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# The Dexamethasone Suppression Test in Depressive and Schizophrenic Patients Under Controlled Treatment Conditions

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**Summary.** Endogenous depressive and schizophrenic patients demonstrated the same frequency of pathological DST results after admission. After 3 weeks of psychopharmacological treatment the percentage of abnormal DST results was significantly reduced in both groups, although the treatment conditions were different. A correlation between the DST non-suppression and intensity of depression was observed only in the depressive group, not in the schizophrenic group. Normalization of DST results in depressive patients was mostly associated with an improvement of depressive scores. Other course patterns of DST results did not seem to be combined with psychopathological changes. From this data it has to be concluded that DST non-suppression is in some part related to depressive symptoms, but is not characteristic or specific for endogenous depression or for depressivity.

**Key words:** Dexamethasone suppression test – Depression – Schizophrenia

## Introduction

The most promising results of neuroendocrine research strategy have come from investigations of the limbic hypothalamus pituitary adrenal axis in depressed patients. In particular the results of dexamethasone suppression tests (DST) have recently gained importance. Non-suppression of cortisol release in this test has been described as a specific biological marker for endogenous depression and the DST was proposed as a useful laboratory test for endogenous depression (Carroll et al. 1981; Greden et al. 1983). In most cases abnormal DST results returned to normal after the episode of depression, indicating that the limbic neuroendocrine disturbance revealed by the test is episode-related rather than a trait marker.

The DST was also suggested by some authors as a predictor for the response to antidepressive drugs in general, or for the response to a specific antidepressant as well as a predictor for safe withdrawal of antidepressant therapy (Amsterdam et al. 1983; Brown et al. 1979; Goldberg 1980; Beckmann et al. 1984). However the body of evidence for this predictive value is weaker than the data basis for the association of depression and pathological DST results.

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An important advantage of this test seems to be that the result is not influenced by most drugs prescribed in psychiatry, especially not by antidepressants, neuroleptics or lithium (Carroll et al. 1981). Nevertheless is has to be considered that the current or recent use of drugs that accelerate the metabolism of dexamethasone (i.e. barbiturates, phenytoin, carbamazepine) and oral contraceptives can lead to spuriously positive DST results, whereas high-dose benzodiazepines (>25 mg/day of diazepam) induce negative results. Thus patients with such a medication should be excluded from studies on the differential diagnostic functions of the DST. Also patients with a weight loss of more than 20% of ideal body weight and patients with severe medical illness or pregnancy, conditions which are known to produce positive DST results, should be excluded (Carroll et al. 1981; Carroll 1982).

Recently the optimism concerning the value of the DST for the diagnosis of depression has been criticized (Berger and Klein (1984); Klein (1986). The specificity of the test as a biological marker for endogenous depression has been questioned among other things by the fact that abnormal DST results could also be demonstrated in a higher proportion of patients with psychiatric disorders, and by experimental results that sleep withdrawal, weight loss and psychological stress are able to induce non-suppression in the DST (Baumgartner 1984; Berger et al. 1982b; Doerr et al. 1980). The percentage of non-suppression in the DST seems to correlate with the score on depression scales (Davis et al. 1981; Kasper and Beckmann 1983; Rush et al. 1982) which provoked the question whether a pathological DST result is really characteristic of endogenous depression or only a sign of severe depression. Selecting only patients with a comparable intensity of depression, Berger et al. (1982a) found no difference concerning DST results between patients with neurotic and endogenous depression.

We applied the DST repeatedly in two studies on endogenous depressive patients and in one study on schizophrenic patients. Our aim was to investigate the frequency and course pattern of pathological DST results and their relationship to changes of psychopathological phenomena under controlled treatment conditions. The main question was whether differences between the frequency of abnormal DST results and the course pattern of DST results could be found between endogenous depressives and schizophrenics. The findings are of interest because they support the critical attitude mentioned

above towards the DST as a biological marker for endogenous depression.

## Methods

The DST was performed under controlled treatment conditions in three clinical studies. Each study was performed to evaluate special treatment procedures: in the two studies on endogenous depressives the combination of antidepressives and haloperidol, in the study on schizophrenic patients the treatment with endorphines or haloperidol. The design, the characteristics of the patients included and the results of the therapeutical evaluation have been reported elsewhere in detail (Kissling et al. in press; Möller et al. 1984b; Möller et al. 1986). Thus the description of methodological aspects can be shortened here to some basic points and the presentation of the results can be focussed on the DST data.

