.17), while the wrist joints displayed the lowest frequency.



L=Left side; R=Right side

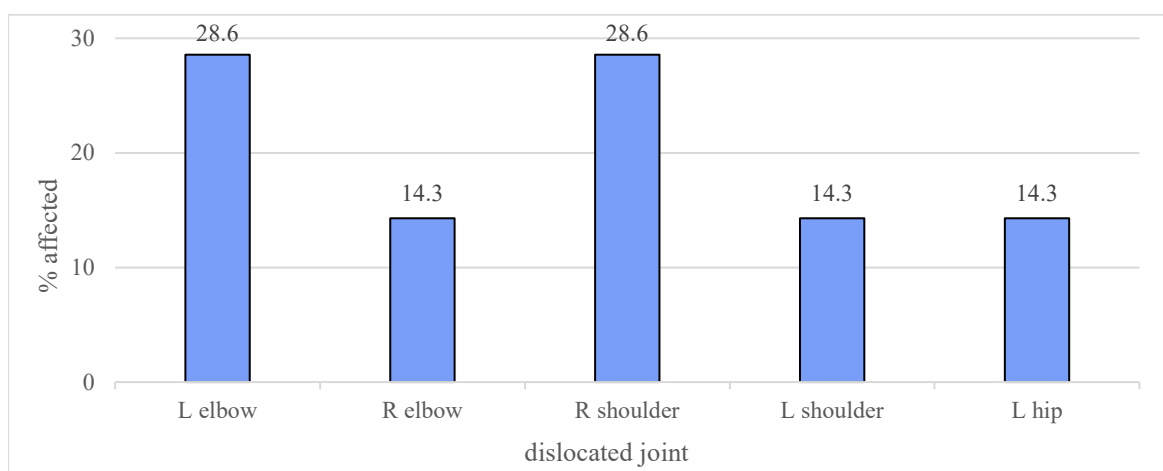
Figure 4.17. Distribution frequency (%) of OA on peripheral joints.

Only 35 individuals (5.9% of cohort) displayed eburnation on their joints (Table 7.20; Appendix 1), with both knee joints and the left elbow being most affected (Fig. 4.18). Dislocated peripheral joints were observed in 1.2% (n=7) of cases (Fig. 4.19) (Table 7.21; Appendix 1), with the upper limbs, especially the right shoulder (see Fig. 4.20A) and left elbow, being most affected. All dislocated joints were observed in SAC males between 21 and 54 years, except for one incidence in a 37-year-old SAB male from the late era. All upper-limb joint dislocations were from the mid-era, with the exception of two cases from the late era. A dislocated left hip was observed in a 66-year-old SAC male from the early time period (Fig 4.20B).



L=Left side; R=Right side

Figure 4.18. Distribution frequency (%) of eburation at peripheral joints.



L=Left side; R=Right side

Figure 4.19. Distribution frequency (%) of dislocation of peripheral joints.

Figures 4.20A-B. Examples of dislocated joints A) shoulder and B) hip.

4.1.4.2 Osteoarthritis: Vertebral joint osteoarthritic (vOA) markings

The vertebrae of the KSC cohort were examined for markings of osteoarthritis Table 4.12). The vertebrae of the males were slightly more affected than the females. However, if the traits were examined separately, females showed a higher correlation to the laminar spurs, costovertebral spurs and the zygapophyseal joints OA. All the traits showed the old adult age group significantly more affected, except for the laminar spurs, where the young adult age group showed the highest frequency. When the population groups were compared, the SAW population shows the highest involvement of vertebral OA followed by the SAC and SAB

population groups respectively, although the SAC showed a higher frequency of laminar spurs compared to the other groups. There was no correlation to a specific time period for each OA trait, although, overall, the mid era shows the highest involvement of osteoarthritic markers on the vertebral column. Wedging of the vertebrae indicated kyphosis, scoliosis, lordosis or a combination of these conditions. Statistically, males and the SAW population group demonstrated the highest frequency of wedged vertebrae, and the old adult age group was mostly affected. A comparison of the time periods, displayed the early and mid eras with statistically noteworthy higher wedged vertebrae, than the late era (Table 4.12).

Table 4.12. Statistical analyses of vertebral joint osteoarthritis (n=511).

Trait	Age Group (%)			p	Population Group (%)			p	Time Period (%)			p	Sex (%)		Total number		
	Y	A	O		SAB	SAC	SAW		EC	MC	LC		F	M			
Vertebral OA	VO	(n=9) 7.3	(n=35) 13.0	(n=38) 30.9	†<0.01	(n=14) 16.9	(n=48) 13.3	(n=20) 28.6	†0.01	(n=9) 19.6	(n=42) 18.1	(n=31) 13.1	0.26	(n=21) 14.0	(n=61) 16.7	0.44	n=82
	laminar	(n=22) 17.9	(n=31) 11.5	(n=9) 7.6	‡0.05	(n=9) 10.8	(n=47) 13.0	(n=6) 9.0	0.58	(n=3) 6.7	(n=20) 8.7	(n=39) 16.5	*0.02	(n=23) 15.4	(n=391) 10.8	0.15	n=62
	costovert	(n=19) 15.5	(n=51) 19.0	(n=31) 26.1	0.11	(n=18) 21.7	(n=68) 18.8	(n=15) 22.4	0.72	(n=5) 11.1	(n=58) 25.3	(n=38) 16.0	*0.02	(n=36) 24.2	(n=65) 18.0	0.11	n=101
	body	(n=45) 36.6	(n=174) 64.7	(n=83) 68.6	†<0.01	(n=36) 44.4	(n=222) 61.0	(n=44) 64.8	†0.01	(n=28) 58.3	(n=150) 65.0	(n=124) 52.5	*0.02	(n=77) 51.7	(n=225) 61.8	†<0.01	n=302
	zjoint	(n=35) 28.5	(n=114) 42.5	(n=69) 58.0	†<0.01	(n=25) 30.9	(n=150) 41.4	(n=43) 64.2	†<0.01	(n=18) 40.0	(n=116) 50.6	(n=84) 53.6	*0.04	(n=71) 48.0	(n=147) 40.6	0.13	n=218
	kissing	(n=10) 7.5	(n=42) 14.8	(n=43) 17.2	*0.04	(n=7) 7.9	(n=52) 13.2	(n=16) 23.2	*0.02	(n=5) 7.8	(n=45) 18.2	(n=25) 10.4	*0.02	(n=21) 12.8	(n=54) 13.9	*0.03	n=75
Vertebral wedging	plana	(n=5) 4.1	(n=39) 14.5	(n=22) 18.5	†<0.01	(n=7) 8.4	(n=51) 14.1	(n=8) 12.0	0.34	(n=7) 15.6	(n=40) 17.5	(n=19) 8.0	†0.01	(n=12) 8.1	(n=54) 14.9	*0.03	n=66
	post	(n=6) 4.5	(n=22) 8.2	(n=13) 10.9	0.21	(n=4) 4.8	(n=31) 8.6	(n=6) 9.0	0.52	(n=6) 13.3	(n=19) 8.3	(n=16) 6.8	0.36	(n=12) 8.1	(n=29) 8.0	0.99	n=41
	lat	(n=5) 4.1	(n=18) 6.7	(n=8) 6.7	0.57	(n=7) 8.4	(n=16) 4.4	(n=8) 11.9	‡0.05	(n=4) 8.9	(n=18) 7.9	(n=9) 3.8	‡0.10	(n=7) 4.7	(n=24) 6.6	0.39	n=31
	ant	(n=13) 10.6	(n=23) 8.6	(n=16) 13.5	0.35	(n=4) 4.8	(n=38) 10.5	(n=10) 14.9	0.11	(n=3) 6.7	(n=24) 10.5	(n=25) 10.6	0.84	(n=11) 7.4	(n=41) 11.3	0.17	n=52

OA=osteoarthritis; VO=vertebrate osteophytosis; lamina=laminar spurs; costovert=costovertebral joint; body=anterior body of vertebra; zjoint=zygapophyseal joint; kiss=kissing osteophytes; post=posterior; lat=lateral; ant=anterior

Y=Young adult; A=Mid adult; O=Old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

°p<0.1; *p<0.05; †p<0.01

Table 4.13. Statistical analyses of vertebral fusion and spondylolysis.

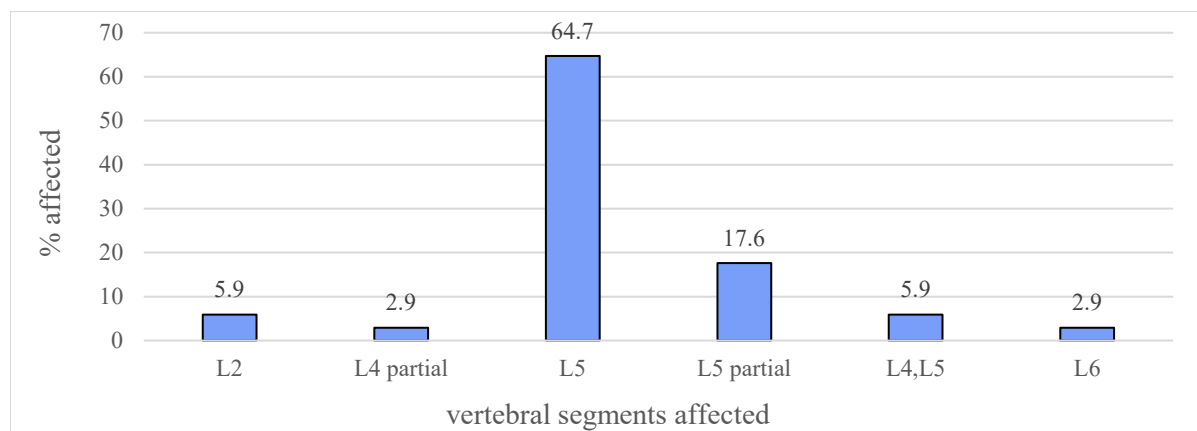
Vertebral Trait	Age Group (%)			<i>p</i>	Population Group (%)			<i>p</i>	Time Period (%)			<i>p</i>	Sex (%)		
	Y	A	O		SAB	SAC	SAW		EC	MC	LC		F	M	<i>p</i>
Spondylolysis	(n=7) 5.7	(n=21) 7.8	(n=6) 5.0	0.52	(n=2) 2.4	(n=27) 7.5	(n=5) 7.4	0.23	(n=5) 6.8	(n=13) 5.7	(n=16) 11.1	0.35	(n=7) 4.7	(n=27) 7.5	n=34 0.23
DISH	(n=0) 0.0	(n=4) 1.3	(n=7) 4.5	†<0.01	(n=2) 2.0	(n=6) 1.4	(n=3) 3.6	0.23	(n=1) 0.7	(n=8) 2.9	(n=2) 1.5	0.13	(n=2) 1.1	(n=9) 2.1	n=11 0.37
SIJ fusion	(n=2) 1.6	(n=10) 3.7	(n=10) 8.3	*0.04	(n=5) 6.0	(n=14) 3.8	(n=3) 4.4	0.58	(n=1) 2.9	(n=14) 6.1	(n=7) 2.2	0.21	(n=6) 4.0	(n=16) 4.4	n=22 0.84
AS	(n=5) 3.4	(n=14) 4.4	(n=16) 10.2	*0.02	(n=3) 2.9	(n=19) 4.3	(n=13) 15.7	†<0.01	(n=4) 4.7	(n=18) 6.5	(n=13) 6.1	0.59	(n=14) 7.5	(n=21) 4.8	n=35 0.19

DISH=diffuse idiopathic skeletal hyperostosis; SIJ=sacroiliac joint; AS=ankylosing spondylitis

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

**p*<0.05; †*p*<0.01

Spondylolysis was present in 6.7% (n=34) of the individuals with a vertebral column (Table 7.22; Appendix 1). The mean age for affected individuals was 48.6 years ranging between 21 to 81 years (SD:12.4). No statistical significant differences among the variables were observed (Table 4.13). The vertebral level that was considerably more involved compared to the other levels, was L5 (82.3%), with either partial or complete spondylolysis (Fig. 4.21).



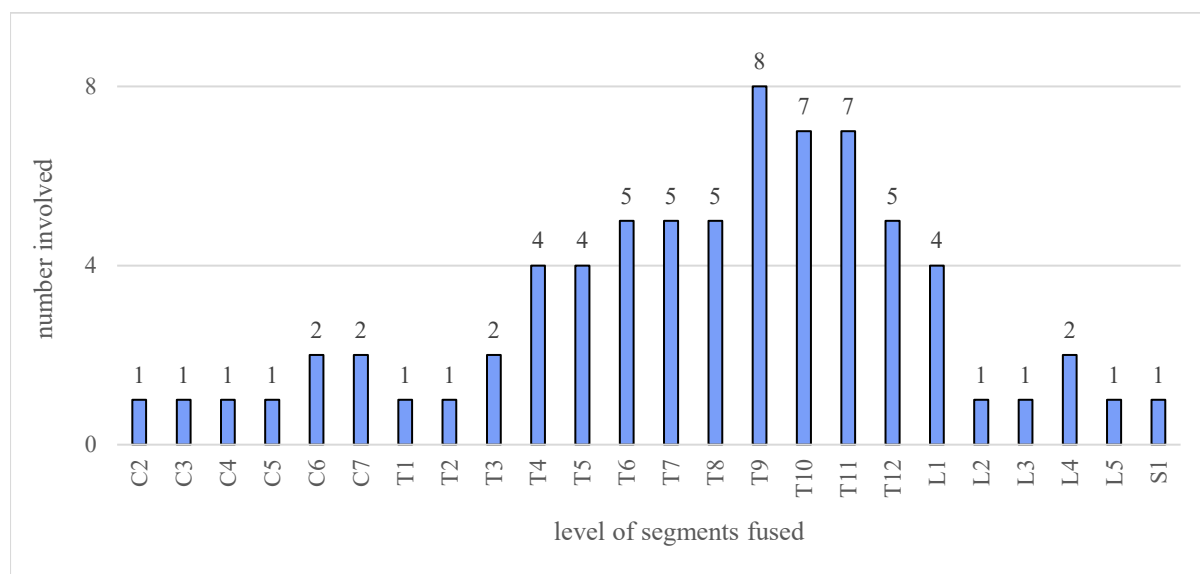
L=lumbar vertebra

Figure 4.21. Vertebral levels with spondylolysis in the cohort (%).

4.1.4.3 Osteoarthritis: Vertebral osteophytosis (VO)

A total of 11/510 (2.2%) individuals in this study showed diffuse idiopathic skeletal hyperostosis (DISH) with four or more vertebral segments fused (Table 7.23; Appendix 1). Although a male predilection at a ratio of 4.5:1 is seen, the bias towards males in the collection should be taken into account (Table 4.12). All the affected individuals were either from the mid-adult (1.3%) or old (4.5%) age groups with a mean age of 61.9 years. The youngest person with DISH was 53 years and the oldest 81 years (SD: 7.8). The SAW population group shows the highest involvement of DISH, although not significantly. In the majority of cases, the lower

thoracic region of the vertebral column was fused, followed by the upper thoracic segments (Fig 4.22).



C=Cervical vertebra; T=Thoracic vertebra; L= Lumbar vertebra; S=Sacral vertebra

Figure 4.22. Vertebral levels involved in fusion in DISH.

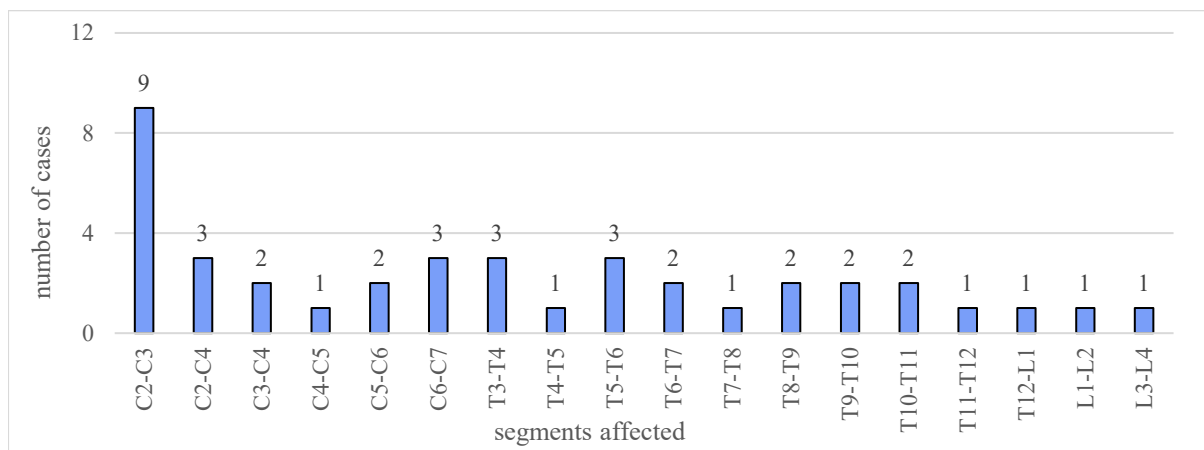
4.1.4.4 Arthropathies

4.1.4.4.1 Sacroiliac joint fusion (SIJ)

Individuals with os coxae (n=537) and sacra (n=510) were evaluated for unilateral or bilateral sacroiliac joint fusion to aid in differential diagnosis of degenerative diseases. A total of 4.1% individuals showed sacroiliac joint fusion arthropathies (Table 7.24; Appendix 1). The old age group were significantly more affected with SIJ compared to the other age groups (Table 4.13). The mean age of individuals with SIJ fusion was 54.7 years ranging from 22 to 73 years (SD:11.4) The side mostly involved was the left side (45.5%), followed by the right side (36.6%), and lastly bilateral involvement (17.9%).

4.1.4.4.2 Sero-negative arthropathies

Ankylosing spondylitis (AS) was seen in 6.9% of the individuals in the KSC cohort (Table 7.25; Appendix 1). The age group mostly affected was the old adult age group, while the population groups mostly affected was the SAW population group. This SAW population was also the group with the oldest individuals, explaining the significant higher frequency of AS of their vertebrae compared (Table 4.13). The two vertebrae mostly affected by fusion were C2-C3 (Fig. 4.23).

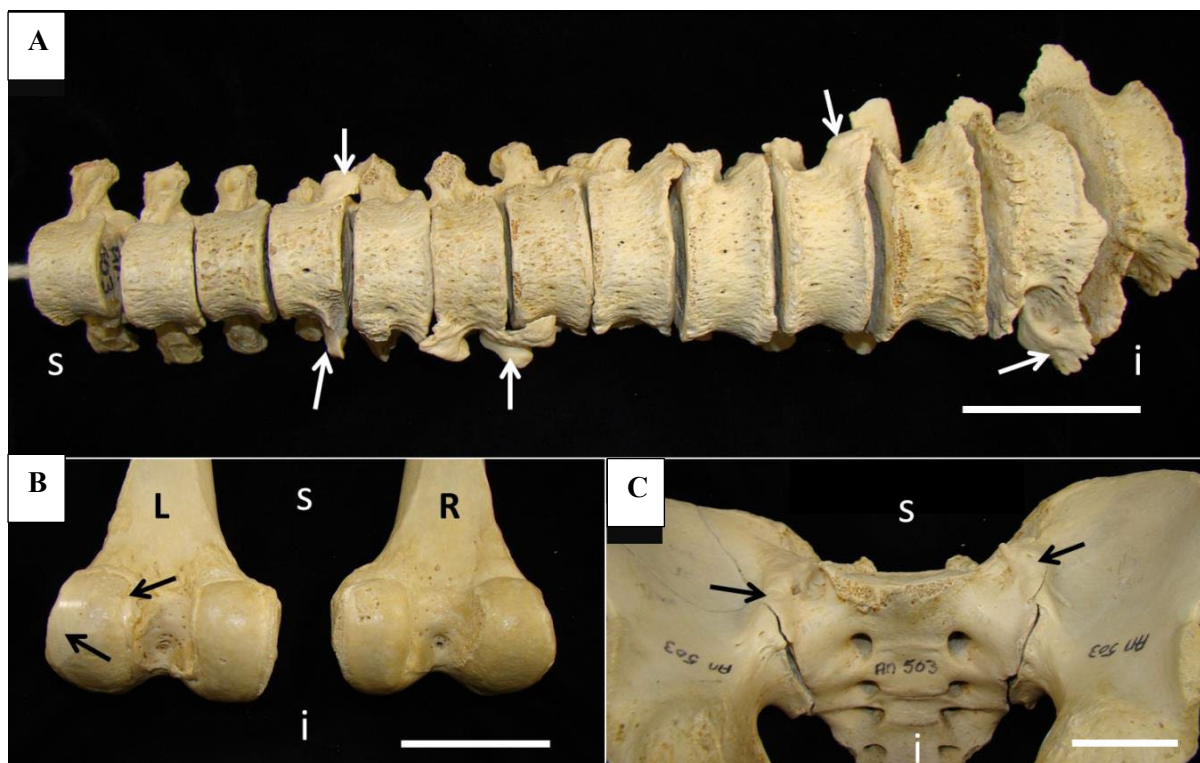


C=Cervical vertebra; T=Thoracic vertebra; L=Lumbar vertebra; S=Sacral vertebra

Figure 4.23. The number and distribution of the fused vertebrae in the KSC cohort.

Nine individuals showed more than one area containing ankylosed vertebrae. In three individuals, more than eight segments were affected, fused in two or three groups, forming asymmetrical areas of ligamentous ossification along the vertebral column. These are also called “skip lesions” and may be associated with Reactive arthritis (ReA).

Two cases of Reactive Arthritis (ReA) were diagnosed in the KSC cohort, showing various traits associated with ReA. A 66 year old male from the mid era (AN 405) was identified with patches of asymmetrical ossification within the ligamentous structures along the vertebral column (T3-T12 and L2-L4), also called “skip lesions”, asymmetric (left side) ossification of the sacro-iliac joint (SIJ), osteophytes on the acetabular rims of the os coxae, and arthritis of the large joints of the lower limbs, in this instance, the knees. Some OA on the right foot phalanges was observed as seen in patients with this condition, since ReA tends to asymmetrically involve the small joints in the feet more than the small joints in the hands. A 73 year old SAC male (AN 503) showing signs of ReA with kissing osteophytes (T8,T9) and ankyloses (T10, T11) along the vertebral column (Fig 4.24A) was identified. Asymmetrical arthritis of the left knee with eburnation (Fig 4.24B) was also observed and bilateral fusion of the SIJ (Fig 4.24C). Although hands are not usually involved, OA on the CMC and IP joints of the left hand and PIP and DIP joints of the right hand were observed. The age of onset for this condition was from adolescence, before 40 years, however, the medical history of these individuals is unknown and can therefore not be confirmed (Table 7.26; Appendix 1).



Photos by: J Walters (AN 503)

Figures 4.24A-C. Reactive arthritis differential diagnostic traits, including A) vertebral ligament ossification, B) unilateral OA and eburnation on the knee; and C) bilateral ossification of the SIJ.

4.1.5 NEOPLASTIC DISEASES

4.1.5.1 Malignant primary neoplasms

Only two (0.3%) individuals in the KSC study showed signs of a malignant primary bone neoplasm, namely a parosteal surface osteosarcoma and a lymphosarcoma (Table 7.27; Appendix 1). The large sclerotic parosteal surface osteosarcoma (AN 464) was seen in the distal metaphysis of the left femur of a 65 year old SAC male from the mid 20th century (Fig. 4.25A,B) at the site of the typical distribution of a parosteal osteosarcoma.

The lymphosarcoma (Fig. 4.26A,B) was antemortem diagnosed in a 65 year old SAC male from the mid era (AN 522) and registered on the death certificate as a reticulum cell lymphosarcoma. On the dry bone, fine spiculed osteoblastic activity on the os coxae and woven bone depositing on the scapula were observed.

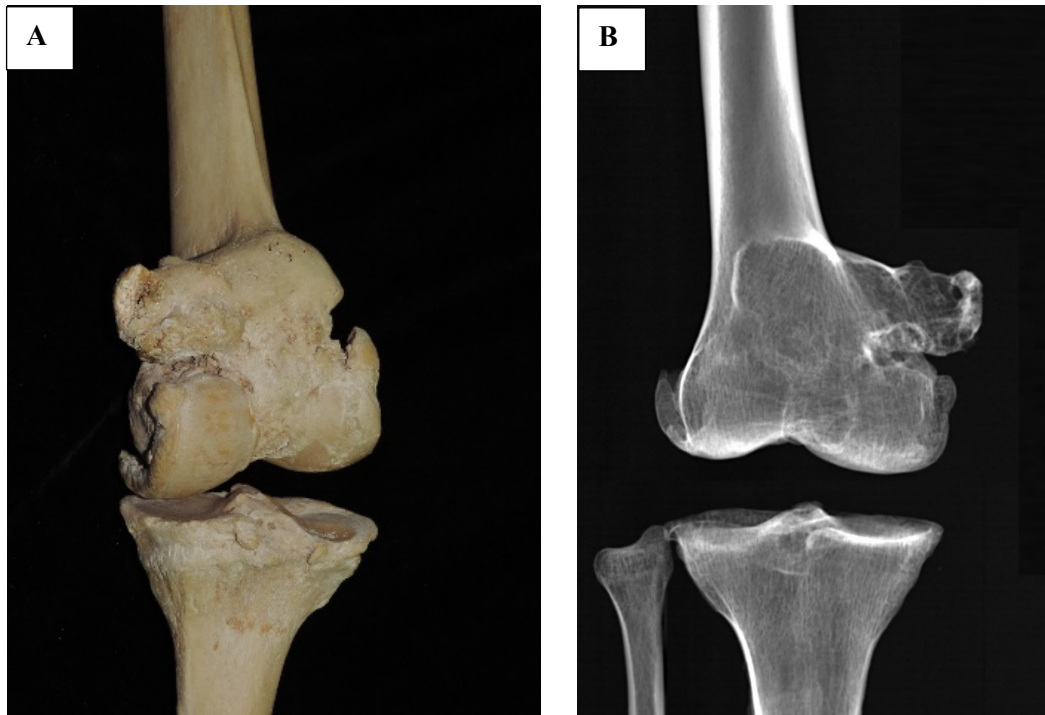
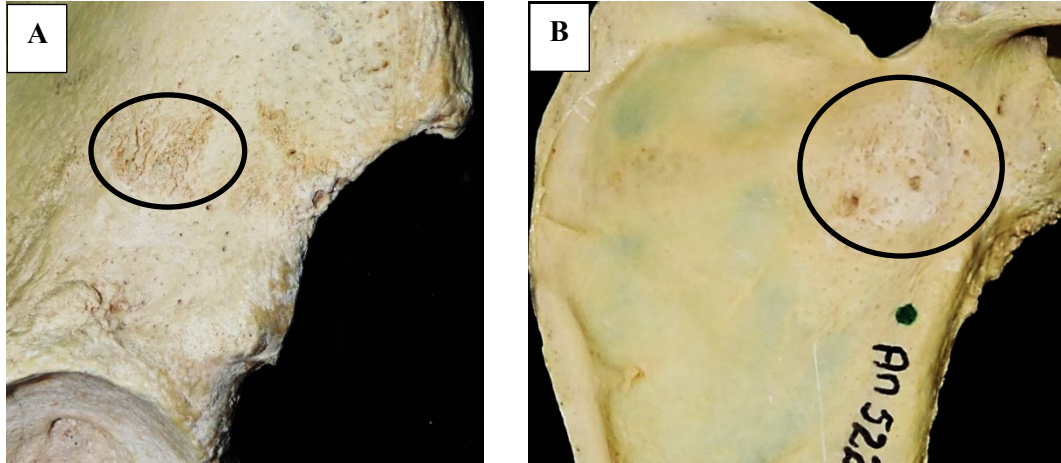


Photo by: A Alblas (posterior view), Lodox[®] scan by J Dempers (A-P view) (AN 464)

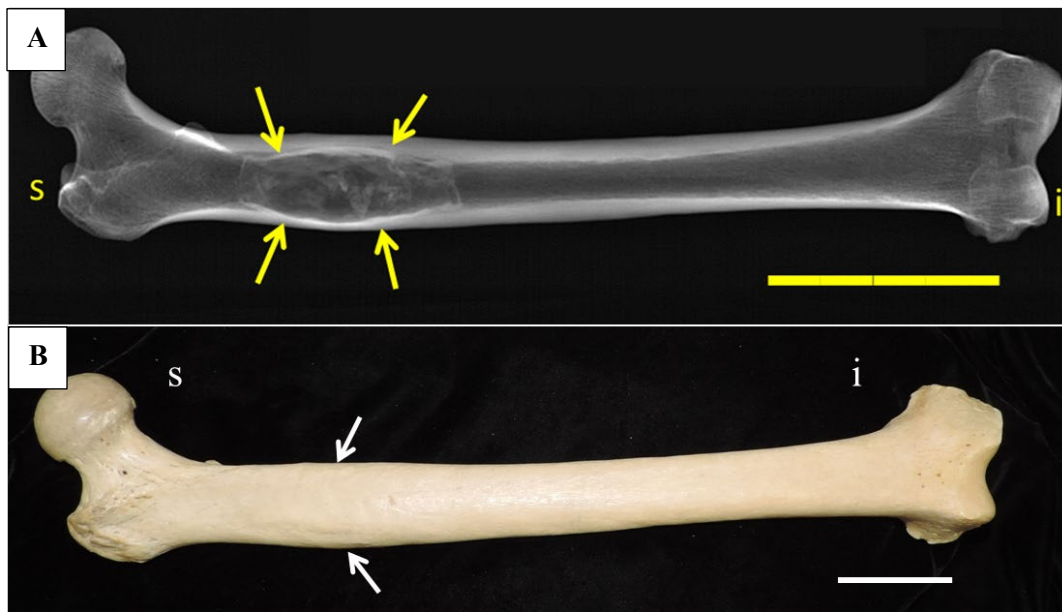
Figures 4.25A-B. Parosteal surface osteosarcoma with a large sclerotic deposit on the distal end of the left femur A) posterior view B) Lodox[®] scan A-P view.



Photos by: A Alblas (AN 522); A) posterolateral view of os coxa and B) anterior view of scapula
 Figures 4.26A-B. Lymphosarcoma with: A) fine spiculated osteoblastic activity on the ileum of os coxa; or B) woven bone depositing on the scapula (encircled).

4.1.5.2 Benign primary neoplasms

A well-defined focal expansile lesion in right proximal femoral diaphysis containing internal chondroid matrix were observed on the Lodox[®] scan of one 50 year old SAC female from the mid era (AN 645). No discernible cortical break was observed and a benign enchondroma was suspected (Fig. 4.27A,B).



Lodox®, (A-P view) and photo (anterior view of right femur) by: J Walters (AN 645)

Figures 4.27A-B. Enchondroma in the proximal shaft of the left femur seen on the A) Lodox® scan; and B) shows expansion of the femur shaft on the dry bone (arrows).

4.1.5.3 Cancer-related cause of death

All the cancer-related deaths indicated on the register as the patients' COD were 139/624 (22.3%) (Table 7.28; Appendix 1). The average age-at-death was 51.4 years and the age range was between 21 and 78 years (SD:11.4). The related cancers were grouped together and plotted on a graph (Fig. 4.28). The pulmonary or respiratory-related cancers showed the highest prevalence (24.5%) followed by oesophagus cancers (13.7%), female cervical cancers and stomach- and colon cancers followed closely (12.2% and 11.5%, respectively).

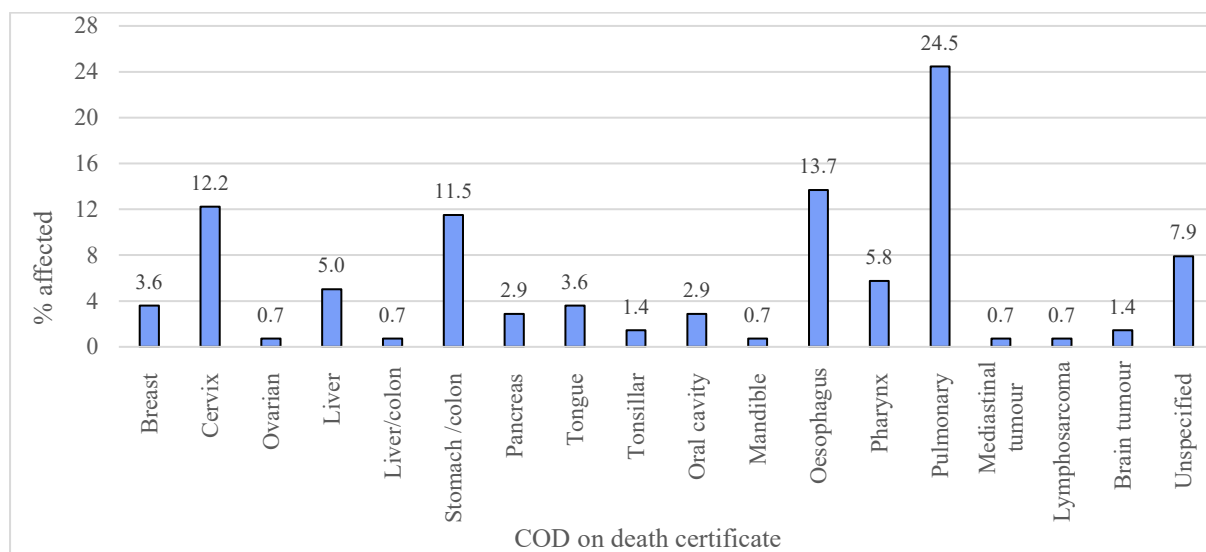


Figure 4.28. Percentage of specific cancers indicated on the death certificates of the individuals in the cohort (%).

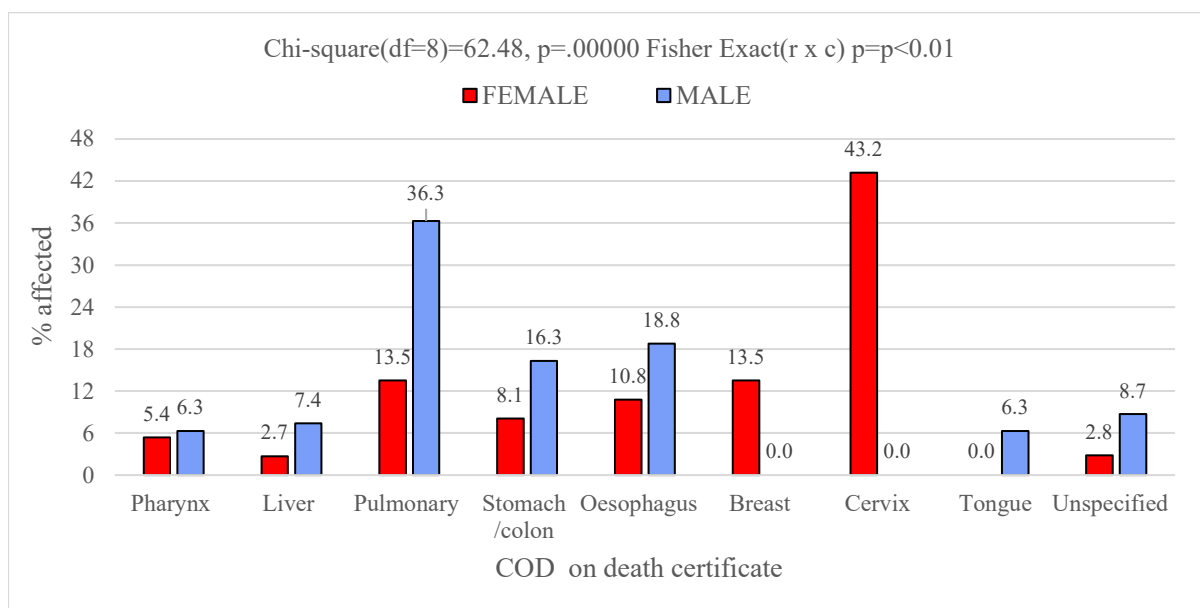


Figure 4.29. Distribution of the type of registered cancers per sex in the cohort (%).

