The Dexamethasone Suppression Test in Depression: A World Health Organisation Collaborative Study

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Abstract

The response to the Dexamethasone Suppression Test (DST) was examined in 543 patients suffering from major depressive illness and 246 healthy controls from 13 research centers, from 12 different countries in a World Health Organisation Collaborative Study. In almost all the centres, the post-dexamethasone plasma cortisol concentration was significantly higher in patients than in controls. There were large and significant variations in DST between centres, which were not related to variables such as age, sex and severity of illness. These factors, however, had a modest, but clinically non-significant contribution to the variation in DST results in the whole population of patients.

Introduction The introduction of the Dexamethasone Suppression Test (DST) for the investigation of the psychobiology of depression and its application in management has been a land-mark in the development of biological psychiatry. There have been hundreds of studies on its use in diagnosis, in management and as a paradigm to investigate the pathophysiology of depressive illness (1,2). The balance of evidence indicates that the DST based on 1 mg dexamethasone given at night, a plasma cortisol concentration estimate in the afternoon of the next day and a cut-off point of 50 mg/ml has satisfactory sensitivity for depressive illness (50%) and high specificity (90%) when its results are compared with those obtained in normal controls and patients with minor psychiatric conditions and chronic schizophrenia (2)

During the sixth exchange of visits of heads of W.H.O. Collaborating centres for Research and Training in Biological Psychiatry in Basle in 1980, the importance of studies on biological approaches to the classification of mental disorders and depression in particular was stressed. This led to the participants of this meeting initiating a multi-centre study on the use of the DST as a biological indicator of depressive illness. Thirteen research centres from twelve countries from Europe, Africa, Asia and North America took part in the study. These centres were located in Basle, Brussels, Budapest, Casablanca, Copenhagen, Epsom, Irvine, Lucknow, Moscow, Munich, Nagasaki, Sapporo and Utrecht. The MRC Neuropsychiatry. Research Centre in Epsom which initiated the study was identified as the lead centre which co-ordinated the study and received all the collected data from all centres, including plasma samples for the estimation of cortisol concentration. The investigators were trained in the use of the Hamilton Rating Scale for Depression (3) and the reliability study of the Newcastle Diagnostic Scale (4) was undertaken by correspondence by the time the study was started. All investigators were sent copies of the Newcastle Scale with definitions of its items and instructions with case summaries on which the Newcastle Scale was completed.

A brief report of the main findings of the study was published (5). The results supported the view that post- dexamethasone plasma cortisol concentrations were higher in depressive patients than in normal controls. There was, however, marked variation between centres in the prevalence of non- suppression whilst the majority of healthy control individuals from various countries responded normally to the DST. These findings confirmed the notion that DST non-suppression is one of the more robust biological markers of depressive illness. The report confined itself to reporting the rate of non-suppression in patients and controls from all centres. In the present report, however, we provide the detailed and extensive analysis of the

data from all centres, including the data obtained from the Standardized Schedule for Assessment of Depressive Disorders (WH0 - SADD) (6) completed on a subgroup of these patients in Basle, Brussels, Copenhagen, Irvine, Lucknow, Moscow, Munich, Nagasaki and Utrecht. Part one of this report provides the results on the diagnostic performance of the DST and examines its relationships with age, sex, classification, severity and other variables. Part two provides the results and detailed analyses on DST status in relation to SADD data.

Patients and Methods All patients from all centres had to fulfill the Research Diagnostic Criteria (RDC) for major depressive disorder (7) before entry into the study. They were consecutively admitted to the study without any pre-selection. The patients were classified according to the International Classification of Diseases - 9 th Revision (ICD-9) (8) and the Newcastle Diagnostic Scale (4). Severity of patients' depressive symptoms was rated on the Hamilton Rating Scale for Depression (HRSD) (3). Information was obtained on the patients' age, sex, presence of physical illness and medication received in the two weeks prior to the administration of dexamethasone. The SADD provided demographic and clinical information including psychiatric history and mental state examination. During the early stages of the study, it became apparent that the small number of abnormal responders previously reported in a healthy population might also vary from country to country and all investigators were asked, if possible, to supply post-dexamethasone plasma from normal controls.

