

The β -adrenoceptor and endocrine abnormalities in juvenile depression

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Summary

Neuro-endocrine disturbances were determined in juvenile depressives and possible correlations with biochemical abnormalities investigated. It was established that, as in adult depressives, the young also showed a high incidence of cortisol non-suppression after dexamethasone administration. In addition, a positive correlation observed between lymphocyte β -adrenoceptor maximal number of binding sites and basal cortisol levels stresses the involvement of the noradrenergic neurotransmission system.

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Psychiatrists have become increasingly aware of the existence of major depressive disorder in children and adolescents. A biological background for these disorders was established when it was shown in several groups that the dexamethasone suppression test (DST) was as specific and sensitive in children as in adults.¹⁻³ As a result, depression in childhood can also be treated by properly administered tricyclic antidepressant drugs, such as imipramine.⁴⁻⁶

In 1984, Siever and Davis⁷ postulated that the fundamental defect contributing to the aetiology of depression was likely to be dysregulation of the noradrenergic neurotransmitter system. In addition, depression is frequently associated with neuro-endocrine disturbances. These include a blunted thyrotrophin (TSH) response to TSH-releasing hormone (TRH)⁸⁻¹⁰ and also hypersecretion of cortisol and non-suppression of serum cortisol levels after administration of the synthetic steroid, dexamethasone; these features are characteristic of patients with major depressive disorder.¹¹⁻¹⁶ In the case of the blunted TSH response to TRH, it is not clear yet at which level of the hypothalamic-pituitary-thyroid axis TSH release is affected; however, it is now known that noradrenaline (NA) inhibits release of this neuropeptide via β -adrenoceptors.¹⁷ Regarding elevated basal cortisol levels and non-suppression of serum cortisol after dexamethasone suppression in patients with depression, it has been suggested that hypothalamic-pituitary-adrenal (HPA) axis activity may be increased due to either central noradrenergic deficiency and thus disinhibition of the axis^{15,18-20} or an excitatory effect of increased noradrenergic activity.²⁰⁻²²

Lymphocytes have been widely used as a model for indirectly evaluating changes in β -adrenoceptor binding and activity in adults with major depressive disorder.²³⁻²⁸ So far as is known,

only one study has investigated β -adrenoceptor binding parameters on lymphocytes of children and adolescents with the disorder.²⁹ This study was extended to investigate possible correlations between these binding parameters and TSH cortisol levels before and after TRH and dexamethasone administration, respectively.

Patients and methods

All patients were examined by a psychiatrist and treated in the Department of Child Psychiatry, Tygerberg Hospital. Each child and parent were assessed by the Interview Schedule for Children (ISC).³⁰ The ISC symptoms and signs contributed towards a particular diagnosis only if they met the operationally defined level of clinical severity, e.g. a rating > 5 on a 0 - 8-point scale.³⁰ The diagnosis of major depressive disorder was made according to criteria in the *Diagnostic and Statistical Manual of Mental Disorders*, 3rd edition.³¹ All children presenting with symptoms of depression or a suicide attempt were evaluated. None of these children had ever received antidepressant treatment. The exclusion criteria were an organic deficit, mental retardation, and childhood schizophrenia or autism. Permission to undertake this study was obtained from the Ethics Committee, Tygerberg Hospital. A volunteer control group of normal, healthy schoolchildren was also evaluated.

Patients and controls were kept drug-free for 3 weeks before the receptor binding studies were performed. Blood samples were always collected between 08h00 and 08h30 to avoid possible circadian variation.

Dexamethasone suppression test (DST). This test^{13,32} involved suppression of cortisol secretion by the administration of either 0.5 mg (for patients \leq 10 years) or 1 mg (adolescents) of dexamethasone taken orally at 23h00. Cortisol levels were determined in blood samples taken at 08h00, 16h00 and 23h00 the following day. Non-suppression was recorded if any of the cortisol levels were > 140 nmol/l.