A breakdown of the noteworthy type of cancers registered on the death certificates of the KSC individuals compared between males and females (Fig. 4.29), showed males with a higher predilection for pulmonary cancers (36.3%) compared to females (13.5%), although females show a statistically higher prevalence of reproductive organ-related cancers (43.2%) with no male-related cancers ($p<0.01$).

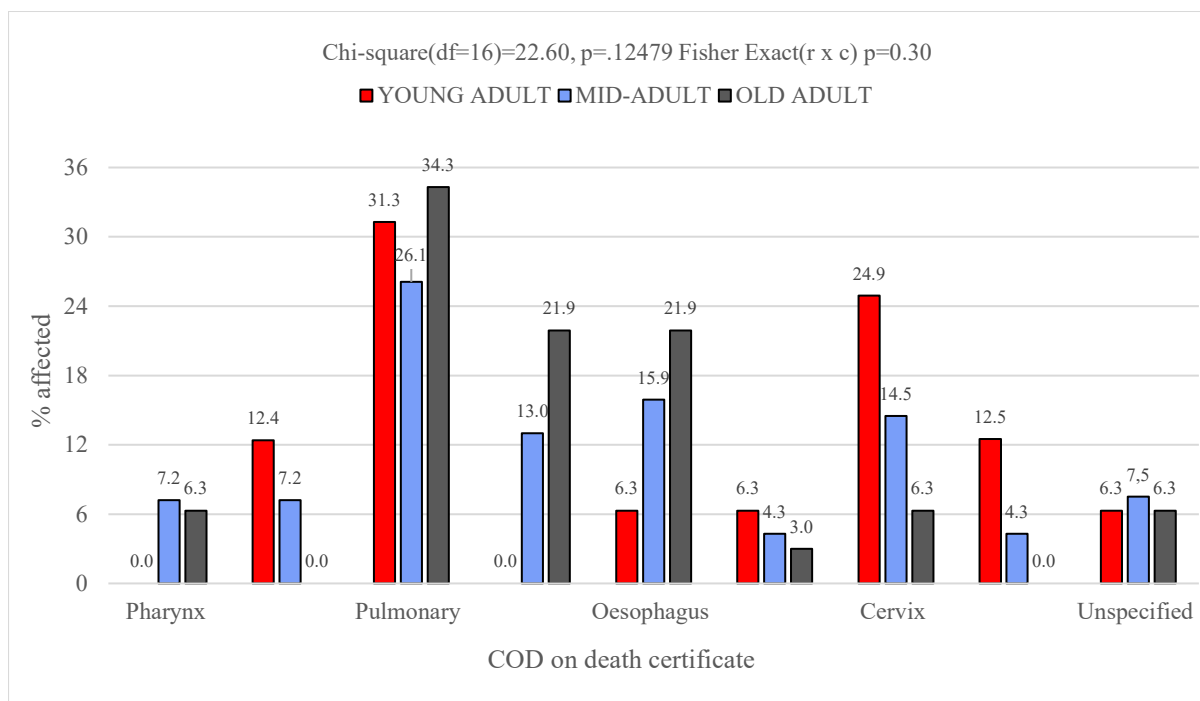


Figure 4.30. Distribution of the type of registered cancers per age group in the cohort (%).

When comparing age groups of these cancers (Fig. 4.30), pulmonary cancers were prominent in all three age groups. The old age group were more affected by gastrointestinal type of cancers, while cervical cancers were high in the young females. Liver and tongue cancers showed a higher prevalence in the young adult age group compared to the other groups.

More individuals of the SAB population groups died of cancer (24/102; 23.5%) compared to the SAC (82/439; 18.7%) and SAW (11/83; 13.2%) population groups, although the total number of individuals in the cohort were more comparable between the SAB and SAW groups than the SAC population group with a much higher number of individuals for comparison. This sample bias towards the SAC population group should therefore be considered when interpreting the graph (Fig. 4.31). The high prevalence of female reproductive system cancers in the SAW population group is however significant, as well as the pharyngeal and liver cancers in the SAC population and tongue and oesophagus cancers in the SAB population group.

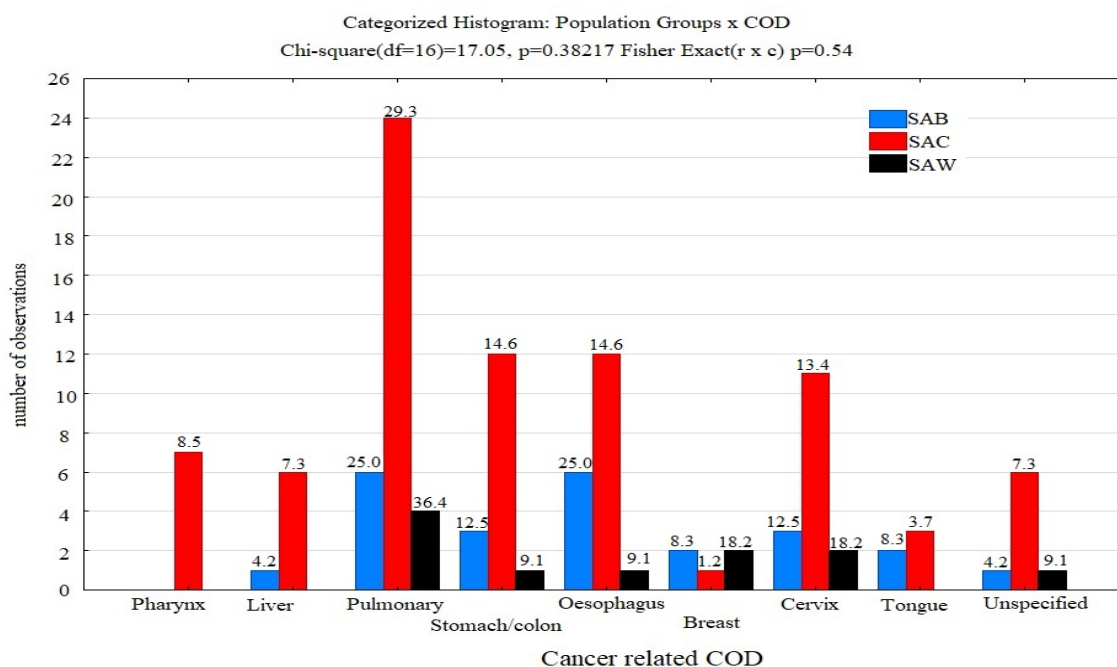


Figure 4.31. Distribution of the type of registered cancers as COD per population group in the cohort.

A comparison of the time periods (Fig. 4.32) showed a high prevalence of pulmonary cancers in all three time periods. The early time period had no cases of pharyngeal, liver, breast and cervical cancers; however, stomach/colon cancers were the highest during this period.

Oesophagus cancers showed a decline in the mid era, however, the prevalence was escalating again in the late era. An increase in oesophagus, liver and cervix cancers towards the late period were observed, while a decline in breast, pharynx and tongue cancers were observed between the mid and the late eras. The low number of individuals representing the early era should be considered in the final interpretation of the graph.

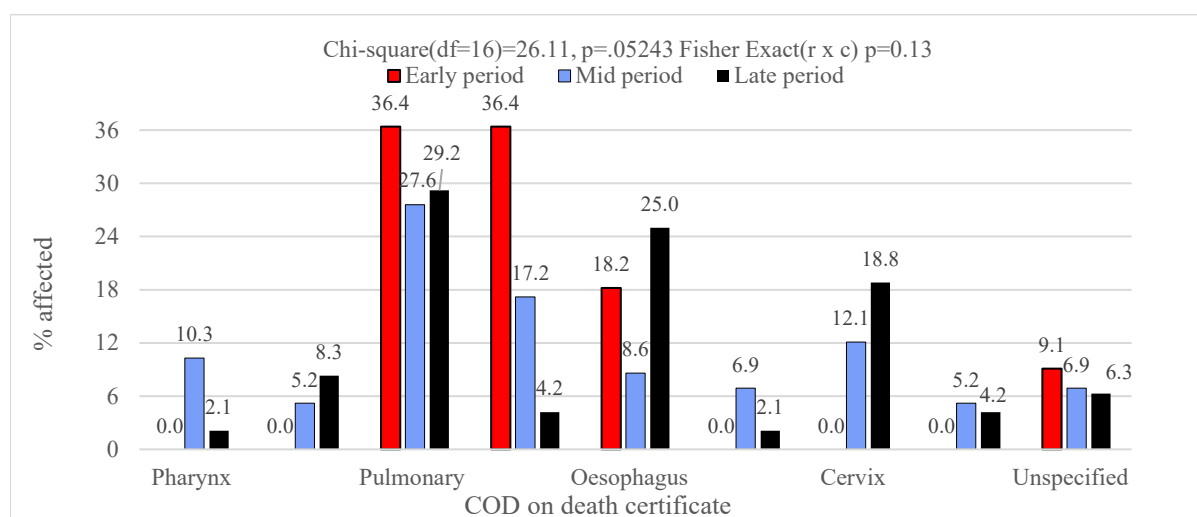


Figure 4.32. Distribution of the type of registered cancers per time period in the cohort (%).

4.1.5.4 Metastatic skeletal lesions

Further statistical analyses were done on samples of the KSC cohort that showed lytic and/or sclerotic lesions on the bone, regardless of the known COD; this total was 117/624 (18.8%) (Table 4.14). Females (23.1%) showed a statistical higher prevalence than males (16.9%; ($p=0.07$)). No other variables showed a statistical significant difference of metastatic lesions.

Table 4.14. Statistical analyses of metastatic lesions on individuals in the KSC cohort.

Various Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total number
	Y	A	O	<i>p</i>	SAB	SAC	SAW	<i>p</i>	E	M	L	<i>p</i>	F	M	<i>p</i>	
Neoplasm	(n=23)	(n=65)	(n=29)		(n=24)	(n=77)	(n=16)		(n=10)	(n=54)	(n=53)		(n=43)	(n=73)		n=117
	15.6	20.4	18.4	0.46	23.5	17.6	19.3	0.40	15.2	19.4	19.0	0.71	23.1	16.9	0.07	

Y=young adult; *A*=mid adult; *O*=old adult; *SAB*=South African Black; *SAC*=South African Coloured; *SAW*=South African White; *E*=Early era; *M*=Mid era; *L*=Late era; *F*=Female; *M*=Male.

^o $p < 0.1$

Each bone element showing either sclerotic, lytic and/or both lesions, were plotted on a graph displaying the pelvis with the highest prevalence of neoplastic lesions, followed by the vertebrae and cranium (Fig. 4.33). A comparison of different types of lesions per sex (Fig.

4.34), show males to have a slightly higher prevalence of lytic lesions, but females have a significantly higher prevalence of mixed lesions and no sclerotic lesions. When the type of lesion was compared to the registered COD, expected lytic lesions were seen for lung carcinomas and mixed lesions for breast cancers, although uncharacteristically, high sclerotic lesions were seen for cervix cancers and high mixed lesions were seen in pulmonary cancers. Gastrointestinal cancers did not show much metastatic lesions on the bone and many of the pulmonary cancers also did not show metastatic lesions visible on bones (Fig. 4.35).

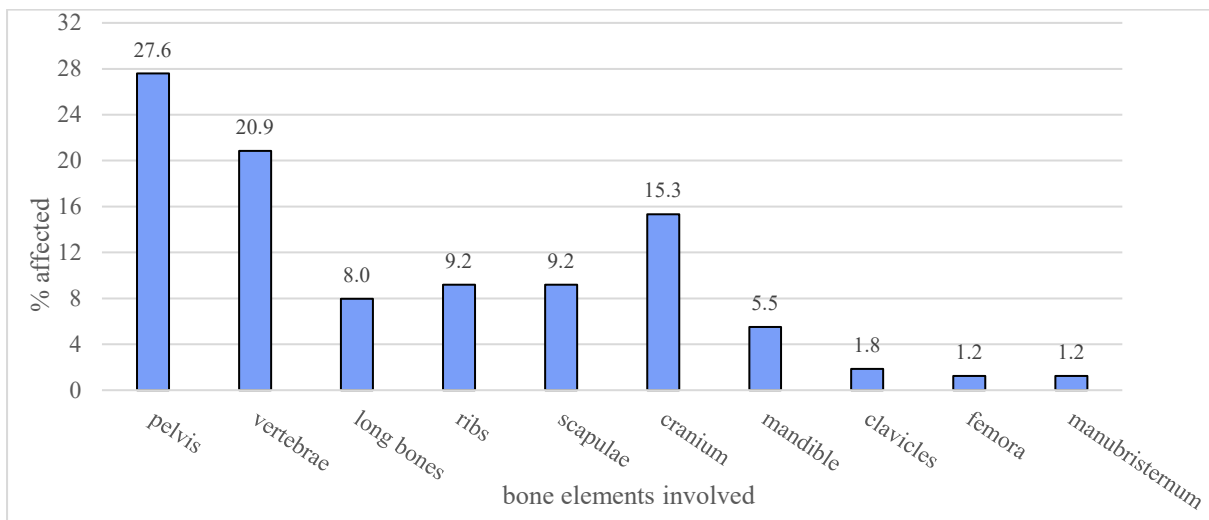


Figure 4.33. Lesions observed on various bone elements in the cohort (%).

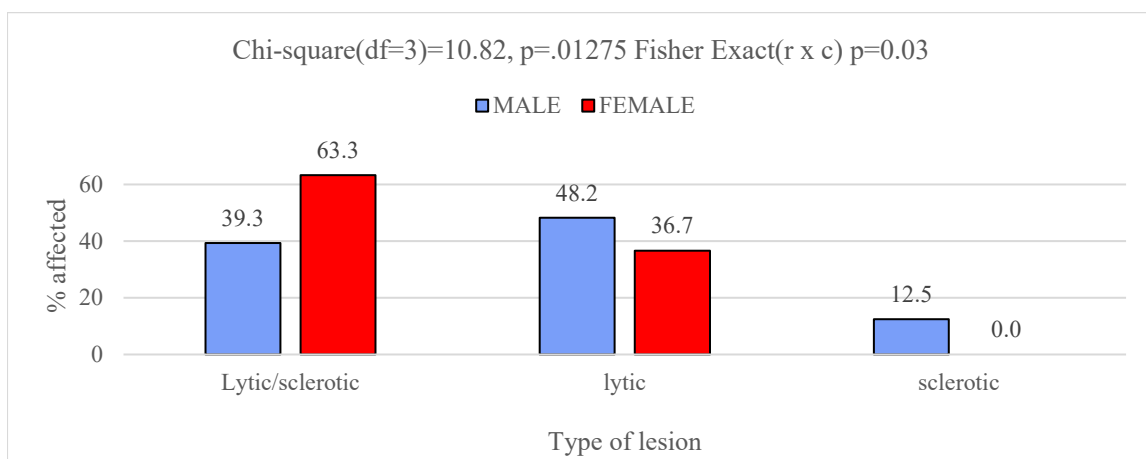


Figure 4.34. Type of lesions observed per sex in the cohort (%).

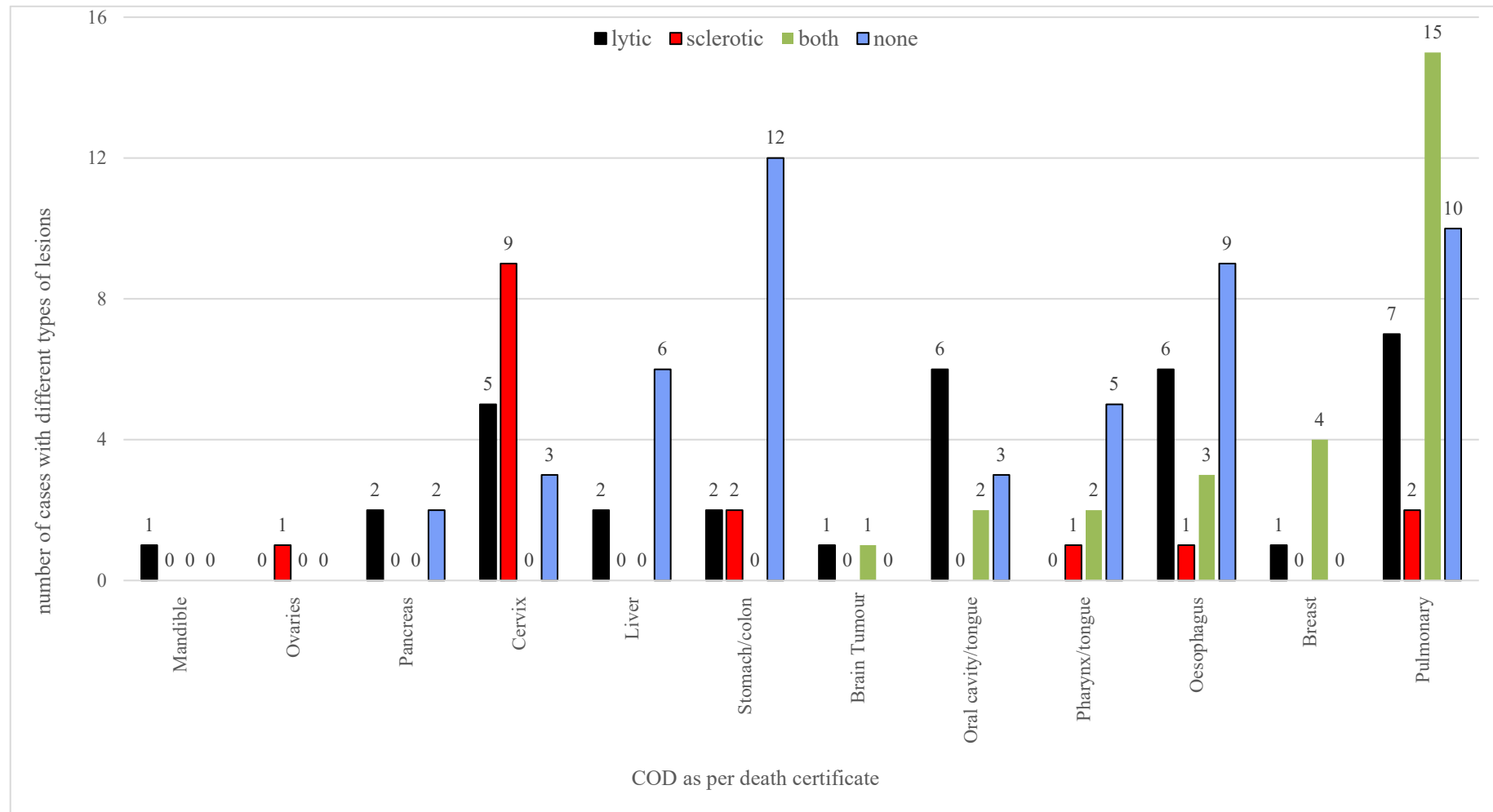


Figure 4.35. Type of lesions observed per bone element compared to COD reported on the death certificates.

4.1.6 OTHER DISEASES/CONDITIONS

Other conditions observed in low numbers on the skeletons included hyperostosis frontalis interna (1.8%), Paget's disease (0.5%; n=3), hypertrophic osteoarthropathy (10.4%) and one case with bone infarctions (0.3%).

Table 4.15. Statistical analyses of HFI and HOA lesions on individuals in the KSC cohort.

Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total number
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p	
	(n=2)	(n=2)	(n=6)		(n=1)	(n=6)	(n=3)		(n=4)	(n=2)	(n=4)		(n=7)	(n=3)		
HFI	1.6	0.7	4.3	*0.03	1.1	1.6	4.0	0.31	8.7	0.8	1.6	†<0.01	4.3	0.8	†<0.01	n=10
	(n=7)	(n=39)	(n=17)		(n=11)	(n=45)	(n=7)		(n=6)	(n=37)	(n=20)		(n=18)	(n=45)		
HOA	4.8	12.2	10.8	*0.02	10.8	10.3	8.4	0.85	9.1	13.3	7.2	0.05	9.7	10.3	0.82	n=63

HFI=Hyperostosis frontalis interna; HOA=hypertrophic osteoarthropathy

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

°p<0.1; *p<0.05; †p<0.01

4.1.6.1 Hyperostosis frontalis interna (HFI)

A total of 541/624 individuals (86.7% of cohort) were examined for signs of HFI on the endocranial frontal bones. Ten cases (1.8%) of HFI were observed (Table 7.29; Appendix 1), with a significantly higher incidence rate in females than males (p<0.01) (Table 4.15). The ages of affected individuals ranged from 31 to 79 years, with a mean age of 56.8 years (SD:17.2). The old-adult age group showed a significantly higher prevalence of HFI than the other age groups (p=0.03). When comparing time periods, the early period showed a significantly higher frequency of HFI than the other periods (p<0.01). No significant difference was observed among population groups.

4.1.6.2 Paget's disease

Signs of Paget's disease of the bone (osteitis deformans) were observed in 3/624 skeletons (0.5% of cohort) (Table 7.30; Appendix 1). Diagnostic criteria comprised hyperostosis of the vault and long bones, as well as unorganised proliferation of other bones, seen radiologically as a 'cotton wool' appearance (Fig. 4.36A-F). No cases of Paget's disease were observed in the young-adult age group and only one case in the mid-adult age group. The two cases from the old-adult age group were both in their 6th decade of life. One individual was from the early time period, while two skeletons were from the mid-20th century. One male and two females were affected. All three affected individuals were from the SAC population group.



Photo by A Alblas (AN 215)



Lodox[®] by J Walters (AN 215)



Photo by A Alblas (AN 215)



Lodox[®] by J Walters (AN 215)

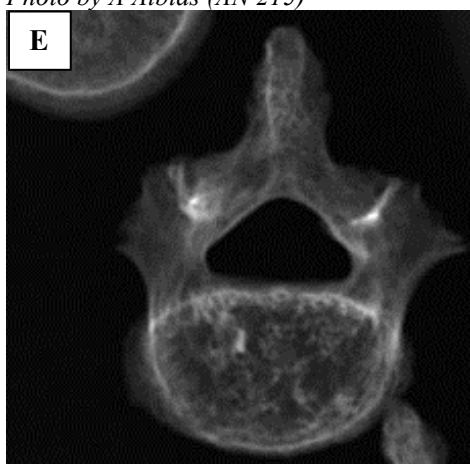
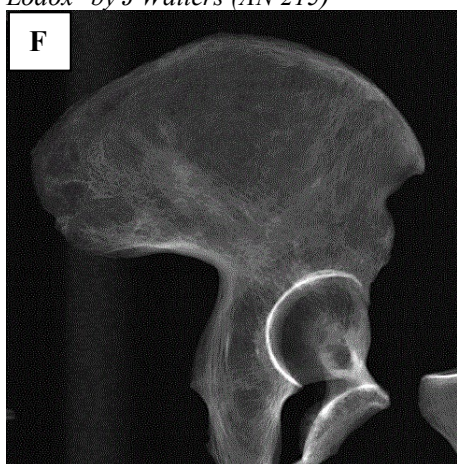


Photo by A Alblas (AN 215)



Lodox[®] by J Walters (AN 215)

Figures 4.36A-F. Signs of Paget's disease on dry bone: A) hyperostosis of calvarium; B) Lodox[®] image; C) disorganised, densely woven bone; D) Lodox[®] image; E) 'cotton wool' effect on a radiological image; and F) disorganised woven bone deposits on a radiological image.

4.1.6.3 Hypertrophic osteoarthropathy (HOA)

Symmetrical periosteal reactions – nodular, striated or irregular – on the tubular bones, as associated with OA of peripheral joints, were regarded as a sign of HOA. Other bones, such as the os coxae, scapulae and ribs, were inspected in a similar manner for systemic periosteal reactions. This condition was observed in 63/603 individuals (10.4% of the cohort) (Table 7.31; Appendix 1). The average age at death of individuals with HOA was 53.6 years (SD:11.4). No significant differences were noted between genders or among population groups; however, the young-adult age group showed a significantly lower prevalence of HOA than the mid- and old-adult groups ($p=0.02$) (Table 4.15). A slightly higher rate of HOA cases were seen in the mid-20th century group, compared to the early and late time periods ($p=0.05$). Extensive involvement of the entire skeleton (excluding the skull) was observed in one individual (AN 1197, male, 60 years). The skeletal elements most affected were the fibulae, followed by the tibiae and the tibiofibular joints (Fig. 4.37).

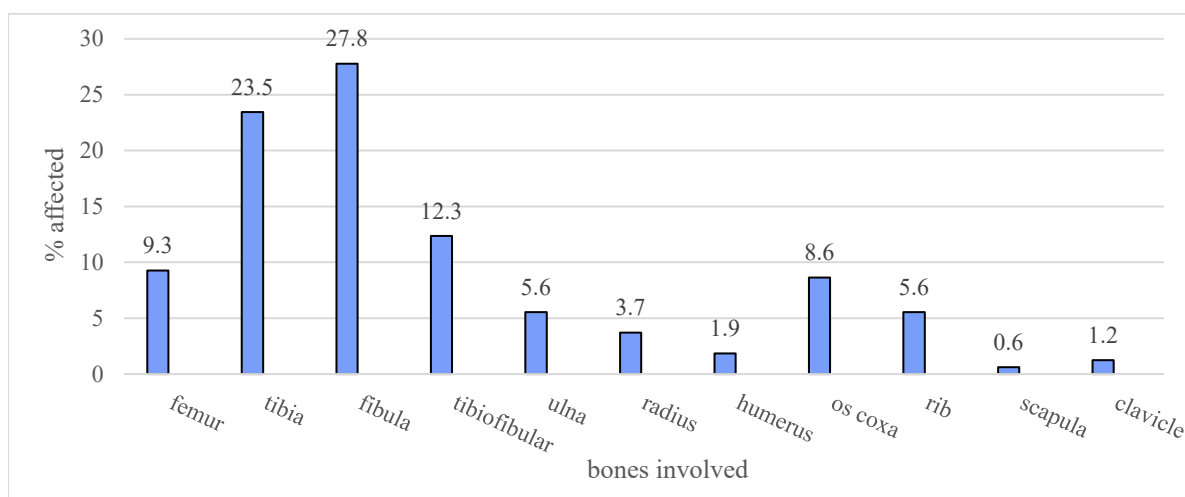
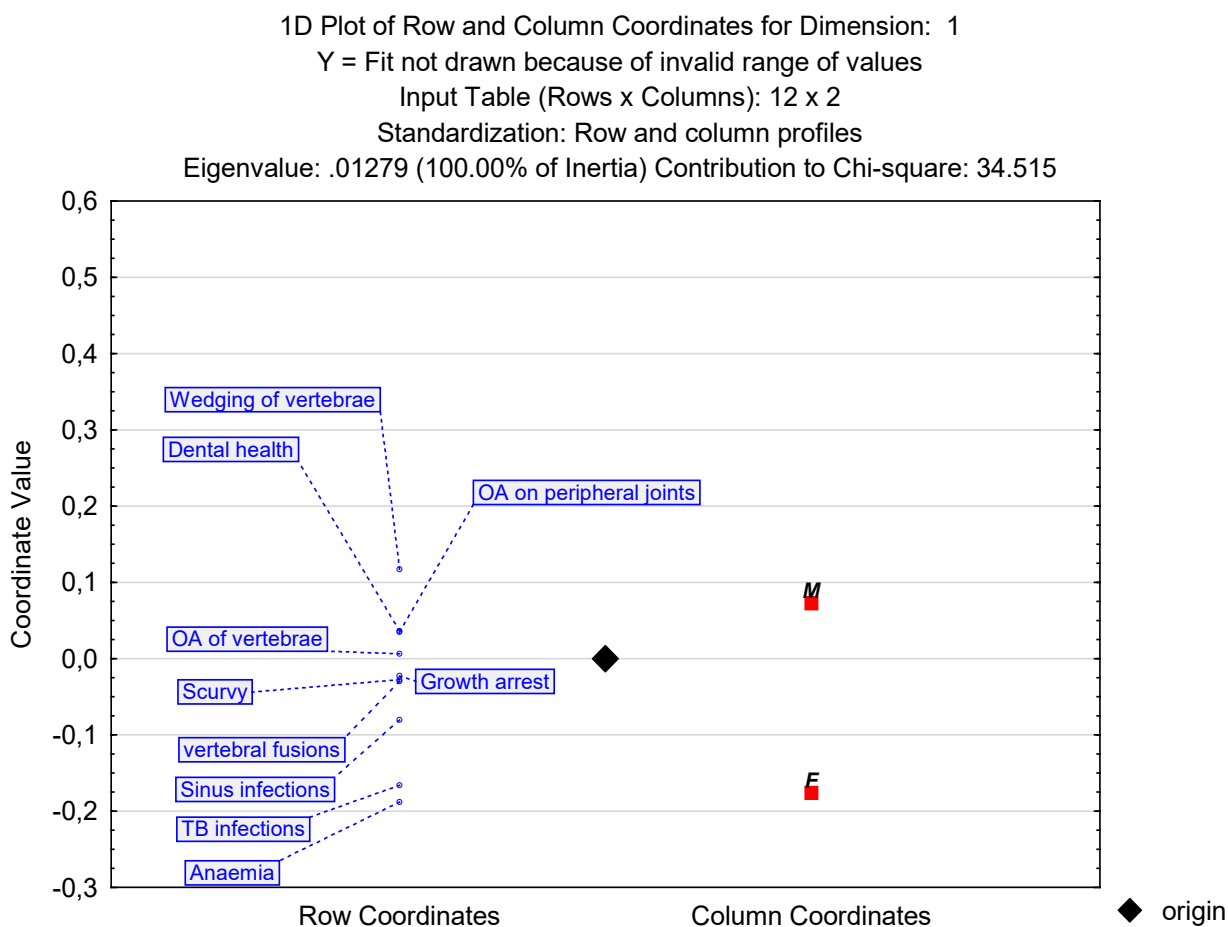


Figure 4.37. The number of skeletal elements with signs of HOA in this cohort.

4.2 CORRESPONDENCE ANALYSES

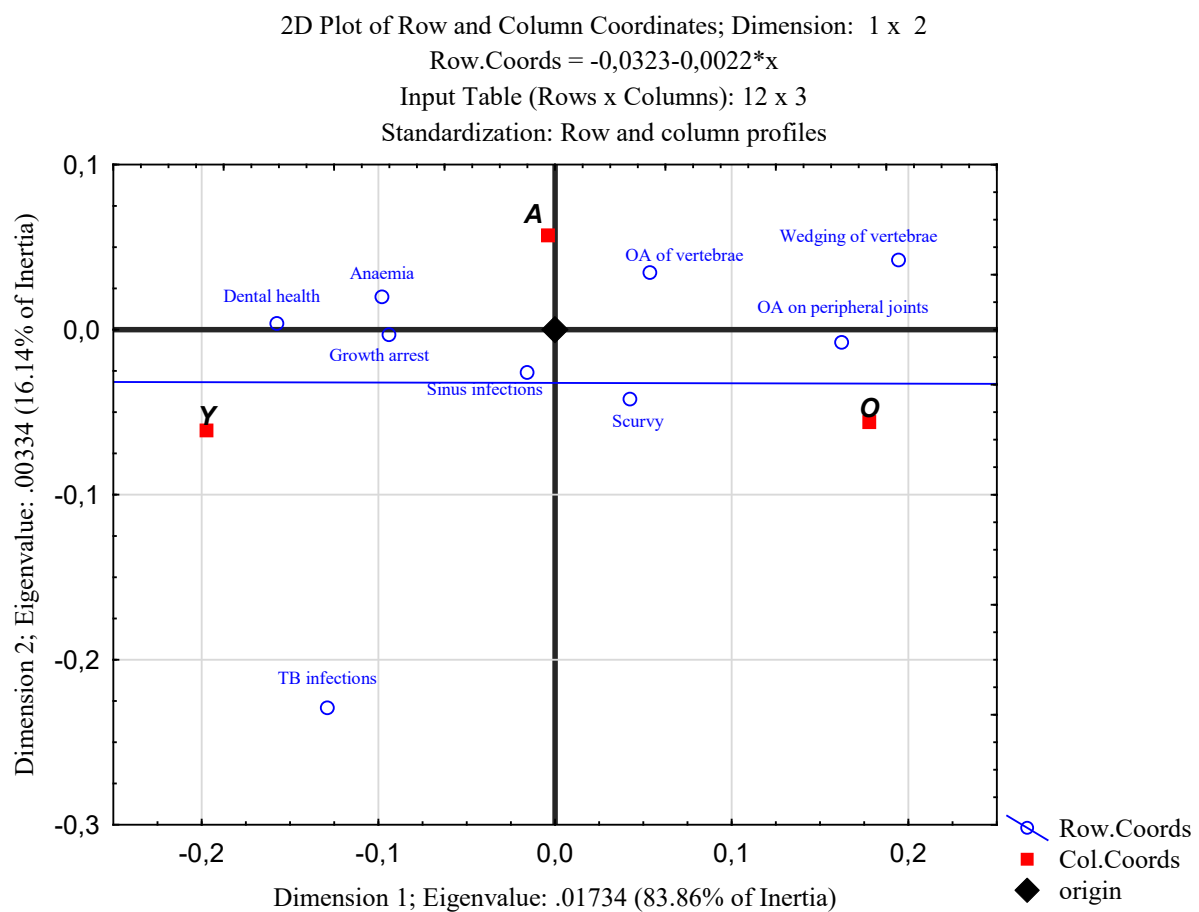
Correspondence analyses were plotted on contingency tables to determine correlations between the various broadly-categorised pathology groups observed in the KSC cohort and sex, age group, population group and time period, respectively. Pathology lesions observed were grouped as signs of: growth arrest, iron deficiency anaemias, osteomalacia (rickets), scurvy, dental health problems, infections (TB and localised infections), and degenerative diseases, further categorised as OA on the peripheral joints and vertebral joint diseases (OA, ankylosed and wedged vertebrae).



OA= osteoarthritis

Figure 4.38. Correspondence analysis of sex (male and female) correlated to broadly categorised pathology groups.