Dexamethasone Suppression Test

One mg of dexamethasone (Oradexon - Organon (R)) was administered to all patients at 23.00 hr (except for the patients from the Epsom Centre who received the dexamethasone at 20.00 hr). Blood samples were collected into heparin tubes from the patients between 15.00 and 16.00 hr the following day; the plasma was obtained by centrifugation; it was stored at -20°C until a sufficient number of samples had been acquired for transport in dry ice by air to the laboratory at the Epsom Centre. Cortisol was estimated using a radioimmunoassay technique (Amerlex(R)). The samples from Epsom were assayed using a radioimmunoassay kit produced by CIS International. It has already been reported that there is an excellent agreement between these two kits (9).

The results of the cortisol assays are expressed for each centre in two ways:(a) as the mean post-dexamethasone plasma cortisol concentration and (b) as the percentage of abnormal responses. A post-dexamethasone cortisol level of <50 ng/ml was taken as a normal response, and an abnormal response was taken as >50 ng/ml of cortisol. The sensitivity of the DST refers to the percentage of patients with major depression who had an abnormal response, whilst the specificity refers to the percentage of normal control subjects who had a normal response. The scientific officer who estimated the cortisol levels was unaware of the clinical details of the patients.

Statistical Methods The data for this analysis were age, sex, HRSD score, Newcastle score, ICD-9 diagnosis, post-dexamethasone cortisol concentration, presence or absence of physical illness and whether the patients received psychotropic or other medication in the two weeks preceding the DST. The frequency distribution of cortisol concentrations in the whole group of patients and the whole group of normal controls were plotted as percentages of suppressors/non-suppressors in both groups. The influence of age, sex, HRSD and the Newcastle scale on the DST results was determined by comparing the mean of post-dexamethasone cortisol concentration in subgroups in the whole patient group, subdivided according to sex (males versus females), age (age < 65 versus age > 65 years), HRSD (HRSD > 16 versus HRSD < 16), Newcastle scale (Newcastle score <5 versus > 6), ICD-9 diagnosis, and the presence or absence of physical illness or whether they received psychotropic or other medication. Product-moment correlations and partial correlations were calculated between age, HRSD, Newcastle scale and cortisol levels. Finally multiple-regression analyses were undertaken to determine the contribution of all variables to DST with post-dexamethasone cortisol

concentration (untransformed and log-10 transformed) as the dependent variable and all the other variables as independent variables.

Results Plasma samples for cortisol analysis were received from 562 patients. All except 31 were in-patients at the time of the test. Nineteen patients had to be omitted from the study: two because their concomitant medication was thought to have affected the DST, three because of a severe physical illness, three because of incomplete clinical data and 11 because their diagnosis did not conform to the RDC criteria for major depressive disorder. The reasons for this non-conformity to RDC criteria are not known. These patients were excluded from the analysis before evaluating their DST results. Plasma was also received and assayed for cortisol from 246 controls.

Table (1) indicates the number of patients in each of the research centres who participated in the study, their sex distribution, their mean age, HRSD and Newcastle scores and percentage with ICD-9 diagnoses of endogenous depression (296.1 and 296.3).

Most centres (all except Basle, Irvine, Moscow and Utrecht) collected and sent post-dexamethasone plasma samples from healthy controls, 246, altogether (Table 2). There was no stipulation that controls should be age or sex matched to the patient group since these variables were not thought to influence the DST.

Table (3) indicates the mean post-dexamethasone plasma cortisol concentration of patients and controls. When the centre included controls in the study, the statistical comparison was made between the patient and the control groups within the centre. Since some centres did not provide controls, a statistical comparison has also been made between the mean for the patients' group in that centre and the total (n = 246) control group. In seven out of the nine centres in which comparison was possible, the mean post-dexamethasone plasma cortisol concentration of the patients was significantly higher than the mean of the control group. In 12 out of 13 centres, the mean post-dexamethasone plasma cortisol concentration of the patients was higher than the mean of the total control group. Table (3) also indicates the percentage of abnormal responses to dexamethasone administration in patients and controls, using the 50 mg/ml cut-off point.