In the first study 20 patients with an endogenous depression according to ICD and RDC were treated for 3 weeks with 150 mg chlorimipramine/day, the patients of the experimental group also received 9 mg haloperidol/day for the first 6 days of the trial (Möller et al. 1984a, b). In the second study 28 patients with endogenous depression according to ICD and RDC were treated with 150 mg maprotiline/day orally, the patients of the experimental group also received 9 mg haloperidol/day for the first 6 days of the trial (Möller et al. 1986). Some 24 patients with an acute manifestation of a schizophrenic disorder (schizoaffective psychoses excluded) according to ICD and DSM-III criteria were treated for the first 14 days of the trial with 3 mg desenkephaline-gamma-endorphine/day i.m. or with 10 mg haloperidol/day orally (Kissling et al. in press). In the following 2 weeks the medication with endorphine was continued if the treatment was successful, or the medication of non-responders was changed to 24 mg haloperidol/day.

All studies were performed under double blind conditions. In none of these studies a significant difference or a relevant discrepancy between the experimental group and the control group with respect to the therapeutic effect could be observed.

The basic characteristics of the patients are shown in Table 1. All depressive patients were treated with antidepressants or combinations of psychotropic drugs (mostly combinations of antidepressants with tranquillizers) over a longer period of time (at least more than 6 weeks) before hospital admission. Similarly most of the schizophrenic patients were on medication (mostly neuroleptics) before admission. In general the pre-medication was interrupted for a wash-out period of 3 days in the depression studies or 7 days in the schizophrenia study, but this was impossible in some of the patients because of the severity of illness.

The patients went through a detailed psychiatric exploration, a comprehensive physical examination and thorough laboratory screening (including blood, liver and urine profiles, thyroid function, chest X-ray, ECG, EEG, any other laboratory tests indicated on the basis of the individual medical history or physical examination) to verify the diagnosis of an endogenous phychosis and to rule out serious medical illness, before they were included into the study. Along with serious medical illness, alcoholism and drug-abuse were exclusion criteria. In female patients the absence of pregnancy had to be proven. The patients had to give informed consent for the psychopharmacological evaluation.

**Table 1.** Basic characteristics of the three samples

| | Patients | Diagnosis | Sex | Age |
|---|---|---|---|---|
| Chlorimipramine study | 20 | Endogenous depression (ICD, RDC) | 16 F<br>4 M | $\bar{x} = 43.8$<br>SD = 12.2 |
| Maprotiline study | 28 | Endogenous depression (ICD, RDC) | 16 F<br>12 M | $\bar{x} = 44.8$<br>SD = 12.7 |
| Endorphine study | 24 | Schizophrenia (ICD, DSM-III) | 17 F<br>7 M | $\bar{x} = 34.4$<br>SD = 13.4 |

The psychopathological state of the patients was assessed before the beginning of the trial and several times during the trial. As psychopathological assessment procedure the Hamilton Rating Scale for Depression was used in the two samples of depressive patients. To measure the psychopathological changes of the schizophrenic patients the Brief Psychiatric Rating Scale (BPRS) and the Comprehensive Psychiatric Rating Scale (CPRS) were applied. Additionally the patients filled out an actual mood self-rating scale (von Zerssen et al. 1976).

The DST was performed once before (generally on the 3rd day of hospital stay) and repeatedly during the trial in a modification suggested by Carroll (1982): 1 mg dexamethasone was administered at 23 h, plasma samples were collected on the following day at 16 h and 23 h. The points of measurement were not completely identical in the three studies, but sufficiently comparable. In the chlorimipramine study the DST was performed before the beginning of the standardized medication, at day 10 and day 21. In the maprotiline study the DST was performed before the trial, and at days 7, 14 and 21. In the endorphine study the points of measurement were before the trial, at day 7 and day 23. These discrepancies were caused by the respective organisational needs of each study. The plasma concentration of cortisol was determined using a radioimmunoassay in the endocrinological laboratory of our Medical Department. Post-dexamethasone plasma cortisol concentrations over 4 μ/dl (110 nm/l) in either of the blood samples were taken as indicating non-suppression (Carroll 1982).

Some patients of the original sample had to be excluded from the DST evaluation because of the known risk factors for a false DST result mentioned above. These included 5 patients of the chlorimipramine study, 4 patients of the maprotiline study and 4 patients of the endorphine/haloperidol study. The DST of the remaining patients are not complete in all cases. Missing data, predominantly at the last point of measurement, are explained by refusal of the patients or by problems of organization.

## Results

The comparison between the DST results of the endogenous depressive patients and the schizophrenic patients before beginning the standardized medication, revealed no difference between these two diagnostic groups. The percentage of pathological (positive) DST results was almost the same: 59% for the depressives, 60% for the schizophrenics (Tables 2 and 3).