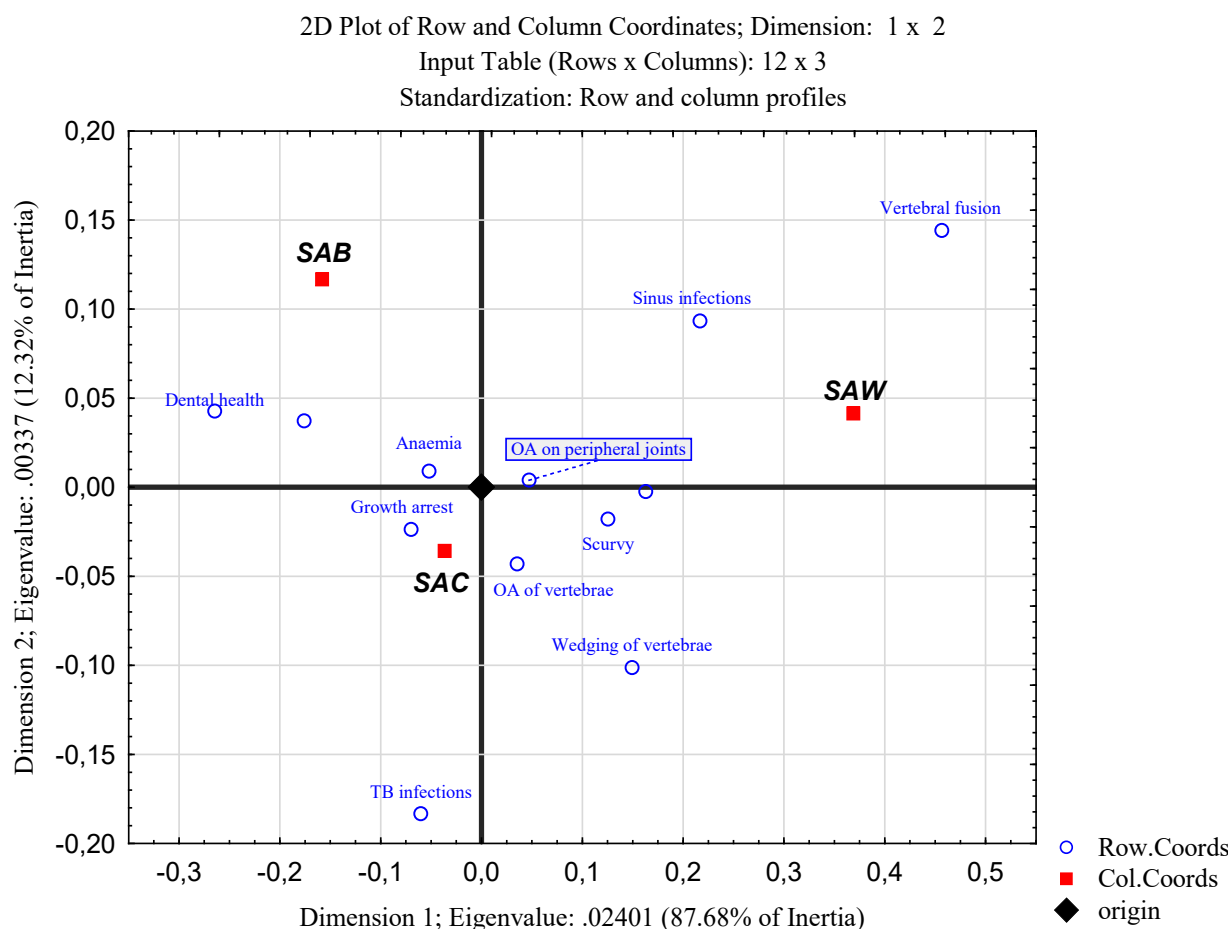
A correspondence analysis for the grouped pathological lesions seen on the skeletal elements revealed associations with both sexes (Fig. 4.38). Although most pathology lesions were associated with both sexes, stronger correlations were observed between females and anaemia, TB infections and localised cranial infections, respectively. On the other hand, osteoarthritic disease markings, such as peripheral joint OA and wedging of vertebrae, were more associated with males than females. Generally, poor dental health was also more frequent among males than females.



OA= osteoarthritis; Row.Coord=row coordinates; Col.Coord= column coordinates

Figure 4.39. Correspondence analysis of age groups (young adult, mid-adult and old adult) correlated to broadly categorised pathology groups.

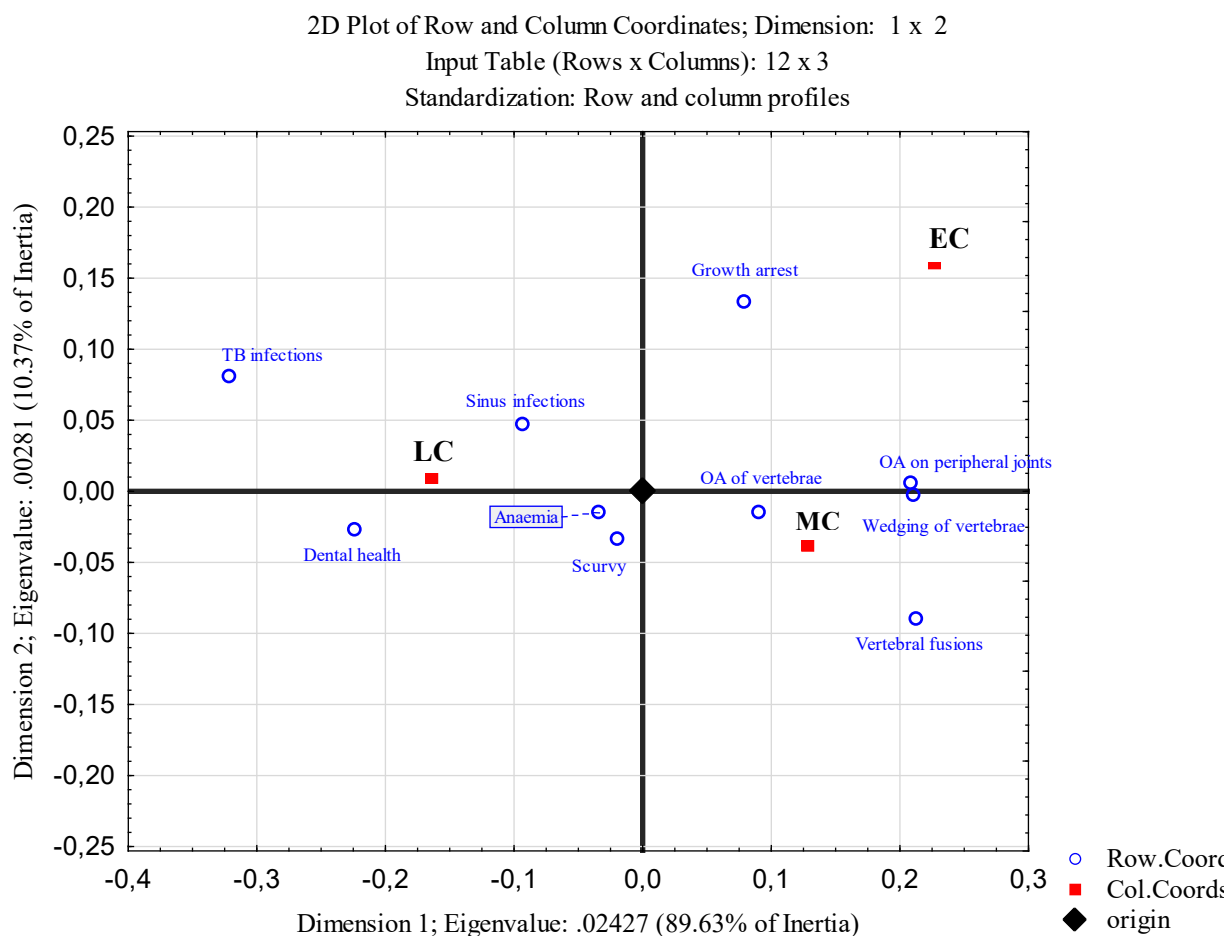
A correspondence analysis for the grouped pathologies seen on skeletal elements was plotted against the three age groups, namely the young adult group (Y=20-39 years), the mid-adult group (A=40-59 years), and the old adult group (O=60 years and older) (Fig. 4.39). Most of the pathology groups were moderately associated with all three population groups; however, skeletal lesions from TB were significantly associated with the young group, while fused vertebrae were more common in the old age group than in the other groups. Wedging was only seen on the vertebral bodies of mid-adult and old-adult skeletons. Osteoarthritis on the peripheral joints correlated closest to old age, while the young-adult age group showed a negative association with this condition. Poor dental health was associated with the young and mid-adult age groups, but negatively correlated with the old group as many of them did not have teeth to compare.



OA= osteoarthritis; Row.Coord=row coordinates; Col. Coord= column coordinates

Figure 4.40. Correspondence analysis of population groups (SAB, SAC and SAW) correlated to broadly categorised pathology groups.

A correspondence analysis plotted the grouped pathologies on the skeletal elements against the three population groups included in the cohort, namely the South African Black (SAB), South African Coloured (SAC) and South African White (SAW) population groups (Fig. 4.40). Most of the pathologies showed an intermediate correlation with all three population groups, although vertebral fusion and local infections were highly associated with the SAW group, TB infections and dental health problems showed a negative correlation with this group. On the other hand, the SAC population group was highly correlated with TB infections as well as growth arrest lines, but negatively associated with vertebral fusion. The SAB group, in contrast, showed a high affinity for dental health problems, but not for vertebral wedging.



OA= osteoarthritis; Row.Coord=row coordinates; Col. Coord= column coordinates

Figure 4.41. Correspondence analysis of 20th century time periods (Early, Mid and Late) correlated to broadly categorised pathology groups.

A correspondence analysis for the grouped pathologies observed on the bones of the cohort was plotted against the three time periods of the 20th century, namely the early era (EC=1928-1940), the mid era (MC=1941-1956), and the late era (LC=1957-1995) (Fig. 4.41). Most of the pathology groups were intermediately correlated to all the time periods. There were, however, some specific associations, such as dental health problems that were highly associated with the late era, but negatively correlated to the early time period. On the other hand, rickets (osteomalacia) correlated with the late and mid-eras, but had a significant negative association with the early time period. In contrast, growth arrest lines were highly associated with the early period but had the weakest correlation to the late period. Vertebral fusion positively correlated with the mid-era and negatively correlated with the early era.

4.3 TRAUMA MARKINGS ON BONE

All trauma lesions observed on individuals in the KSC cohort are summarised in Table 7.32, Appendix 1.

4.3.1 CRANIAL TRAUMA

Some individuals in the cohort did not have all cranial elements present. A total of 536/624 (85.6%) calvaria, 549/624 (88.0%) crania and 525/624 (84.1%) mandibles were available for analyses.

Healed cranial and mandibular traumas were observed in 429/624 individuals (68.8% of cohort). The highest frequency of fractures in the cranial bones was observed in the nasal bones (31.0%), followed by the maxilla (14.6%), and the zygoma (10.9%). The cranial bone with the lowest frequency of fractures was the occipital bone, with only one SAC individual (0.0%) presenting with a blunt blow on the posterior occipital bone. The left side showed a higher prevalence of trauma signs than the right side for all bones, except the parietal and temporal bones where the right side displayed a higher rate of trauma. Healed trauma was highly prevalent on both nasal bones. The maxilla showed similar rates of bilateral trauma and trauma on the left side only (41.7% vs. 41.8%), but a lower frequency of trauma on the right side only (16.5%) (Table 4.16). The mandible showed signs of trauma in 3.0% of the sample, with the left side slightly more affected than the right side.

Table 4.16. Frequency (n) of antemortem cranial trauma per bone, side and type of trauma.

	Female			Male			Total			Total fx	% fx	Total number
	L	R	L,R	L	R	L,R	L	R	L,R			
Frontal	7	3	1	21	14	2	28	17	3	48	9.0	536
Parietal	2	4	0	17	21	2	19	25	2	46	8.6	536
Temporal	0	0	0	3	4	0	3	4	0	7	1.3	549
Zyg arch	5	4	2	25	15	19	30	19	11	60	10.9	549
Maxilla	9	4	3	25	9	30	34	13	33	80	14.6	549
Nasal	2	6	32	15	9	106	17	15	138	170	31.0	549
Mandible	2	1	0	8	5	0	10	6	0	16	3.0	525

L=left; Right=right; L,R=both sides; Zyg arch=zygomatic arch; fx= fracture

The occurrence of sharp force trauma (SFT) or pathological fractures was insignificant (less than 2% in all bones) compared to blunt force trauma (BFT).

No significant differences in the frequency of trauma lesions in the cranial bones were observed among age groups (Table 4.17). Although males showed a higher prevalence of trauma markings on all cranial bones than females, these differences were not statistically significant, except in the case of the zygomatic arches and maxillae, where a weak significance was noted ($p=0.02$) and the parietal and nasal bones where a difference of strong significance between the sexes was seen ($p<0.01$). Males and females from the SAC population group displayed the highest rate of cranial trauma, with the exception of the parietal bone where a slightly higher trauma rate was observed in the SAB male population group. The SAW population group had the lowest trauma rate on all cranial bones. The zygomatic arches and the nasal bones were significantly more affected in the SAC population group ($p<0.01$). A difference in trauma rates (weakly significant) among the three time periods was seen on the zygomatic arches, with a greater incidence rate in the late era than the other time periods ($p=0.09$). In the case of the nasal bones, significantly less trauma was seen in the early period than in the other periods ($p<0.01$).

Table 4.17. Statistical analyses of trauma lesions on cranial elements in the KSC cohort.

Trauma Trait	Age Group (%)			<i>p</i>	Population Group (%)			<i>p</i>	Time Period (%)			<i>p</i>	Sex (%)			<i>p</i>
	Y	A	O		SAB	SAC	SAW		EC	MC	LC		F	M		
frontal	(n=10) 8.2	(n=26) 9.5	(n=12) 8.6	0.90	(n=6) 7.7	(n=36) 9.4	(n=6) 8.0	0.84	(n=4) 8.9	(n=23) 9.7	(n=21) 8.3	0.87	(n=11) 6.5	(n=37) 10.1	0.20	n= 48
parietal	(n=10) 8.3	(n=23) 8.4	(n=13) 9.4	0.93	(n=6) 7.7	(n=38) 10.0	(n=2) 2.7	0.10	(n=4) 8.9	(n=20) 8.5	(n=22) 8.7	1.00	(n=6) 3.6	(n=40) 11.0	†<0.01	n= 46
temporal	(n=0) 0.0	(n=5) 2.0	(n=2) 1.6	0.46	(n=2) 2.9	(n=5) 1.4	(n=0) 0.0	0.32	(n=1) 2.9	(n=3) 1.4	(n=3) 1.3	0.57	(n=0) 0.0	(n=7) 2.1	0.10	n= 7
zygoma	(n=7) 6.5	(n=37) 14.6	(n=16) 12.8	0.73	(n=4) 5.8	(n=54) 15.5	(n=2) 2.9	†<0.01	(n=2) 5.7	(n=21) 9.7	(n=37) 15.7	0.09	(n=11) 7.1	(n=49) 14.8	*0.02	n= 60
maxilla	(n=13) 12.0	(n=49) 19.4	(n=18) 14.4	0.17	(n=8) 11.6	(n=64) 18.4	(n=8) 11.6	0.17	(n=3) 8.6	(n=31) 14.4	(n=46) 18.6	0.16	(n=16) 10.4	(n=64) 19.3	*0.02	n= 80
nasal	(n=34) 31.5	(n=95) 37.6	(n=41) 32.8	0.45	(n=15) 21.7	(n=143) 41.1	(n=12) 17.4	†<0.01	(n=5) 14.3	(n=84) 38.9	(n=81) 34.5	†0.01	(n=40) 26.0	(n=130) 39.2	†<0.01	n=170
mandible	(n=3) 2.5	(n=9) 3.4	(n=4) 3.0	0.95	(n=1) 1.3	(n=15) 4.1	(n=0) 0.0	0.14	(n=0) 0	(n=9) 3.8	(n=7) 2.9	0.55	(n=3) 1.8	(n=13) 3.6	0.41	n= 16

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

° $p<0.1$; * $p<0.05$; † $p<0.01$

One 56-year-old SAW male presented with multiple traumas to the skull, including a depression fracture to the left parietal and left zygomatic arches, the nasal bones and the anterior aspect of both maxillary bones. Four SAB males presented with extensive facial

traumas, including multiple traumas to the parietal, zygomatic, maxillary and nasal bones. A total of 48 SAC males showed multiple cranial traumas, with combinations of involvement of the parietal, temporal, zygomatic, nasal, maxillary, and mandible bones. When examining extensive cranial traumas on female cranial bones, one SAW female showed multiple traumas on the nasal, zygomatic, and maxillary bones, while eight females showed signs of trauma to the nasal, zygomatic, parietal and maxillary bones. No SAB female showed traumas to multiple cranial bones.

4.3.2 POST-CRANIAL TRAUMA

4.3.2.1 General post-cranial lesions

A total of 624 individuals were analysed for antemortem trauma markings on the post-cranial bones, including the long bones (humeri, ulnae, radii, femora, tibiae, fibulae, clavicaulae), scapulae, ribs, and ossa coxae. The prevalence (%) of the trauma lesions on each bone is indicated in Fig. 4.42. The ribs demonstrated the highest rate of injury (39.8%), followed by the ulnae (17.7%), and fibulae (12.0%). The least injured skeletal elements were the scapulae (1.5%).

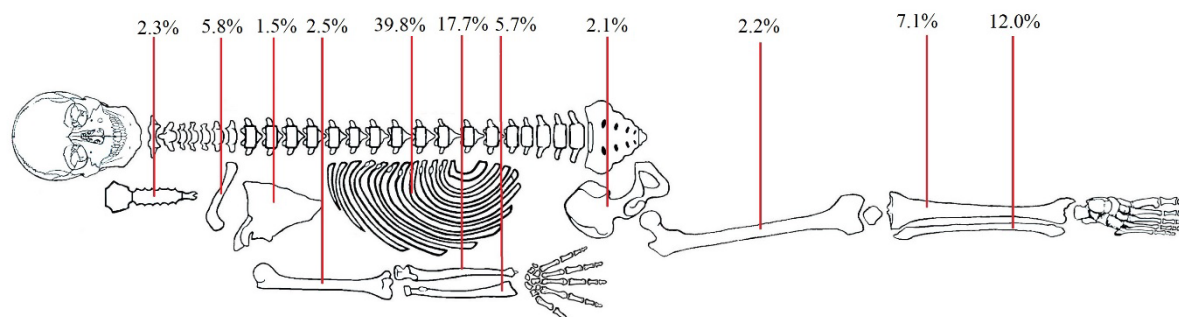


Figure 4.42. Prevalence (%) of post-cranial trauma observed per bone in KSC cohort.

The number of post-cranial trauma lesions were compared between sexes, and among different population groups, age groups, and time periods, respectively (Table 4.18).

Table 4.18. Trauma per bone (%) in different age groups, population groups, eras and sexes.

Trait	Age Group (%)				Population Group (%)				Time Period (%)				Sex (%)			Total Number
	Y	A	O	p	SAB	SAC	SAW	p	EC	MC	LC	p	F	M	p	
clavicle	(n= 4) 2.7	(n=20) 6.3	(n= 8) 5.1	0.45	(n= 6) 5.9	(n=22) 5.0	(n= 4) 4.8	0.55	(n= 6) 9.1	(n=15) 5.4	(n=11) 3.9	0.41	(n=9) 4.8	(n=23) 5.3	0.82	n=32
humerus	(n= 4) 2.7	(n= 8) 2.5	(n= 2) 1.3	0.57	(n= 1) 1.0	(n=12) 2.7	(n= 1) 1.2	0.28	(n= 0) 0.0	(n=10) 3.6	(n= 4) 1.4	†<0.01	(n=4) 2.2	(n=10) 2.3	0.65	n=14
ulna	(n= 9) 8.2	(n=30) 17.2	(n=20) 20.9	†<0.01	(n= 8) 14.7	(n=36) 18.7	(n=14) 3.6	†<0.01	(n= 8) 19.7	(n=21) 16.5	(n=29) 14.7	0.53	(n=18) 18.8	(n=40) 14.8	0.44	n=58
radius	(n=11) 4.1	(n=27) 4.7	(n=20) 7.0	0.23	(n= 8) 3.9	(n=39) 6.2	(n=12) 1.2	0.18	(n= 8) 4.6	(n=22) 6.8	(n=29) 3.6	0.34	(n=18) 7.5	(n=41) 4.1	0.22	n=59
femur	(n= 1) 0.7	(n= 7) 2.2	(n= 4) 13.3	0.56	(n= 0) 0.0	(n=10) 2.3	(n= 2) 2.4	0.14	(n= 1) 1.5	(n= 4) 1.4	(n= 7) 2.5	0.33	(n= 4) 2.2	(n= 8) 1.8	0.89	n=12
tibia	(n= 8) 5.5	(n=22) 6.9	(n=10) 6.3	0.92	(n= 8) 7.8	(n=28) 6.4	(n= 4) 4.8	0.26	(n= 2) 3.0	(n=16) 5.7	(n=22) 7.9	0.29	(n= 8) 4.3	(n=32) 7.3	0.35	n=40
fibula	(n= 12) 8.2	(n=36) 11.3	(n=18) 11.4	0.17	(n=14) 13.7	(n=49) 11.2	(n= 3) 3.6	†0.01	(n= 6) 9.1	(n=27) 9.7	(n=33) 11.8	0.53	(n=14) 7.5	(n=52) 11.9	0.24	n=66
scapula	(n= 3) 2.1	(n= 2) 0.6	(n= 3) 1.9	0.16	(n= 3) 2.9	(n=4) 0.9	(n= 1) 1.2	‡0.06	(n= 0) 0.0	(n= 4) 1.4	(n= 4) 1.4	†<0.01	(n=0) 0.0	(n=8) 1.8	0.17	n= 8
os coxa	(n= 2) 1.4	(n= 5) 1.6	(n= 4) 2.5	0.66	(n= 0) 0.0	(n= 9) 2.1	(n= 2) 2.4	0.13	(n= 1) 1.5	(n= 3) 1.1	(n= 7) 2.5	†<0.01	(n= 2) 1.1	(n= 9) 2.1	0.57	n=11

Y=young adult; A=mid adult; O=old adult; SAB=South African Black; SAC=South African Coloured; SAW=South African White; EC=Early era; MC=Mid era; LC=Late era; F=Female; M=Male.

‡p<0.1; *p<0.05; †p<0.01

In general (Table 4.18), males demonstrated a higher rate of antemortem trauma lesions on all bone elements, except the arms (humerus, ulna and radius). The young-adult age group had the lowest rate of post-cranial trauma on all bone elements, with the exception of the humerus and scapula, for which this age group showed the highest frequency of fractures. For all other bone elements, except the clavicle and tibia, the old adult group showed the highest frequency of callus formation due to accumulation of lesions throughout the individuals' lives. In the cases of the clavicle and tibia, the mid-adult group displayed the highest rate of fractures. On the whole, the SAW population group showed the lowest fracture rate on their post-cranial bones. However, in the cases of the os coxa and sternum, this group showed the highest fracture rate. With regards to the femur, equal fracture rates were seen in the SAW and SAC population groups and no fractures in the SAB population group. In the cases of the shoulder (clavicle and scapula) and lower limbs (tibia and fibula) the highest fracture rate was seen in the SAB group, while the SAC population group demonstrated the highest fracture rate in the arms (humerus, ulna, radius). Defence fractures (forearm) were more prevalent in the early time period, while trauma to the pelvis and lower limbs were more prevalent in the late era. There were no humerus or scapula fractures during the early era, while the mid-era showed a significantly

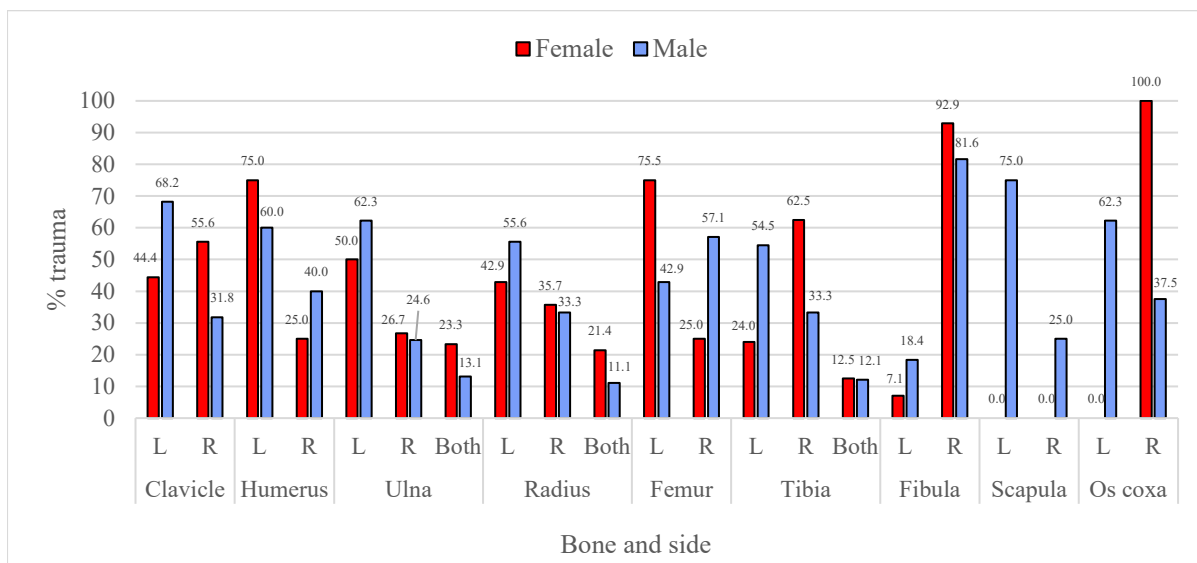
higher rate of humeral fractures than the other time periods ($p < 0.01$). A higher rate of radial fractures was also seen for the mid-era, although not statistically significant.

Table 4.19. Trauma per post-cranial bone (%) compared between sexes and among population groups.

(%)	Clavicle	Humerus	Ulna	Radius	Femur	Tibia	Fibula	Scapula	Os coxa	Sternum
Male SAB	n=5 6.0	n=1 1.2	n=12 14.3	n=3 3.6	n=0 0	n=7 8.3	n=13 15.5	n=3 3.6	n=0 0	n=0 0
Female SAB	n=1 5.6	n=0 0	n=3 16.7	n=1 5.6	n=0 0	n=1 5.6	n=1 5.6	n=0 0	n=0 0	n=0 0
Male SAC	n=14 4.6	n=8 2.6	n=50 16.5	n=14 4.6	n=6 2.0	n=21 6.9	n=36 11.9	n=4 1.3	n=8 2.6	n=8 2.6
Female SAC	n=8 5.9	n=4 2.9	n=32 23.5	n=13 9.6	n=4 2.9	n=7 5.1	n=13 9.6	n=0 0	n=1 0.7	n=1 0.7
Male SAW	n=4 7.8	n=1 2.0	n=3 5.9	n=1 2.0	n=2 3.9	n=4 7.8	n=3 5.9	n=1 2.0	n=1 2.0	n=1 2.0
Female SAW	n=0 0	n=0 0	n=0 0	n=0 0	n=0 0	n=0 0	n=0 0	n=0 0	n=1 3.1	n=1 3.1
Total	n=32	n=14	n=100	n=32	n=12	n=40	n=66	n=8	n=11	n=11

SAB=South African Black; SAC=South African Coloured; SAW=South African White

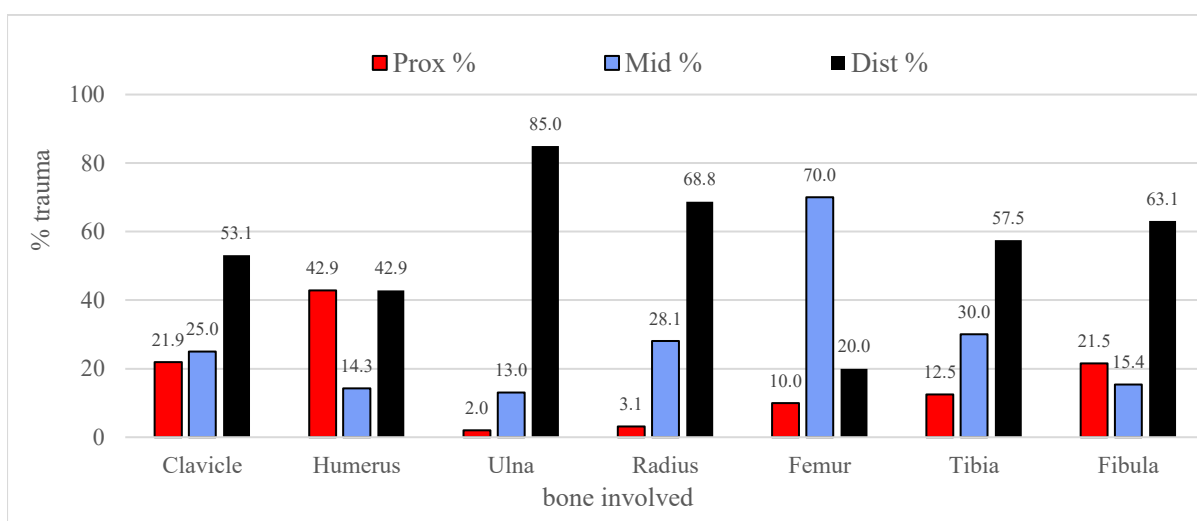
The SAW females had a lower antemortem trauma rate than any of the other sex and population groups (Table 4.19). There was higher prevalence of ulnar fractures in the SAC population group than in the SAB and SAW population groups, with ulnar fractures being especially prevalent among SAC females (Table 4.19). Femur and pelvis fractures were most prevalent in the SAW male population group, while shoulder and lower limb injuries were most prevalent in the SAB population group. A strong association was observed between fibular fractures and this population group, while a weak correlation was seen in the case of the scapular fractures. However, when the antemortem trauma rate per sex was compared among population groups, the SAW males had the highest rate of clavicular fractures, while such fractures were completely absent among SAW females (Table 4.19). Sternal trauma was mainly seen in the SAW population, including both males and females.



L=left; R=right, Both=both sides

Figure 4.43. Frequency (%) of post-cranial trauma observed per side, sex and bone in KSC cohort.

A comparison of post-cranial trauma between the two sides per sex for each long bone involved (Fig. 4.43), indicates a higher frequency of trauma on the left side in most of the post-cranial bones. No cases of scapular trauma were seen among females. Trauma to the fibula was biased to the right side for both sexes. Females showed a higher prevalence of right-side fractures in the clavicle, tibia and os coxa, while males displayed a higher trauma rate on the right side in the case of the femur, and on the left side in the case of the tibia.



Prox=proximal third of long bone; Mid=middle third of long bone; Dist=distal third of long bone.

Figure 4.44. Percentage post-cranial trauma observed per long bone section.

When comparing the three sections per long bone, namely the proximal third, mid-third and distal third, involved in trauma (Fig. 4.44), the distal third of all the bone shafts had a higher rate of healed trauma; however, the femur showed more midshaft fractures and the humerus showed the same involvement for the proximal and distal parts of the shaft.

Table 4.20. Trauma complication (n) per post-cranial bone.

Trait	Skeletal element (n)						total	total %
	deform	fusion	infect.	non-union	surg.	traum arthr.		
Clavicle	5	0	0	2	0	5	12	2.3
Humerus	1	0	2	0	0	3	6	1.1
Ulna	1	1	3	2	1	4	12	2.6
Radius	0	2	1	0	0	1	4	0.8
Femur	0	1	0	0	5	3	9	1.7
Tibia	1	6	3	0	3	0	13	2.5
Fibula	0	6	4	0	1	0	11	2.3
Scapula	2	0	0	1	0	2	5	0.9
Os coxa	7	2	0	0	0	2	11	2.1

deform=bone not aligned properly after the fracture; fusion= fusion of two bone elements after excessive remodelling of a fracture; infect.= infective periostitic response after trauma; non-union= parts of bones missing/damaged, where the fracture failed to heal; surg= orthopaedic wires, screws, plates and implants; traum.arthr=dislocation, traumatic arthritis.

Trauma complications of post cranial elements after a fracture were uncommon and only a small number of individuals presented with complications after a traumatic incident (Table 4.18).

4.3.2.2 Rib fractures

Some individuals in the cohort did not have all their ribs present, but 616/624 (98.7%) had at least one rib present. The mean number of ribs present in the cohort was 13.6 of the possible 24 (SD:7.06) (Fig. 4.45). The ribs present in the highest number were ribs #1 and #12, while ribs #3 to #6 were present in the lowest number (due to cremation of ribs by previous curators). Due to the missing ribs, the frequency of rib trauma could not be accurately determined. A total of 245/616 individuals (39.8% of individuals with at least one rib) had healed fractures on their ribs. Among these individuals, the mean number of ribs with trauma were 1.5 (SD:2.34). No signs of trauma were seen on ribs #1 and #3 on either side, while ribs #9 to #11 showed the highest frequency of healed fractures (Fig. 4.46). In 19 individuals more than 50% of their available ribs were fractured, while 44 individuals displayed trauma signs on between 30% and 49% of their ribs. The rest had signs of healed trauma on less than 30% of their available ribs. The maximum number of antemortem rib fractures in one individual was 12.

