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Abnormal Dexamethasone Suppression Test in Normal Females

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Summary: Eighty women taking part in a population study were subjected to a dexamethasone suppression test (DST) intended as a diagnostic aid for melancholia. The women were selected systematically from two age strata, 38 and 50 years. Fifteen subjects (19 per cent) were found to be non-suppressors. High post-dexamethasone serum cortisol concentrations were not the result of elevated concentrations of the main cortisol binder, transcortin. There were no differences between suppressors and non-suppressors as regards depressive symptoms, strain experience, body mass, gynaecological history, drug use, smoking, erythrocyte sedimentation rate, number of leucocytes, activity of serum aminotransferases and γ-glutamyltransferase, serum iron, bilirubin, ferritin content, serum growth hormone or serum prolactin. However, the non-suppressors reported a significantly lower ($P < 0.01$) orgasmic capacity in a questionnaire inquiry about two weeks before the DST. The outcome of the study indicates that DST as the presently recommended procedure for out-patients has a lower specificity for melancholia than has been reported previously.

The dexamethasone suppression test (DST) has been reported to be of diagnostic value to differentiate melancholia from other types of depression and other psychiatric disorders (for recent studies, see Brown *et al*, 1979; Nuller and Ostroumova, 1980; Schlesser *et al*, 1980; Carroll *et al*, 1981). The DST sensitivity has typically been quoted as 40–65 per cent and its specificity as higher than 90 per cent. A pathological outcome of the DST has, however, been rather common in a few other psychiatric syndromes, such as schizo-affective disorders and anorexia nervosa (Gerner and Gwirtsman, 1981) and it is regularly found in Cushing's syndrome which is sometimes associated with severe psychiatric symptoms. False positive results may be obtained in some conditions: the most important of these is reported (Carroll *et al*, 1981) to be current or recent use of drugs that induce accelerated metabolism of dexamethasone in the liver.

Nearly all investigations on DST have been directed at psychiatric populations. To our knowledge there have been no studies reported on representative samples of a general population. This paper will report the findings from such a study. Our aims were (1) to establish the rate of non-suppression of post-dexamethasone serum cortisol concentration in a female population sample performing the test as recommended for out-patients, (2) to relate the DST outcome to depressive symptoms, and (3) to relate the outcome to the physical health of the subjects, use of drugs, smoking habits, etc. in order to identify factors causing abnormal test results.

Subjects and Methods

Subjects

Eighty women were subjected to a dexamethasone suppression test, 47 subjects aged 38 and 33 aged 50. They were participants in a comprehensive population study of women in Göteborg, Sweden. This was the third phase of a longitudinal study taking place in 1980–1981. The group of 800 women studied was a systematically selected sub-sample of the participants. Pregnancy was the single exclusion criterion. By virtue of the sampling procedure and the high participation rate, the subjects studied are considered to be representative of women of the same age in the general population. Further details about the population

study, including the sampling procedure, have been given previously (Bengtsson *et al*, 1973*a*; Hällström, 1973; Bengtsson *et al*, 1978).

Dexamethasone suppression test (DST)

The subjects were informed of the purpose of the present study. Instructions were given orally and in writing and each participant gave information on a questionnaire brought by her to the laboratory. One mg of dexamethasone (Decadron®) was taken at 10.00 p.m. One blood sample for serum cortisol determination was obtained at 3.00 p.m. the following day. The blood samples were collected by experienced technicians using a vacuum tube technique, following the routine for out-patients at the Department of Clinical Chemistry. The first DST was performed about two weeks after the medical and psychiatric investigations. The non-suppressors were interviewed about their compliance with the instructions: all reported that they had taken the dexamethasone tablet at the exact time requested.

A second DST was performed after about three months on the non-suppressors, i.e. those with a serum cortisol concentration greater than 140 nmol/l (5 μg/dl). The persistent non-suppressors were subjected to a third test after about six months.

Questionnaire data

The Comprehensive Psychopathological Rating Scale (CPRS) (Åsberg *et al*, 1978) was modified into a self-assessment questionnaire. Twelve items were used for measuring the occurrence and intensity of the following depressive symptoms: sadness, pessimistic thoughts, tearfulness, suicidal thoughts, lassitude, inability to feel, indecision, concentration difficulties, inner tension, autonomic disturbances, muscular tension and hostile feelings. The scores from the self-assessment questionnaire correlate well with the original interview version of CPRS (Spearman's ϱ = 0.80).