TRH-TSH stimulation test. This test involved the serum TSH response to an intravenous test dose of TRH 500 μ g. Blood samples were collected immediately before and at 20 minutes, 60 minutes and 90 minutes after the injection. Normally there is a marked increase in TSH release, peaking about 30 minutes after TRH administration. A blunted response was described as a Δ TSH (maximum increase in serum TSH) < 7 μ U/ml.³³

Preparation of lymphocyte membranes. This was done by the method of Davies and Lefkowitz,³⁴ slightly modified by Carstens *et al.*^{28,29}

Binding of ³H-dihydro-alprenolol to lymphocyte membranes. The method used to measure ³H-dihydro-alprenolol (³H-DHA) binding to lymphocyte membranes was essentially that of Davies and Lefkowitz,³⁴ as described by Carstens *et al.*^{28,29}

Analysis of data. The apparent dissociation constant (K_d) and the maximal number of binding sites (B_{max}) were determined for lymphocyte β -adrenoceptors of each subject by means of linear transformation of the ³H-DHA binding data.^{29,35} For the statistical evaluation of the data, the Spearman rank-correlation test³⁶ was used, and also the Mann-Whitney *U*-test.³⁷

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Results

The correlations between parameters of ^3H -DHA binding to lymphocyte membranes and serum TSH and cortisol levels of normal controls and children and adolescents with major depressive disorder (MDD) were investigated. Since a large number of the young patients had made a suicide attempt, the population was divided into those with (MDDS) and those without (MDD) a suicide attempt.

Table I shows the β -adrenoceptor-binding parameters of the controls and subpopulations of young depressives.²⁹ No significant differences were observed between K_d values of any of the subpopulations (Mann-Whitney U -test). However, the B_{\max} values of the subpopulation with MDD, as well as the total patient population were significantly elevated compared with controls ($P < 0,02$ and $P < 0,005$, respectively; Mann-Whitney U -test).

In Tables II and III the different thyroid and cortisol levels, respectively, are given for the control and patient populations. Although the free thyroxine (T_4) values of all controls and patients were well within the normal range, the patient subgroups had values significantly higher compared

with controls (Table II — MDD $P < 0,05$; MDDS $P < 0,02$; total patient population $P < 0,005$; Mann-Whitney U -test). Interestingly, a blunted TSH response to TRH administration was observed in 17% of the controls, 17% of the MDDs and 15% of the MDDSs. No correlation between any of the thyroid parameters and the lymphocyte β -adrenoceptor-binding parameters was observed.

As far as the DST was concerned, so significant differences were observed between any of the control and patient parameters (Table III). In 35% of the controls there was no suppression of cortisol levels after dexamethasone administration, whereas 50% and 69%, respectively, of the MDD and MDDS patients showed dexamethasone non-suppression.

In order to investigate possible correlations in the controls and different patient subpopulations between the lymphocyte β -adrenoceptor-binding parameters and TSH or cortisol values of these children, a Spearman-rank correlation test was performed.

As far as the controls were concerned, a positive correlation was observed between the β -adrenoceptor B_{\max} and basal cortisol values (Spearman-rank coefficient, $r = 0,46884$; $P = 0,02$). No correlation was found in either patient subpopu-

TABLE I. K_d AND B_{\max} VALUES OF ^3H -DHA BINDING TO LYMPHOCYTE MEMBRANES OF NORMAL HEALTHY CONTROLS AND CHILDREN WITH MAJOR DEPRESSIVE DISORDER

Population	No. of patients	Age (yrs) mean \pm SD	K_d (nM)		B_{\max} (fmol/mg protein)	
			Mean \pm SD	Median	Mean \pm SD	Median
Controls	23	16,4 \pm 1,0	0,79 \pm 0,40	0,60	42,5 \pm 9,8	38,0
MDD	12	13,4 \pm 2,5	0,68 \pm 0,26	0,63	50,8 \pm 10,9*	51,3
MDDS	13	15,8 \pm 2,8	0,64 \pm 0,54	0,44	47,1 \pm 9,61	48,5
MDD + MDDS	25	14,6 \pm 2,9	0,66 \pm 0,42	0,56	48,8 \pm 10,2**	49,1

Neither a seasonal nor an age-dependent variation was observed for either the K_d or the B_{\max} values of the patient subpopulations (one-way analysis of variance²⁹).