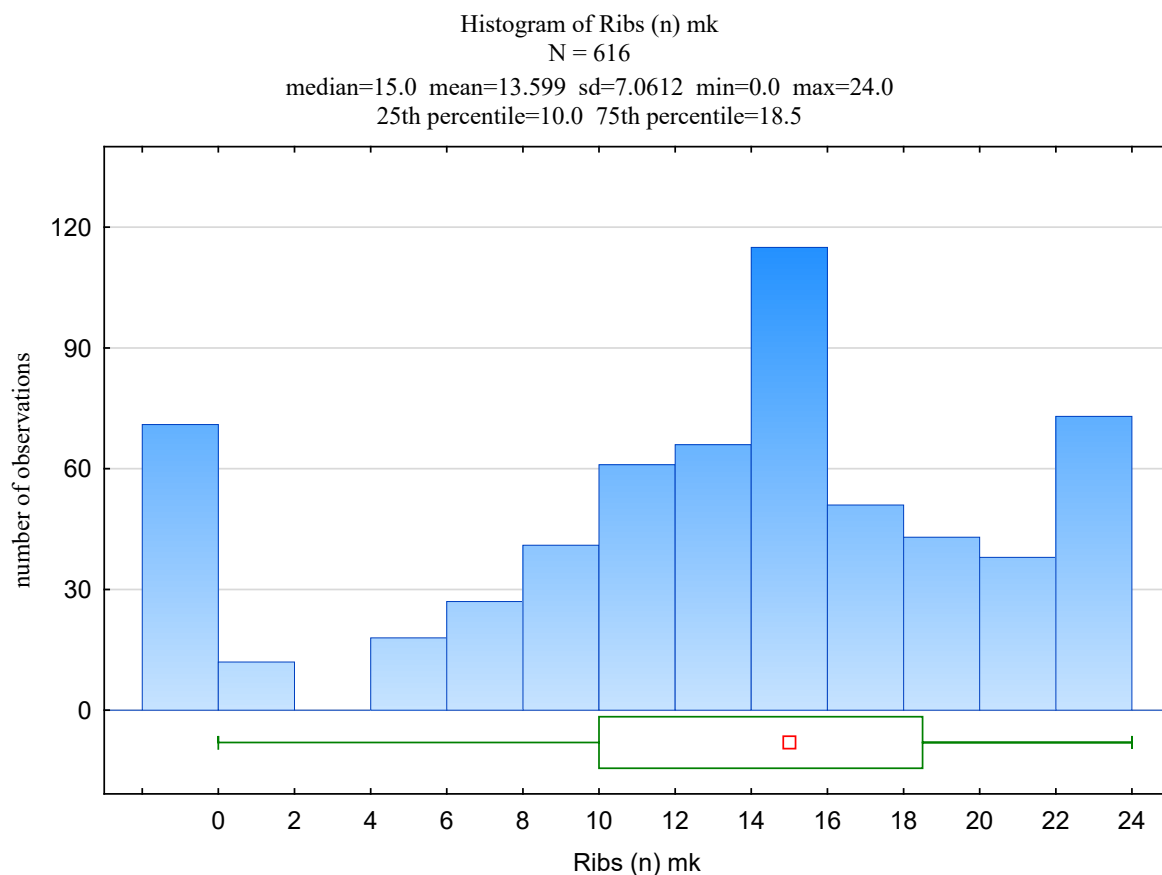
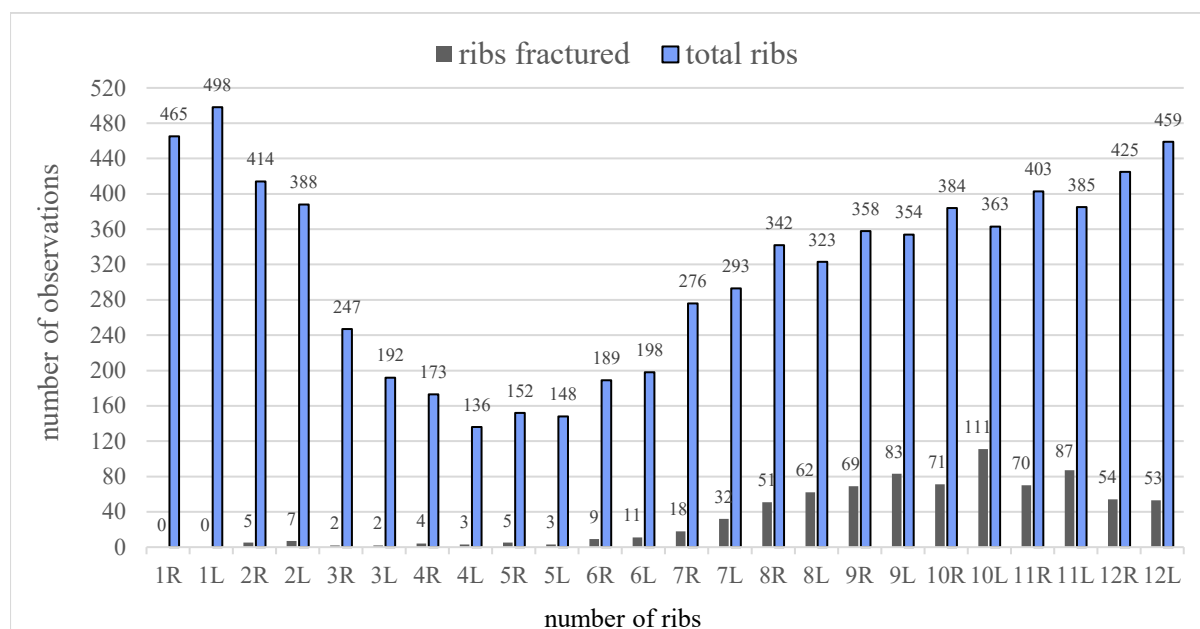


Figure 4.45. The number of ribs present in the cohort.



L=left; R=right

Figure 4.46. The number of ribs present and the number of healed fractures on the ribs.

4.4.3 TOTAL TRAUMA

A total of 448/624 individuals (71.8% of cohort) displayed signs of cranial and/or post-cranial trauma (Table 7.32, Appendix 1). The ribs showed the highest prevalence of antemortem trauma (39.8%), followed by the nasal bones (31.0%). The rest of the bones showed a much lower frequency of fractures. The occipital bone (0.0%) and the temporal bone (1.3%) showed the lowest trauma rates (Fig. 4.47). A comparison between the sexes showed that rib fractures were more common among females (41.3%), while nasal fractures were more common among males (39.2%). Cranial fractures were mostly a male phenomenon, while forearm (ulna and radius) fractures occurred mostly in females. No fractures were present on the female scapulae or the temporal bones (Fig. 4.48). The young-adult age group showed a slightly higher rate of scapula and humerus fractures, while the mid-adult and old-adult age groups showed a very high prevalence of nasal and ulnar fractures. Maxillary fractures were mainly seen in the mid-adult age group (Fig. 4.49). The SAW population groups showed a low prevalence of trauma on most bones and no trauma on the occipital, temporal and mandible bones. The SAB group showed a low prevalence of trauma on the mandible and no trauma on the femur; however, the frequency of trauma to the shoulder (clavicle and scapula) and lower legs (fibula and tibia) were the highest in this population group. The trauma rate on most of the cranial bones (especially the nasal bones) and the arm (humerus, ulna and radius) was highest in the SAC population group (Fig. 4.50). Clavicle and ulna fractures were most prevalent on bones from the early time period, whereas nasal and radius fractures occurred most frequently in the mid-era and zygomatic, maxillary and lower-leg fracture most frequently in the late time period (Fig. 4.51).

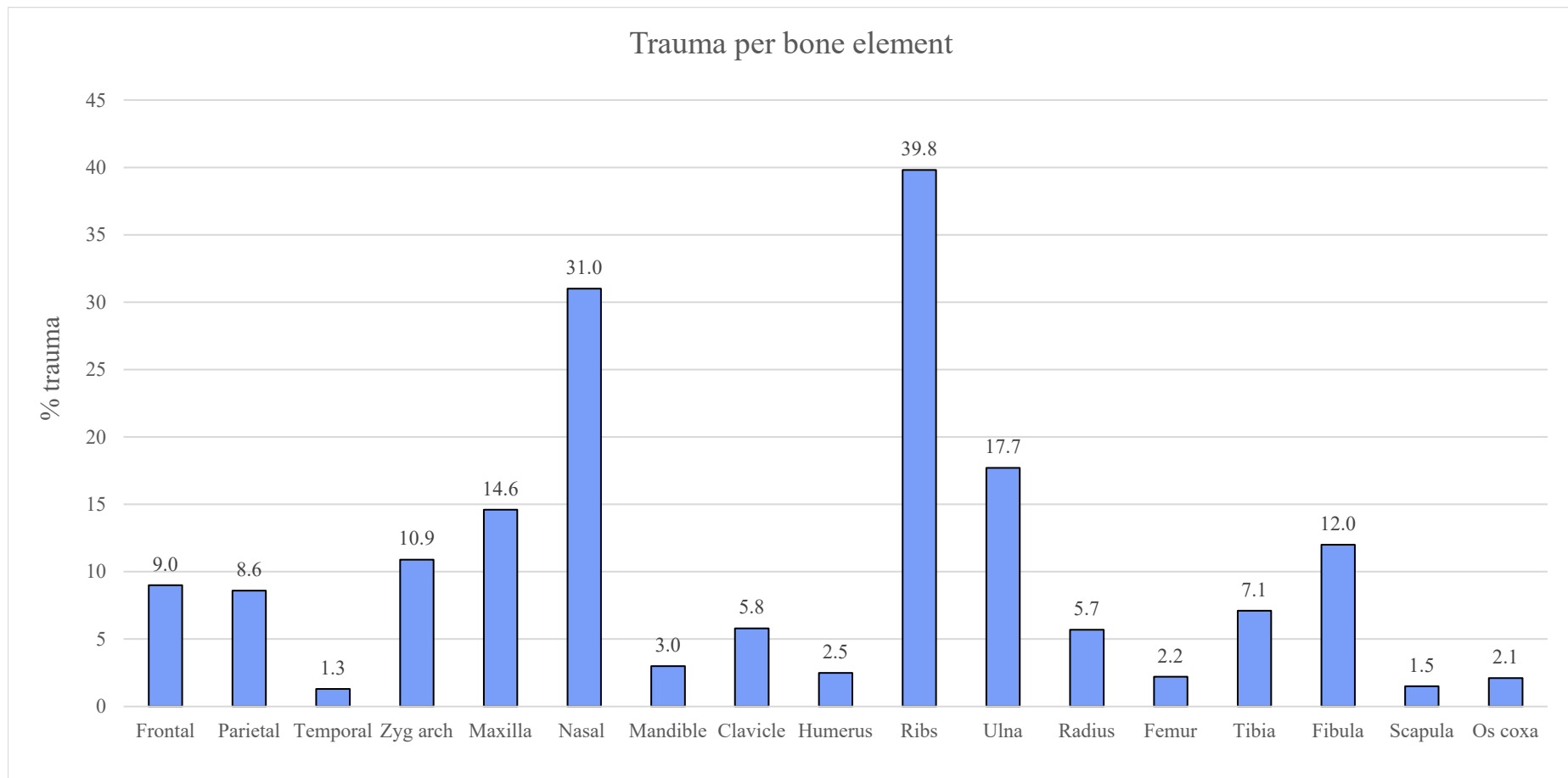


Figure 4.47. Percentage of healed trauma per cranial and post-cranial bones analysed

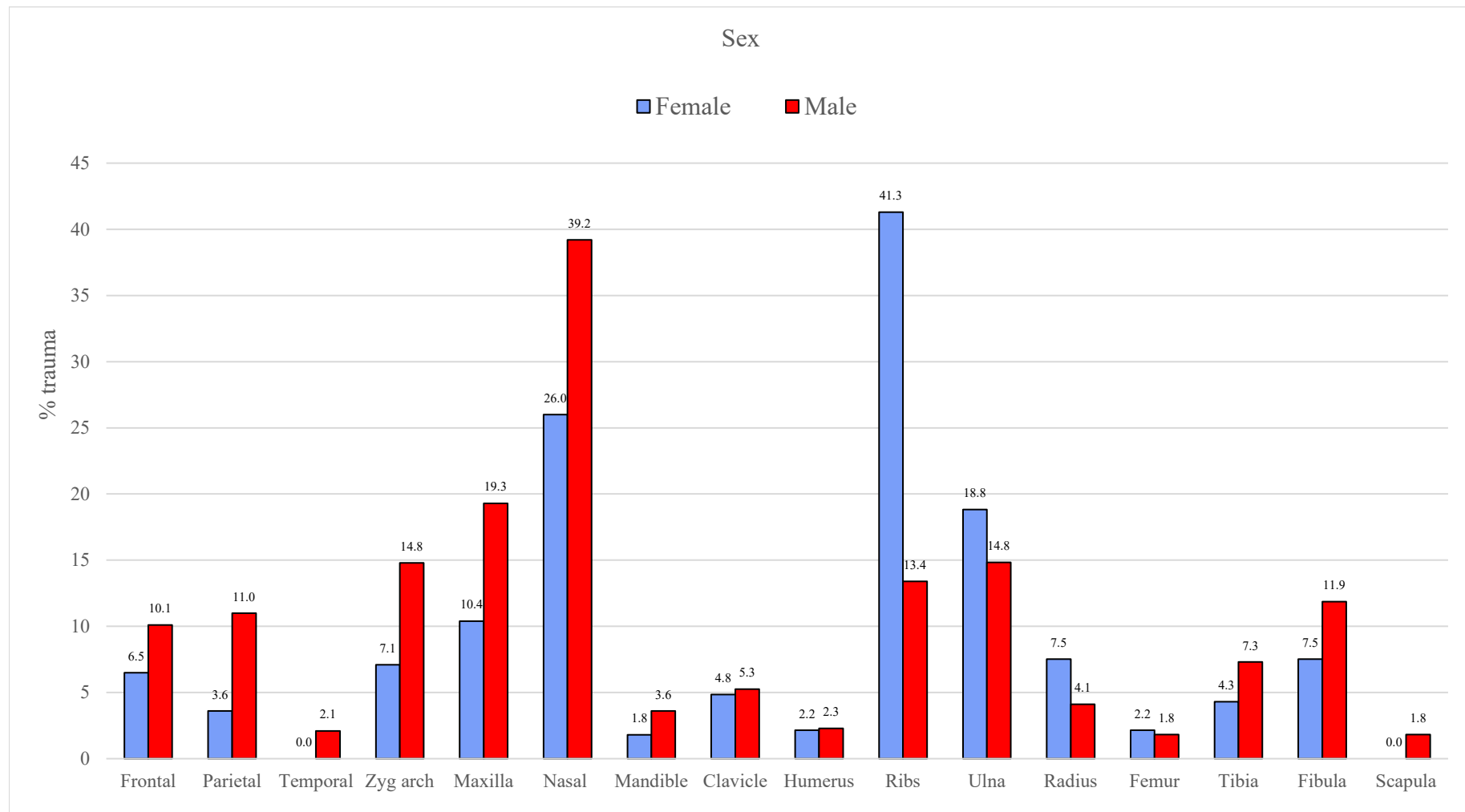


Figure 4.48. Percentage of healed trauma per sex.

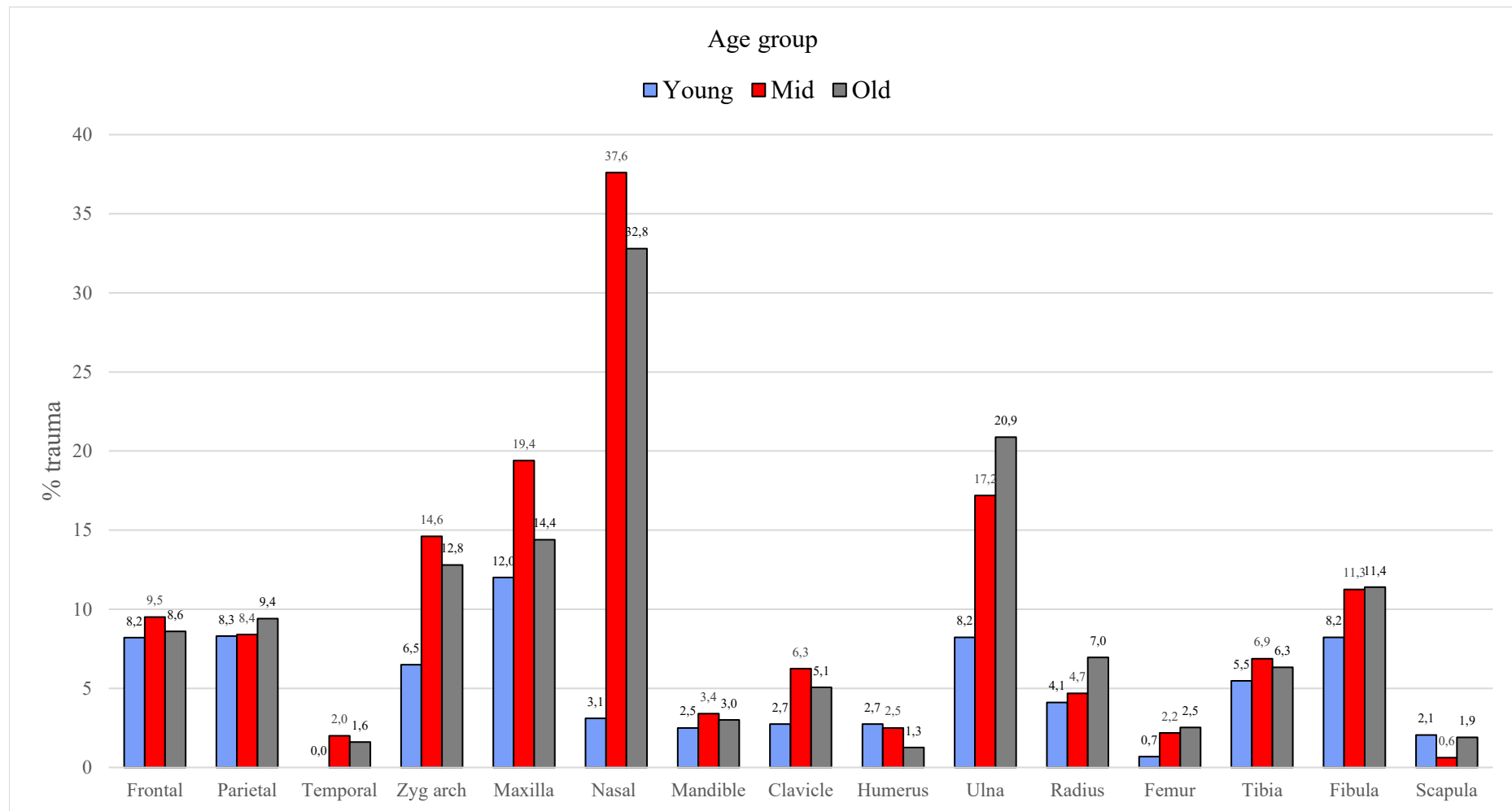


Figure 4.49. Percentage of healed trauma per age group.

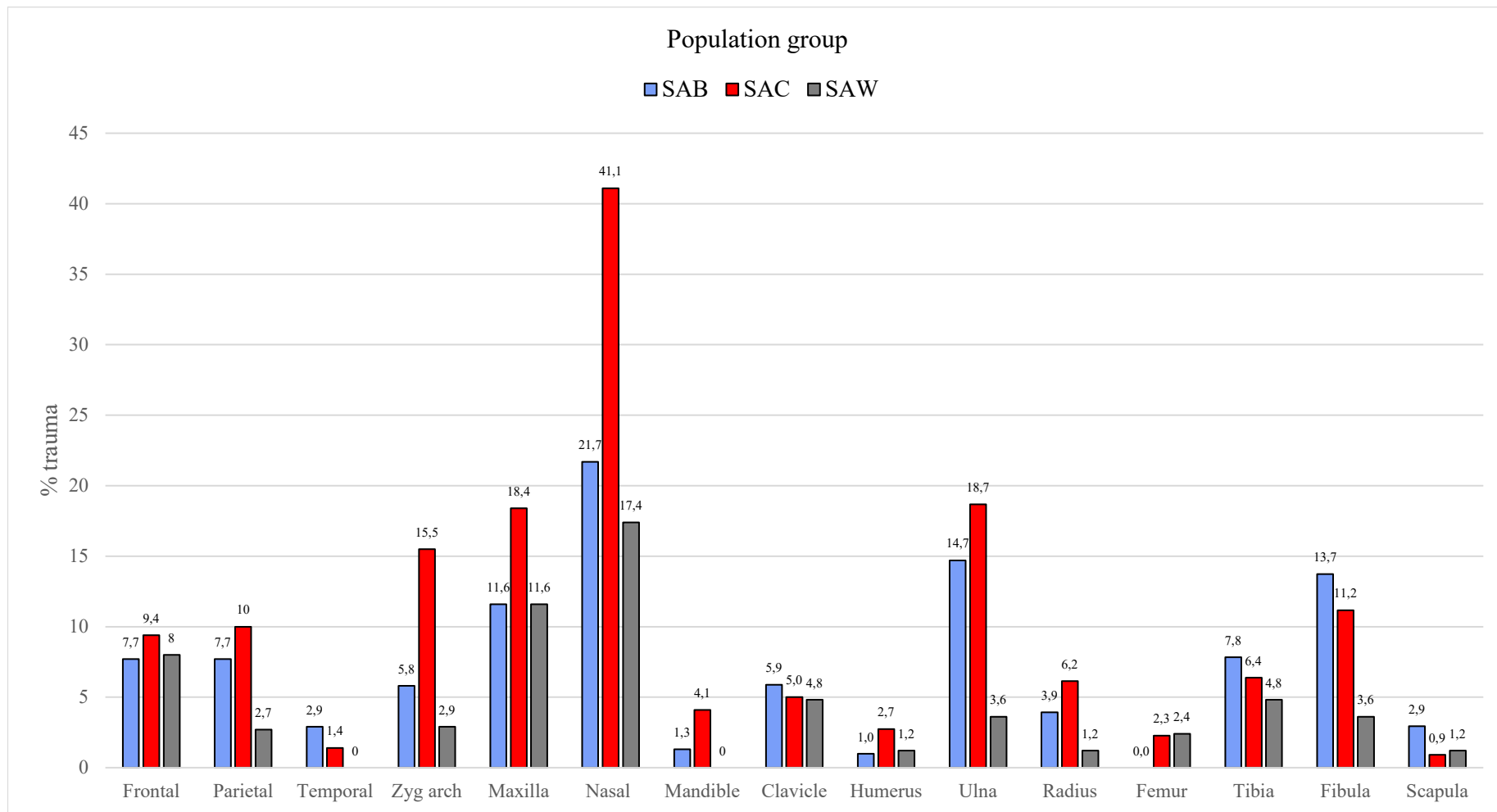


Figure 4.50. Percentage of healed trauma per population group.

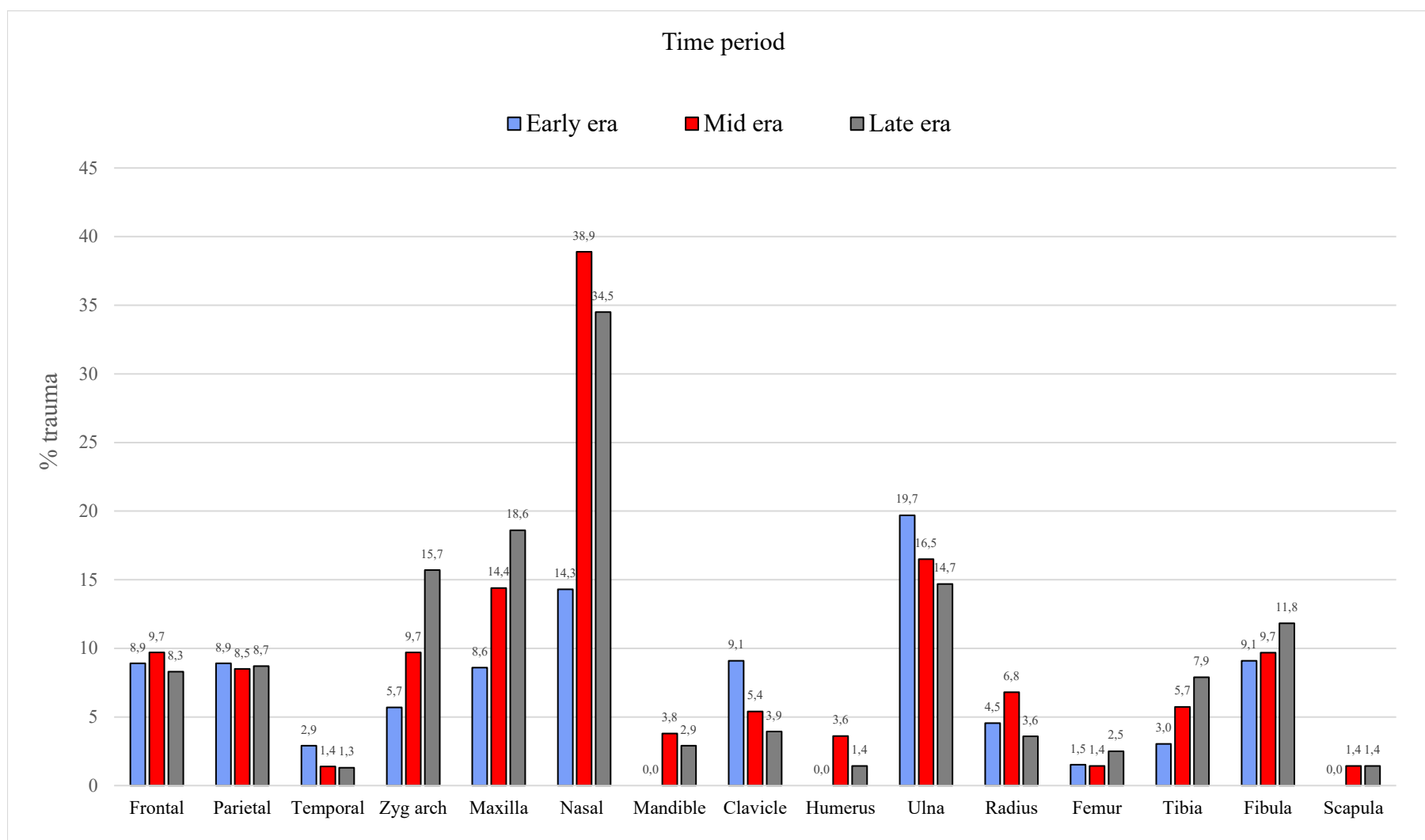


Figure 4.51. Percentage of healed trauma per time period in the 20th century.

CHAPTER 5: DISCUSSION

This study aimed to describe skeletal indicators of congenital variants, metabolic disorders, degenerative conditions, neoplasms and antemortem traumatic injuries of a reference skeletal collection, known as the Kirsten Skeletal Collection. The largest sample of this collection, and therefore the cohort of this study, comprises of the unique heterogenous mixed population group of South Africa, otherwise referred to as the South African Coloured (SAC) population group. Furthermore, this study aimed to assess the demographic associations of such indicators, and to determine any correlation with the 20th century timeline.

5.1 CONGENITAL/ GENETIC INFLUENCES

The prevalence of congenital anomalies among population groups differ, as was demonstrated by this study, where the SAW population group showed a dominance in terms of anomalies such as KFS and SBO. These differences may be due to either environmental influences (Sharma et al., 2011), or a genetic predilection in the population itself (Barnes, 1994). Although a genetic study of the population represented in the KSC is beyond the scope of this study, it is worth noting that closely-related populations and population groups tend to resemble each other in the incidence of anomalies on a genetic basis, while less-related groups show significant differences in the patterns of these variations. External factors, such as dietary influences (for example, a lack of folic acid in a diet), should not be excluded, as it may cause serious pathologies (neural tube defects (NTDs) in the case of folic acid deficiency) (Wald & Sneddon, 1991). When specific congenital anomalies have a higher prevalence rate in one population group than another in the same geographical region, it has both clinical and forensic anthropological implications. Therefore, the prevalence of congenital anomalies such as KFS, cleft neural arches or SBO, and LSTV in this KSC cohort, comprised of individuals from various population groups in the WCP, provides insightful information. It should, however, be kept in mind that the study sample is not a true representation of the inhabitants of the WCP, but a biased cadaver population, derived mainly from the northern suburbs of Cape Town.

The clinical relevance of congenital anomalies lies in accurate interpretation of the normal anatomy for surgical and other interventions (Kumar et al., 2007). Forensic relevance includes individual characteristics seen on dry bone, which can be compared to antemortem records (X-ray, CT and MRI films), where available, to facilitate victim identification in medico-legal circumstances (Vergauwen et al., 1997; Sharma et al., 2011; Kubavat et al., 2012).

KFS is a rare congenital anomaly, with the highest level of fusion in cervical vertebrae occurring in the occiput to C3 region (Gray et al., 1964), especially between C2 and C3 (Bonola, 1956; Baird et al., 1967; Somartzis et al., 2006). Although previous studies reported a female predilection (Gray et al., 1964; Helmi & Pruzansky, 1980; Pizzutillo et al., 1994, Thomsen et al., 1997; Larson et al., 2001; Fernandes & Costa, 2007; Paradowska et al., 2007; Samartzis et al., 2007), the current study showed a higher prevalence of KFS among males; however, the low number of KFS cases in the cohort, along with the large male bias in the sample, could have influenced this predisposition to males. Population-specific prevalence of KFS is unknown, but this KSC study showed a significantly higher prevalence among adult SAW males.

The occurrence of open or cleft neural arches in vertebrae is a fairly common congenital malformation, resulting from developmental failure of the ectodermal neural tube (Aufderheide & Rodríguez-Martín, 2011). In the current study, 11.0% of the sample presented with one or more vertebrae with open neural arches of the vertebral spinous process. The first sacral vertebra showed the highest prevalence rate, a finding that concurs with various other studies (Boone et al., 1985; Botto et al., 1999; Shin et al., 2008; Groza et al., 2012). The second highest prevalence rate was seen in the atlas and both the S1 and S2 segments combined. The SAW population group had the highest frequency of cleft arches (with a weak statistical significance) compared to the other two population groups. This finding is in agreement with studies by Shin et al. (2008) and Eubanks & Cheruvu (2009). Although previous research has shown a higher prevalence of open or cleft neural arches in males than in females (Lorber & Levick, 1967; Vannier et al., 1981; Fidas et al., 1987), this study revealed a slightly higher incidence rate among females than among males.

A review of various studies on LSTV indicated that the prevalence in specific population groups varies between 3.6% and 38.0%, with a mean frequency of 16.5%. This is comparable to the 16.3% prevalence rate found in the current KSC study. The wide range described in literature may be due to differences in genetic inheritance, inclusion conditions, diagnostic criteria, and imaging techniques used (e.g. the use of plain antero-posterior radiographs versus macroscopic dry bone observations) (Bron et al., 2007). In the current KSC study, lumbarisation (51.8%) and sacralisation (48.2%) were present in nearly equal frequencies, with a slight dominance (30.1%) of Type III sacralisation (according to Castellvi's classification) over the other types described in the classification system. Irrespective of type, the general

prevalence rates of sacralisation and lumbarisation in the study sample are comparable to those described by other authors (Hsieh et al., 2000; Bron et al., 2007; Konin & Walz, 2010). Although LSTV appears to be fairly common, specific statistics for the various South African population groups are insufficient.

5.2 PATHOLOGICAL LESIONS

5.2.1 SEX

Although most pathological lesions showed very little bias towards a specific sex, a strong female dominance in terms of TB infections and localised cranial infections was observed. This finding is in contrast to the global trend for males to have a higher probability of developing PTB (Caracta, 2003; Austin et al., 2004; Balasubramanian et al., 2004; Neyrolles & Quintana-Murci, 2010; WHO 2011). However, the higher rate of extrapulmonary TB lesions among females, is in agreement with studies by Musellim et al. (2005) and Sreeramareddy et al. (2008). The higher female infection rate may be attributed to differences in environmental exposure. For example, informal houses in squatter camps and townships generally have poor structural integrity with damp interiors that promote certain chronic illnesses. Together with overcrowding, this contributes to the dissemination of tuberculosis and infections by other bacteria (Govender et al., 2011). Poor employment prospects for females in the 20th century due to low levels of education, poor job opportunities, and limited child care options (Byrnes, 1996), suggests that more women stayed at home in these adverse housing conditions, increasing their chances of compromised immunity. Caring for sick children, the aged (often considered a woman's job) could also promote infections to spread. Compromised immune systems and adverse environmental conditions among females may also explain the female predilection for dental diseases such as abscesses and caries, although this finding stands in contrast to findings by other authors, who showed a male bias for periapical cysts (Avelar et al., 2009). On the other hand, males in this KSC study showed a higher prevalence for periodontal deposits, such as stains and formation of calculi, suggesting a general lack of dental hygiene.

Regarding deficiency diseases, no differences between sexes were observed in the KSC study, except for signs of anaemia (collectively seen as CO and PH) on dry bone, where females showed a higher prevalence than males, as was found in most other studies (Bharati & Basu, 1990; Brabin & Brabin, 1992; Scholl & Hediger, 1994). Rosso & Lederman (1982) suggested

that developing countries, like SA, have an anaemic bias towards young females due to a tendency for pregnancies at a young age, malnutrition, and poor access to health care facilities. The SAC population group increased at a rate of 0.5% between 1951 and 1975 (Van Rensburg & Mans, 1982), suggesting higher pregnancy rates. Furthermore, males have a larger iron storage ability than females due to slower growth in late adolescence, thereby improving their iron level status (Cohen & Armelagos, 1984), supporting the higher prevalence of iron-deficiency anaemia in females seen in this study. The current study showed a slight male predilection for CO, which may be due to a more complicated etiology of CO, such as subperiosteal bleeding associated with nutritional deficiencies, and complicated by a high alcohol intake (Mensforth et al., 1978; Steckel & Rose, 2002; Walker et al., 2009). A number of studies confirmed substantial alcohol consumption in communities in and around Cape Town (Pluddemann et al., 2004; Parry et al., 2005; Schneider et al., 2007; Richard et al., 2010; Ward et al., 2012; Corrigan & Matzopoulos, 2013; Schuurman, et al., 2015), seen especially in males (Peltzer & Ramlagan, 2009).

Although some studies indicate no sex bias in the prevalence of DJDs (Bachmann et al., 1999; Bonsell et al., 2000), osteoarthritic disease markings on the peripheral joints (pOA), and vertebral joint osteoarthritis (vOA), spondylolysis and DISH were slightly more associated with males than females in the current study. Furthermore, degenerative diseases of the vertebrae that are directly related to age (Ortner, 2003), such as osteophytes anterior on the bodies of the vertebrae (VO) and overall wedging of the vertebrae, showed a significant male predilection in the KSC study. However, due to the larger number of males and older people in the KSC collection, this male bias may be regarded as insignificant.

Primary neoplastic conditions in the KSC were so few in number that no interpretation of the prevalence in the sexes could be attempted. Both malignant primary bone tumours present in the cohort were observed in males, while one benign primary bone tumour was observed in a female. Statistically, secondary metastatic lesions that present on the skeleton showed a weak higher prevalence in females, compared to males. A comparison of CODs registered on the death certificates indicated that females showed a statistically higher prevalence for sex hormone-related cancers than males (breast and cervical cancers in females vs. prostate cancers in males), which would most likely present with metastatic bone lesions (Huben, 1992; Sone & Yano, 2007; Zhang et al., 2009; Hofbauer et al., 2014). This may be attributed to differences in hormones and hormonal regulation. Furthermore, males showed a higher predilection for

pulmonary cancers than females in this KSC study. This may be due to a higher number of smokers (Sieminska & Jassem, 2014) as there is a known correlation between smoking and lung cancer (Ozlu & Bülbül, 2005). A higher frequency of alcohol consumption among males may contribute to a higher rate of pulmonary cancers as suggested by some studies (Addolorato et al., 1998; Rehm et al., 2010; Praud et al., 2016),

5.2.2 AGE

The young-adult age group showed a significantly higher rate of periosteal lesions on the visceral surface of their ribs, indicative of TB infections or pulmonary diseases, than the mid- and old-adult age groups. Studies have shown a correlation between the risk of PTB and low SES, including poor housing, overcrowding, poor sanitation, and high alcohol consumption (Addolorato et al., 1998; Davies, 2005; Rehm et al., 2010). In cadaver studies by Geldenhuys (2014) and Geldenhuys et al. (2016), statistically noteworthy associations were observed between the prevalence of PTB and liver disease associated with heavy intake of alcohol. Heavy alcohol consumption and smoking are more prevalent in young males (Chan-Yeung et al., 2002; Peltzer & Ramlagan, 2009), therefore the association with PTB is higher in young males. Furthermore, researchers found that the prevalence of TB in poor SES areas in the WCP is extremely high and that PTB is currently inclined to develop at younger ages in SA, compared to a number of years ago (Lawn et al., 2006; Wood et al., 2010). In contrast, HOA, known to have an underlying association with pulmonary or pleural pathology (Carroll & Doyle, 1974; Geldenhuys, 2014), showed a significantly lower prevalence in the young-adult age group compared to the mid- and old-adult groups. Literature indicates that individuals presenting with HOA are usually in their 5th to 7th decades of life (Hammarstan & O'Leary, 1957; Ali et al., 1980; Segal & Mackenzie, 1982; Halcrow et al., 2014). However, inhabitants from the Western Cape with HOA tend to have a below average age at death. Geldenhuys (2014), for example, reported a median age of 45.2 years at death for SAC individuals from the WCP who presented with HOA. Similarly, the average age at death of individuals with HOA in the current study was 53,6 years. The younger age at death can be attributed to the high burden of PTB in the Western Cape. The WCP is one of the highest TB-burdened regions in the world (WHO, 2011), with a disease rate of up to 60 times higher than many developed countries, including the USA and Europe (Fourie, 2011; Geldenhuys, 2014).