Table 1: Clinical characteristics of patients

	n		Age	HRS-D	Newcastle score		% ICD endogenous
	M	F	years		< 5	< 6	
Basle	13	26	57.1+2.3	24.2+1.0	14	25	74.3
Brussels	20	16	47.7+2.2	24.8+1.1	14	22	100
Budapest	4	44	53.6+1.9	24.3+0.8	1	47	100
Casablanca	8	4	38.3+2.7	28.5+1.3	2	10	91.7
Copenhagen	4	10	49.4+3.6	15.0+1.0	9	5	92.8
Epsom	28	91	59.4+1.3	22.2+0.4	41	78	86.8
Irvine	12	14	38.2+2.9	21.6+1.4	4	22	96.1
Lucknow	16	9	37.9+1.5	25.3+1.0	5	20	82.6
Moscow	9	45	48.2+2.1	21.4+0.7	5	49	90.7
Munich	11	50	48.6+1.8	21.4+0.6	25	36	68.8
Nagasaki	17	11	43.7+2.4	17.1+2.0	3	25	100
Sapporo	31	21	46.8+1.8	19.6+0.8	22	30	78.8
Utrecht	9	20	45.0+2.1	22.7+1.1	10	19	82.7

Results expressed as means + s.e.m.

Table 2: Control Subjects

Centre	n	M	F	Age (years)
Brussels	8	4	4	29.0+2.2
Budapest	27	10	17	31.3+1.3
Casablanca	22	16	6	28.2+8.0
Copenhagen	11	3	8	46.9+2.8
Epsom	79	27	52	45.1+1.6
Lucknow	25	25	0	-
Munich	26	9	17	35.2+2.7
Nagasaki	15	8	7	40.9+2.8
Sapporo	33		7	39.2+1.5

Table 3: Post-dexamethasone plasma cortisol concentration (mg/ml) and percentage of abnormal responses of patients and controls

	Patients		Controls	
	Plasma	% abnormal concentration responses	Plasma	% abnormal concentration responses
Basle	82.2+10.9!	59	-	-
Brussels	92.6+12.3*	69	13.4+2.4	0
Budapest	65.8+7.2*!	54	21.3+3.3	7
Casablanca	37.4+12.1	25	26.2+8.0	14
Copenhagen	116.5+23.2*!	71	16.5+1.8	0
Epsom	101.0+6.7!	70	26.2+2.6	11
Irvine	52.6+9.0!	42	-	-
Lucknow	46.8+8.2!	48	48.9+9.4	40
Moscow	22.5+4.2	15	-	-
Munich	91.1+10.1*!	62	25.0+3.5	8
Nagasaki	42.0+10.9*	21	13.5+1.0	0
Sapporo	39.3+6.8*	23	16.3+2.0	3
Utrecht	66.8+11.7!	41	-	-

Results expressed as means + s.e.m.

* Patients significantly different (P<0.02) from controls from same centre.

! Patients significantly different (P<0.02) from controls from all controls.

Table 4: Suppressor/non-suppressor status in relation to ICD and Newcastle endogenous/non-endogenous status.

	ICD (%)		Newcastle (%)	
	Endo	Non-endo.	Endo	Non-endo.
Suppressors	234 (51.2)	30 (48.4)	190 (49.9)	83 (52.9)
Non-Suppressors	223 (48.8)	32 (51.1)	191 (50.1)	74 (47.1)

Table 5: Predications of post-dexamethasone cortisol by multiple regression analyses

Regression analysis: Non stepwise

Dependent variable: Cortisol

Variable entered	B	SE B	Beta	T	P
Age	.90	.27	193	3.37	.0009
Sex	14.15	8.04	.098	1.76	.0795
Hamilton Score	.21	.68	.019	.31	.7627
Newcastle Score	-.71	1.63	-.028	-.44	.6643
ICD	3.06	1.63	.111	1.89	.0602
Antidepressants	21.72	7.58	.164	2.87	.0044
Neuroleptics	.71	8.69	.005	.081	.9355
Anxiolytics	3.40	9.39	.021	.362	.7176
Benzodiazepine	-3.42	8.51	-.023	-.402	.6879
Lithium	20.56	18.82	.061	1.092	.2755
Other Medication	14.69	10.33	.080	1.423	.1558
Physical Illness	17.33	16.46	.060	1.054	.2927