Also the course of DST results during the standardized treatment was very similar in the two diagnostic groups (Ta-

**Table 2.** DST results in the two samples of endogenous depressives

| DST | Before trial | Day 7/10 | Day 21 |
|---|---|---|---|
| Non-suppressors | 23 | 13 | 7 |
| Suppressors | 16 | 24 | 23 |
| Total | 39 | 37 | 30 |
| Percentage of non-suppressors | 59% | 35% | 23% |

**Table 3.** DST results in the sample of schizophrenics

| DST | Before trial | Day 7 | Day 23 |
|---|---|---|---|
| Non-suppressors | 12 | 7 | 6 |
| Suppressors | 8 | 12 | 7 |
| Total | 20 | 19 | 13 |
| Percentage of non-suppressors | 60% | 37% | 46% |

bles 2 and 3), although the treatment was totally different. The frequency of pathological DST results was markedly reduced 7 to 10 days after the standardized treatment, while there was no marked difference between this point of measurement and the end of the trial. In contrast to the depressive group, where the reduction in pathological DST results continued, a small increase in pathological DST results was observed in the schizophrenic group. This increase might perhaps be explained as a sampling artefact, caused by a reduction in the number of patients. Apart from this special problem it should be underlined that the most impressive change concerning the frequency of pathological DST results took place in the first 7 to 10 days of the study, when clinical improvement usually is insufficient and that the percentage of non-suppression at this time was not significantly decreased during the following time of the trial.

The comparison of the two groups of depressive patients showed that there were remarkable differences concerning the DST results. The first group (chlorimipramine study) had a much higher proportion of non-suppression than the second group (maprotiline study): 73% vs 50% at the beginning, 42% vs 13% at the end of the trial. These differences demonstrated the relevance of sampling effects and perhaps accidental factors. They cannot be explained by the intensity of depression, because the mean value of the Hamilton Depression Scale at the beginning of the study was lower (21.8) in the chlorimipramine group than in the maprotiline group (27.8).

To identify the psychopathological correlation of the hypercortisolism, indicated by the pathological DST results, 16-h cortisol values of the depressive patients were correlated separately for each day of measurement with the total score and some selected items of the Hamilton Depression Scale as well as with the Actual Mood Scale. The correlation coefficients revealed a significant but not very substantial (e.g. $r$ = 0.33 at the beginning of the trial) relationship between hypercortisolism and intensity of depression, which was stable at different points of measurement. This relationship was only found in the ratings performed by the physician, not in the self-ratings of by the patients. A similar degree of correlation was found between the item weight loss of the Hamilton Depression Scale and hypercortisolism ($r$ = 0.33), whereas the correlations concerning the items anxiety, suicidal ideas and sleep disturbances were on a lower level ($r$ = 0.10–0.17) not reaching statistical significance.

In the same fashion the total scores of the BPRS and the CPRS, which were applied for the schizophrenic sample, were correlated with the 16-h cortisol concentration. Instead of the expected positive correlation, we found a negative one ($r$ about −0.50). The correlations between the BPRS subscore anxious depression and the cortisol concentration gave no consistant results at the different points of measurement.

On the single case level different course patterns of DST results could be differentiated. In the depressive patients, normalization of pathological DST results was found in 32% of the patients with a complete DST series. Among these the quick normalization after 7 to 10 days of treatment was the most frequent one. The persistence of pathological DST results was only found in 10% of the patients. The persistence of normal test results during the whole trial occurred in 28% of the patients. In 32% of the patients the originally normal results either became pathological or demonstrated unstable fluctuations. This course pattern has to be considered as problematical under the aspect of retest stability, especially when the fluctuations cannot be explained by psychopathological changes. The course patterns of DST results in the schizophrenic group had a similar distribution.

The question whether changes in DST results at the single case level are associated with psychopathological changes, was likewise analysed. In the group of depressive patients DST normalization seemed to be rather closely associated with the reduction of Hamilton Depression scores, especially if we accept a time lag between DST normalization and clinical improvement (Table 4). Of special interest was the fluctuation in DST results which were normal at the beginning of this trial. No relationship between these changes in DST results and the psychopathological situation in terms of depression scores appeared to exist (Table 5). Also the persistence of abnormal DST results did not seem to correlate well with the psychopathological state. The analogous single case analyses for the group of schizophrenics did not detect any relationships between DST results and psychopathological state,