Information about current sexual behaviour was obtained by questionnaire. The following items were self-assessed: strength of sexual desire (strong/moderate/weak/none), coital frequency, orgasmic capacity during intercourse (always/usually/seldom/never).

Information about current use of drugs was obtained by questionnaires, at the time of the original screening and at the time of DST.

Interview data

Information about subjective feelings of stress or strain was obtained in an interview. The subjects were asked whether they had had a feeling of stress or strain for a month or longer—including tension, fear, anxiety, or sleep disturbances—in connection with conflicts in the family, at work, etc. The amount of strain was rated according to the following six-point scale: never/occasionally/occasionally during the last five years/several times during the last five years/continuously during the last year/continuously during the last five years (Bengtsson *et al*, 1973*b*).

Premenstrual symptoms were rated according to the CPRS. The following items were assessed: autonomic disturbances, hostile feelings, sadness, fatiguability and concentration difficulties.

The following gynaecological variables were analyzed: menstrual status at the time of DST (follicular phase, luteal phase, post-menopausal status), menstrual cycle length, amount of menstrual flow, parity (sum of number of pregnancies, abortions and extrauterine pregnancies).

Smoking habits (number of cigarettes per day) and body mass loss during the last four months were recorded in an interview.

Physical examination

Comprehensive physical examinations were performed, including measurement of body height and body mass. Body mass index was defined as

$$\left[\frac{\text{body mass (kg)}\times 100}{\text{height (cm)}-100}\right]$$

Biochemical methods

Serum cortisol was measured with reagents from Diagnostic Products Corp., Los Angeles, USA. Information may be obtained from the authors (G.L.) as to the bias of this method, judged from results obtained through participating in an international immunoassay control programme. The assays were carried out in duplicate. Intra-assay coefficients of variation, calculated from the differences between the duplicates, were 3.3–7.2 per cent (mean 4.3 per cent) in the interval 0–280 nmol/l in nine consecutive assay series during a 3-month period at the time of the first DST series. The total (inter- plus intra-) coefficients of variation, calculated from the mean values from duplicate analyses of four serum pools, were 7.0 per cent (M = 83 nmol/l, n = 9), 4.7 per cent (M = 380 nmol/l, n = 9), 3.8 per cent (M = 440 nmol/l, n = 9), and 4.8 per cent (M = 890 nmol/l, n = 9).

The results obtained were compared with results from another radioimmunoassay with reagents from Farmos, Turku, Finland. A correlation coefficient of 0.994 was obtained (regression line $y = 1.06x - 10$, where x represents the results presented in this study). Growth hormone was analysed as described by Balldin *et al* (1982). Serum prolactin was determined with reagents from Diagnostic Products Corp., Los Angeles, USA. The properties of this assay in our hands

will be described in another paper (Lundberg *et al,* to be published), as well as those for serum transcortin assay carried out by electroimmunoassay with an antiserum against human transcortin prepared in this laboratory. The results were expressed in relation to the concentration in a human pregnancy pool. Urinary cortisol was determined by a transcortin-binding technique (modified from Müller *et al,* 1974; Horn *et al,* 1975). Other laboratory tests were carried out according to methods used at the Chemical Central Laboratory at Sahlgren's Hospital, University of Göteborg.

Follow-up study

The persistent non-suppressors were subjected to a further follow-up to test for endocrinological disease. The follow-up comprised a clinical examination, TRH-test (200 μg of thyroliberin, sampling after −10, 0, +10, +20, +30, +45 and +60 min, with analyses of TSH, prolactin and growth hormone), determination of basal concentration of serum T_4, serum T_3, serum free T_4 and urinary 24-hour excretion of cortisol.

Statistical methods

To test the hypothesis of no differences between supressors and non-suppressors Student's *t*-test was used when the independent variable was of interval scale type: otherwise Mann-Whitney's U-test or Fisher's exact test was performed. The differences were considered statistically significant for values of $P < 0.05$ (two-tailed test). Spearman's ρ was used for correlation between two variables. A regression coefficient was calculated for the association between the depression score and the post-dexamethasone serum concentration of cortisol in non-suppressors. Significance testing was done in this case by use of the *F*-test.