F. value overall = 2.3 $p<0.01$ n = 330 multiple R^2 0.08 (i.e. 8% of variance explained)

B = Coefficient

SE B = Standard error of B

Beta = Standardised coefficient

T = Student t

P = Probability

Table 6: Patient group comparisons on age, DST, Hamilton and Newcastle scores

Group	Comparisons
DST status Suppressors vs. Non-suppressors	Age p<0.001, HRDS p<0.01
Sex Males vs. Females	Age p<0.01, Cortisol p<0.001
Severity HRSD < 15 vs. HRSD > 16	Newcastle p<0.001
Age Groups < 65 yrs vs. > 65 yrs	Cortisol P<0.01, Newcastle p <0.01
Newcastle Classification Endogenous V Non-endogenous	Age p <0.001, HRSD p<0.001
ICD Classification Endogenous vs. Non-endogenous	Age p<0.01, HRSD p<0.01, Newcastle p<0.001

Table 7: Product-moment correlations of age, Hamilton, Newcastle scores and post-dexamethasone cortisol (n = 521)

	Age	HRSD	Newcastle	Cortisol
Age	1.00	0.09*	0.16***	0.17***
HRSD	0.09*	1.00	0.31***	0.11**
Newcastle	0.16***	0.31***	1.00	0.06
Cortisol	0.17***	0.11**	0.06	1.0

Partial correlations of age, Hamilton score, Newcastle score, and Cortisol concentrations

Age/Newcastle	controlling for Cortisol	r = 0.16, P<0.001
Age/Cortisol	controlling for Newcastle	r = 0.17, P<0.001
Newcastle/Cortisol	controlling for Age	r = 0.03, NS
Newcastle/Hamilton	controlling for Cortisol	r = 0.31, P<0.001
Cortisol/Hamilton	controlling for Newcastle	r = 0.1, P<0.03
Newcastle/Cortisol	controlling for Hamilton	r = 0.02, NS
Age/Hamilton	controlling for Cortisol	r = 0.07, NS
Age/Cortisol	controlling for Hamilton	r = 0.17, P<0.001
Cortisol/Hamilton	controlling for Age	r = 0.1, P<0.03

In Controls

Age / Cortisol	r = -0.12, P<0.05
*	P< 0.05
**	P <0.01
***	P < 0.001

Table 8: Characteristics of the main ICD-diagnosis: age, Hamilton, Newcastle scores and cortisol concentrations (results mean +50)

ICD-9	n	Age (yrs)	HRSD Score	Newcastle Score	Cortisol (ng/ml)	% NS
Controls	248	37.6+11.7	-*	-**	24.9+25.9	10.9
296.1	392	51.7+15.1	22.3+6.2	6.9+2.2	70.1+69.6	48.2
296.3	65	48.8+13.2	23.8+5.5	8.0+1.6	71.2+58.6	52.3
300.4	46	40.8+12.3	19.4+4.3	3.1+1.8	60.6+65.5	45.7
309.1	16	50.9+11.1	21.2+6.1	3.4+1.5	92.5+77.5	68.8

296.1 Manic depressive psychosis, depressed type

296.3 Manic depressive circular currently depressed

300.4 Neurotic depression

309.1 Prolonged depressive reaction

NS Non-suppressors

* Significantly lower than all groups P<0.001

** Significantly lower than 296.1, 296.3 and 300.4 P<0.0001 and 309.1 P<0.05

Table 9: Cortisol concentrations and concurrent medication in the whole population (mean + SD).

		Cortisol (ng/ml)		
Medication	n	on medication	n	no medication
Antidepressants	195	57.9+67.8	227	65.6+61.0
Neuroleptics	91	60.2+62.6	331	62.6+64.8
Lithium	13	53.6+43.1	409	62.4+64.9
Hypnotics	97	62.4+67.6	325	61.9+63.4
Anxiolytics	63	66.7+61.1	359	61.3+64.9
Other Medication	50	61.5+48.9	292	68.2+68.1
Physical Illness	20	47.5+45.8	322	68.4+66.6

Analysis of Differences between Centres

One-way analysis of variance showed statistically significant differences between centres on all variables: age, HRSD, Newcastle score, Cortisol (PCO.OO1). Cross tabulation and chi- square analysis of the distribution of sex, ICD categories, Newcastle groups and the presence or absence of all psychotropic and nonpsychotropic medication also showed significant differences between centres (p<O.OOl).