**Table 4.** Relationship between normalization of DST results and change of depression score. NS = non-suppression, S = suppression

| Chlorimipramine study | Before trial | Day 10 | Day 21 | |
|---|---|---|---|---|
| HAMD | 34 | 19 | 24 | |
| DST | NS | S | S | |
| | 25 | 3 | 0 | |
| | NS | NS | S | |
| | 24 | 18 | 12 | |
| | NS | S | S | |
| | 30 | 4 | 8 | |
| | NS | S | S | |
| | 11 | 4 | — | |
| | NS | S | — | |
| **Maprotiline study** | **Before trial** | **Day 7** | **Day 14** | **Day 21** |
| HAMD | 31 | 20 | 12 | 4 |
| DST | NS | S | S | S |
| | 30 | 18 | 10 | 6 |
| | NS | S | S | S |
| | 26 | 11 | 8 | 8 |
| | NS | S | S | S |
| | 29 | 16 | 17 | 12 |
| | NS | NS | S | S |

**Table 5.** Relationship between fluctuation of DST results and change of depression score

| Chlorimipramine study | Before trial | Day 10 | Day 21 | |
|---|---|---|---|---|
| HAMD | 26 | 19 | 13 | |
| DST | NSD | S | NS | |
| | 25 | 0 | 0 | |
| | S | NS | NS | |
| | 25 | 9 | 23 | |
| | S | S | NS | |
| **Maprotiline study** | **Before trial** | **Day 7** | **Day 14** | **Day 21** |
| HAMD | 43 | 32 | 28 | 35 |
| DST | S | NS | S | S |
| | 22 | 11 | 11 | 3 |
| | S | NS | S | S |
| | 31 | 16 | 7 | 0 |
| | NS | S | S | NS |
| | 24 | 6 | 20 | 7 |
| | NS | S | NS | S |
| | 24 | 19 | 23 | 29 |
| | S | NS | NS | S |
| | 35 | 19 | 32 | 23 |
| | NS | S | NS | — |

**Table 6.** Relationship between DST results before trial and response to antidepressants at day 21

| Before trial | Day 21 | |
|---|---|---|
| | Responder | Non-responder |
| Non-suppression ($n = 20$) | 13 (65%) | 7 (35%) |
| Suppression ($n = 15$) | 9 (60%) | 6 (40%) |

**Table 7.** Relationship between DST result and response to antidepressants at day 21

| | | |
|---|---|---|
| Responder | Suppression | 15 |
| Responder | Non-suppression | 6 |
| Non-responder | Suppression | 7 |
| Non-responder | Non-suppression | 2 |

either for the total score of BPRS and CPRS, or for the BPRS subscore for anxious depression. In particular there was no association with regard to high scores of schizophrenic symptoms and non-suppression.

With respect to prognostic considerations the following question can be answered: whether the DST result before the controlled trial conditions is predictive for the response to antidepressants after 3 weeks of treatment. Response to antidepressive treatment was defined as a reduction of the Hamilton score below 10 or of 50% at the end of the trial. The data showed no prognostic relationship (Table 6). This result was independent of the sample taken into accout: the chlorimipramine sample, the maprotiline sample, or the whole sample of depressive patients.

The relationship between DST results and response to antidepressive treatment at the end of the trial is shown in Table 7. The predominant combination (52%) was treatment response and normal DST result. In 6 of 21 responders the DST result was normal. About the same proportion (2 of 9) of pathological DST results were found in the group of non-responders.

## Discussion

Based on our findings and on the results reported in the literature (Berger and Klein 1984), the following conclusions might be drawn.

The function of abnormal DST results as a biological marker for endogenous depression has to be questioned. In this study endogenous depressive patients and schizophrenic patients demonstrated the same frequency of pathological DST results after admission. This interesting result cannot be confounded by affective symptoms in the schizophrenic sample, because schizoaffective disorders had been excluded and because the DSM-III criteria of schizophrenia are very restrictive concerning affective symptoms. Also the result cannot be explained by drug withdrawal stress (Greden et al. 1983) because after 7 to 10 days of standardized treatment there was no difference between endogenous depressives and schizophenics concerning the frequency of non-suppression.

The percentage of pathological DST results in the group of endogenous depressive patients in this study was almost the same as reported in reviews on DST results in endogenous depressive patients: 40% (Berger and Klein 1984) to 60% (Carroll 1982). In concordance with our DST findings on schizophrenic patients, other authors have described DST non-suppression in schizophrenic patients at a frequency of up to about 50% (Bloodworth 1982; Carman et al. 1981; Dewan et al. 1982; Greden et al. 1981). In several other severe psychiatric disorders rather high percentages of abnormal DST results have also been observed, e.g. in senile dementia (Raskind et al. 1982; Spar and Gerner 1982), in alcoholism (Oxenkrug 1978; Schwartz and Dunner 1982) and in manic patients (Graham et al. 1981), whereas another study found normal DST values in mania (Schlesser et al. 1979). Thus there is a large body of evidence implying that abnormal DST results can be generally found in large percentage of severe psychiatric disorders and that they are not characteristic or specific for endogenous depression.