Results

Fig 1 shows the distribution of the post-dexamethasone serum cortisol concentrations obtained in the first study of the 80 women. Fifteen subjects (19 per cent) were non-suppressors as defined by a serum cortisol concentration greater than 140 nmol/l (5 μg/dl). Of these, only five had a post-dexamethasone serum cortisol concentration greater than 140 nmol/l after a second DST three months later. A third DST was performed in these five subjects after another six months; four had high post-dexamethasone serum cortisol concentration.

Thirteen women (16 per cent) fulfilled the medical exclusion criteria proposed by Carroll *et al* (1981): four were on high-dose oestrogens, four were taking barbiturates or meprobamate, four reported a body mass loss of 4 kg or more in the last four months and one subject suffered from chronic anorexia nervosa

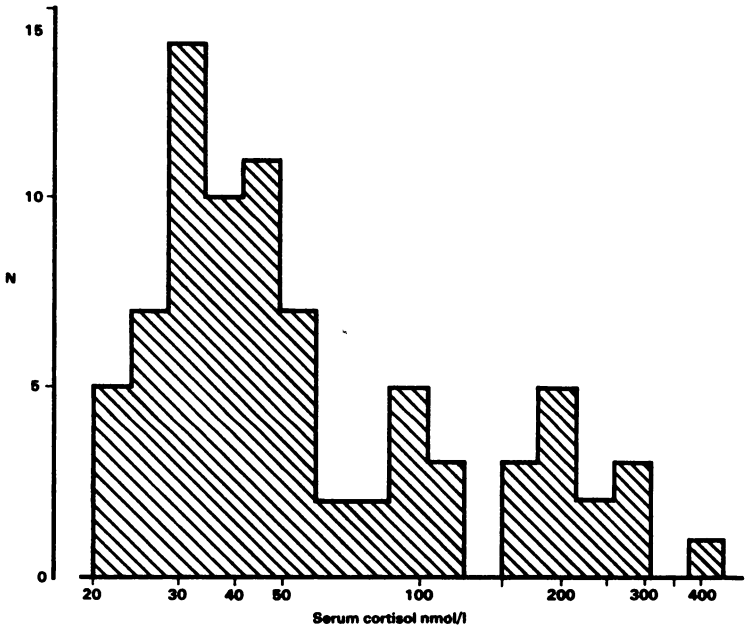

FIG 1.— Distribution of post-dexamethasone serum cortisol concentrations in 80 females from a general population.

and had a body mass index of less than 80. Of these women, three were non-suppressors. When these 13 subjects were excluded, 12 out of 67 (18 per cent) were non-suppressors. The following account is based on the total sample unless otherwise is stated.

Six subjects (8 per cent) presented a depressive syndrome of a mild or medium degree (CPRS depression score ≥10). All of these were suppressors. One of them fulfilled the diagnostic criteria for melancholia. By using Carroll's medical exclusion criteria the specificity of the DST for melancholia is thus 54:66 = 82 per cent±9 (95 per cent confidence limits).

Mean depression scores (CPRS) in the suppressors and non-suppressors (first test series) are shown in Table I. There were no statistically significant differences between the two groups, although there were slight trends in all variables towards increased symptom load in the suppressors. In contrast to this finding, there was a significant association between depression score and post-dexamethasone cortisol concentration in non-suppressors ($r = 0.59$, $P < 0.05$) as is shown in Fig 2.

Twelve women had a body mass index of less than 80; three were non-suppressors and nine were suppressors, *i.e.* they were distributed proportionally between the two groups. Those who reported a body mass loss of 4 kg or more were all suppressors. One of the non-suppressors had a history of anorexia nervosa. There was no difference in amount of strain experienced between non-suppressors and suppressors, nor was there any difference in smoking habits between the non-suppressor group and the suppressor group (five smokers in the former group and 28 in the latter). One out of 15 non-suppressors was living on an altered day–night cycle: she was a night-shift worker.