The mid-adult age group showed a slightly higher rate of non-specific periosteal reactions than the old age group. One would expect immune-compromised older individuals to have a higher prevalence rate. However, it could be that older individuals with a high infection rate (seen as non-specific periosteal reactions) were removed from the population before bone markings developed, and therefore only the individuals strong enough to survive are represented. The young population group, on the other hand, demonstrated a low frequency of periosteal reactions as their resistance against infections should be considerably higher due to stronger immune systems.

Poor dental health (calculi, abscesses and caries) was associated with both the young- and mid-adult age groups, but could not be correlated with the old group, as a large number of individuals from this group were edentulous, mainly due to age-related antemortem tooth loss. These findings were in agreement with those from other authors (e.g. Avelar et al., 2009). Geldenhuys (2014) found a higher abscess rate among a 30-39 years age group in a WCP cadaver cohort. Individuals from the older age group showed a predilection for staining when teeth were present, as well as a higher prevalence of dental work, compared to the mid and young adult age groups. This can be explained by the fact that, in general, the need for dental work increases with age.

Although a statistical difference in age groups was seen among the three individuals with pectus carinatum (indicating a possible vitamin D deficiency), the number of affected individuals was too small to contribute to a meaningful result. In terms of signs of vitamin C deficiency, no significant difference was seen among age groups, although the old age group was marginally more affected. Due to an abundance of ascorbic acid in many available foodstuffs, scurvy is not a primary deficiency in contemporary populations and may therefore be encountered only as a secondary deficiency among the elderly and/or in chronic alcoholics (Kumar et al., 2007).

The old age group demonstrated the highest frequency of OA on all the peripheral and vertebral joints. This is to be expected, as studies have confirmed that age is the single biggest factor involved in OA (Mann & Murphy, 1990). The old-adult age group was more likely to show fused vertebrae (AS) than any of the other groups, while wedging was observed on the vertebral bodies of both mid-adult and old-adult skeletons. Furthermore, DISH was observed in the vertebrae of mostly older individuals (after their 6th decade of life). This is in concurrence with most other studies reporting an increased prevalence after the 5th decade of life

(Aufderheide & Rodríguez-Martín, 2011). Geldenhuys (2014) found DISH (n=12) in a WCP cadaver cohort to occur at a mean age of 65.1 years.

5.2.3 POPULATION GROUPS

Most of the pathologies were moderately associated with all three population groups in the cohort. Analyses of COD during the 20th century showed high numbers of infections, particularly PTB, present in the SAC communities in the Cape Flats (Van Rensburg & Mans, 1982; Coovadia, 2009), mostly due to their adverse environment, including malnutrition, poor sanitation and susceptibility to infections due to overcrowding (Van Rensburg & Mans, 1982). On the dry bones, the SAC population group showed the highest prevalence of osteomyelitis in the KSC cohort, while no osteomyelitis markings were seen on the skeletons of individuals from the SAW population group. Non-specific periosteal reactions were similarly more prevalent in the SAC and SAB population groups and significantly lower in the SAW group. The PTB infection rate observed on the dry bones of the KSC individuals by examining visceral rib lesions, weight bearing joints or vertebrae, were higher in the SAC population, with both the SAB and SAW populations affected at lower rates. Geldenhuys (2014) found that 84.0% of the cadavers (in a study including mainly SAC individuals from the WCP) were affected by PTB. Differences in infection rates among the three population groups can be ascribed to an uneven distribution of health services among population groups during the 20th century, with most advanced health care facilities and drugs being more accessible to the privileged SAW population group, and not to the ‘non-white’ populations of the Cape Flats (Van Rensburg & Mans, 1982). Although the SAB population group lived in similar, or even worse conditions, than the SAC population during the apartheid era in the WCP, the prevalence of markings from infectious diseases on their skeletons were lower than observed in the SAC population group. This can, firstly, be explained by a population bias resulting from a large number of skeletons from the SAC population group and a low number from the SAB population in the KSC, mainly due to the fact that fewer members of the SAB population lived in the ‘catchment’ area of cadaver donations to Tygerberg hospital (Whittingdale, 1973, Friedling, 2004). Secondly, the SAB communities would, traditionally, rather make use of traditional/tribal healers or sangomas, who do not use modern drugs/antibiotics (Van Rensburg & Mans, 1982). Therefore, the limited health care facilities in their communities were poorly visited and, due to premature death, infections did not have a chance to advance to a chronic stage that would present on the skeleton.

The under-presentation of signs of dental disease, such as abscesses and caries, seen in the SAW population group can be explained by the relatively easy access to dental health facilities available to the privileged SAW population during the 20th century. This is in contrast to the ‘non-white’ communities who lived in impoverished settings and did not have the access to preventative health care (Govender et al., 2010; Geldenhuys, 2014). Dental work was markedly more prevalent in the SAW population group, most likely because qualified dentists were easily accessible to the SAW population group in comparison to the ‘non-white’ population groups. In 1946, for example, the dentist to population ratio was 1:13958, with most of the dental practices in ‘white’ urban areas, and not accessible to the ‘non-white’ population (Van Rensburg & Mans, 1982).

Although rickets and osteomalacia are ancient diseases, they can still be present in developing countries due to an imbalanced nutritional intake, malnutrition (Winzenberg & Jones, 2013), or lack of sunlight (Thacher et al., 2013). There is no lack of vitamin D in the WCP, since abundant sunlight and sources of vitamin D-rich fish are available, supporting the low 2.4% prevalence of signs of osteomalacia on bones of the KSC sample. During the apartheid regime, managerial positions, which were mostly indoor positions, were reserved for the more privileged white minority (Bundy, 2016), which could explain the higher rate of vitamin D deficiency markings seen in the SAW population group. Although a darker skin pigmentation is generally prone to vitamin D deficiency, ‘non-white’ population groups were, at the time, mostly employed outdoors as labourers (Bundy, 2016), exposing them to more sunlight.

The SAB population group showed the highest prevalence of anaemia signs when all the traits were pooled, a finding similar to that of Zakai et al. (2008). The second highest anaemia rate was seen in the SAC population group. Malnutrition, parasitic pathogens and poor SES were common among the SAC and SAB populations during the 20th century and have been associated with anaemias (Cohen & Armelagos, 1984; Walker et al., 2009). Unlike the other traits, CO on its own showed a slightly higher prevalence in the SAW population than in the other two population groups. Age, diabetes, and vascular disease were determined to have a stronger correlation with anaemia among a SAW group than a SAB group in a study by Zakai et al. (2008). In the current study, the SAW population group showed a much higher prevalence of cardiovascular disease than the SAB and SAC population groups, which may be the reason for the SAW group having a higher CO rate in the KSC population.

The SAW population group statistically demonstrated the highest frequency of scurvy markings, with lower prevalence in both the SAC and SAB population groups. This may be due to cultural behaviour, as the SAW population group is inclined to be ‘meat eaters’ (including traditional ‘droëwors’ and ‘biltong’) and may have a deficiency of fruit and vegetables in their diet (Coetzee, 1977). According to a study by Hampl et al. (2004), non-Hispanic black males, smokers, and people who did not use supplements, showed higher risks of vitamin C deficiency, while Mexican-Americans showed lower risks.

With regards to DJD, the SAW population group showed the highest frequency of OA. The reason for this is that the age of the cadaver intake for the SAW group in the collection was generally more advanced, with more individuals in the old-adult category. The average age at death for males in this group was in their early 60s, and for females in their late 60s, while the other two groups both showed an average age at death of in the 50s for males and in the 40s for females. Diet and cultural behaviour are factors that may have an influence on the prevalence of degenerative diseases, such as DISH. A study by Rogers & Waldron (2001), for example, found the prevalence of DISH to be higher in populations with a protein-rich diet. In the current KSC study, the SAW population group showed the highest prevalence of DISH, followed by the SAB and SAC population groups, respectively. This finding is in concurrence with a study by Adebajo & Davis (1994). The SAW population group, in general, had access to good protein sources during the 20th century, compared to ‘non-whites’ from low socio-economic circumstances whose diets consisted mostly of low-cost, starch-based foods (Coetzee, 1977). This may explain the high prevalence of DISH in SAW individuals in the KSC. Compared to spondylolysis, Kiss et al. (2002) found that DISH patients were more likely to report a history of diabetes mellitus, weighed more at a younger age, put on more weight, and had a greater BMI than patients with spondylolysis. These risk factors cannot be verified in this skeletal study, but the SAC and SAW population groups showed an almost equal prevalence of spondylolysis, followed by a much lower rate in the SAB group.

5.2.4 TIME PERIODS

General infections, such as localised cranial infections, HOA, periostitis and osteomyelitis seen on bone, occurred the least during the early 20th century, representing the time period between 1928 and 1940. Before the advance of antibiotics, these localised infections and associated complications had a severe impact on the daily lives of individuals (Flohr & Schultz,

2009), often resulting in death, thereby explaining the low infection frequency in the KSC sample during this time period. After the introduction of antibiotics in the 1940s, infections were treated successfully, resulting in a longer life span, but also chronic diseases as result of recurring infections (Steyn et al., 2013). Such chronic infections can result in permanent osseous changes in the bones (Flohr & Schultz, 2009), explaining the increase in skeletal lesions seen in the mid- and late 20th century. However, it should also be taken in consideration that the early period is under-represented in the KSC sample compared to the mid- and late 20th century periods. The highest infection rate on the bones occurred during the mid-20th century, representing the time between 1941 and 1956. This can be explained by the displacement of the 'non-whites' who lived in Cape Town to squatter camps and townships in the Cape Flats area after the Group Areas Act was passed. These townships were overcrowded, with poor sanitation, poor health care access, and a high infection risk (Bundy, 2016). Malnutrition may have contributed to compromised immunity, which, in turn, may have further contributed to a higher infection burden (Cluver, 1959).

Although deaths due to PTB were recorded throughout the 20th century (Cluver, 1959; Van Rensburg & Mans, 1982; Coovadia, 2009), visceral rib lesions and extrapulmonary TB markings on bone in this study were observed mostly during the late 20th century, representing the time period between 1957 and 1995. Steyn et al. (2013) reported an increase in the frequency of TB lesions observed on skeletons over time, especially after the introduction of antibiotics in the 1940s. According to the authors, TB lesions on bones increased from 21% to 38%, suggesting a longer life span as a result of treatment, and therefore more time for these lesions to manifest on the skeletal elements. When they compared earlier years to the period after 1985, when the first multidrug-resistant TB (MDR-TB) case was reported (Sissolak, et al., 2010), a statistically significant increase of 41% in TB lesions was noted. Steyn et al. (2013) suggested that this increase was due to extended treatment with drugs, causing the bacteria to gradually gain a drug resistance, providing more opportunity for skeletal lesions to develop.

Dental health problems were highly associated with the late time period, but negatively correlated with the early time period, suggesting a decrease in dental health over the 20th century as environmental and social conditions deteriorated. In addition, dental work became more prevalent in the late time period, since the dentist to population ratio gradually improved towards the late 20th century (Van Rensburg & Mans, 1982).

Deficiency diseases did not show a significant difference between the three time periods of the 20th century. Weak correlations with the early time period for growth arrest lines were observed, while rickets (osteomalacia) was positively correlated with the late and mid-eras, but negatively associated with the early period. Signs of anaemia on the skeletal material were present in equal rates during the mid- and late eras, although a much lower prevalence was observed in the early period. Geldenhuys (2014) found a weak association between PTB infections and anaemia, which may explain the higher involvement of CO and PH in the later 20th century when a higher TB infection rate was seen.

DJD highly correlated with age, rather than time period. However, with regards to mid- and lateral wedging in the vertebral bodies, the late time period was statistically less affected than the early and mid-periods, whereas vertebral fusion, such as AS and DISH, were less prevalent during the early time period, although not significantly. Osteoarthritic markings on peripheral joints were mostly similar among time periods, except for markings on the shoulders and elbow joints, that showed a statistically higher prevalence during the mid-era, and the lowest prevalence in the late era. Although OA on synovial joints is an age-related condition, OA in the shoulder and elbow joints may be the result of chronic mini traumas from overuse, caused by strenuous activity during the industrial revolution (Hall, 2003) in the mid-20th century. The prevalence of sclerotic and/or lytic lesions, indicative of metastatic neoplasms, in the skeletal cohort were similar for all three time periods. A comparison of cancer-related causes of death registered on the death certificates of individuals in the KSC cohort, showed a very low prevalence of cancers in the early era and a much higher prevalence during the mid- and late time periods. This lower prevalence may be ascribed to undiagnosed cancer-related deaths during a time when the doctor to patient ratio, especially for 'non-white' individuals, was highly inadequate and very few health care facilities were available for underprivileged people (Van Rensburg & Mans, 1982).

5.3 TRAUMA MARKINGS

The KSC mostly represents vulnerable residents from low socio-economic areas around Cape Town. These areas are burdened with high levels of unemployment, poor living conditions, gang-related activities, alcohol abuse and drug addiction (Schneider et al., 2007; Inwood & Masakure, 2013), resulting in high levels of interpersonal violence (IPV). Skeletal signs of antemortem trauma were observed in 71.8% of individuals assessed in this study. The

skeletal elements showing the highest prevalence of healed trauma were the ribs (39.8%) and nasal bones (31.0%), followed by the ulna (17.7%) and fibula (12.0%). Isolated rib fractures (Lovell, 1997; Brickley, 2006), nasal bone fractures (Lee, 2009) and forearm fractures (Kilgore et al., 1997; Alvrus, 1999; Judd, 2008) are often related to incidences of IPV, although accompanying injuries to multiple skeletal elements may be indicative of MVAs and accidental falls (McCoy et al., 1988; Lovell, 1997). Evaluation of the ribs was done with particular caution due to the difficulty in determining the cause of injury. Although the prevalence of callus formation on the ribs was high, the total involvement of the ribs could not be established, as most of the ribs between 3 and 8 were missing from the individuals in the collection. These ribs were sectioned during undergraduate student dissections and the fragments were consequently cremated, deemed of little value by previous curators of the KSC. Healed trauma was highly prevalent on both nasal bones, suggesting direct frontal blows to the nose, or numerous blows on various occasions. The ulna was found to be the most frequently injured long bone in the current study, similar to findings from other studies (Kilgore et al., 1997; Alvrus, 1999; Judd, 2008). The left side was more affected than the right, possibly attributable to defensive wounds against a right-handed attacker (Norman et al., 2007). The distal ulnar shaft showed the highest prevalence of fractures (85.0%). Fractures to the distal third of the ulnar shaft are known as parry fractures, and have been associated with a defence position, when a person is fending off a blow to the head (Ali, 2003; Judd, 2008). The prevalence rates of fractures to the proximal and mid regions of the ulna were much lower (2.0% and 13.0%, respectively), and may be the result of falls, as suggested by Lovell (1997).

In the current KSC study, the ribs and upper limbs, specifically the humerus, radius, and ulna, showed a higher prevalence of fractures in females than in males. This is in line with findings by Van Staa (2001). Fractures on the ribs and forearms are known to be associated with intimate partner violence (IPV). Although South Africa has an existing culture of domestic violence against women and children (Krug, 2002; Jewkes et al., 2002; Abrahams et al., 2006), it is difficult to gather accurate statistical data, as such incidents are generally underreported. This was especially true during the 20th century, when domestic violence was generally regarded a private matter (Wood & Jewkes, 2001). Prior to democracy (1994), no law existed in SA to protect victims of IPV (Usdin et al., 2000). IPV is reported to be fairly common in SA, and abuse against women is generally tolerated. According to Statistics South Africa (StatsSA, 2012), one in 40 SA women from different population groups believe it is acceptable for men to physically assault women. During the 20th century, SA was a patriarchal society,

deeming both SAB women from traditional African societies and SAW women from a settler society inferior to men. The men took all major decisions, both in society and within the home. Furthermore, a recent study by Teitelman et al. (2017) found binge drinking to be associated with IPV, especially in males with a history of exposure to violence, who were more likely to show violence towards a partner than males without a history of exposure to violence.

Ten cases in the current study showed injuries involving both the left ulna and radius, while two cases had similar injuries on the right only. In cases where both the radius and ulna are involved on the same side, an accidental fall (Judd, 2008), MVA or physical conflict (Lovell, 1997; Hertel & Rothenfluh, 2010) may be the cause, although larger forces are needed to result in injury to both skeletal elements. Thus, it is proposed that the fractures involving the middle or distal ulna and associated proximal or middle radius observed in this study, may have resulted from IPV or from MVAs. The main causes of the fibular fractures and trauma to this region in the current study, were either a direct impact (as seen in sports and MVAs), or indirect trauma caused by a fall. Tibial fractures may occur in conjunction with fibular fractures, especially in a PVA, as the vehicle bumper usually hits an upright adult at the level of the lower limbs (McCoy et al., 1988), although team sport games such as football (rugby) have also been implicated in fibular trauma (Werner et al., 2017). The prevalence of trauma to the distal third of the fibula with accompanying tibial injuries in the current study, is presumably not a result of IPV, but rather due to a fall, PVA or MVA, as suggested in literature (Court-Brown & McBirnie, 1995; Lovell, 1997; Alvrus, 1999).

Overall, males showed a higher fracture rate than females in this KSC study. Similar findings were reported in previous studies (Butchart & Brown, 1991; Judd, 2008; Murphy et al., 2010). Males are more commonly involved in gang-related violence than females according to the 2003 South African National Victims of crime survey (Peterson et al., 2004). The fact that gang-related activity in the WCP is much more advanced and sophisticated than in other provinces (Kinnes, 2000), may provide an explanation for the higher prevalence of skeletal trauma, specifically IPV-related trauma, in males (Van Wyk & Theron, 2005). Furthermore, it is acknowledged that the WCP of SA has a major problem with high alcohol consumption, seen as a cultural reaction to their socio-economic challenges (Schneider et al., 2007). The higher rate of trauma on most of the male skeletal elements may be associated with binge drinking and subsequent violent encounters between males (Corrigall & Matzopoulos, 2013; Schuurman et al., 2015).

The prevalence of trauma observed on the left side of the cranial bones was higher than on the right side for most bones. It is common for the left side of the face or body to be more affected during IPV, since an assailant will most likely be right-handed, and a blow from the right will result in trauma to the left side of the victim (Sauer, 1998; Norman et al., 2007). Post-cranially, trauma to the long bones was mostly localised to the left side. However, both sexes showed a higher prevalence of trauma to the right tibia, revealing a right-handed dominance in the case of sports-related injuries or injuries due to kicking (Lovell, 1997; Alvrus, 1999).

Although the mechanism of the injury (either blunt or sharp force trauma) is important in the interpretation of skeletal trauma, the findings in a cadaver-derived skeletal collection may be biased towards blunt force injuries. Sharp-edged trauma may more likely result in immediate death, leading to autopsy rather than donation to a medical school. Nevertheless, Brink et al. (1998), found blunt force trauma to skeletal material to be the main cause of injury in assault victims, while Fox (2011) estimated that blunt force trauma occurs in 31.1% of IPV cases. In the present study, less than 2% of skeletal trauma was caused by sharp force, suggesting that IPV may be the main cause of injuries in the KSC cohort. However, trauma caused by IPV can be indistinguishable from work- or sports-related injuries, and in the case of multiple traumas it is not always evident whether the injuries were acquired simultaneously or on separate occasions (Brickley, 2006).

Trauma was more prevalent in the SAC population group than in the other population groups. This finding is in concordance with those of an epidemiological study by Butchart & Brown (1991), as well as a study by Silber & Geffen (2009), both finding violence to be more prevalent in the Cape Flats SAC communities around the WCP, where gang-related activities and low socio-economic circumstances are prominent. Van Rensburg & Mans (1982) reported homicide or wilful injury by others to be common CODs among SAC males during the 20th century. In 1976, for example, homicide was recorded as the highest COD among males of the SAC population group (Van Rensburg & Mans, 1982). Furthermore, the high trauma lesions in the SAC population groups may be explained by the high alcohol abuse patterns, a legacy of the “dopstelsel” in this population group (London, 1999; Parry et al., 2005; Schneider et al., 2007; Peltzer & Ramlagan, 2009; Gossage et al., 2014). The SAW females show the lowest rate of antemortem trauma of all the sex and population groups. The SAW group, in general, showed a high rate of ischaemic heart diseases during the 20th century (Cluver, 1959; Van Rensburg & Mans, 1982) and cardiopulmonary resuscitation or, in one case, open heart

surgery, may explain the higher prevalence of sternal fractures in the SAW population, for both males and females. However, compared to the ‘non-white’ population groups, trauma to the pelvis and femur were more prevalent in males of the SAW population group. Trauma to these skeletal elements are uncommon during IPV and rather occur during a frontal collision MVA, where compressive pelvic injuries (McCoy et al., 1988) and/or an axial load midshaft fracture of the femur (Kolmert & Wulff, 1982) may occur. This may be explained by the fact that the more privileged SAW population group, particularly SAW males, had a much higher risk of MVA accidents as they had access to private motor vehicles, while other population groups were generally more dependent on public transport.

Most of the injuries on the skeletal elements were observed in the old age group, as would be expected. No medical records of individuals in the KSC cohort were available for clinical assessment. Therefore, it can only be speculated that the increase in signs of skeletal trauma with age may result either from an increased risk or, more likely, from an accumulation of injuries over the course of life. The age distribution of skeletal trauma in the KSC cohort may therefore not be a true reflection of a ‘prime age’ for trauma, but rather an indication of the accumulation of trauma throughout an individual’s life.

5.4 CONCLUSION

Congenital, pathological and traumatic lesions manifesting on bones were analysed in a skeletal sample from the KSC, a reference collection that broadly aims to represent individuals who lived in the WCP during the 20th century. Distinct differences were observed in the prevalence and distribution of diseases and trauma patterns among the various age groups, population groups, and time period, as well as between males and females, reflecting differences in cultural variables, political influences, fragmented health services, and socio-economic risk factors.

5.4.1 CONGENITAL ANOMALIES DURING THE 20TH CENTURY

Congenital variation found in this study may be population specific, as KFS and SBO were both found to be more prevalent in the SAW population group. Analysis from a genetic perspective falls outside the scope of this study; however, further studies comparing genetic susceptibility per population group and possible external factors that may play a role, could shed light on the incidence of anomalies on a genetic basis.

5.4.2 PATHOLOGICAL LESIONS DURING THE 20TH CENTURY

Throughout the entire 20th century, the young adult age group showed a high frequency of TB-related infections, whereas the mid-adult age group showed a higher rate of non-specific periostitis and osteomyelitis infections, and the old age group showed more OA lesions.

Although dental enamel defects are more favoured to HL for assessing growth disruptions, HL in the current study showed a greater prevalence on the skeletal elements compared the DEH on the teeth due to the lack of teeth in many KSC individuals. The maxillary incisors and the mandibular canines are the best teeth to be examined for DEH according to Goodman and his colleagues (1980); however, the current study showed many individuals without these teeth, either due to cultural extraction of specifically the incisors and canines, or total edentulous mandibles and maxillae.

The SAW population group represented in the KSC study, showed the lowest prevalence of signs of anaemias, growth arrest signs, poor dental health and infection, confirming that the higher SES societies that escaped the unsanitary, overcrowded, and poor living conditions, are more successful in buffering themselves from malnutrition and exposure to pathogens.

5.4.2.1 Early-time period

During the early 20th century, before 1940, after the devastation of World War I, the various infective disease epidemics, and in the midst of the Great Depression, SA battled to keep the economy afloat, ultimately bringing about political change. At the time, the poor economic outlook caused many white South Africans to become destitute and the ruling white minority party contemplated apartheid laws to ensure the economic upliftment of the poor 'white' people, at the expense of the 'non-white' majority in the country. The proclaimed laws lead to substandard education, housing, sanitation, health care, and economic opportunities for the 'non-white' SAB and SAC communities of SA. This was also the period before antibiotics were available to treat infectious diseases, such as cholera, tuberculosis, and venereal diseases.

Individuals in the KSC representing this early time period showed the least signs of infection on their bones, as people would die from an aggravating infection before it could leave bone markings. Signs of growth arrest (Harris' lines) were slightly higher during the early period for both 'non-white' population groups, suggesting a lack of medical attention in these groups.

5.4.2.2 Mid-time period

During the mid-period (between 1941 and 1956), at the end of WWII and the start of apartheid, large scale urbanisation due to the industrial revolution became a challenge for the SA government. Overcrowded and unhygienic low-cost informal settlements became common in the WCP. Although the advancement in vaccines and the development of antibiotics greatly improved quality of life and the outcome of infective conditions globally, poverty-related diseases brought on by malnutrition, overcrowding and poor hygiene persisted in the disadvantaged communities of the Cape Flats, evident in this study as a high rate of signs of non-specific periosteal reactions on the skeletons of the ‘non-white’ population groups during this mid time period.

5.4.2.3 Late-time period

In the time period after 1957, at the height of apartheid and the ensuing political turmoil, statistically more signs of TB were observed on bones of SAC individuals in the KSC cohort, suggesting high infection rates in the overcrowded “Coloured” townships established around the industrial parks of the northern suburbs of Cape Town. Successful treatment of infections with antibiotics also contributed to a higher frequency of infection markings on bones in the KSC cohort during the mid- and late 20th century. This finding suggests an increase in the life-span of people, thus providing more time for lesions to manifest on bones. After the introduction of antibiotics, females manifested with increasingly more lesions on the skeleton (resulting from both specific and non-specific infections) than males, with the exception of osteomyelitis. This may suggest that females were more immune-compromised than their male counterparts. Further studies are needed to investigate the interactions between, and changes in disease agent resistance and host immune response concomitant with the spread of HIV/Aids in the transformed late 20th century environment.

The differences between higher and lower social categories were clearly seen in skeletons from the late time period of this study, encapsulating the apartheid era, when specific laws were put into place to uplift the minority SAW population group. The consequences of this regime began to show in specimens from the 1960’s onwards, when growth arrest lines (DEH) and scurvy became more prevalent than in any of the other eras.

5.4.3 TRAUMATIC LESIONS DURING THE 20TH CENTURY

This study provides valuable information regarding socio-economic risk factors as well as the prevalence of IPV in the WCP during the 20th century. The KSC does not represent the entire Western Cape population, as the individuals in the KSC were mainly donated from the poor SAC communities whose members often served as labourers in the industrial parks of that era. The SAC group in the study can be regarded as a subgroup of a modern SAC society, with relatively high levels of physical aggression. The SAC population group in the study showed high rates of trauma from assault, abuse, and other violent crimes, mostly due to risk factors such as political (apartheid), socio-economic (low SES), environmental (unemployment) and cultural (alcohol or drug abuse) stressors. Macroscopic analysis of the sample indicated that most of the skeletal traumas in the cohort resulted from cumulative incidents. Observed injuries showed none of the distinct patterns usually seen in MVAs, rather suggesting IPV, with trauma clustering mostly seen on the nasal bones, ribs (isolated, or in combination with long bone injuries), and the middle and distal third regions of the ulnae (so-called parry fractures). Males from the SAC population group may have been at higher risk for IPV as they showed the highest prevalence of trauma in this study, although the bias towards SAC males in the collection should be taken into consideration. The high incidence of parry fractures, especially among SAC females is noteworthy, probably reflecting violence related to a patriarchal society, as was prevalent in many SA communities during the 20th century. In addition to a culture of violence in SA, a sense of disempowerment due to unemployment among males, who were traditionally expected to be the breadwinners, may have contributed to a violent display of power against females.

5.4.4 RELEVANCE OF SKELETAL COLLECTIONS TO RESEARCH

Studies on skeletal collections rely on the assumption that the remains represent a past community, population group, or specific region, and can be used as a valid comparative reference for reconstructing different aspects of skeletal biology of past people who lived in that population. The KSC cohort has limitations in its application potential because it relies on body donations or retention of adult unclaimed bodies under South African law and the statutes of the Inspector of Anatomy, resulting in a biased sample. The fact that no medical histories of these donated individuals are known makes interpretation of the lesions observed on the skeletal material in the KSC not exact. Furthermore, the limitations of the three conceptual problems noted in the osteological paradox paper described by Wood and colleagues in 1992

should be considered before skeletal lesions as indicators of a healthy or diseased society are interpreted. A deeper understanding of the part that cultural context play in determining heterogenous susceptibility to disease and death, and the level of selective mortality of a population can be attempted with data generated from known medical skeletal collections.

The age at death represented in the KSC is unequal with, for example, only nine sub-adults and no infants represented in the collection, while older ages are over-represented, especially in the case of the SAW population group where more individuals belonged to the old-adult category (older than 60 years), with males mostly being in their early 60s and females in their late 60s at the time of death. Although the KSC includes both sexes, males are overrepresented as observed in donated medical skeletal collections. Resulting from unclaimed, as well as bequeathed cadaver origin, various socio-economic standards are represented; however, low socio-economic environments are over-represented. The KSC represents three major South African population groups, suggesting population variation; however, it is a highly unequal representation, with mainly individuals from the heterogenous mixed population group (SAC) that lived in and around the northern townships of Cape Town. Accompanying mandatory death certificates documented the cause of death for the majority of individuals, and most individuals also have a last-known address, confirming region-specific traits seen in the study.

We acknowledge that substantial biases and limitations exist, but the overall representation of various comparison groups in the KSC deems it useful for future studies at honours, masters and PhD level, to refine regional and population-specific reference data, and to play a supporting role in the research and training of specialists. Suggested data to be collected and interpreted include estimation of demographic parameters (age, sex, and ancestry origin), as well as human variation, genetic influence, trauma biomechanics, and pathological conditions.

If the existing biases in the collection are acknowledged and accounted for via suitable methodology, the KSC holds the potential to add a wealth of future research projects in anthropological studies.

CHAPTER 6: REFERENCE LIST

- Abou-Raya S, Abou-Raya A. 2006. Spinal tuberculosis: overlooked? *Journal of Internal Medicine* 260:160-163.
- Abrahams N, Jewkes R, Laubscher R, Hoffman M. 2006. Intimate partner violence: prevalence and risk factors for men in Cape Town, South Africa. *Violence and Victims* 21(2):247-264.
- Acheson RM, Blanco RA, Canosa C, Salomon JB. 1974. Height, weight and lines of arrested growth in young Guatemalan children. *American Journal of Physical Anthropology* 40:39-48.
- Acsadi G, Nemeskéri J. 1970. *History of human life span and mortality*. Budapest: Akadémia Kiadó pp346.
- Addolorato G, Capristo E, Greco AV, Stefanini GF, Gasbarrini G. 1998. Influence of chronic alcohol abuse on body weight and energy metabolism: is excess ethanol consumption a risk factor for obesity or malnutrition? *Journal of Internal Medicine* 244:387-395.
- Adebajo A, Davis P. 1994. Rheumatic diseases in African blacks. *Seminars in Arthritis and Rheumatism* 24(2):139-53.
- Adeyemo WL, Ladeinde AL, Ogunlewe MO, James O. 2005. Trends and characteristics of oral and maxillofacial injuries in Nigeria: a review of the literature. *Head and Face Medicine* 1(1):1-9. Doi:10.1186/1746-160X-1-7.
- Adi M, Ogden GR, Chisholm DM. 1990. An analysis of mandibular fractures in Dundee, Scotland (1977-1985). *British Journal of Oral Maxillofacial Surgery* 28:194-199. Doi:10.1016/0266-4356(90)90088-3
- Agaja SB, Ayorinde RO. 2008. Chronic osteomyelitis in Ilorin, Nigeria. *South African Journal of Surgery* 46(4):116-118.
- Agur AMR, Lee ML. 1999. *Grant's Atlas of Anatomy*, 10th ed. Baltimore: Lippincott Williams & Wilkins.
- Aihara T, Takahashi K, Ogasawara A, Itadera E, Ono Y, Moriya H. 2005. Intervertebral disc degeneration associated with lumbosacral transitional vertebrae: A clinical and anatomical study. *Journal of Bone and Joint Surgery [Br]* 87(5):687-91.
- Aktas D, Kalcioglu MT, Kutlu R, Oncel S. 2003. The relationship between the concha bullosa, nasal septal deviation and sinusitis. *Rhinology* 41(2):103-106.

- Albanese J. 2003. Identified skeletal reference collection and the study of human variation. Unpublished doctoral dissertation. Ontario: McMaster University, Canada.
- Alblas A, Greyling LM, Geldenhuys E. 2018. Composition of the Kirsten Skeletal Collection at Stellenbosch University. *South African Journal of Science* 114(1):1–6. Doi:10.17159/sajs.
- Albrecht TL, Scutter SD, Henneberg M. 2007. Radiographic method to assess the prevalence of sacral spina bifida occulta. *Clinical Anatomy* 20:170-174.
- Ali A, Tetalman MR, Fordham EW, Turner DA, Chiles JT, Patel SL, Schmidt KD. 1980. Distribution of hypertrophic pulmonary osteoarthropathy. *American Journal of Radiology* 134:771-780.
- Alvi A, Doherty T, Lewen G. 2003. Facial fractures and concomitant injuries in trauma patients. *Laryngoscope* 113: 102-106.
- Alvrus A. 1999. Fracture patterns among Nubians of Semna South, Sudanese Nubia. *International Journal of Osteoarchaeology* 9:417-429.
- Ameen S, Staub L, Ulrich S, Vock P, Ballmer F, Anderson S. 2005. Harris lines of the tibia across centuries: a comparison of two populations, medieval and contemporary in central Europe. *Skeletal Radiology* 34:279–284.
- Andrews JR, Shah NS, Gandhi N, Moll T, Friedland G. 2007. Multidrug-resistant and extensively drug-resistant tuberculosis: Implications for the HIV epidemic and antiretroviral therapy rollout in South Africa. *The Journal of Infectious Diseases* 196:482-490.
- Angel J. 1966. Porotic hyperostosis, anemias, malarias, and marshes in the prehistoric Eastern Mediterranean. *Science* 153(3737):760-763. Doi:10.1126/science.153.3737.760.
- Ansari MH. 2004. Maxillofacial fractures in Hamedan province, Iran: a retrospective study (1987-2001). *Journal of Craniomaxillofacial Surgery* 32:28-34.
- Arbuckle J, Olsen L, Howard M, Brullman J, Anctil C, Sklar D. 1996. Safe at home? Domestic violence and other homicides among women in New Mexico. *Annals of Emergency Medicine* 27(2):210-215.
- Armit I, Shapland F, Montgomery J, Beaumont J. 2015. Difference in Death? A lost neolithic inhumation cemetery with Britain's earliest case of rickets, at Balevullin, Western Scotland. *Proceedings of the Prehistoric Society* 81, 199–214. doi:10.1017/ppr.2015.7

- Armstrong DJ, McCausland EMA, Wright GD. 2007. Hypertrophic pulmonary osteoarthropathy (HPOA) (Pierre Marie-Bamberger syndrome): two cases presenting as acute inflammatory arthritis. Description and review of the literature. *Rheumatology International* 27:399-402.
- Atkins A. 1997. The illegal drugs trade and development in South Africa: Some observations. *Catholic Institute for International Relations*, London.
- Attiah M, Macyszyn L, Cahill P, 2014. Management of spondylolysis and spondylolisthesis in the pediatric population: A review. *Seminars in Spine Surgery* 26(4):230–237. Doi:10.1053/j.semss.2014.09.005.
- Aufderheide AC, Ragsdale B, Buikstra J, Ekber F, Vinh TN. 1997. Structure of the radiological “sunburst” pattern as revealed in an ancient osteosarcoma. *Journal of Paleopathology* 9:101-106.
- Aufderheide AC, Rodríguez-Martín C. 2011. *The Cambridge Encyclopedia of Human Paleopathology*, 1st ed. Cambridge University Press, Cambridge.
- Austin JF, Dick JM, Zwarenstein M. 2004. Gender disparity amongst TB suspects and new TB patients according to data recorded at the South African Institute of Medical Research laboratory for the Western Cape region of South Africa. *International Journal of Tuberculosis and Lung Disease* 8:435-439.
- Avelar RL, Antunes AA, Carvalho RWF, Bezerra PGCF, Neto PJO, Andrade ESS. 2009. Odontogenic cysts: a clinicopathological study of 507 cases. *Journal of Oral Science* 51:581-586.
- Avrahami E, Frishman E, Fridman Z, Azor M. 1994. Spina bifida occulta of S1 is not an innocent finding. *Spine* 19:12–15.
- Bachmann GF, Basad E, Rauber K, Damian MS, Rau WS. 1999. Degenerative joint disease on MRI and physical activity: a clinical study of the knee joint in 320 patients. *European Radiology* 9:145-152.
- Baird PA, Robinson GC, Buckler WS. 1967. Klippel–Feil syndrome. A study of mirror movement detected by electromyography. *American Journal of Diseases in Children* 113:546–551.