Student - Newman - Keuls tests (5% level of significance) showed patients from Moscow, Sapporo and Nagasaki to have significantly lower cortisol (untransformed) than Utrecht,

Epsom, Munich, Copenhagen, Brussels and Basel, whilst Casablanca also showed lower cortisol levels than Epsom, Munich, Brussels and Basel. For log 10 transformed cortisol from patients, Moscow and the two Japanese centres showed lower values (P<0.05) than all the other centres except for Casablanca which in turn had significantly lower values than Budapest, Epsom, Utrecht, Munich, Brussels and Basel.

The percentage of suppressors/non-suppressors (cut-off point >50 ng/ml) in the endogenous and non-endogenous diagnostic groups (ICD and Newcastle) were not significantly different (Table 4).

The contribution of all variables (independent) excluding the centone as variable to the variance in cortisol in the whole patient population (dependent) was examined by multiple regression analysis (Table 5). Variation of all these variables accounted for 8% of the variation in cortisol. Stepwise analysis, showed that older age (2% of variance) being drug-free from antidepressants (4.3% of variance) and having nonendogenous depressions on ICD (5.5% of variance) had a statistically significant contribution to the variance in cortisol values.

The frequency distribution of post-dexamethasone cortisol concentrations were plotted in both the whole group of patients and the whole group of controls (Figure 2) with a percentage of non-suppressors of 49.2% in patients and 10.9% in controls, a statistically highly significant difference (P<.001).

Table 6 shows comparisons between patient groups on age, cortisol, HRSD and Newcastle scores. The group of non- suppressors was significantly older and had a significantly greater mean HRSD than suppressors. Non-suppressors, however, had a similar mean Newcastle score to suppressors. Female patients were significantly older and had significantly higher post-dexamethasone cortisol concentrations than males. Males and females had similar HRSD and Newcastle scores. Patients with HRS >16 had higher mean Newcastle scores than those with HRS <15 but no significant differences for cortisol or age. Older patients (age > 65 years) had significantly higher post-dexamethasone cortisol concentrations than patients age of less than 65 years and had significantly higher Newcastle scores than younger patients. Patients who were classified as endogenous on the Newcastle Scale had significantly higher mean age and HRSD than those classified as non-endogenous but no significant difference for cortisol concentrations. These associations were confirmed by product - moment correlations of age, HRSD, Newcastle scores and 'J post-dexamethasone cortisol levels (Table 7). Table 7 also shows partial correlations of these variables with the largest and most significant correlation between age and cortisol level (P<0.001) after excluding the influence of HRSD whilst the correlation between cortisol and HRSD after excluding the influence of age is a modest one (P<.03). In the control group, age showed a modest negative correlation with cortisol levels (P<.05). Table 8 shows the data for the main ICD diagnostic subgroups in comparison with the whole group of normal controls: the normal controls group was significantly younger than the patients groups, except for the patients with ICD diagnosis 300.4 (neurotic depression) and had a significantly lower mean cortisol level than all the four patient groups (P<0.001). The percentage of non suppressors was significantly greater in all depressive groups than controls (P<0.001). There were no significant differences between the four patient groups in cortisol concentrations. The two patient groups with endogenous depression (ICD 296.1 and 296.3) had significantly greater mean Newcastle score than the nonendogenous groups (ICD - 9 300.4 and 309.1). There were no significant differences in mean cortisol levels between patients who received psychotropic or other medication and those without medication or between those who had serious physical illness and those without physical illness (Table 9).

Discussion These results show marked variations in the rates of non- suppression in groups of depressive patients from various countries and ethnic groups. The sensitivity of the DST varied between 15 in Moscow and 71 in Copenhagen with an overall sensitivity of 49.2% in the whole population if a criterion of non-suppression of cortisol >50 ng/ml was considered. The specificity of the DST varied between 100 and 60 with an overall specificity of 89.1% in normal controls. These results are similar to the overall results obtained in the world literature which indicates a sensitivity of 47% and a specificity of 90% for depressive

illness (1). DST non-suppression was not associated with endogenicity, as endogenous and non-endogenous depressives defined according to the ICD and the Newcastle Scale had similar rates of non-suppression and there was no significant correlation between cortisol levels and Newcastle scores in the whole population. This finding is not in harmony with the notion that the DST shows higher sensitivity for endogenous depression than neurotic depression (10), a claim that has not been substantiated by many other investigators (2).