Table II presents the distribution of age and menstrual status between non-suppressors and suppressors. The majority of the non-suppressors belonged to the 38-year-old group. Of the 50-year-old non-suppressors all three menstruated regularly, thus we found no post-menopausal non-suppressor. The number of subjects is, however, small and no statistically valid conclusions may be drawn as to differences in dexamethasone suppression before and after the menopause. No difference was seen between the 38-year-old women studied in the follicular phase and those studied in the luteal phase of the menstrual cycle. Women using sex-steroids and the woman with the diagnosis of anorexia nervosa were excluded from this

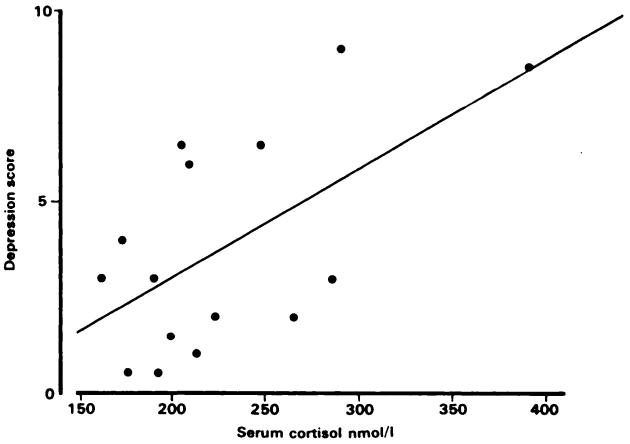

FIG 2.—Total depression score graphed against post-dexamethasone serum cortisol concentration in fifteen non-suppressors. Regression line: $y = 0.028\chi - 2.6$; $r = 0.59$; $F(1, 13) = 7.03$; $P < 0.05$.

TABLE I

Mean depression scores (CPRS) in dexamethasone suppressors and non-suppressors

Depressive syndrome component	Non-suppressors (N = 15)		Suppressors (N = 65)	
	Mean	Median	Mean	Median
Depressive thoughts[1]	1.7	1.1	2.2	1.0
Retardation[2]	2.7	1.2	3.1	2.0
Anxiety[3]	3.1	1.6	3.5	2.1
Total depression score	*7.6*	*5.5*	*8.9*	*6.2*

[1] Sum of scores for sadness, pessimistic thoughts, tearfulness and suicidal thoughts.
[2] Sum of scores for lassitude, inability to feel, indecision and concentration difficulties.
[3] Sum of scores for inner tension, autonomic disturbances, muscular tension and hostile feelings.
The differences in score between suppressors and non-suppressors are not statistically significant.

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analysis. There were no differences between non-suppressors and suppressors regarding parity (number of pregnancies, abortions and extra-uterine pregnancies), menstrual cycle length, amount of menstrual flow, premenstrual symptom scores, frequency of sexual intercourse or sexual desire. However, the non-suppressors reported a significantly lower orgasmic capacity (Table III).

Thirty-nine out of 80 reported use of one or more drugs at the time of the dexamethasone suppression test (Table IV). However, we decided to disregard the use of a small dose of salicylates and the use of antacids, vitamins, iron or laxatives. Nine (60 per cent) of the non-suppressors were drug-users as compared to 30 (46 per cent) of the others, the differences not being statistically significant.

As shown by Table V, there were no mean differences between non-suppressors and suppressors as regards erythrocyte sedimentation rate, number of leucocytes in peripheral blood, activity of serum aminotransferases, γ-glutamyltransferase, serum iron, bilirubin or ferritin content. Neither was there any difference between non-suppressors and suppressors as regards percentage of individuals with raised γ-glutamyltransferase activity or ferritin content. There was, however, a significant difference ($P < 0.05$) in alkaline phosphatase activity between the two groups. In the analysis of transcortin concentration data, subjects taking sex steroids were excluded. The concentration in the non-suppressor group (68.8 per cent±SD 5.8) was significantly higher ($P < 0.05$) than in the suppressor group (64.1 per cent±SD 7.3). Determination of serum growth hormone showed no correlation to the post-dexamethasone serum cortisol concentrations or to dexamethasone suppression/non-suppression, neither was there any relationship between serum prolactin and serum cortisol.

Table VI shows clinical and laboratory data from the four women who had persistently abnormal DST results in three consecutive tests. One of these (Case 2) suffered from a chronic course of anorexia nervosa. None of the others showed clinical signs of hypercortisolism or other endocrine disease. However, Case 1 had a raised basal serum prolactin concentration, Cases 2 and 3 displayed a growth hormone (GH) peak after TRH administration, and Case 3 also had a slightly raised urinary 24 h cortisol concentration.