- Balasubramanian R, Garg R, Santha T, Gopi PG, Subramani R, Chandrasekaran V, Thomas A, Rajeswari R, Anandkrishan R, Perumal M, Niruparani C, Sudha G, Jaggarajamma K, Frieden TR, Narayanan PR. 2004. Gender disparities in tuberculosis: report from a rural DOTS program in south India. *International Journal of Tuberculosis and Lung Disease* 8:323-332.
- Banks E, Joshy G, Weber MF, Liu B, Grenfell R, Egger S, Paige E, Lopez AD, Sitas F, Beral V. 2015. Tobacco smoking and all-cause mortality in a large Australian cohort study: findings from a mature epidemic with current low smoking prevalence. *BMC Medicine* 13(1):38. Doi:10.1186/s12916-015-0281-z.
- Barber G, Watt I, Rogers J. 1997. A comparison of radiological and palaeopathological diagnostic criteria for hyperostosis frontalis interna. *International Journal of Osteoarchaeology* 7:157-164.
- Barker DJP. 1981. The epidemiology of Paget's disease. *Metabolic Bone Disease and Related Research* 4&5:231-234.
- Barnes E. 1994. *Developmental Defects in the Axial Skeleton in Paleopathology*. University Press of Colorado, Niwot.
- Batson E. (ed.). 1941. Series of reports and studies issued by the Social Survey of Cape Town: The poverty datum line (Report No. SS3). Department of Social Sciences, University of Cape Town.
- Beckles VLL, Wynn Jones H, Harrison WJ. 2010. Chronic haematogenous osteomyelitis in children. A retrospective review of 167 patients in Malawi. *Journal of Bone and Joint Surgery* 92B:1138-1143.
- Bellamy R, Ruwende C, Corrah T, McAdam KPWJ, Whittle HC, Hill AVS. 1998. Variations in the NRAMP1 gene and susceptibility to tuberculosis in West Africans. *The New England Journal of Medicine* 338:640-644.
- Bennike P. 1999. Facts or myths? A re-evaluation of cases of diagnosed tuberculosis in the past in Denmark. In: G Pálfi, O Dutour, J Deák, I Hutás (eds). *Tuberculosis: past and present. Budapest-Szeged*. Golden Book/Tuberculosis Foundation pp. 511-518.
- Berberi EF, Steckelberg JM, Osmon DR. 2010. Osteomyelitis. In: GLMandell, JE Bennett, R Dolin (eds). *Mandell, Douglas and Bennett's principles and practice of infectious diseases*, 7th ed. Philadelphia, PA: Churchill Livingstone pp. 1457–1468.

- Beresheim AC, Pfeiffer SK, Grynepas MD, Alblas A. 2018. Sex-specific patterns in cortical and trabecular bone microstructure in the Kirsten Skeletal Collection, South Africa. *American Journal of Human Biology* 30(3):e23108. Doi:10.1002/ajhb.23108.
- Bertolotti M. 1917. Contributo alla conoscenza dei vecchi differenziazione regionale del rachida con speciale regards all asimilazione sacrale della v. lombare. *Radiologique Medica* 4:113-144.
- Bertoni F, Bacchini P, Staals EL, Davidovitz P. 2005. Dedifferentiated parosteal osteosarcoma: The experience of the Rizzoli Institute. *Cancer* 103:2373–82.
- Bharati P, Basu A. 1990. Fertility, mortality and maternal anaemic status in a village population of West Bengal, India. *Annals of Human Biology* 17:331-335.
- Bhat S, Heurich AE, Vaquer RA, Dunn EK, Strashun AM. 1989. Hypertrophic osteoarthropathy associated with *Pneumocystis carinii* pneumonia in AIDS. *Chest* 96:1208-1209.
- Binder M, Roberts C, Spencer N, Antoine D, Cartwright C. 2014. On the antiquity of cancer: evidence for metastatic carcinoma in a young man from ancient Nubia (c. 1200BC). *PLoS ONE* 9(3): e90924. Doi:10.1371/journal.pone.0090924.
- Blom DE, Buikstra JE, Keng L, Tomczak P, Shoreman E, Stevens-Tuttle D. 2005. Anemia and childhood mortality: latitudinal patterning along the coast of pre-Columbian Peru. *American Journal of Physical Anthropology* 127:152-169.
- Bodavula P, Liang SY, Wu J, Van Tassell P, Marschall J. 2015. Pressure ulcer-related pelvic osteomyelitis: A neglected Disease? *Open Forum Infectious Diseases* 2(3):ofv112. Doi:10.1093/ofid/ofv112.
- Boeseken AJ. 1977. *Slaves and free Blacks at the Cape 1658-1700*; Tafelberg Publishers, Cape Town.
- Boldsen JL, Milner GR, Konigsberg LW, Wood JW. 2002. Transition Analysis: A new method for estimating age from skeletons. In: RD Hoppa & JW Vaupel (eds.). *Paleodemography: Age distributions from skeletal samples*. Cambridge University Press, Cambridge pp. 73-106.
- Bonola A. 1956. Surgical treatment of the Klippel–Feil syndrome. *Journal of Bone Joint Surgery [Br]* 38B:440–449.

- Bonsell S, Pearsall AW, Heitman RJ, Helms CA, Major NM, Speer KP. 2000. The relationship of age, gender and degenerative changes observed on radiographs of the shoulder in asymptomatic individuals. *Journal of Bone and Joint Surgery* 82B: 1135-1139.
- Boone D, Parsons D, Lachmann SM, Sherwood T. 1985. Spina bifida occulta: lesion or anomaly? *Clinical Radiology* 36:159-161.
- Boston HC, Dahlin DC, Ivins JC, Cupps RE. 1974. Malignant lymphoma (so called reticululum cell sarcoma) of bone. *Cancer* 34:1131-1137.
- Botto L, Moore C, Khoury MJ, Ericksen JD. 1999. Neural tube defects. *New England Journal of Medicine* 341(20):1509-19. Doi:10.1056/NEJM199911113412006.
- Bothwell TH, Seftel H, Jacobs P, Torrance JD, Baumslag N. 1964. Iron overload in Bantu subjects. Studies on the availability of iron in Bantu beer. *The American Journal of Clinical Nutrition* 14(1):47-51. Doi: 10.1093/ajcn/14.1.47.
- Bowman RM, Mathews S, Myers J. 2010. Applying upstream interventions for interpersonal violence prevention: an uphill struggle in low- to middle-income contexts. *Health Policy* 97: 62-70.
- Brabin L, Brabin BJ. 1992. The cost of successful adolescent growth and development in girls in relation to iron and vitamin A status. *American Journal of Clinical Nutrition* 55: 955-958.
- Braithwaite VS, Freeman R, Greenwood CL, Summers DM, Nigdikar S, Lavy CB, Offiah AC, Bishop NJ, Cashman J, Prentice A. 2016. The etiology of rickets-like lower limb deformities in Malawian children. *Osteoporosis International* 27(7):2367–2372.
- Braun J, Sieper J. 2007. Ankylosing spondylitis. *Lancet* 369:1379-1390.
- Brickley M. 2006. Rib fractures in the archaeological record: A useful source of sociocultural information? *International Journal of Osteoarchaeology* 16:61-75.
- Brickley M, Ives R. 2006. Skeletal manifestations of infantile scurvy. *American Journal of Physical Anthropology* 129:163-172.
- Brickley M, Ives R. 2008. *The bioarchaeology of metabolic bone disease*. Academic Press, San Diego. Elsevier Science and Technology, Oxford.

- Brickley M, Smith M. 2006. Culturally determined patterns of violence: Biological anthropological investigations at a historic urban cemetery. *American Anthropologist* 108(1):163-177.
- Brickley MB. 2018. Cribra orbitalia and porotic hyperostosis: A biological approach to diagnosis. *American Journal of Physical Anthropology*. Doi:10.1002/ajpa.23701
- Bringhurst FR, Demay MB, Kronenberg HM. 2008. Hormones and disorders of mineral metabolism. In: HM Kronenberg, S Melmed, KS Polonsky, PR Larsen (eds.). *Williams Textbook of Endocrinology* 11th ed. Philadelphia: Elsevier.
- Brink O, Vesterby A, Jersen J. 1998. Pattern of injuries due to interpersonal violence. *Injury* 29(9):705-709.
- Bron JL, Van Royen BJ, Wuisman PIJM. 2007. The clinical significance of lumbosacral transitional anomalies. *Acta Orthopaedica Belgica* 73:687-695.
- Brook A. 2009. Multilevel complex interactions between genetic, epigenetic and environmental factors in the etiology of anomalies of dental development. *Archives of Oral Biology* 54:3-17.
- Brooks A, Barker P. 2003. Missile and explosive wounds. *Surgery* 21(8):190-192.
- Brooks A, Macnab C, Boffard K. 1999. South Africa. *Trauma Quarterly* 14(3):301-310.
- Brooks S, Suchey JM. 1990. Skeletal age determination based on the os pubis: A comparison of the Acsadi-Nemeskéri and Suchey-Brooks methods. *Human Evolution* 5(3): 227 -238.
- Brothwell DR (ed.) 1968. *The skeletal biology of earlier human populations*. Pergamon Press.
- Brothwell DR. 1981. *Digging up bones the excavation, treatment, and study of human skeletal remains*. (3rd ed.), Ithaca, N.Y, London: Cornell University Press. British Museum (Natural History).
- Brown M, Ortner DJ. 2011. Childhood scurvy in a medieval burial from Macvanska Mitrovica, Serbia. *International Journal of Osteoarchaeology* 21:197-207.
- Buckberry JL, Chamberlain AT. 2002. Age estimation from the auricular surface of the ilium: A revised method. *American Journal of Physical Anthropology* 119: 231- 239.

- Butchart A, Brown DSO. 1991. Non-fatal injuries due to interpersonal violence in Johannesburg-Soweto: incidence, determinants and consequences. *Forensic Science International* 52:35-51.
- Buikstra JE, Ubelaker DH. 1994. Standards: For data collection from human skeletal remains. *Arkansas Archaeological Survey Research Series No. 44*. New York: Wiley-Liss.
- Bundy C. 2016. *A Jacana Pocket History – Poverty in South Africa: past and present*. Jacana Media 176pp.
- Byers S. 1991. Calculation of age at formation of radiopaque transverse lines. *American Journal of Physical Anthropology* 85(3):339–343. Doi:10.1002/ajpa.1330850314.
- Byrnes, RM. 1996. (ed.) *South Africa: A country study*. Washington: GPO for the Library of Congress [Online]. [n.d.]. Available: <http://countrystudies.us/south-africa> [Accessed: 2018, May 6].
- Caffey J. 1978. *Pediatric X-ray Diagnosis: Textbook for students and practitioners of pediatrics, surgery and radiology*. 7th ed. Yearbook Medical Publishers Inc., Chicago.
- Calhoun KH, Waggenpack GA, Simpson CB, Hokanson JA, Bailey BJ. 1991. CT evaluation of the paranasal sinuses in symptomatic and asymptomatic patients. *Otolaryngology-Head and Neck Surgery* 104:480–483.
- Cammisa M, De Serio A, Guglielmi G. 1998. Diffuse idiopathic skeletal hyperostosis. *European Journal of Radiology* 27:S7-S11.
- Campillo D. 2005. Palaeoradiology. III: Neoplastic conditions. *Journal of Paleopathology* 17: 93–135.
- Cao Y, Willett WC, Rimm EB, Stampfer MJ, Giovannucci EL. 2015. Light to moderate intake of alcohol, drinking patterns, and risk of cancer: results from two prospective US cohort studies. *BMJ* 351: h4238.
- Capasso LL. 2005. Antiquity of cancer. *International Journal of Cancer* 113(1): 2–13.
- Caracta CF. 2003. Gender differences in pulmonary disease. *The Mount Sinai Journal of Medicine* 70:215-224.
- Carlson BM. 2014. *Human Embryology and Developmental Biology* (5th ed). Elsevier, Mosby Inc. Philadelphia, USA.

- Carroll KB, Doyle L. 1974. A common factor in hypertrophic osteoarthropathy. *Thorax* 29:262-264.
- Casselbrant ML, Mandel EM, Kurs-Lasky M, Rockette HE, Bluestone CD. 1995. Otitis media in a population of black American and white American infants, 0–2 years of age. *International Journal of Pediatric Otorhinolaryngology* 33(1):1–16.
- Castellvi AE, Goldstein LA, Chan DK. 1984. Lumbosacral transitional vertebra and their relationship with lumbar extradural defects. *Spine* 9(5):493-495.
- Cawley WD, Paine RR. 2015. Skeletal indicators of reactive arthritis: A case study comparison to other skeletal conditions, such as rheumatoid arthritis, ankylosing spondylitis, ankylosing sero-negative SpA, and DISH. *International Journal of Paleopathology* 11:70–74. Doi: 10.1016/j.ijpp.2015.10.001
- Chaljub G, Johnson RF, Sitton CW. 1999. Unusually exuberant hyperostosis frontalis interna: MRI. *Neuroradiology* 41(1), 44-45. Doi: 10.1007/s002340050703.
- Chan-Yeung M, Noertjojo K, Chan SL, Tam CM. 2002. Sex differences in tuberculosis in Hong Kong. *International Journal of Tuberculosis and Lung Disease* 6:11-18.
- Chen Y, Olckers A, Schurr TG, Kogelnik AM, Huoponen K, Wallace DC. 2000. mtDNA variation in the South African Kung and Khwe - and their genetic relationships to other African populations. *American Journal of Human Genetics*. 66:1362-1383. <https://doi.org/10.1086/302848>.
- Chhem RK, Brothwell DR. 2008. *Paleoradiology. Imaging Mummies and Fossils*. Springer-Verlag Berlin Heidelberg.
- Chrcanovic BR, Freire-Maia B, Napier de Souza L, de Oliveira Araujo V, de Abreu MHNG. 2004. Facial fractures: a 1-year retrospective study in a hospital in Belo Horizonte. *Brazilian Oral Research* 18(4): 322-328.
- Christopher AJ. 2001. Urban segregation in post-apartheid South Africa. *Urban Studies* 38:449–466.
- Chung KC, Spilson SV, Arbor A. 2001. The frequency and epidemiology of hand and forearm fractures in the United States. *The Journal of Hand Surgery* 26A:908-915.

- Clark S. 1978. Markers of metabolic insult: The association of radiopaque transverse lines, enamel hypoplasias and enamel histopathologies in a prehistoric skeletal sample. Unpublished dissertation. University of Colorado, Boulder.
- Clarke S. 1982. The association of early childhood enamel hypoplasia and radiopaque transverse lines in a culturally diverse perihistoric skeletal sample. *Human Biology* 54:77–84.
- Cluver EH. 1959. *Public health in South Africa*, 6th ed. Central News Agency Ltd. 364 p.
- Cocheton JJ, Fineltain L, Poulet J. 1974. Le syndrome de Morgagni-Morel, mythe ou realite? *Sem Hop* 50(47–48):2945–2950.
- Coelho M, Sequeira F, Luiselli D, Belezza S, Rocha J. 2009. On the edge of Bantu expansions: mtDNA, Y chromosome and lactase persistence genetic variation in southwestern Angola. *BMC Evolutionary Biology* 9:80. Doi: 10.1186/1471-2148-9-80.
- Coetzee R. 1977. *The South African culinary tradition*. C. Struik Publishers, Cape Town, South Africa.
- Cohen MN, Armelagos GJ (eds). 1984. *Paleopathology at the origins of agriculture*. New York: Academic Press. pp 615.
- Coleman RE. 2006. Clinical features of metastatic bone disease and risk of skeletal morbidity. *Clinical Cancer Research* 12(20):6243s–6249s. Doi:10.1158/1078-0432.ccr-06-0931.
- Coovadia H, Jewkes R, Barron P, Sanders D, McIntyre D. 2009. Health in South Africa. The health and health system of South Africa: historical roots of current public health challenges, *Lancet* 374: 817–34.
- Corrigall J, Matzopoulos R. 2013. Violence, alcohol misuse and mental health: gaps in the health system's response. In: A Padarath & R English (eds.). *South African health review 2012/13*. Cape Town, South Africa: Health Systems Trust pp.103-14.
- Cosgrove KP, Wang S, Kim S-J, McGovern E, Nabulsi N, Gao H, Labaree D, Tagare HD, Sullivan JM, Morris ED. 2014. Sex Differences in the brain's Dopamine signature of cigarette smoking. *Journal of Neuroscience* 34(50):16851–16855. Doi:10.1523/jneurosci.3661-14.2014.
- Court-Brown CM, McBirnie J. 1995. The epidemiology of tibial fractures. *The Journal of Bone and Joint Surgery* 3,77B:417-421.

- Cox M, Mays S. 2000. *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media.
- Cukurova I, Yaz A, Gumussoy M, Yigitbasi OG, Karaman Y. 2012. A patient presenting with concha bullosa in another concha bullosa: a case report. *Journal of Medical Case Reports* 6:87. Doi: 10.1186/1752-1947-6-87.
- Cummings SR, Nevit MC. 1994. Non-skeletal determinants of fractures: The potential importance of the mechanism of falls. *Osteoporosis International* 1:67-70.
- Dalby G. 1994. The palaeopathology of middle ear and mastoid disease: a comparison of methods of investigation and results of the examination of the temporal bones of skeletal material from Romano-British, Anglo-Saxon and late medieval cemeteries. Unpublished doctoral dissertation. Bradford: The University of Bradford; England.
- Daya M, van der Merwe L, Galal U, Möller M, Salie M, Chimusa ER, Galanter JM, van Helden PD, Henn BM, Gignoux CR, Hoal E. 2013. A panel of ancestry informative markers for the complex five-way admixed South African coloured population. *PLoS One* 20;8(12):e82224. Doi: 10.1371/journal.pone.0082224.
- Dayal MR, Kegley ADT, Strkalj G, Bidmos MA, Kuykendall KL. 2009. The history and composition of the Raymond A. Dart Collection of human skeletons at the University of the Witwatersrand, Johannesburg, South Africa. *American Journal of Physical Anthropology* 140:324-335. Doi.org/10.1002/ajpa.21072.
- De Filippo C, Barbieri C, Whitten M, Mpoloka MS, Gunnarsdottir ED, Bostoen K, Nyambe T, Beyer K, Schreiber H, de Knijff P, Luiselli D, Stoneking M, Pakendorf B. 2011. Y-chromosomal variation in sub-Saharan Africa: Insights into the history of Niger-Congo groups. *Molecular Biology and Evolution* 28(3):1255-1269. Doi:10.1093/molbev/msq312.
- De La Cova C. 2010. Cultural patterns of trauma among 19th-century-born males in cadaver collections. *American Anthropologist* 112(4):589-606. Doi: 10.1111/j.1548-1433.201.01278.x.
- De Wit E, Delpont W, Rugamika CE, Meintjes A, Moller M, van Helden PD, Seoghe C, Hoal EG. 2010. Genome-wide analysis of the structure of the South African Coloured population in the Western Cape. *Human Genetics* 128(2):145–153. Doi:10.1007/s00439-010-0836-1.

- Delanghe JR, Langlois MR, De Buyzere ML, Torck, MA. 2007. Vitamin C deficiency and scurvy are not only a dietary problem but are codetermined by the haptoglobin polymorphism. *Clinical Chemistry* 53:1397–1400. Doi:10.1373/clinchem.2007.088658.
- Delpont EG, Cucuzzella TR, Kim N, Marley J, Pruitt C, Delpont AG. 2006. Lumbosacral transitional vertebrae: incidence in a consecutive patient series. *Pain Physician* 9(1):53-6.
- Den Boon S, Van Lill SWP, Borgdorff MW, Enarson DA, Verver S, Bateman ED, Irusen E, Lombard CJ, White NW, de Villiers C, Beyers N. 2007. High prevalence of tuberculosis in previously treated patients, Cape Town, South Africa. *Emerging Infectious Diseases* 13:1189-1194.
- Devriendt W, Piercecchi-Marti MD, Adalian P, Sanvoisin A, Dutour OJ, Leonetti G. 2005. Hyperostosis frontalis interna: forensic issues. *Journal of Forensic Sciences* 50(1):143-6.
- Dharati K, Nagar SK, Ojaswini M, Dipali T, Paras S, Sucheta P. 2012. A study of sacralisation of fifth lumbar vertebra in Gujarat. *National Journal of Medical Research* 2:211-213.
- Dickinson CJ. 1993. The etiology of clubbing and hypertrophic osteoarthropathy. *European Journal of Clinical Investigation* 23:330-338.
- Doll R, Peto R, Wheatley K, Gray R, Sutherland I. 1994. Mortality in relation to smoking: 40 years' observations on male British doctors. *BMJ* 309(6959):901-911.
- Dorfman HD, Czerniak B. 1998. *Bone Tumors*. Mosby, Inc. St. Louis.
- Dreizen S, Currie C, Gilley E, Spies T. 1964. The influence of age and nutritional status on bone scar formation in the distal end of the growing radius. *American Journal of Physical Anthropology* 22:375-377.
- Dubey P, Ha CS, Besa PC, Fuller L, Cabanillas F, Murray J, Hess MA, Cox JD. 1997. Localized primary malignant lymphoma of bone. *International Journal of Radiation Oncology, Biology, Physics* 37:1087-1093.
- Edwards JK, Thiongo A, Van den Bergh R, Kizito W, Kosgei RJ, Sobry A, Reid AJ. 2014. Preventable but neglected: Rickets in an informal settlement, Nairobi, Kenya. *Public Health Action* 4(2):122–127.
- Eisenstein S. 1978. Spondylolysis: a skeletal investigation of two population groups. *Journal of Bone and Joint Surgery* 60B:488-494.

- Elder CJ, Bishop NJ. 2014. Rickets. *Lancet* 383 (9929): 1665–1676.
- Eldeirawi K, Persky VW. 2004. History of ear infections and prevalence of asthma in a national sample of children aged 2 to 11 years: The third national health and nutrition examination survey, 1988 to 1994. *Chest* 125(5):1685–1692.
- Eldridge W, Holm G. 1940. Incidence of hyperostosis frontalis interna in female patients admitted to a mental hospital. *American Journal of Roentgenology and Radiation Therapy* 43(3):356-359.
- El-Najjar MY. 1976. Maize, malaria and the anemias in the Pre-Columbian New World. *Yearbook of Physical Anthropology* 20:329-337.
- El Titi A, El Zain M, Attar D. 1987. Tuberculosis of bones and joints at the Riyadh Armed Forces Hospital. *Saudi Medical Journal* 8:147–54.
- Endere ML. 2002. The reburial issue in Argentina: a growing conflict. In: C Fforde, J Hubert, P Turnbull (eds.). *The dead and their possessions: repatriation in principle, policy and practice*. London: Routledge pp. 266–283.
- Erasmus Z. 2012. Apartheid race categories: Daring to question their continued use. *Transformation: Critical Perspectives on Southern Africa* 79(1):1-11. Doi:10.1353/trn.2012.0008.
- Eubanks JD, Cheruvu VK. 2009. Prevalence of sacral spina bifida occulta and its relationship to age, sex, race, and the sacral table angle. *Spine* 34(15): 1539–1543. Doi:10.1097/brs.0b013e3181a98560.
- Facchini F, Rastelli E, Brasili P. 2004. Cribra orbitalia and cribra cranii in Roman skeletal remains from the Ravenna area and Rimini (I-IV Century AD). *International Journal of Osteoarchaeology* 14(2):126–136. Doi:10.1002/oa.717.
- Fain O. 2005. Musculoskeletal manifestations of scurvy. *Joint Bone Spine* 72: 124–128.
- Favus M. (ed.). 1999. Primer on the metabolic bone diseases and disorders of mineral metabolism. 4th ed. Philadelphia: Lippincott William & Wilkins.
- Fennell KJ, Trinkaus E. 1997. Bilateral femoral and tibial periostitis in the La Ferrassie 1 Neanderthal. *Journal of Archaeological Science* 24:985–99.

- Fernandes T, Costa C. 2007. Klippel-Feil syndrome with other associated anomalies in a medieval Portuguese skeleton (13th-15th century). *Journal of Anatomy* 211: 681-685.
- Fibiger L, Ahlström T, Bennike P, Schulting RJ. 2013. Patterns of violence-related skull trauma in Neolithic Southern Scandinavia. *American Journal of Physical Anthropology* 150:190-202.
- Fidas A, MacDonald HL, Elton RA, Wild SR, Chisholm GD, Scott R. 1987. Prevalence and patterns of spina bifida occulta in 2707 normal adults. *Clinical Radiology* 38:537–542.
- Flohr S, Schultz M. 2009. Osseous changes due to mastoiditis in human skeletal remains. *International Journal of Osteoarchaeology* 19: 99–106
- Fourie B. 2011. *The burden of tuberculosis in South Africa*. [Online]. Available: <http://www.sahealthinfo.org/tb/tburden.htm>. [Accessed: 2018, May 5].
- Fox T. 2011. Domestic violence as a major cause of trauma in Western Province, Papua New Guinea. *Pacific Health Dialog* 17(1):65 - 76.
- Frederickson BE, Baker D, McHolick WJ, Yuan HA, Lubicky JP. 1984. The natural history of spondylolysis and spondylolisthesis. *Journal of Bone and Joint Surgery* 66:699-707.
- Friedling LJ. 2004. Dental modification practices on the Cape Flats in the Western Cape. Unpublished MSc thesis. Cape Town: University of Cape Town; South Africa.
- Friedling LJ. 2007. Grave Tales: An osteological assessment of health and lifestyle from 18th and 19th century burial sites around Cape Town. Unpublished doctoral dissertation. Cape Town: University of Cape Town; South Africa.
- Friedling LJ, Morris AG. 2007. Pulling teeth for fashion: dental modification in modern day Cape Town, South Africa: scientific. *South African Dental Journal* 62(3):106-108.
- Garn S, Silverman F, Hertzog K, Rothmann G. 1968. Lines and bands of increased density, their growth implications to growth and development. *Medical Radiographs and Photographs* 44:58–89.
- Gassner R, Tuli T, Hächl O, Rudisch A, Ulmer H. 2003. Cranio-maxillofacial trauma: a 10 year review of 9,543 cases with 21,067 injuries. *Journal of Cranio-Maxillo-Facial Surgery* 31:51-61.

- Gay RE, Ilharreborde B, Zhao K, Zhao C, An K. 2006. Sagittal plane motion in the human lumbar spine: Comparison of the in vitro quasistatic neutral zone and dynamic motion parameters. *Clinical Biomechanics* 21:914-19.
- Geber J, Murphy E. 2012. Scurvy in the Great Irish Famine: Evidence of vitamin C deficiency from a mid-19th century skeletal population. *American Journal of Physical Anthropology* 148(4):512–524. Doi:10.1002/ajpa.22066.
- Geldenhuis E. 2014. Morphological description and distribution of tuberculous lesions. Unpublished doctoral dissertation, Cape Town: Stellenbosch University; South Africa.
- Geldenhuis E, Burger, EH, Alblas A, Greyling LM, Kotzé SH. 2016. The association between healed skeletal fractures indicative of interpersonal violence and alcoholic liver disease in a cadaver cohort from the Western Cape, South Africa. *Alcohol* 52:41-48. Doi:10.1016/j.alcohol.2016.02.003.
- Gennari L, Di Stefano M, Merlotti D, Giordano N, Martini G, Tamone C, Zatteri R, De Lucchi R, Baldi C, Vattimo A, Capoccia S, Burrioni L, Geraci S, De Paola V, Calabro A, Avanzati A, Isaia G, Nuti R. 2005. Prevalence of Paget's disease of bone in Italy. *Journal of Bone and Mineral Research* 20:1845-1850.
- Gershon-Cohen J, Schraer H, Blumberg N. 1955. Hyperostosis frontalis interna among the aged. *American Journal of Roentgenology* 73:396-397.
- Giliomee H, Mbenga B. 2007. *New history of South Africa*. Tafelberg Publishers, Cape Town.
- Gindhart P. 1969. The frequency of appearance of transverse lines in the tibia in relation to childhood illnesses. *American Journal of Physical Anthropology* 31:1722.
- Glab H, Szostek K, Kaczanowski K, 2006. Hyperostosis frontalis interna, a genetic disease?: Two medieval cases from Southern Poland. *Homo* 57(1):19–27.
- Glucksmann A. 1981. *Sexual dimorphism in human and mammalian biology and pathology*. London: Academic Press.
- Goepferd S, Flaitz C. 1981. Enamel hypoplasia associated with congenital hypoparathyroidism. *Pediatric Dentistry* 3:196–200.
- Goldblatt M, Cremin J. 1978. Osteoarticular tuberculosis: its presentation in Coloured races. *Clinical Radiology*. 29:669-677.

- Goodman AH, Armelagos G. 1988. Childhood stress and decreased longevity in a prehistoric population. *American Anthropology* 90:936–944.
- Goodman AH, Armelagos G, Rose J. 1980. Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Human Biology* 52(3):515–528.
- Goodman AH, Clark G. 1981. Harris lines as indicators of stress in prehistoric Illinois populations. In: D Martin & M Blumsted (eds.). *In biocultural adaptation: Comprehensive approaches to skeletal analysis. Research Reports* 20:3547.
- Goodman AH, Rose JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *American Journal of Physical Anthropology* 83(Suppl 11):59-110.
- Goodman AH, Thomas RB, Swedlund AC, Armelagos GJ. 1988. Biocultural perspectives on stress in prehistoric, historical and contemporary population research. *Yearbook of Physical Anthropology* 31:169–202.
- Goosen J, Bowley DM, Degiannis E, Plani F. 2003. Trauma care systems in South Africa. *Injury* 34:704–708.
- Gordeuk VR, McLaren CE, MacPhail AP, Deichsel G, Bothwell TH. 1996. Associations of iron overload in Africa with hepatocellular carcinoma and tuberculosis: Strachan’s 1929 thesis revisited. *Blood* 87(8):3470- 3476.
- Gossage PJ, Snell CL, Parry CDH, Marais AS, Barnard R, de Vries M, Blankenship J, Seedat S, Hasken JM, May PA. 2014. Alcohol use, working conditions, job benefits, and the legacy of the “dop” system among farm workers in the Western Cape Province, South Africa: Hope despite high levels of risky drinking. *International Journal of Environmental Research and Public Health* 11(7), 7406–7424.
- Govender T, Barnes JM, Pieper CH. 2010. Living in low-cost housing settlements in Cape Town, South Africa – the epidemiological characteristics associated with increased health vulnerability. *Journal of Urban Health* 87:899-911.
- Govender T, Barnes JM, Pieper CH. 2011. Housing conditions, sanitation status and associated health risks in selected subsidized low-cost housing settlements in Cape Town, South Africa. *Habitat International* 35:335-342.
- Gray P. 1967. Radiography of ancient Egyptian mummies. *Medical Radiology* 43:3444.

- Gray SW, Romaine CB, Skandalakis JE. 1964. Congenital fusion of the cervical vertebrae. *Surgery, Gynecology Obstetrics* 118:373-385.
- Greenberg D, Givon-Lavi N, Broides A, Blancovich I, Peled N, Dagan R. 2006. The contribution of smoking and exposure to tobacco smoke to *Streptococcus pneumoniae* and *Haemophilus influenzae* carriage in children and their mothers. *Clinical Infectious Diseases* 42(7): 897–903.
- Gregg JB, Steele JP, Holzhueter A. 1965. Roentgenographic evaluation of temporal bones from South Dakota Indian burials. *American Journal of Physical Anthropology* 23: 51–62.
- Gregory JK, Lachman N, Camp CL, Chen LP, Pawlina W. 2009. Restructuring a basic science course for core competencies: an example from anatomy teaching. *Medical Teacher* 31:855-861.
- Griffith TE. 1979. Epidemiology of otitis media - an interracial study. *Laryngoscope* 89(1):22-30.
- Groenewald P, Msemburi W, Morden E, Zinyakatira N. 2014. *Western Cape mortality profile 2011*. Cape Town, South Africa: South African Medical Research Council. ISBN: 978-1-920618-23-0.
- Groza V, Simalecsik A, Bejenaru L. 2012. Frequency of spina bifida occulta and other occulta spinal dysraphisms in the medieval population of Iași City: Skeleton paleopathology in the Necropolis discovered in the eastern part of the Princely Court (“Curtea Domneasă”), 17th Century. *Biologie animală* LVIII:195-204.
- Guañabens N, Garrido J, Gobbo M, Morales-Piga A, del Pino J, Torrijos A, Descalzo MA, García FJB, Cros JRR, Carbonell J, Pérez MR, Tornero J, Carmona L. 2008. Prevalence of Paget's disease of bone in Spain. *Bone* 43:1006–1009.
- Hackett CJ. 1975. An introduction to diagnostic criteria of syphilis, treponarid and yaws (treponematoses) in dry bones and some implications. *Virchows Archaeology. Anatomical Pathology Anatomy and Histology* 368: 229 - 341.
- Haglund WD, Sorg MH. (eds.). 2002. *Advances in Forensic Taphonomy: Method, Theory and Archaeological perspectives*. CRC Press, Inc., Boca Raton.
- Halcrow SE, Harris NJ, Beavan N, Buckley HR. 2014. First bioarchaeological evidence of probable scurvy in Southeast Asia: Multifactorial etiologies of vitamin C deficiency in a

tropical environment. *International Journal of Paleopathology* 5:63–71. Doi:10.1016/j.ijpp.2014.01.004.

Hammarstan JF, O’Leary J. 1957. The features and significance of hypertrophic osteoarthropathy. *Archives of Internal Medicine* 99:431-441.

Hampel JS, Taylor CA, Johnston CS. 2004. Vitamin C deficiency and depletion in the United States: The third national health and nutrition examination survey, 1988 to 1994. *American Journal of Public Health* 94(5):870–875.