Under clinical drug treatment conditions – in our study the drug trials lasted about 3 weeks – the frequency of abnormal DST results was reduced. In this respect too, a significant difference between the group of endogenous depressive patients and the group of schizophrenic patients was not observed. This seems of special interest, because the treatment conditions of depressives and schizophrenics were totally different. The main reduction in pathological DST results took place in the first 7 to 10 days of the standardized drug treatment, i.e. during an interval, in which the clinical improvement in general is not so impressive. A normalization of pathological DST results during a rather short episode after clinical admission has also been reported by other authors (Berger et al. 1984; Coccora et al. 1983; Haskett et al. 1983). This was interpreted as a consequence of clinical admission and its stress factors which disappear after a short period of time. But our data show that DST normalization in depressive patients is associated in most cases with a marked reduction of depressive symptoms, both of which occur to a certain extent even in the early phase of clinical treatment. At the end of the 3-week trial 30% of the depressive patients still had a pathological DST result. This percentage is higher than reported from the

studies with unlimited treatment duration (Greden et al. 1983; Holsboer et al. 1982). This discrepancy can be explained by the fact that a longer treatment period increases the probability of clinical improvement and with this the probability of DST normalization.

As in other studies (Davis et al. 1981; Papacostas 1981; Reus et al. 1982; Rush et al. 1982; Kasper and Beckmann 1983) a correlation between non-suppression and intensity of depression was observed. There was also a correlation between non-suppression and weight loss, demonstrating that perhaps weight loss is one of the most important background variables for non-suppression in depressive patients (Berger et al. 1982b). The important rule of weight loss concerning DST results has also been shown in patients with anorexia nervosa (Fichter et al. 1981) and in fasting volunteers (Berger et al. in press). When the course of single cases was analysed, a certain association of pathological DST results and depressive symptomatology in depressive patients was observed. Normalization of DST results was combined mostly with an improvement in depression scores, a finding which has also been reported by other authors who applied serial DST (Albala et al. 1980; Gold et al. 1980; Greden et al. 1983; Holsboer et al. 1982; Rothschild and Schatzberg 1982). But not all patients showing improvement of their depression also reached a normalization of pathological DST results, a finding also described by the authors mentioned above. Clinical improvement of depression without DST normalization was interpreted by others as a predictor of early relapse after discontinuation of antidepressive drugs (Goldberg 1980), a hypothesis which could not be tested in our study.

About one-third of the depressive patients showed a course pattern of DST results which we called flucturation: Although they started with normal DST results subsequent DSTs demonstrated non-suppression or fluctuated between abnormal and normal results. This course pattern, problematic with respect to re-test stability, generally could not be explained by psychopathological changes. In one case the fluctuation corresponded to a switch into mania. In most cases other background variables have to be hypothesized, e.g. the stress factors mentioned above.

The DST result at the beginning of the standardized treatment was not predictive for the response to antidepressants after 3 weeks in our study. This result is in disagreement with Brown et al. (1979), but in accordance with the findings of Baumgartner (1984). Following differentiation between chlorimipramine treatment and maprotiline treatment a predictive function of the DST result could not be found. The results in the literature describing the DST result as an indicator for a better response to noradrenergic or serotonergic antidepressants (Amsterdam et al. 1983; Beckmann et al. 1984; Peselow et al. 1983) could not be reproduced.

In the group of schizophrenics a positive relationship to severity of schizophrenic symptoms or depressive symptoms was not found. Unexpectedly there was a negative correlation between the severity of schizophrenic symptoms and non-suppression, for which an interpretation is needed. Hitherto specific background variables for DST non-suppression in schizophrenia are unknown. Thus the non-specific factors mentioned have to be taken into account.

In summary DST non-suppression seems to be in some part related to depressive symptoms, but it does not seem characteristic or specific for endogenous depression or for depressivity. As is known from experimental studies very many variables, which taken together might be viewed as stress inducing factors, are able to provoke pathological DST results. From our viewpoint, a pathological DST result found in a high percentage of patients with severe psychiatric disorders and without medical illness can only be interpreted as a non-specific indicator for a disturbance of the limbic hypothalamic pituitary adrenal system. A pathological DST is hypothesized to be secondary either to a decrease of noradrenergic activity (van Loon et al. 1971) or to an increased cholinergic activity in the CNS (Garver and Davis 1979).

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