TABLE II

DST results compared by age and menstrual status

	Non-suppressors	Suppressors
Aged 38, (all premenopausal, N = 47)	12	35
Aged 50, premenopausal (N = 20)	3	17
Aged 50, postmenopausal (N = 13)	0	13

TABLE III

Orgasmic capacity compared with DST result for 41 38-year-old women

Frequency of orgasm during intercourse	Non-suppressors (N = 11)	Suppressors (N = 30)
Always	1	5
Usually	1	21
Sometimes	7	2
Never	2	2

The difference between suppressors and non-suppressors is statistically significant ($Z = -3.23$, $P < 0.01$).

TABLE IV

Drug intake compared with DST result

Drug	Non-suppressors (N = 15)	Suppressors (N = 65)
Drugs of any kind (N = 39)	*9*	*30*
Psychotropic drugs (N = 18)	4	14
barbiturates, meprobamate (N = 4)	1	3
benzodiazepines (N = 12)	4	8
antidepressants (N = 3)	0	3
neuroleptics (N = 4)	1	3
Sex steroids (N = 10)	3	7
Levo-thyroxine (N = 5)	2	3

Discussion

Our study showed a surprisingly high rate of nonsuppression of serum cortisol 17 hours after ingestion of 1 mg of dexamethasone, i.e. the procedure for DST recommended for out-patients (Carroll, 1982). The application of the medical exclusion criteria proposed by Carroll *et al* (1981) did not substantially decrease this rate. Nuller and Ostroumova (1980)

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TABLE V

Laboratory test data (analysis of serum if not indicated otherwise) compared by DST result

	Non-suppressors (N = 15)		Suppressors (N = 65)		Statistical significance
	Mean	SD	Mean	SD	
ESR (mm/h)	12	6.7	11	6.7	–
Blood leucocytes (10^9/l)	5.4	1.8	5.7	1.5	–
Iron (μmol/l)	19	7.3	22	7.7	–
Aspartate aminotransferase (μkat/l)	0.30	0.06	0.32	0.09	–
Alanine aminotransferase (μkat/l)	0.27	0.10	0.30	0.15	–
Alkaline phosphatases (μkat/l)	1.8	0.45	2.2	0.67	$P < 0.05$
Bilirubin (μmol/l)	9.9	4.0	11	4.3	–
γ-Glutamyltransferase (μkat/l)	0.24	0.09	0.27	0.15	–
Ferritin (μg/l)	31	25	36	36	–

reported a rate of 9 per cent non-suppressors out of 85 healthy controls, while Carroll (1982) when using the out-patient procedure found only 4 per cent non-suppressors among 70 'euthymic subjects with no current psychiatric disorder'. Gold *et al* (1981) likewise found one non-suppressor (4 per cent) in 25 normal control subjects and Tourigny-Rivard *et al* (1981) reported one non-suppressor (5 per cent) in 20 normal subjects. The test–retest reliability is generally said to be high (0.94) among normal individuals and psychiatric patients not suffering from endogenous depression, and only sporadic false positives can thus be expected (Carroll, 1981, personal communication). To our knowledge there have so far been no other reports on the reproducibility of abnormal test results in normal individuals: the high rate of non-suppressors in the present study of normal females is thus not in accordance with other studies. We also found test–retest reliability to be low in the non-suppressors.

We cannot be absolutely certain that the subjects took the dexamethasone tablet. However, we know that they carefully followed the instructions of other examinations in the population study, and they reported in a special interview after the test that they had actually taken the tablets. We therefore feel convinced of a high compliance in this group of women.

The abnormally high post-dexamethasone serum cortisol concentrations were not a result of elevated carrier-protein concentration. The slight but significant difference between the groups might, however, indicate a difference in plasma-protein homeostasis. The possibility remains that subjects with serum cortisol greater than 140 nmol/l had a more rapid elimination rate of dexamethasone than the suppressors. However, there is no evidence from analysis of serum γ-glutamyltransferase activity that the non-suppressors had abnormally high liver enzymatic activity, nor had they any evidence of disturbed liver cell integrity. This is in line with the findings of Carroll *et al* (1980) that non-suppression is not explained by unusually rapid clearance of dexamethasone from plasma. The finding of normal γ-glutamyl transferase activity supports statements given by the subjects as regards absence of intake of phenobarbital or other drugs in large quantity.

It is conceivable that the high post-dexamethasone cortisol concentration in the subject with night-shift work is caused by an altered circadian rhythm. It is possible that in this case the spontaneous cortisol secretion had not reached its minimum in the late evening when dexamethasone was taken.