Hannallah D, White A, Goldberg G, Albert T. 2007. Diffuse idiopathic skeletal hyperostosis. *Operative Techniques in Orthopaedics* 17: 174-177.

Harris H. 1933. *Bone growth in health and disease*. London: Oxford Medical Publications.

Hart P, Aldred M, Crawford P, Wright N, Hart T, Wright J. 2002. Amelogenesis imperfecta phenotype-genotype correlations with two amelogenin gene mutations. *Archives of Oral Biology* 47(4):261–265.

Hatipoğlu HG, Çetin MA, Yüксе E. 2005. Concha bullosa types: their relationship with sinusitis, ostiomeatal and frontal recess disease. *Diagnostic and Interventional Radiology*, 11: 145-149.

Hatch JW, Willey PS, Hunt EE. 1983. Indicators of status-related stress in Dallas society: Transverse lines and cortical thickness in long bones. *Midcontinental Journal of Archaeology* 8:49-72.

Helmi C, Pruzansky S. 1980. Craniofacial and extracranial malformations in the Klippel–Feil syndrome. *Cleft Palate Journal* 17:65–88.

Henneberg RJ, Henneberg M. 1999. Variation in the closure of the sacral canal in the skeletal sample from Pompeii, Italy, 79 AD. *Perspectives in Human Biology* 4:177–188.

Henschen F. 1949. *Morgagni's Syndrome*. London: Oliver and Boyd.

Hensinger RN, Lang JE, MacEwen GD. 1974. Klippel–Feil syndrome. A constellation of associated anomalies. *Journal of Bone and Joint Surgery* 56:1246–1253.

Hershkovitz I, Greenwald C, Rothschild BM, Latimer B, Dutour O, Jellema LM, Wish-Baratz. 1999. Hyperostosis frontalis interna: an anthropological perspective. *American Journal of Physical Anthropology* 109:303-325.

- Hertel R, Rothenfluh DA. 2010. Fractures of the shafts of the radius and ulna. In: RW Bucholz, J Heckman, CM Court-Brown (eds.). *Rockwood and Green's fractures in adults*, 6th ed. Philadelphia: Lipincott Williams and Wilkins.
- Heyning FH, Hogendoorn PC, Kramer MH. 1999. Primary non-Hodgkin's lymphoma of bone: a clinicopathological investigation of 60 cases. *Leukemia* 13:2094-98.
- Hofbauer LC, Rachner TD, Coleman RE, Jakob F. 2014. Endocrine aspects of bone metastases. *Lancet Diabetes Endocrinology* 2(6):500–12. Doi:10.1016/S2213-8587(13)70203-1.
- Hofman K, Primack A, Keusch G, Hrynkow S. 2005. Addressing the growing burden of trauma and injury in low-and middle-income countries. *American Journal of Public Health* 95:13–17. Doi: 10.2105/AJPH.2004.039354.
- Hofmann G, Gonschorek O, Hofmann GO, Buhren V. 1997. Stabilisierungsverfahren bei osteomyelitis. *Osteosynthesis International* 5:226–231.
- Hogendoorn PCW. 2010. Bone sarcomas: ESMO Clinical practice guidelines for diagnosis, treatment and follow-up. *Annals of Oncology* 21 (Suppl 5): v204v213. Doi:10.1093/annonc/mdq223.
- Hogler W. 2015. Complications of vitamin D deficiency from the foetus to the infant: One cause, one prevention, but who's responsibility? Best practice and research. *Clinical Endocrinology & Metabolism* 29(3):385–398.
- Holland TD, O'Brien MJ. 1997. Parasites, porotic hyperostosis, and the implications of changing Perspectives. *American Antiquity* 62(2)183-193.
- Hsieh CJ, Vanderford JD, Moreau SR, Prong T. 2000. Lumbosacral transitional segments: classification, prevalence, and effect on disk height. *Journal of Manipulative and Physiological Therapeutics* 23:483-489.
- Huben RP. 1992. Hormone therapy of prostatic bone metastases. *Advanced Experimental Medical Biology* 324: 305-16. Doi:10.1007/978-1-4615-3398-6_33.
- Hughes RJ, Saifuddin A. 2006. Numbering of lumbosacral transitional vertebrae on MRI: role of the iliolumbar ligaments. *AJR American Journal Roentgenology* 187(1):59-65.
- Hunt D, Albanese J. 2005. History and demographic composition of the Robert J. Terry Anatomical Collection. *American Journal of Physical Anthropology* 127:406-417.

- Huss-Ashmore R, Goodman AH, Armelagos GJ. 1982. Nutritional inference from paleopathology. In: MB Schiffer (ed.), *Advances in Archaeological Method and Theory*, vol. 5. New York: Academic Press pp 395-474.
- Hussain K, Wijetunge DB, Grubnic S, Jackson IT. 1994. A comprehensive analysis of craniofacial trauma. *The Journal of Trauma* 36:34-47.
- Infante P, Gillespie G. 1974. An epidemiological study of linear enamel hypoplasia of deciduous anterior teeth in guatemalan children. *Archives of Oral Biology* 19:1055–1061.
- Inwood K, Masakure O. 2013. Poverty and physical well-being among the coloured population in South Africa. *Economic History of Developing Regions* 28(2):56–82.
- Isaacson C, Bothwell TH, MacPhail AP, Simon MO. 1985. The iron status of urban black subjects with carcinoma of the oesophagus. *South African Medical Journal* 67:591-593.
- İşcan MY, Loth SR, Wright RK. 1985. Age estimation from the rib by phase analysis: White females. *Journal of Forensic Sciences* 30(3):853-863.
- İşcan MY, Kennedy KAR. (eds.). 1989. *Reconstruction of life from the skeleton*. Alan R. Liss, New York.
- İşcan MY, Steyn M. 2013. *The human skeleton in forensic Mmdicine*, 3rd ed. Springfield: Charles C Thomas Publisher, Illinois, USA.
- Ito T, Goto K, Yoh K, Niho S, Ohmatsu H, Kubota K, Nagai K, Miyazaki E, Kumamoto T, Nishiwaki Y. 2010. Hypertrophic pulmonary osteoarthropathy as a paraneoplastic manifestation of lung cancer. *Journal of Thoracic Oncology* 5:976-980.
- Jacobson CK, Amoateng AY, Heaton TB. 2004. Inter-racial marriages in South Africa. *Journal of Comparative Family Studies* 35:443.
- Jewkes R, Levin J, Penn-Kekana L. 2002. Risk factors for domestic violence: findings from a South African cross-sectional study. *Social Sciences and Medicine* 55:1603-1617.
- Jha P. 2009. Avoidable global cancer deaths and total deaths from smoking. *Nature Reviews Cancer* 9(9):655-664. Doi:10.1038/nrc2703.
- Jin X, Wang B, Antony B, Zhu Z, Han W, Cicuttini F, Wluka A, Winzenberg T, Blizzard L, Jones G, Ding C. 2017. Associations between endogenous sex hormones and MRI structural

changes in patients with symptomatic knee osteoarthritis. *Osteoarthritis and Cartilage* 25: 1100-1106.

Jones KDJ, Hachmeister CU, Khasira M, Lorna Cox L, Schoenmakers I, Munyi C, Nassir HS, Hüntten-Kirsch B, Prentice A, Berkley JA. 2017. Vitamin D deficiency causes rickets in an urban informal settlement in Kenya and is associated with malnutrition. *Maternal and Child Nutrition* 14(1):e12452. Doi:10.1111/ mcn.12452.

Jordana X, Galtés I, Couto AR, Gales L, Damas M, Lima M, Bruges-Armas J. 2009. The coexistence of ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis—a postmortem diagnosis. *Clinical Rheumatology* 28:353-356.

Judd MA. 2004. Trauma in the City of Kerma: Ancient versus modern injury patterns. *International Journal of Osteoarchaeology* 14:34–51. Doi: 10.1002/oa.711.

Judd MA. 2006. Continuity of interpersonal violence between Nubian communities. *American Journal of Physical Anthropology* 131:324-333.

Judd MA. 2008. The parry problem. *Journal of Archaeological Science* 35:1658-1666.

Jurmain RD, Kilgore L. 1995. Skeletal evidence of osteoarthritis: a palaeopathological perspective. *Annals of the Rheumatic Diseases* 54: 443-450.

Kakaliouras AM. 2012. An anthropology of repatriation. *Current Anthropology* 53(5):S210-221. Dio:10.1086/662331.

Kalichman L, Kim DH, Li L, Guermazi A, Berkin V, Hunter DJ. 2009. Spondylolysis and spondylolisthesis: prevalence and association with low back pain in the adult community-based population. *Spine* 34:199-205.

Kalyan CK, Venumadhav N, Siddaraju KS, Pandey SN. 2013. Morphological study on lumbosacral transitional vertebra in adult Indian sacra and its clinical implications. *International Journal of Applied Biology and Pharmaceutical Technology* 4(4):340-346.

Kanis JA. 1998. *Pathophysiology and treatment of Paget's disease of bone*. 2nd ed. Martin Dunitz, London, UK.

Kaplan GJ, Fleshman K, Bender TR. 1973 Long-term effects of otitis media: A ten-year cohort study of Alaskan eskimo children. *Pediatrics* 52:577-585.

- Kayikcioglu E, Aydin AA, Onder AH, Sayiner A, Suren D, Ozturk B. 2017. An extremely rare neoplasm, histiocytic sarcoma: A report of two cases with an aggressive clinical course. *Journal of Oncological Sciences* 3:84-86.
- Keleş B, Öztürk K, Ünalı D, Arbağ H, Özer B. 2010. Is there any relationship between nasal septal deviation and concha bullosa? *European Journal of General Medicine* 7:359–364.
- Kellgren JH, Lawrence JS. 1957. Radiological assessment of osteo-arthritis. *Annals of Rheumatic Diseases* 16:494-502.
- Kendell SD, Collins MS, Adkins MC, Sundaram M, Unni K. 2004. Radiographic differentiation of enchondroma from low-grade chondrosarcoma in the fibula. *Skeletal Radiology* 33(8):458–466.
- Kent S. 1986. The Influence of sedentism and aggregation on porotic hyperostosis and anaemia: A case study. *Royal Anthropological Institute of Great Britain and Ireland* 21(4):605-636.
- Khan MA. 2002. Update on spondyloarthropathies. *Annals of Internal Medicine* 136:896-907.
- Kilgore L, Jurmain R, Van Gerven D. 1997. Paleoepidemiological patterns of trauma in a medieval Nubian skeletal population. *International Journal of Osteoarchaeology* 7:103 - 114.
- Kim MJ, Lee IS, Kim Y-S, Oh CS, Park JB, Shin MH, Shin DH. 2012. Diffuse idiopathic skeletal hyperostosis cases found in Joseon Dynasty human sample collection of Korea. *International Journal of Osteoarchaeology* 22:235-244.
- Kim RY, Weppelmann B, Salter MM, Brascho DJ. 1987. Skeletal metastases from cancer of the uterine cervix: Frequency, patterns, and radiotherapeutic significance. *International Journal of Radiation Oncology, Biology, Physics* 13:705-708.
- Kim SK, Choi BR, Kim CG, Chung SH, Choe JY, Joo KB, Bae SC, Yoo DH, Jun JB. 2004. The prevalence of Diffuse Idiopathic Skeletal Hyperostosis in Korea. *Journal of Rheumatology* 31:2032-2035.
- Kinnes I. 2000. From urban street gangs to criminal empires: the changing face of gangs in the Western Cape. *Institute for Security Studies (ISS). Monograph 28, Halfway House.*
- Kiss C, Szilágyi M, Paksy A, Poór G. 2002. Risk factors for diffuse idiopathic skeletal hyperostosis: a case-control study. *Rheumatology* 41(1):27–30. Doi:10.1093/rheumatology/41.1.27.

- Klales AR, Ousley SD, Vollner JM. 2012. A revised method of sexing the human innominate using Phenice's nonmetric traits and statistical method. *American Journal of Physical Anthropology* 149:104-114.
- Kolmert L, Wulff K. 1982. Epidemiology and treatment of distal femoral fractures in adults. *Acta Orthopaedica Scandinavica* 53:957-962.
- Komar D, Grivas C. 2008. Manufactured populations: What do contemporary reference skeletal collections represent? A comparative study using the Maxwell Museum documented Collection. *American Journal of Physical Anthropology* 137:224-233. Doi:10.1002/ajpa.20858.
- Konin GP, Walz DM. 2010. Lumbosacral transitional vertebrae: classification, imaging findings, and clinical relevance. *American Journal of Neuroradiology* 31:1778-1786.
- Kotzé SH, Mole CG, Greyling LM. 2012. The translucent cadaver: an evaluation of the use of full body digital X-ray images and drawings in surface anatomy education. *Anatomical Sciences Education* 5:287-294.
- Kozak J, Krenz-Niedbala M. 2002. The occurrence of cribra orbitalia and its association with enamel hypoplasia in a medieval population from Kolobrzeg, Poland. *Variability and Evolution* 10:75–82.
- Krogman WM, İşcan MY. (eds.). 1986. *The Human Skeleton in Forensic Medicine*, 2nd ed. Springfield, Charles C Thomas Publisher, Illinois, USA.
- Krug EG. (ed.). 2002. World Report on violence and health. Geneva, Switzerland: *World Health Organization* pp. 1-21.
- Krug EG, Sharma GK, Lozano R. 2002. The Global burden of disease. *American Journal of Public Health* 90:523-526.
- Kubavat DM, Nagar SK, Malukar O, Dipali T, Paras S, Suchetas P. 2012. A Study of sacralisation of fifth lumbar vertebra in Gujarat. *National Journal of Medical Research* 2(2):211-13.
- Kumar A, Khan SA, Sampath Kumar V, Sharma MC. 2014. Bizarre parosteal osteochondromatous proliferation (Nora's lesion) of phalanx in a child. *BMJ Case Report*, pii: bcr2013201714. doi:10.1136/bcr-2013-201714.

- Kumar V, Abbas AK, Fausto N, Mitchell RN. 2007. *Robbins Basic Pathology*, 8th ed. WB Saunders, Philadelphia, Elsevier pp. 946.
- Kundu ZS. 2014. Classification, imaging, biopsy and staging of osteosarcoma. *Indian Journal of Orthopaedics* 48(3):238–246. Doi:10.4103/0019-5413.132491.
- Kunitz SJ, Euler RC. 1972. *Aspects of Southwestern Paleoepidemiology*, Vol. 7. Prescott, Arizona: Prescott College Press pp. 55.
- Kwiatkowska B, Gawlikowska-Sroka A, Szczurowski J, Nowakowski D, Dzieciolowska-Baran E. 2011. A case of concha bullosa mucopyocele in a Medieval human skull. *International Journal of Osteoarchaeology* 21:367-370.
- Kyriacou DN, Anglin D, Taliaferro E, Stone S, Tubb T, Lindin JA, Muelleman R, Barton E, Krauss JF. 1999. Risk factors for injury to women from domestic violence. *The New England Journal of Medicine* 341(25):1892-1898.
- La Fond EM. 1958. An analysis of adult skeletal tuberculosis. *Journal of Bone Surgery* 40A:346–64.
- L'Abbé EN, Loots M, Meiring JH. 2005. The Pretoria Bone Collection: A modern South African skeletal sample. *HOMO* 56:197-205. Doi:10.1016/j.jchb.2004.10.004
- Labuschagné BCJ, Mathey B. 2000. Cadaver profile at University of Stellenbosch medical school, South Africa, 1956-1996. *Clinical Anatomy* 13:88-93. Doi:10.1002/(sici)1098-2353.
- Ladhani S, Srinivasan L, Buchanan C, Allgrove J. 2004. Presentation of vitamin D deficiency. *Archives of Disease in Childhood* 89(8):781–784.
- Lam WW, Liang EY, Woo JK, Van Hasselt A, Metreweli C. 1996. The etiological role of concha bullosa in chronic sinusitis. *European Radiology* 6:550 –552.
- Lambert PM. 2002. Bioarchaeology at Coweeta Creek: continuity and change in native health and lifeways in protohistoric western North Carolina. *Southeastern Archaeology* 21:36-48.
- Larsen CS. 1997. *Bioarchaeology interpreting behavior from the human skeleton*. Cambridge, New York: Cambridge University Press.
- Larsen CS. 2001. (ed.). *Bioarchaeology of Spanish Florida: The Impact of Colonialism*, Gainesville: University Press Florida.

- Larsen CS, Hutchinson DL, Stojanowski CM. 2007. Health and lifestyle in Georgia and Florida: agricultural origins and insesification in regional perspective. In: MN Cohen & GMM Crane-Kramer (eds.). *Ancient Health: Skeletal Indicators of Agricultural and Economica Intensification*. Gainesville, Florida: University Press of Florida.
- Larsen CS, Shavit R, Griffin MC. 1991. Dental caries evidence for dietary change: an archaeological eon- text. In: MA Kelly & CS Larsen (eds.). *Advances in Dental Anthropology* New York: Wiley-Liss.
- Larson AR, Josephson KD, Pauli RM, Opitz JM, Williams MS. 2001. Klippel-Feil anomaly with Sprengel anomaly, omovertebral bone, thumb abnormalities, and flexion-crease changes: novel association or syndrome? *American Journal of Medical Genetics* 101:158-162.
- Lazer E. 1994. The fat hairy women of Pompeii. *New Scientist*, September 22.
- Lazzarini L, Mader JT, Calhoun JH. 2004. Osteomyelitis in long bones. *Journal of Bone and Joint Surgery [Am]* 86-A(10):2305-18.
- Lebowitz RA, Brunner E, Jacobs JB. 1995. The agger nasi cell: radiological evaluation and endoscopic management in chronic frontal sinusitis. Operative techniques. *Otolaryngology-Head and Neck Surgery* 6:171–175.
- Ledesma-Montes C, Hernández-Guerrero JC, Garcés-Ortíz M. 2000. Clinico-pathologic study of odontogenic cysts in a Mexican sample population. *Archives of Medical Research* 31(4):373-6.
- Lee KH, Snape L, Steenberg LJ, Worthington J. 2007. Comparison between interpersonal violence and motor vehicle accidents in the etiology of maxillofacial fractures. *ANZ Journal of Surgery* 77: 695-698.
- Lee KH. 2009. Interpersonal violence and facial fractures. *Journal of Oral and Maxillofacial Surgery* 67:1878-1883.
- Legge S. 2004. Klippel-feil syndrome: Examples from two skeletal collections of Alaskan Natives. *Proceedings of the 6th annual conference of the British Association for Anthropology and Osteoarchaeology* 10-11 September 2004, Bristol, United Kingdom pp 59-63.
- Lew DP, Waldvogel FA. 2004. Osteomyelitis. *Lancet* 364(9431):369–379.

- Li Y, Navia JM, Bian JY. 1995. Prevalence and distribution of developmental enamel defects in primary dentition of Chinese children 3-5 years old. *Community Dentistry Oral Epidemiology* 23(2):72-9.
- Limb D, Dreghorn C, Murphy JK, Mannion R. 1994. Primary lymphoma of bone. *International Orthopedics* 18:180-183.
- Lind T, Krøner K, Jensen J. 1989. The epidemiology of fractures of the proximal humerus. *Archives of Orthopaedic and Trauma Surgery* 108:285-287.
- London L. 1999. The “dop” system, alcohol abuse and social control amongst farm workers in South Africa: A public health challenge. *Social Science and Medicine* 48(10):1407–1414.
- Lorber J, Levick K. 1967. Spina bifida cystica: incidence of spina bifida occulta in parents and in controls. *Archives of Disease in Childhood* 42:171–173.
- Lothrop HA. 1903. The anatomy of the inferior ethmoidal turbinate bone with particular reference to cell formation: surgical importance of such ethmoid cells. *Annals of Surgery* 38:233–255.
- Lovejoy CO, Meindl RS, Pryzbeck TR, Mensforth RP. 1985. Chronological metamorphosis of the auricular surface of the ilium: A new method for the determination of adult skeletal age at death. *American Journal of Physical Anthropology* 68:15-28.
- Lovell NC. 1997. Trauma analysis in Paleopathology. *Yearbook of Physical Anthropology* 40:139-170.
- Lowry RB. 1977. The Klippel-Feil anomalad as part of the fetal alcohol syndrome. *Clinical Teratology* 16(1) 53-56. Doi:10.1002/tera.1420160109.
- Lukacs JR. 1989. Dental Paleopathology: Methods for reconstructing dietary patterns. In İşcan MY, Kennedy KAR. (eds.). *Reconstruction of life from the human skeleton*. New York: Alan R. Liss, Inc. pp 261-286.
- Luoma K, Vehmas T, Raininko R, Luukkonen R, Riihimaki H. 2004. Lumbosacral transitional vertebra: Relation to disk degeneration and low back pain. *Spine* 29(2):200-205.
- Maat GJR. 1984. Dating and Rating of Harris’s Lines. *American Journal of Physical Anthropology* 63:291–299.

- Maat GJR. 2004. Scurvy in adults and youngsters: the Dutch experience. A review of the history and pathology of a disregarded disease. *International Journal Osteoarchaeology* 14:77–81.
- Mabunda M, Swart L, Seedat M. 2008. Magnitude and categories of pedestrian fatalities in South Africa. *Accident Analysis and Prevention* 40(2):586-593.
- Mahato NK. 2010. Morphological traits in sacra associated with complete and partial lumbarisation of first sacral segment. *Spine Journal* 10(10):910-915.
- Malanga GA, Cook PM. 2004. Segmental anomaly leading to wrong level disc surgery in cauda equina syndrome. *Pain Physician* 7(1):107-110.
- Mann RW, Murphy SP. 1990. *Regional atlas of bone disease: A guide to pathological and normal variation in the human skeleton*. Charles C Thomas Publisher, Illinois, USA.
- Mantzaris EA. 1995. Labour struggles in South Africa. *The forgotten pages* 1903 - 1921.
- Manzi G, Censi L, Sperduti A, Passarello P. 1989. Linee di Harris e ipoplasia dello smalto nei resti scheletrici delle popolazioni umane di Isola Sacra e Lucus Feroniae (Roma). *Rivista Di Antropologia* 67:129–148.
- Marais JS. 1939. *The Cape Coloured People, 1652-1937*. Longmans Green & Co. pp 286.
- Marlet JJ. 1974. Development of cranial hyperostosis. *Radiologia Clinica et Biologica* 43:473–82.
- Masniková S, Beňuš R. 2003. Developmental anomalies in skeletal remains from the Great Moravia and Middle Ages cemeteries at Devín (Slovakia). *International Journal of Osteoarchaeology* 13:266-274.
- Mason RJ, Broaddus VC, Martin TR, King Jr TE, Schraufnagel D, Murray JF, Nadel JA. 2010. *Murray and Nadel's textbook of respiratory medicine: 2-volume set*. Elsevier Health Sciences.
- Matos V, Santos AL. 2006. On the trail of pulmonary tuberculosis based on rib lesions: Results from the Human Identified Skeletal Collection from the Museu Bocage (Lisbon, Portugal). *American Journal of Physical Anthropology* 130:190-200.
- Matsuo K, Hirohata T, Sugioka Y, Ikeda M, Fukuda A. 1988. Influence of alcohol intake, cigarette smoking, and occupational status on idiopathic osteonecrosis of the femoral head. *Clinical Orthopaedics and Related Research* 234:115-23.

- Mays S. 2008. A likely case of scurvy from Early Bronze Age Britain. *International Journal of Osteoarchaeology* 18:178–187.
- Mays S. 2012. Nasal septal deviation in a mediaeval population. *American Journal of Physical Anthropology* 148(3):319-326.
- Mays S, Vincent S, Snow M, Robson-Brown K. 2011. Concha bullosa: a neglected condition in palaeopathology. *International Journal of Paleopathology* 1:184-187.
- Mays S, Mavrogordato M, Lambert J, Sofaer J. 2012. The prevalence and health implications of concha bullosa in a population from Mediaeval England. *International Journal of Osteoarchaeology* 24(5):614-622. Doi:10.1002/oa.2246.
- McCoy GF, Johnstone RA, Nelson IW, Kenwright J, Duthie RB. 1988. Incidence and consequences of ejection in motor vehicle accidents. *BMJ* 297(6658):1244-1245.
- McDade AM, McNicol RD, Ward-Booth P, Chesworth J, Moos KF. 1982. The etiology of maxillo-facial injuries, with special reference to the abuse of alcohol. *International Journal of Oral Surgery* 11:152-155.
- McKern TW, Stewart TD. 1957. Skeletal changes in young American males, analysed from the standpoint of identification. *Natick, MA: Headquarters Quartermaster Research and Engineering Center, USA.*
- McHenry H. 1968. Transverse lines in long bones of prehistoric California Indians. *American Journal of Physical Anthropology* 29:1-17.
- Meindl RS, Lovejoy CO. 1985. Ectocranial suture closure: A revised method for the determination of skeletal age at death based on the lateral-anterior sutures. *American Journal of Physical Anthropology* 68:57-66.
- Mensforth RP, Lovejoy CO, Lallo JW, Armelagos GJ. 1978. Part two: the role of constitutional factors, diet, and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in infants and children. *Medical Anthropology* 2:1–59.
- Moore S. 1955. *Hyperostosis Cranii*. CC Thomas, Springfield.
- Moore BH, Illinois C. 1925. Sacralisation of the fifth lumbar vertebra. *Journal of Bone and Joint Surgery* 7:271-8.

- Morgan B, Coakley F, Finlay DB, Belton I. 1996. Hypertrophic osteoarthropathy in staging skeletal scintigraphy for lung cancer. *Clinical Radiology* 51:694-697.
- Morris PS. 1998. A systematic review of clinical research addressing the prevalence, etiology, diagnosis, prognosis and therapy of otitis media in Australian Aboriginal children. *Journal of Paediatrics and Child Health* 34(6): 487–497. Doi:10.1046/j.1440-1754.1998.00299.x
- Mould RF. 1983. *Cancer Statistics*. Bristol: Adam Hilger.
- Mountain A. 2003. *The First People of the Cape*. David Philip Publishers, Cape Town, South Africa.
- Mulligan ME, McRae G, Murphey MD. 1999. Imaging features of primary lymphoma of bone. *American Journal of Roentgenology* 173:1691-1697.
- Murray RO, Jacobson HG. 1977. *The radiology of skeletal disorders*, 2nd ed. Edinburgh: Churchill Livingstone.
- Murphy MS, Gaither C, Goycochea E, Verano JW, Cock G. 2010. Violence and Weapon-Related Trauma at Puruchuco-Huaquerones, Peru. *American Journal of Physical Anthropology* 142:636–649. Doi:10.1002/ajpa.21291.
- Museum of London Collections. St. Bride's lower churchyard photographs. [Online]. Available: <https://www.museumoflondon.org.uk/collections> [Accessed: 2018, May 18].
- Neidengard L, Carter TE. 1978. Klippel-Feil malformation complex in fetal alcohol syndrome. *American Journal of Diseases of Children* 132:929–930.
- Neyrolles O, Quintana-Murci L. 2009. Sexual inequality in tuberculosis. *PLoS Medicine* 6:e1000199. Doi:10.1371/journal.pmed.1000199.
- Nelson DE, Jarman DW, Rehm J, Greenfield TK, Rey G, Kerr WC, Miller P, Shield KD, Ye Y, Naimi TS. 2013. Alcohol-attributable cancer deaths and years of potential life lost in the United States. *American Journal of Public Health* 103:641–8. Doi:10.2105/AJPH.2012.301199.
- Nordqvist A, Petersson CJ. 1995. Incidence and causes of shoulder girdle injuries in an urban population. *Journal of Shoulder and Elbow Surgery* 4:107-112.
- Norman R, Bradshaw D, Schneider M, Jewkes R, Mathews S, Abrahams N, Matzopoulos R, Vos T, South African Comparative Risk Assessment Collaborating Group. 2007. Estimating

- the burden of diseases attributable to interpersonal violence in South Africa in 2000. *South African Medical Journal* 97(8):653-656.
- Nowak O, Piontek J. 2002. The frequency of appearance of transverse (Harris) lines in the tibia in relationship to age at death. *Annals of Human Biology* 28:314-32.
- O'Gradaigh D, Conway R. 2017. Bone Disease. In: P Kumar & M Clark (eds.). *Kumar and Clark's Clinical Medicine*. Elsevier Ltd. pp. 707-719
- ODCCP, 1999. United Nations Office for Drug Control and Crime Prevention, South Africa: Country Profile and Drugs and Crime. Global Illicit Drug Trends (ODCCP Studies on Drug and Crime, Statistics), Vienna 1999. https://www.unodc.org/documents/southafrica/sa_drug.pdf [Accessed: 2018, April 14].
- Odhav B, Naicker V. 2002. Mycotoxins in South African traditionally brewed beers. *Food Additives and Contaminants* 19(1): 55–61. Doi:10.1080/02652030110053426.
- Oettlé AC, Steyn M. 2000. Age estimation from sternal ends of ribs by phase analysis in South African Blacks. *Journal of Forensic Sciences* 45(5):1071-1079.
- Oji C. 1999. Jaw fractures in Enugu, Nigeria, 1985-95. *The British Journal of Oral and Maxillofacial Surgery* 37:106-109.
- Okada K, Frassica FJ, Sim FH, Beabout JW, Bond JR, Unni KK. 1994. Parosteal osteosarcoma. A clinicopathological study. *The Journal of Bone and Joint Surgery [Am]* 76:366–78.
- Olivieri I, D'Angelo S, MD, Palazzi C, Padula A, Mader R, Khan MA. 2009. Diffuse Idiopathic Skeletal Hyperostosis: Differentiation from ankylosing spondylitis. *Current Rheumatology Reports* 11:321–328.
- Olvi L.G., Santini-Araujo E. 2015. Bone Infarct. In: E Santini-Araujo, R Kalil, F Bertoni, YK Park (eds.). *Tumors and Tumor-Like Lesions of Bone*. Springer, London. Doi.org/10.1007/978-1-4471-6578-1_65.
- Ortner DJ. 1999. Scurvy: Its skeletal manifestations and prevalence in North and South American skeletal samples. *American Journal of Physical Anthropology* 28:216.
- Ortner DJ. 2003. *Identification of pathological conditions in human skeletal remains*, 2nd ed. Academic Press, New York.

- Ortner DJ, Butler W, Cafarella J, Milligan L. 2001. Evidence of probable scurvy in subadults from archeological sites in North America. *American Journal of Physical Anthropology* 114:343-351.
- Ortner DJ, Ericksen MF. 1997. Bone changes in the human skull probably resulting from scurvy in infancy and childhood. *International Journal Osteoarchaeology* 7:212–220.
- Ortner DJ, Kimmerle EH, Diez M. 1999. Probable evidence of scurvy in subadults from archeological sites in Peru. *American Journal of Physical Anthropology* 108:321–331.
- Ortner DJ, Ponce P, Ogden A, Buckberry J. 2010. Multicentric osteosarcoma associated with DISH, in a 19th century burial from England. *International Journal of Osteoarchaeology* 22(2): 245–252. Doi:10.1002/oa.1196.
- Ortner DJ, Putschar WG. 1985. *Identification of pathological conditions in human skeletal remains*. Washington DC: Smithsonian Institution Press.
- Ostermann PA, Seligson D, Henry SL. 1995. Local antibiotic therapy for severe open fractures. A review of 1085 consecutive cases. *Journal of Bone and Joint Surgery [Br]* 77:93–97.
- Ósz B, Hajnal K, Marcsik, A, Fogas, O, Horváth, F, Zádori P, Kelemen, K, Vandulek, C, Schultz, M, Márk L, Molnár E, Pálfi G. 2009. Preliminary report on the paleopathological research of the skeletal material from the Szeged medieval castle excavation. *Acta Biologica Szegediensis* 53(2):125-138.
- Ozlu T, Bülbül Y. 2005. Smoking and lung cancer. *Tuberkuloz ve Toraks* 53(2):200-209.
- Papageorgopoulou C, Suter SK, Rühli FJ, Siegmund F. 2011. Harris lines revisited: Prevalence, comorbidities, and possible etiologies. *American Journal of Human Biology*, 23(3): 381–391. doi:10.1002/ajhb.21155
- Paradowska A, Szelag J, Slawicki K. 2007. Klippel-Feil Syndrome – Review of the literature. *Dental and Medical Problems* 44:491-494.
- Park E. 1964. The imprinting of nutritional disturbances on the growing bone. *Pediatrics* 33:815–862.
- Park E, Richter C. 1953. Transverse lines in bones: the mechanism of their development. *Bulletin of the Johns Hopkins Hospital* 93:234–248.

- Parry CD, Pluddemann A, Steyn K, Bradshaw D, Norman R, Laubscher R. 2005. Alcohol use in South Africa: Findings from the first demographic and health survey (1998). *Journal of Studies on Alcohol* 66:91–97.
- Pascoe L, Kim Seow W. 1994. Enamel hypoplasia and dental caries in Australian Aboriginal children: prevalence between the two diseases. *Pediatric Dentistry* 16(3):193–199.
- Patterson N, Petersen DC, Van der Ross RE, Sudoyo H, Glashoff RH, Marzuki S, Reich D, Hayes VM. 2010. Genetic structure of a unique admixed population: Implications for medical research. *Human Molecular Genetics* 19:411-419. Doi:10.1093/hmg/ddp505.
- Peckmann TR. 2002. Dialogues with the dead: An osteological analysis of the palaeodemography and life history of the 18th and 19th century northern frontier in South Africa. Unpublished doctoral dissertation. Cape Town: University of Cape Town; South Africa.
- Peden M, McGee K, Sharma G. 2002. The injury chart book: A graphical overview of the global burden of injuries. Geneva, Switzerland: World Health Organization.
- Peltzer K, Ramlagan S. 2009. Alcohol use trends in South Africa. *Journal of Social Sciences* 18:1-12.
- Perou ML. 1964. *Cranial Hyperostosis*. Charles C. Thomas, Springfield, IL.
- Perruccio AV, Yip C, Badley EM, Power JD. 2017. Musculoskeletal Disorders: A neglected group at public health and epidemiology meetings? *American Journal of Public Health* 107(10):1584–1585. Doi:10.2105/ajph.2017.303990.
- Pestalozza G. 1984. Otitis media in newborn infants. *International Journal of Pediatric Otorhinolaryngology* 8:109-124.
- Petersen DC, Libiger O, Tindall EA, Hardie R-A, Hannick LI, Glashoff RH, Mukerji M, Fernandez P, Haacke W, Schork NJ, Hayes VM, Indian Genome Variation Consortium, 2013. Complex patterns of genomic admixture within southern Africa. *PLoS Genetics* 9:e1003309. Doi:10.1371/journal.pgen.1003309.
- Peterson CK, Bolton J, Hsu W, Wood A. 2005. A cross-sectional study comparing pain and disability levels in patients with low back pain with and without transitional lumbosacral vertebrae. *Journal of Manipulative Physiological Therapy* 28(8):570–574.