MDD = major depressive disorder; MDDS = major depressive disorder with a suicide attempt.

*Significantly different from controls ($P < 0,02$).

** $P < 0,005$.

TABLE II. DIFFERENT THYROID PARAMETERS FOR CONTROLS AND DEPRESSED CHILDREN

Population	No. of patients	Free T_3 (pmol/l)	Free T_4 (pmol/l)	TSH basal (mU/l)	Δ TSH (mU/l)
Controls	23	7,3 \pm 1,52	14,6 \pm 3,21	2,0 \pm 0,69	13,7 \pm 6,8
MDD	12	7,9 \pm 1,55	16,8 \pm 1,99*	1,5 \pm 0,78	14,6 \pm 12,6
MDDS	13	6,7 \pm 2,08	17,5 \pm 3,01**	2,0 \pm 1,24	14,6 \pm 12,9
MDD + MDDS	25	7,2 \pm 1,91	17,1 \pm 2,54***	1,8 \pm 1,05	14,6 \pm 12,5

*Significantly different from controls ($P < 0,05$).

** $P < 0,02$.

*** $P < 0,005$.

Normal values: $T_3 < 20$ years 4,2 - 10,4 pmol/l; T_4 8,8 - 23,0 pmol/l; basal TSH 0,4 - 4,0 mU/l.

TABLE III. DIFFERENT CORTISOL PARAMETERS FOR CONTROLS AND DEPRESSED CHILDREN

Population	No. of patients	Cortisol (nmol/l)			
		Basal	08h00	16h00	23h00
Controls	23	455 \pm 144	75 \pm 43	138 \pm 141	110 \pm 112
MDD	12	619 \pm 209	111 \pm 85	98 \pm 55	81 \pm 52
MDDS	13	428 \pm 185	161 \pm 179	171 \pm 102	161 \pm 165
MDD + MDDS	25	499 \pm 211	136 \pm 139	138 \pm 93	123 \pm 129

Normal 08h00 cortisol levels 193 - 690 nmol/l.

lations. However, treating the patients as a single population, revealed a similar positive correlation between the β -adrenoceptor B_{max} and basal cortisol values (Spearman-rank coefficient, $r = 0,60294$; $P 0,02$). No correlations were observed in any of the groups between the β -adrenoceptor K_d values and the other parameters measured.

Discussion

In this study it was found that, although the thyroid functions of both the control and patient groups were clinically normal, the patients with MDD, those with MDDS, as well as the total population had T_4 values significantly higher compared with controls. In addition, the controls and both patient subgroups showed a similar incidence of blunted TSH response to TRH administration. No correlation between the different thyroid functions and the lymphocyte β -adrenoceptor-binding parameters was observed.

Although in the case of the DST the different patient subgroups did not differ significantly from the controls regarding the basal and postdexamethasone cortisol levels, a high incidence of cortisol non-suppression was observed in both patient subgroups, compared with controls. In addition, a positive correlation was observed between the β -adrenoceptor B_{max} and basal cortisol values of the controls. As far as the different patient subgroups were concerned, a similar correlation was found only in the total patient population.