One of the subjects suffered from anorexia nervosa (Table VI, Case 2). This case excepted, determinations of relative body mass, recent body mass loss, erythrocyte sedimentation rate, blood leucocyte and serum iron content provided no evidence that the non-suppressors had active disease at the time of the study: thus in the 15 non-suppressors no common denominators emerged as regards clinical or laboratory variables. The same applies to those four subjects who remained non-suppressors after the second and third challenge: the laboratory findings in these persistent non-suppressors are of unclear clinical significance. Until more experience is gained with the DST it seems appropriate to monitor persistent unexplained non-suppressors regularly for the presence of endocrine disease.

As shown by the depression scores, there was no correlation between non-suppressibility and depression. Of the depressives, however, only one fulfilled the criteria for melancholia. This finding is not discordant with a sensitivity of around 50 per cent for the DST in identifying cases of melancholia. The claims for specificity higher than 90 per cent seem more dubious, however, given the results of the present study.

The association between depression score and

TABLE VI

Clinical and laboratory data of persistent DST non-suppressors: TRH-tests carried out about eight months after the first DST-test. Reference interval between brackets

Case no	Age yrs	Height cm	Body mass kg	Urinary cortisol nmol/24 h (<300)	Serum TSH mU/l (≤3.5)	Serum T_3 nmol/l (1.5–2.8)	Serum T_4 nmol/l (70–160)	Serum free T_4 pmol/l (10.8–23)	Serum prolactin µg/l (≤25)	TRH-test
1	38	166	51	80	2.4	2.2	120	24.0	33	ΔTSH: 27.0 mU/l ΔPRL: 45 µg/l Gradual decrease of GH during the test (−10 min 19 mU/l; +60 min 3.4 mU/l)
	Clinical remarks: Cholecystectomy 1976, after this lost 10 kg of body mass in 6 months to present body mass. Menstruates regularly, 2-para.									
2	38	171	55	210	0.5	1.6	110	26.4	14	ΔTSH: 0 mU/l ΔPRL: 49 µg/l GH peak after 45 min (0 min: 37 mU/l; ΔGH: 24.8 mU/l)
	Clinical remarks: 1959 anorexia nervosa and amenorrhoea, suspicion of hypothyroidism. Thyroxine substitution which was discontinued briefly 1968. PBI when lowest 2.2 µg %. Menstruates regularly since 1970, 2-para. Still anorectic behaviour.									
3	38	163	62	370	2.1	1.6	90	17.1	9.0	ΔTSH: 18.6 mU/l ΔPRL: 110 µg/l GH peak after 20 min (0 min: 4.0 mU/l; ΔGH: 3.4 mU/l)
	Clinical remarks: Previously healthy, menstruates regularly, 2 para.									
4	50	168	68	210	3.1	2.2	110	19.4	12	ΔTSH: 16.4 mU/l ΔPRL: 63 µg/l No change in GH during the test (0 min: 2.2 mU/l; ΔGH: 0 mU/l)
	Clinical remarks: Previously somatically healthy. Menstruates regularly, 2-para. Generalized anxiety disorder since adolescence.									

serum cortisol concentration in the non-suppressors remains to be explained. A similar finding in a clinical sample of depressive patients has been reported by Holsboer *et al* (1980), but it is not easy to see why this correlation should exist in this non-clinical group of non-suppressors, who were all essentially free from depression at the time of investigation.

The neuroendocrine disturbance shown up by the DST may be a marker of a hypothalamic–limbic system dysfunction. This dysfunction may in itself reflect abnormalities in central neurotransmission which need not necessarily be tied to the clinical syndrome of melancholia. A disturbed monoaminergic functioning may operate not only in melancholia but also in anorexia nervosa (Casper *et al*, 1977) and in sexual disturbances of different kinds (Everitt, 1977; Nieschlag, 1977). Our finding of prevalent orgasmic dysfunction in the non-suppressors might be easier to interpret along the lines of abnormalities in monoamine neurotransmission rather than by hypothesizing a highly melancholia-specific property of the DST. This may be relevant in studies of samples where the prevalence for melancholia is low, e.g. in the general population. In such samples the predictive value of the DST for melancholia is very low, but our findings suggest that the specificity also seems to be lower than has been reported previously.

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