- Peterson D, Taylor TJ, Esbensen F. 2004. Gang membership and violent victimization. *Justice Quarterly* 21(4):793-815.
- Petridou E, Browne A, Lichter E, Dedoukou X, Alexe D, Dessypris N. 2002. What distinguishes unintentional injuries from injuries due to intimate partner violence: A study in Greek ambulatory care settings. *Injury Prevention* 8:197-201.
- Pettit CK, Zukerberg LR, Gray MH, Ferry JA, Rosenberg AE, Harmon DC. 1990. Primary lymphoma of bone. A B cell neoplasm with a high frequency of multilobated cells. *American Journal of Surgical Pathology* 14:329-34
- Pfeiffer S, Heinrich J, Beresheim A, Alblas M. 2016. Cortical bone histomorphology of known-age skeletons from the Kirsten Collection, Stellenbosch University, South Africa. *American Journal of Physical Anthropology* 160:137-147. Doi:10.1002/ajpa.22951.
- Phenice TW. 1969. A newly developed visual method of sexing the os pubis. *American Journal of Physical Anthropology* 30:297-302.
- Picci P. 2007. Osteosarcoma (Osteogenic sarcoma). *Orphanet Journal of Rare Diseases* 2:6. Doi:10.1186/1750-1172-2-6.
- Pigrau-Serrallach C, Rodríguez-Pardo D. 2013. Bone and joint tuberculosis. *European Spine Journal* 22 (Suppl 4):556-66. Doi: 10.1007/s00586-012-2331-y.
- Pizzutillo PD, Woods M, Nicholson L, MacEwen GD. 1994. Risk factors in Klippel–Feil syndrome. *Spine* 19:2110–2116
- Platt HS, Stewart RJC, Platt BS. 1963. Transverse trabeculae in the bones of malnourished children. *Proceedings of the Nutrition Society* 22:xxix-xxx.
- Plaza S, Salas A, Calafell F, Corte-Real F, Bertranpetit J, Carracedo A, Comas, D. 2004. Insights into the western Bantu dispersal: mtDNA lineage analysis in Angola. *Human Genetics* 115(5):439-47. Doi:10.1007/s00439-004-1164-0.
- Pluddemann A, Parry C, Donson H, Sukhai A. 2004. Alcohol use and trauma in Cape Town, Durban and Port Elizabeth, South Africa: 1999-2001. *International Journal of Injury Control and Safety Promotion* 11(4):265–267. Doi:10.1080/156609704/233/289599.
- Piontek J, Jerszynska B, Nowak O. 2001. Harris lines in subadult and adult skeletons from the mediaeval cemetery in Cedynia, Poland. *Variability and Evolution* 9:33–43.

- Poór G, Donáth J, Fornet B, Cooper C. 2006. Epidemiology of Paget's disease in Europe: the prevalence is decreasing. *Journal of Bone and Mineral Research* 21:1545-1549.
- Popovich D, McAlhany A, Adewumi AO, Barnes MM. 2009. Scurvy: forgotten but definitely not gone. *Journal of Pediatric Health Care* 23:405–415.
- Post RH. 1966. Pilot study: population differences in the frequency of spina bifida occulta. *Eugen Q* 13(4):341-52.
- Postacchini F, Gumina S, De Santis P, Albo F. 2002. Epidemiology of clavicle fractures. *Journal of Shoulder and Elbow Surgery* 11(5):452-456.
- Potdar GG. 1970. Primary reticulum-cell sarcoma of bone in Western India. *British Journal of Cancer* 24:48-55.
- Praud D, Rota M, Rehm J, Shield K, Zatonski W, Hashibe M, La Vecchia C, Boffetta P. 2016. Cancer incidence and mortality attributable to alcohol consumption. *International Journal of Cancer* 138:1380–7.
- Prinsloo M. (ed.). 2007. A profile of fatal injuries in South Africa: 7th annual report of the national injury mortality surveillance system 2005. *Cape Town: Medical Research Council. University of South Africa Crime, Violence and Injury Lead Programme.*
- Quintana-Murci L, Harmant C, Quach H, Balanovsky O, Zaporozhchenko V, Bormans C, van Helden PD, Hoal EG, Behar DM. 2010. Strong maternal Khoisan contribution to the South African Coloured population: A case of gender-biased admixture. *The American Journal of Human Genetics* 86(4). Doi:10.1016/j.ajhg.2010.02.014.
- Ragsdale BD. 1993. Morphological analysis of skeletal lesions: correlation of imaging studies and pathological findings. In: R Weinstein (ed.). *Advances in Pathology and Laboratory Medicine*. Mosby, New York.
- Ragsdale BD, Campbell RA, Kirkpatrick C. 2018. Neoplasm or not? General principles of morphologic analysis of dry bone specimens. *International Journal of Paleopathology* 21:27–40. Doi: 10.1016/j.ijpp.2017.02.002
- Raikos A, Paraskevas GK, Yusuf F, Kordali P, Meditskou S, Al-Haj A, Brand-Saberi B. 2011. Etiopathogenesis of hyperostosis frontalis interna: a mystery still. *Annals of Anatomy* 193(5):453-8. Doi:10.1016/j.aanat.2011.05.004.

- Ramachandran Nair PN. 2003. Non-microbial etiology: periapical cysts sustain post-treatment apical periodontitis. *Endodontic Topics* 6:96-113.
- Rathbun TA, Mallin R. 1977. Middle ear disease in a prehistoric Iranian population. *Bulletin of the New York Academy of Medicine* 53: 901–905.
- Rehm J, Baliunas D, Borges GLG, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M, Room R, Samokhvalov AV, Taylor B. 2010. The relation between different dimensions of alcohol consumption and burden of disease: An overview. *Addiction* 105:817-843.
- Reichman EF. (ed.). 2013. *Emergency Medicine Procedures*, 2nd ed. MCGraw Hill Education.
- Reid IR, Miller P, Lyles K, Fraser W, Brown JP, Saidi Y, Mesenbrink P, Su G, Pak J, Zelenakas K, Luchi M, Richardson P, Hosking D. 2005. Comparison of a single infusion of zoledronic acid with risedronate for Paget's disease. *New England Journal of Medicine*. 353:898-908.
- Resnick D, Guerra J, Robinson CA and Vint VC. 1978. Association of diffuse idiopathic skeletal hyperostosis (DISH) and calcification and ossification of the posterior longitudinal ligament. *American Journal of Roentgenology* 131:1049-1053.
- Resnick D. 1995. *Diagnosis of Bone and Joint Disorders*, vol 4. 3rded. WB Saunders, Philadelphia.
- Resnick D, Niwayama G. 1995. Infectious diseases. In: D Resnick (ed.). *Diagnosis of Bone and Joint Disorders*, 3rd ed. WB Saunders, Philadelphia pp. 2323–2448.
- Rieder HL, Snider DE, Cauthen GM. 1990. Extrapulmonary tuberculosis in the United States. *American Review of Respiratory Disease* 141:347-351.
- Rivera F & Mirazon Lahr MB. 2017. New evidence suggesting a dissociated etiology for cribra orbitalia and porotic hyperostosis. *American Journal of Physical Anthropology*, 164 76-96. Doi:10.1002/ajpa.23258.
- Roberts C, Lucy D, Manchester K. 1994. Inflammatory lesions of ribs: an analysis of the Terry Collection. *American Journal of Physical Anthropology* 95:169-182.
- Roberts, CA, Boylston A, Buckley L, Chamberlain AC, Murphy EM. 1998. Rib lesions and tuberculosis: the palaeopathological evidence. *Tubercle and Lung Disease* 79(1):55-60.

- Roberts CA, Manchester K. 2005. *The Archaeology of Disease*, 3rd ed. Stroud: Sutton Publishing.
- Robinson HBG, Miller AS. 1983. *Color Atlas of Pathology*, 4th ed. Philadelphia JB Lippincott.
- Rockhold, L.A. and Herrmann, N.P. 1999. A case study of a vehicular hit-and-run fatality: Direction of force. In: *Broken Bones: Anthropological Analysis of Blunt Force Trauma*. Charles C Thomas Publisher, Illinois, USA pp. 35-62.
- Rogers J, Shepstone L, Dieppe P. 2004. Is osteoarthritis a systemic disorder of bone? *Arthritis Rheumatology* 50(2):452-7.
- Rogers J, Waldron T, Dieppe P, Watt I. 1987. Arthropathies in palaeopathology: the basis of classification according to most probably cause. *Journal of Archaeological Science* 14:179-193.
- Rogers J, Waldron T. 2001. DISH and the monastic way of life. *International Journal of Osteoarchaeology* 11(5):357–365. Doi:10.1002/oa.574.
- Rosenberg NJ, Bargar WL, Friedman B. 1981. The incidence of spondylolysis and spondylolisthesis in nonambulatory patients. *Spine* 6:35-38.
- Rosso P, Lederman SA. 1982. Nutrition in pregnant adolescent. *Current Concepts in Nutrition* 11:47-62.
- Rothschild BM, Heathcote GM. 1993. Characterization of the skeletal manifestations of the treponemal disease Yaws as a population phenomenon. *Clinical Infectious Diseases* 17:198-203.
- Rothschild BM, HersHKovitz I, Dutour O. 1998. Clues potentially distinguishing lytic lesions of multiple myeloma from those of metastatic carcinoma. *American Journal of Physical Anthropology* 105: 241–250.
- Rothschild BM, Rothschild C. 1995. Comparison of radiologic and gross examination for detection of cancer in defleshed skeletons. *American Journal of Physical Anthropology* 97: 357–363.
- Rothschild BM, Rothschild C. 1995. Treponemal disease revisited: Skeletal discriminators for yaws, bejel and venereal syphilis. *Clinical Infectious Disease* 20:1402 - 1408.

- Rothschild BM, Rothschild C. 1998. Recognition of hypertrophic osteoarthropathy in skeletal remains. *Journal of Rheumatology* 25:2221e7.
- Rovers MM, De Kok IM, Schilder AG. 2006. Risk factors for otitis media: An international perspective. *International Journal of Pediatric Otorhinolaryngology* 70:1251–6.
- Rubin V. 1975. *Cannabis and Culture*. Walter de Gruyter. 612pp.
- Salmi A, Voutilainen A, Holisti LR, Unnerus CE. 1962. Hyperostosis cranii in a normal population. *American Journal of Roentgenology* 87:1032-1040.
- Saluja PG. 1988. The incidence of spina bifida occulta in a historic and a modern London population. *Journal of Anatomy* 158:91–93.
- Samara TR. 2011. *Cape Town after Apartheid: Crime and governance in the divided city*. University of Minnesota Press, Minneapolis.
- Samartzis D, Herman J, Lubicky JP, 2006. Classification of congenitally fused patterns in Klippel-Feil patients: epidemiology and the role in the development of cervical spine-related symptoms. *Spine* 31(21):E7898-804.
- Samartzis D, Kalluri P, Herman J, Lubicky JP, Shen FH. 2007. Superior odontoid migration in the Klippel–Feil patient. *European Spine Journal* 16:1489–1497. Doi:10.1007/s00586-006-0280-z.
- Sarzi-Puttini P, Atzeni F. 2004. New developments in our understanding of DISH (diffuse idiopathic skeletal hyperostosis). *Current Opinions in Rheumatology* 16:287-292.
- Savage C. 2005. Lumbosacral transitional vertebrae: classification of variation and association with low back pain. Unpublished MA thesis: Columbia: University of Missouri, USA Available: <https://mospace.umsystem.edu/xmlui/handle/10355/4318> [2018, July 18].
- Schilgen M, Loeser, H. 1994. Klippel-Feil anomaly combined with fetal alcohol syndrome. *European Spine Journal* 3(5):289-290.
- Schlebusch CM, Skoglund P, Sjödin P, Gattepaille LM, Hernandez D, Jay F, Li S, Jongh MD, Singleton A, Blum MGB, Soodyall H, Jakobsson M, 2012. Genomic variation in seven Khoesan groups reveals adaptation and complex African history. *Science* 338:374e379. Doi:10.1126/science.1227721.

- Schmitt SK. 2017. Osteomyelitis. *Infectious Disease Clinics of North America* 31(2):325–338. Doi:10.1016/j.idc.2017.01.010.
- Schmidt HGK, Kranz HW, Siebert CH. 1997. Die behandlung von langstreckigen infekt-defekt-pseudarthrosen mit segmenttransport im ringfixateur. *Osteosynthesis Internal* 5:212–220.
- Schneider M, Norman R, Parry CD, Bradshaw D, Pluddemann A. 2007. Estimating the burden of disease attributable to alcohol use in South Africa in 2000. *South African Medical Journal* 97:664-672.
- Schoeman K. 2007. *Early slavery at the Cape of Good Hope 1652 –1717*. Protea Publishers, Pretoria.
- Scholl TO, Hediger ML. 1994. Anemia and iron-deficiency anemia: compilation of data on pregnancy outcome. *American Journal of Clinical Nutrition* 59:492S-501S.
- Schultz M. 2001. Paleohistopathology of bone: A new approach to the study of ancient diseases. *American Journal of Physical Anthropology* 33:106-147.
- Schuster SC, Miller W, Ratan A, Tomsho LP, Giardine B, Kasson LR. 2010. Complete Khoisan and Bantu genomes from southern Africa. *Nature* 463:943-947. Doi:10.1038/nature08795.
- Schuurman N, Cinnamon J, Walker BB, Fawcett V, Nicol A, Hameed SM, Matzopoulos R. 2015. Intentional injury and violence in Cape Town, South Africa: an epidemiological analysis of trauma admissions data. *Global Health Action* 8: 27016. Doi:10.3402/gha.v8.27016.
- Schwameis E, Abdolvahab F, Wurnig C. 1996. Osteomyelitis. *Radiologe* 36:823–833.
- Schweitzer ME, Balsam D, Weiss R. 1993. Spina bifida occulta. *Spine* 18:785–786.
- Segal AM, Mackenzie AH. 1982. Hypertrophic osteoarthropathy: a 10-year retrospective. *Seminars in Arthritis and Rheumatism* 12(2):220-232. Doi: 10.1016/0049-0172(82)90062-2.
- Seow W, Humphreys C, Tudehope D. 1987. Increased prevalence of developmental defects in low birthweight, prematurely-born children: a controlled study. *Pediatric Dentistry* 9:221–223.
- Sharma A, Sharma DK, Shulda CK. 2011. Osteogenic study of lumbosacral transitional vertebra in central India region. *Journal of Anatomical Society of India* 60(2):212-7.
- She R, Szakacs J. 2004. Hyperostosis frontalis interna: case report and review of literature. *Annals of Clinical and Laboratory Science* 34:206-208.

- Shell RC-H. 1997. *Children of bondage – A social history of the slave society at the Cape of Good Hope 1652 – 1838*. Witwatersrand University Press, Johannesburg.
- Sherman R, Steyn M. 2009. E-race-ing the line: South African interracial relationships yesterday and today. In: M Steyn & M van Zyl (eds.). *The prize and the price: Shaping sexualities in South Africa*. HSRC Press, Cape Town, South Africa.
- Shin M, Besser LM, Correa A. 2008. Prevalence of spina bifida among children and adolescents in Metropolitan Atlanta. *Birth Defects Research Part A. Clinical and Molecular Teratology* 82(11):748-54. Doi: 10.1002/bdra.20530.
- Sieminska A, Jassem E. 2014. The many faces of tobacco use among women. *Medical Science Monitor. International Medical Journal of Experimental Clinical Research* 20:153-162. Doi:10.12659/MSM.889796.
- Silber G, Geffen N. 2009. Race, class and violent crime in South Africa: Dispelling the ‘Huntley thesis’. *SA Crime Quarterly* 30:35-43.
- Silk H. 2014. Diseases of the mouth. *Primary Care: Clinics in Office Practice* 41(1), 75–90. doi:10.1016/j.pop.2013.10.011
- Silvennoinen U, Ilzuka T, Lindqvist C, Oikarinen K. 1992. Different patterns of condylar fractures: An analysis of 382 patients in a 3-year period. *Journal of Oral and Maxillofacial Surgery* 50:1032-1037.
- Simarli M, Türüt H, Topçu S, Gülhan E, Yazici Ü, Kaya S, Taştepe I. 2003. A comprehensive analysis of traumatic rib fractures: morbidity, mortality and management. *European Journal of Cardio-thoracic Surgery* 24:133-138. Doi: 10.1016/S1010-7940(03)00256-2.
- Singer FR, Krane SM. 1998. Paget’s Disease of Bone. In: LV Avioli & SM Krane (eds.). *Metabolic Bone Disease*, 4th ed. San Diego, CA: Academic Press.
- Sissolak D, Bamford CM, Mehtar S. 2010. The potential to transmit *Mycobacterium tuberculosis* at a South African tertiary teaching hospital. *International Journal of Infective Diseases* 14:e423e8.
- Sitas F, Urban M, Bradshaw D, Kielkowski D, Bah S, Peto R. 2004. Tobacco attributable deaths in South Africa. *Tobacco Control* 13:396-399.

- Skinner M, Goodman AH. 1992. Anthropological uses of developmental defects of enamel. In: Saunders & Katzenberg (eds). *New Skeletal Biology of Past Peoples: Research Methods*. York Wiley-Liss, Inc. pp. 153-174.
- Soler T, Calderón C. 2000. The prevalence of spondylolysis in the Spanish elite athlete. *The American Journal of Sports Medicine* 28:57-62.
- Solgaard S, Peterson VS. 1985. Epidemiology of distal radius fractures. *Acta Orthopaedica Scandinavica* 56:391-393.
- Solomon L. 1981. Idiopathic necrosis of the femoral head: pathogenesis and treatment. *Canadian Journal of Surgeons* 24:573-8.
- Sone S, Yano S. 2007. Molecular pathogenesis and its therapeutic modalities of lung cancer metastasis to bone. *Cancer Metastasis Review* 26(3-4): 685-9. Doi:10.1007/s10555-007-9081-z.
- Song WS, Jeon DG, Kong CB, Cho WH, Lee SY. 2011. Outcome of reexcision for intralesionally treated parosteal osteosarcoma. *Journal of Surgical Oncology* 103:264-8.
- Sontag L, Comstock G. 1938. Striae in the bones of a set of monozygotic triplets. *American Journal of Disease of Children* 56:301-308.
- South African National Department of Health. 2004. National Policy for Health Act, 2003 (Act No. 61 of 2003). *Government Gazette* 469:66-79.
- South African History Online [Online]. [n.d.]. Available: <http://www.sahistory.org.za/archive/map/> [accessed: 2018, January 18].
- Southam JC, Soames JV. 1993. 2. *Dental Caries. Oral pathology*, 2nd ed. Oxford: Oxford University Press.
- Souza SM. 2002. Rib periosteal reactions in skeletons from Atacama, Chile: tuberculosis. In: Departamento de Antropologia da Universidade de Coimbra. (ed.). 14th European meeting of the Palaeopathology Association: program-abstracts. *Coimbra: Departamento de Anthropologia* p.156.
- Stallman S, Lobo JN, Som PM. 2004. The incidence of concha bullosa and its relationship to nasal septal deviation and paranasal sinus. *American Journal of Neuroradiology* 25:1613-1618.

- Standaert CJ, Herring SA. 2000. Spondylolysis: a critical review. *British Journal of Sports Medicine* 34:415-422.
- Statistics South Africa. 2012. Census 2011: Statistical release. Pretoria: Statistics South Africa.
- Stead WW, Senner JW, Reddick WT, Lofgren JP. 1990. Racial differences in susceptibility to infection by *Mycobacterium tuberculosis*. *The New England Journal of Medicine* 322:422-427.
- Steckel RH, Rose JC. 2002. *The backbone of history: Health and nutrition in the western hemisphere*. New York: Cambridge University Press.
- Stein ME, Kuten A, Gez E, Rosenblatt KE, Drumea K, Ben-Shachar M, Zidan J, Haim N, Epelbaum R. 2003. Primary lymphoma of bone—a retrospective study. *Oncology* 64:322-7.
- Steinbock RT. 1976. *Paleopathological Diagnosis and Interpretation: Bone Diseases in Ancient Human Populations*. Charles C Thomas Publisher, Illinois, USA.
- Stenseth JK, Clagett OT, Woolner LB. 1967. Hypertrophic pulmonary osteoarthropathy. *Chest* 52:62-68.
- Steyn M, İşcan MY, De Kock M, Kranioti EF, Michalodimitrakis M, L'Abbé EN. 2010. Analysis of antemortem trauma in three modern skeletal populations. *International Journal of Osteoarchaeology* 20:561–571. Doi:10.1002/oa.1096.
- Steyn M, Scholtz Y, Botha D, Pretorius S. 2013. The changing face of tuberculosis: Trends in tuberculosis-associated skeletal changes. *Tuberculosis* 93:467-474.
- Strebel PM, Kuhn, L, Yach D. 1989. Smoking practices in the black township population of Cape Town. *South African Medical Journal* 75(9), 428–431.
- Stroud G, Kemp RL. 1993. *The Human Bones in Cemeteries of St Andrew Fishergate*. York: C.B.A. Publications.
- Stuart-Macadam P. 1985. Porotic hyperostosis: representative of a childhood condition. *American Journal of Physical Anthropology* 66:391-398.
- Stull KE, Kenyhercz MW, L'Abbé EN. 2014. Ancestry estimation in South Africa using craniometrics and geometric morphometrics. *Forensic Science International* 245:206.e1-206.e7. <https://doi.org/10.1016/j.forsciint.2014.10.021>
- Sukhai A, Van Niekerk A. 2002. Transport-related deaths. In: R Matzopoulos (ed.). A profile of fatal injuries in South Africa 2001: third annual report of the national injury mortality

surveillance system (NIMSS). *Cape Town: MRC/UNISA Crime, Violence and Injury Lead Programme.*

Swerdlow AJ, Peto R, Doll RS. 2010. Epidemiology of cancer. In: DA Warrel, TM Cox, JD Firth (eds.). *Oxford Textbook of Medicine*. Oxford, UK: Oxford University Press 299-332. Doi: 10.1093/med/9780199204854.003.0601

Symes SA, L'Abbé EN, Chapman EN, Wolff I, Dirkmaat DC. 2012. Interpreting traumatic injury from bone in medicolegal investigations. In: Dirkmaat DC. (ed.). *A Companion to Forensic Anthropology*, pp. 540-590. London: Wiley-Blackwell.

Tassabehji M, Fang ZM, Hilton EN, McGaughran J, Zhao Z, de Bock CE, Howard E, Malass M, Donnai D, Diwan A, Manson FDC, Murrell D, Clarke RA. 2008. Mutations in GDF6 are associated with vertebral segmentation defects in Klippel-Feil syndrome. *Human Mutations* 29:1017-1027.

Teele DW, Klein JO, Rosner BA. 1984. Otitis media with effusion during the first three years of life and development of speech and language. *Pediatrics* 74(2):282-7.

Teitelman AM, Bellamy SL, Jemmott JB 3rd, Icard L, O'Leary A, Ali S, Ngwane Z, Makiwane M. 2017. Childhood sexual abuse and sociodemographic factors prospectively associated with intimate partner violence perpetration among South African heterosexual men. *Annals of Behavioral Medicine* 51(2):170-178.

Thacher TD, Fischer PR, Pettifor JM, Lawson JO, Isichei CO, Reading JC, Chan GM. 1999. A comparison of calcium, vitamin D, or both for nutritional rickets in Nigerian children. *New England Journal of Medicine* 19:341(8):563-568.

Thacher TD, Fischer PR, Tebben PJ, Singh RJ, Cha SS, Maxson JA, Yawn, BP. 2013. Increasing incidence of nutritional rickets: A population-based study in Olmsted County, Minnesota. *Mayo Clinic Proceedings* 88(2): 176–183. Doi:10.1016/j.mayocp.2012.10.018

Thawait G, Thawait SK, Chhabra A, Fayad JA. 2012. An epidemiological imaging study of spinal segment variations in a nonsymptomatic population. *EPOS*, 1865.

Thompson LA. 2013. *History of South Africa*. New Haven, CT: Yale University Press. Doi:10.1163/18757421-90000065

Thomsen MN, Schneider U, Weber M, Johannisson R, Niethard FU. 1997. Scoliosis and congenital anomalies associated with Klippel-Feil syndrome types I–III. *Spine* 22: 396–401

- Throckmorton T, Kuhn JE. 2007. Fractures of the medial end of the clavicle. *Journal of Shoulder and Elbow Surgery* 16:49-54. Doi: 10.1016/j.jse.2006.05.010.
- Thwaites GF, Chau TTH, Mai NTH, Drobniewski F, McAdam K, Farrar J. 2000. Tuberculous meningitis. *Journal of Neurology, Neurosurgery and Psychiatry* 68:289-299.
- Tiemann AH, Hofmann GO. 2009. Principles of the therapy of bone infections in adult extremities. Are there any new developments? *Strategies Trauma Limb Reconstruction* 4(2): 57–64. Doi:10.1007/s11751-009-0059-y.
- Tishkoff SA, Reed FA, Friedlaender FR, Ehret C, Ranciaro A, Froment A, Hirbo JB, Awomoyi AA, Bodo JM, Doumbo O, Ibrahim M, Juma AD, Kotze MJ, Lema G, Moore JH, Mortensen H, Nyambo TB, Omar SA, Powell K, Pretorius GS, Smith MW, Thera MA, Wambebe C, Weber JL, Williams SM. 2009. The genetic structure and history of Africans and African Americans. *Science* 324:1035–1044. Doi:10.1126/science.1172257.
- Todd TW, Lyon Jr DW. 1924. Endocranial suture closure. Part I. Adult males of white stock. *American Journal of Physical Anthropology* 7:325-384.
- Todd TW, Lyon Jr DW. 1925. Cranial suture closure. Part II. Ectocranial suture closure in adult males of white stock. *American Journal of Physical Anthropology* 8:23-43.
- Tracy MR, Dormans JP, Kusumi K. 2004. Klippel-Feil syndrome: clinical features and current understanding of etiology. *Clinical Orthopaedics and Related Research* 424: 183-190.
- Ugboko VI, Odusanya AS, Fagade OO. 1998. Maxillofacial fractures in semi-urban Nigeria teaching hospital. A review of 442 cases. *International Journal of Oral Maxillofacial Surgery* 27(4):286-289.
- Underwood C. 2004. How can we best deliver an inclusive health service? *Primary Health Care* 14(9):20–21.
- Ünlü HH, Akyar S, Caylan R. 1994. Concha bullosa. *Journal of Otolaryngology* 23(1): 23-27.
- Unni KK, Dahlin DC, Beabout JW. 1976. Periosteal osteogenic sarcoma. *Cancer* 37:2476–85.
- Usdin S, Christofides N, Malepe L, Maker A. 2000. The value of advocacy in promoting social change: Implementing the new domestic violence act in South Africa. *Reproductive Health Matters* 8(16):55-67.

- Usher B. 2002. Reference samples: the first step in linking biology and age in the human skeleton. In: R Hoppa & J Vaupel (eds.). *Paleodemography: age distributions from skeletal samples*. London: Cambridge University Press.
- Van der Merwe AE. 2007. Human skeletal remains from Kimberley: an assessment of health in a 19th century mining community. Unpublished MSc Thesis, Pretoria: University of Pretoria. South Africa.
- Van der Ross RE. 2005. *Up from slavery: Slaves at the Cape: their origins, treatment and contribution*. Cape Town: Ampersand Press in association with the University of the Western Cape, Kenilworth [South Africa].
- Van der Spuy JW. 2000. Trauma, alcohol and other substances. *South African Medical Journal* 90(3): 244-46.
- Van Rensburg HCJ, Mans, A. 1982. *Profile of disease and healthcare in South Africa*. H&R Academica, Pretoria.
- Van Staa TP, Dennison EM, Leufkens HGM & Cooper C. 2001. Epidemiology of fractures in England and Wales. *Bone* 29(6):517-522.
- Van Wyk BE, Theron WH. 2005. Fighting gangsterism in South Africa: a contextual review of gang and anti-gang movements in the Western Cape Criminological and Victimological Society of Southern Africa (CRIMSA) 18:51-60.
- Vannier JP, Lefort J, Cavellier B, Ledosseur P, Assailly C, Feingold J. 1981. Spina bifida cystica families X-ray examination and HLA typing. *Pediatric Research* 15:326–329. Doi:10.1203/00006450-198104000-00007.
- Vazquez E, Castellote A, Piqueras J, Mauleon S, Creixell S, Pumarola F, Figueras C, Carreño J-C, Lucaya J. 2003. Imaging of complications of acute mastoiditis in children. *Radiographics* 23(2): 359-372. Doi: 10.1148/rg.232025076.
- Verdy M, Guimond J, Fauteux P, Aube M, 1978. Prevalence of hyperostosis frontalis interna in relation to body weight. *American Journal of Clinical Nutrition* 31(11):2002–2004.
- Vergauwen S, Parizel PM, Van Breusegem L, Van Goethem JW, Nackaerts Y, Van den Hauwe L, De Schepper AM. 1997. Distribution and incidence of degenerative spine changes in patients with a lumbosacral transitional vertebra. *European Spine Journal* 6(3):168-172. Doi:10.1007/BF01301431.

- Veselka B, Hoogland MLP, Waters-Rist AL. 2013. Rural Rickets: Vitamin D Deficiency in a Post-Medieval Farming Community from the Netherlands. *International Journal of Osteoarchaeology* 25(5):665–675. Doi:10.1002/oa.2329.
- Vigorita VJ. (ed.). 1999. *Orthopaedic Pathology*. Lippincott Williams and Wilkens Philadelphia.
- Vos JA, Abbondanzo SL, Barekman CL, Andriko JW, Miettinen M, Aguilera NS. 2005. Histiocytic sarcoma: a study of five cases including the histiocyte marker CD163. *Modern Pathology* 18(5):693–704. Doi:10.1038/modpathol.3800346.
- Wald N, Sneddon J. 1991. Prevention of neural tube defects: Results of the Medical Research Council Vitamin Study. *Lancet* 338 (8760) p131.
- Waldron T. 2009. *Palaeopathology*. Cambridge University Press, Cambridge/New York.
- Walker PL. 2001. A bioarchaeological perspective on the history of violence. *Annual Review of Anthropology* 30:573-96.
- Walker PL. 2008. Sexing skulls using discriminant function analysis of visually assessed traits. *American Journal of Physical Anthropology* 136:39-50.
- Walker PL, Bathurst RR, Richman R, Gjerdrum T, Andrushko VA. 2009. The causes of porotic hyperostosis and cribra orbitalia: a reappraisal of the iron deficiency-anemia hypothesis. *American Journal of Physical Anthropology* 139:109–125.
- Walters J. 2014. Morphological assessment of disease and metabolic disorders in a Western Cape skeletal population. Unpublished MSc thesis, Cape Town: Stellenbosch University, South Africa.
- Ward CL, Artz L, Berg J, Boonzaier F, Crawford-Browne S, Dawes A, Foster D, Matzopoulos R, Nicol A, Seekings J, Van As AB, Van der Spuy E. 2012. Violence, violence prevention, and safety: a research agenda for South Africa. *South African Medical Journal* 102:215-18.
- Webb S. 1984. Intensification, population and social change in Southeastern Australia: The skeletal evidence. *Aboriginal History* 8:154–172.
- Weinfeld RM, Olson PN, Maki DD. 1997. The prevalence of diffuse idiopathic skeletal hyperostosis (DISH) in two large American Midwest metropolitan hospital populations. *Skeletal Radiology* 26:222–225.

- Weiss E, Jurmain R. 2007. Osteoarthritis Revisited: A contemporary review of aetiology. *International Journal of Osteoarchaeology* 17:437-450.
- Wells C. 1967. A new approach to paleopathology: Harris lines. In Brothwell D, Sandison A. (ed.). *Diseases in Antiquity*. Charles C Thomas Publisher, Illinois, USA. pp. 390–404.
- Werner BC, Mack C, Franke C, Barnes RP, Warren RF, Rodeo S. 2017. Distal fibula fractures in National Football League athletes. *Orthopaedic Journal of Sport Medicine* 5(9). Doi: 10.1177/2325967117726515.
- Weston D. 2012. Nonspecific infection in paleopathology: interpreting periosteal reaction. In: AL Grauer (ed.). *A Companion to Paleopathology*. Blackwell Publishing pp 492-511.
- White TD, Black MT, Folkens PA. 2012. *Human Osteology*, 3rd ed. Academic Press, San Diego.
- Whittingdale J. 1973. The development and location of industries in greater Cape Town 1652-1972. Unpublished MA (Geography) Thesis. Cape Town: University of Cape Town; South Africa.
- Wilczak CA, Jones EB. (ed.). 2012. *Osteoware™ Software Manual: Volume II Pathology Module*. Smithsonian Institution: Washington, DC.
- Winzenberg T, Jones G. 2013. Vitamin D and bone health in childhood and adolescence. *Calcified Tissue International* 92(2):140-50. Doi: 10.1007/s00223-012-9615-4.
- Winther B, Alper CM, Mandel EM, Doyle WJ, Hendley JO. 2007. Temporal relationships between colds, upper respiratory viruses detected by polymerase chain reaction, and otitis media in young children followed through a typical cold season. *Pediatrics* 119:1069–75.
- Wright J. 1991. The Drugs Situation in South Africa - Drug Trafficking in South Africa and Trends in the Southern Sub-Region of Africa. *Drugs Arena* 12/91.
- Wood JW, Milner GR, Harpending HC, Weiss KM. 1992. The Osteological Paradox: Problems of inferring prehistoric health from skeletal samples. *Current Anthropology* 33 (4): 343-358.
- Wood K, Jewkes R. 2001. 'Dangerous' love: reflections on violence among Xhosa township youth. In: R Morrell (ed.). *Changing men in Southern Africa*. University of Natal Press: Pietermaritzburg and Zed Books Ltd, London.

World Health Organization. 2011. Global tuberculosis control. WHO Report. Geneva, Switzerland: World Health Organization, http://www.who.int/tb/publications/global_report/. [accessed: 23 June 2016].

Yach D, McIntyre D, Saloojee Y. 1992. Smoking in South Africa: The health and economic impact. *Tobacco Control* 1(4), 272–280.

Yao Q, Altman RD, Brahn E. 2009. Periostitis and hypertrophic pulmonary osteoarthropathy: Report of 2 cases and review of the literature. *Seminars in Arthritis and Rheumatism* 38(6):458–466. Doi:10.1016/j.semarthrit.2008.07.001.

Yoshida C, Takeuchi M. 2008. Histiocytic sarcoma: Identification of its histiocytic origin using immunohistochemistry. Case report. *Internal Medicine* 47(3):165-169. Doi: 10.2169/internalmedicine.47.0386.

Zakai NA, McClure LA, Prineas R, Howard G, McClellan W, Holmes CE, Newsome BB, Warnock DG, Audhya P, Cushman M. 2008. Correlates of anemia in American Blacks and Whites. *American Journal of Epidemiology* 169(3): 355–364. Doi:10.1093/aje/kwn355.

Zakhari S. 2015. Chronic alcohol drinking: liver and pancreatic cancer? *Clinics and Research in Hepatology and Gastroenterology* 39: S86–91.

Zhang XH, Wang Q, Gerald W, Hudis CA, Norton L, Smid M, Foekens FA, Massague J. 2009. Latent bone metastasis in breast cancer tied to Src-dependent survival signals. *Cancer Cell* 16(1):67–78. Doi:10.1016/j.ccr.2009.05.017.

Zinzani PL, Carrillo G, Ascani S, Barbieri E, Tani M, Paulli M. 2003. Primary bone lymphoma: experience with 52 patients. *Haematologica* 88:280-285.

CHAPTER 7: APPENDIX