TSH secretion from the anterior pituitary may be influenced by several factors acting both centrally and peripherally.³⁸⁻⁴¹ It has been suggested that the central noradrenergic system exerts both stimulatory and inhibitory influences on TSH secretion.^{38,42,43} In a recent study¹⁷ it was shown that the α -adrenoceptor agonist, clonidine, caused an increase in rat serum TSH levels, while the β -adrenoceptor agonist, isoproterenol, significantly decreased these levels. Noradrenergic disturbances have been implicated in major depressive disorder.^{7,44} In addition, this affective disorder is also associated with abnormalities in the TSH response to TRH administration.^{8,45}

Both patient subgroups studied revealed noradrenergic disturbances, since their lymphocyte β -adrenoceptor B_{max} values were found to be significantly elevated compared with controls.²⁸ However, since the blunted TSH response to TRH administration observed in the controls was comparable to that found in the young depressives, it does not seem possible that the disturbed noradrenergic function plays a role in the TSH response at a young age. The elevated T_4 levels of the depressives cannot at this stage be explained.

As mentioned before, a large number of juvenile patients with major depressive disorder do not experience suppressed serum cortisol levels after dexamethasone administration.¹⁻³ Disturbances in central noradrenergic activity have also been implicated in this observation.^{18,19,22} In the present study, patients with MDD, and also those with MDDS showed high incidences of cortisol non-suppression compared with controls. The fact that the young depressives had significantly higher lymphocyte β -adrenoceptor B_{max} values compared with controls, and also abnormal DST responses, emphasises the noradrenergic abnormality in these young depressives. Further support for noradrenergic control stems from the observation that, although only found in the total population of depressives, the positive correlation between the basal cortisol and β -adrenoceptor B_{max} values is still retained. Unfortunately, the results from this study do not distinguish between depression with and without suicidal behaviour.

In conclusion, biochemical abnormalities exist in juvenile major depressive disorder, which, as is the case with adult depressives, are accompanied by endocrine disturbances.

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Motion sickness — vestibular habituation with the centrifuge

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Summary

People who are prone to motion sickness have a directional preponderance of nystagmus to the left. Centrifuging will change this preponderance to the right in most people, and at the same time reduce their tendency towards motion sickness but only with regard to air travel.

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Previous studies^{1,2} at the Institute of Aviation Medicine, Pretoria, showed that personnel in the Air Force and Navy who were exposed to vestibular stimulation in the course of their duties fell into three groups: (i) those who were not prone to motion sickness at all; (ii) those who were prone but adapted after a while; and (iii) those who seemingly did not adapt at all and were unable to work satisfactorily because of motion sickness.

Personnel from groups 1 and 2 had a directional preponderance of nystagmus to the right when tested electro-nystagmographically, but those in group 3 and a control group of 395 persons^{1,2} randomly selected showed that the majority had a directional preponderance of nystagmus to the left.

A study was undertaken to further investigate the efficacy of centrifuging in the prevention of motion sickness by habituation.

Subjects and methods

Forty-eight military personnel (20 from the Air Force, 28 from the Navy), who all had a history of motion sickness that prevented them from fulfilling their duties, were selected for the study. They were all subjected to physical and vestibular examination, including a torsion swing test with electro-nystagmographic monitoring. The torsion swing test^{3,4} is well suited to assessing directional preponderance of nystagmus. The stimulus elicits good oculomotor response and provides the type of stimulation that closely mimics the physiological conditions encountered in daily situations. The test is carried out in a semi-darkened room and the subject maintains mental alertness by doing mental calculations while the directional preponderance of nystagmus is calculated. The total number of nystagmus beats both to the left and right are counted during 6 oscillations of the torsion swing and the following formula is used to express the directional preponderance of nystagmus in per cent:

$$\frac{(L - R)}{(L + R)} \times 100.$$

The results before centrifuging are shown in Table I.^{4,5}

The personnel were then exposed to repeated vestibular stimulation by centrifuging and a daily programme in which increments of 0,1 g occurred was instituted until a total of 1,5 g was reached. Subjects were seated in a gondola facing the

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TABLE I. NYSTAGMUS RESULTS BEFORE CENTRIFUGING

Air Force (N = 20)		Navy (N = 28)	
Left	Right	Left	Right
14	6	21	7