DST non-suppression was, however, associated with increased severity on the HRSD, greater age, female sex and having no antidepressant results which are consistent with the findings in a number of studies (11). The correlation between age and post- dexamethasone cortisol levels in the control group was, however, a negative one. Previous investigators have found a similar trend for an association between increased age and DST non- suppression in depressive patients and normal controls (11). It is uncertain whether differences in age between depressive patients and controls in most centres have contributed to differences in DST results.

The single most important contributing variable to the variance in cortisol was the centre from which the patients were recruited. The patients from various centres showed statistically significant differences on all the demographic and clinical variables: there were significant differences between centres in cortisol, age, sex, ICD, Newcastle diagnosis, HRSD and the administration of psychotropic medication. Moscow, the two Japanese centres of Sapporo and Nagasaki and Casablanca had significantly lower levels of cortisol in comparison with the other centres. These differences did not, however, appear to be related to differences in age, sex distribution and HRSD. The Japenese centres had a relatively lower percentage of female patients than the other centres, but similar mean HRSD and age. It is notable that the Japanese control groups also had lower cortisol levels than the other control groups. The Moscow group had one of the highest ratios of females to male patients, whilst Casablanca had a relatively low ratio of female to male patients and lower mean age. Copenhagen showed the lowest mean HRSD and relatively fewer endogenous patients, yet had the highest rate of non-suppression. Overall, demographic and clinical characteristics accounted for no more than 5.8% and 8.7% of untransformed and log-10 transformed cortisol values respectively, which indicates that most of the variance in cortisol values remains unaccounted for.

There were no technical contributing factors to the variation in the cortisol values since all assays were carried out at the same laboratory by the same technician using the same radioimmuno-assay. The contribution of compliance with dexamethasone could not be ascertained as plasma dexamethasone concentrations were not determined. It is likely, however, that compliance was high since 531 out of 562 patients studied were inpatients at the time of the DST.

The overall rate of abnormal DST response masks a large variation between patients from various centres similar to the results obtained in the world literature (1). The single most contributing factor to this variation is related to the centre of investigation reflecting differences in depressive populations from different countries and ethnic groups, differences that are not accounted for by age, sex severity and endogenicity.

Kleinman (12) has criticised the strong trend in World Collaborative Studies to emphasize similarities between clinical populations and overlook or ignore differences in search for universal clinical entities.

This study shows that populations with depressive illness diagnosed in accordance with strict diagnostic criteria show variation on a biological marker such as the DST, probably reflecting biological heterogeneity in depression. The DST may, therefore, provide a criterion to indicate more homogeneous sub- groups in depressive illness and whether this sub-group of patients with an abnormal DST response represents a homogeneous category with regard to treatment or to other clinical and biological characteristics remains to be demonstrated by further research.

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إختبار تثبيط الديكساميتازون فى مرض الاكتئاب

(دراسة تجميعية لمنظمة الصحة العالمية)

تمت دراسة مدى الاستجابة لاختبار تثبيط الديكساميتازون على ٥٤٣ مريضا يعانون من إضطراب الاكتئاب الوجدانى فى مقابل ٢٤٦ شخصا سليما كعينة ضابطه تم تجميعهم من ثلاثة عشر مركزا من مراكز البحوث فى إثنى عشر قطرا تحت إشراف منظمة الصحة العالمية.

وقد بينت النتائج فى مراكز الدراسة أن نسبة الكورتيزول بالدم بعد اختبار الديكساميتازون أعلى بصورة داله إحصائيا لدى مرضى الاكتئاب من الأشخاص الأسوياء (العينه الضابطه). وكانت هناك إختلافات كبيرة ودالة بين النتائج فى المراكز المختلفة ليس لها علاقة بالعمر أو الجنس أو شدة المرض مما يرجح أن مثل هذه العوامل ليست ذات تأثير كبير فى نتائج اختبار تثبيط الديكساميتازون لدى مجموع مرضى الاكتئاب.