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A decade of spontaneous long-term course of psychogenic impairment in a community population sample

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Abstract *Background*: Our epidemiological study demonstrates the spontaneous long-term course of predominantly psychosocially influenced (''psychogenic'') disorders (neurotic spectrum disorders, personality disorders, stress reactions and somatoform disorders) in a representative community sample of the normal adult population of Mannheim, an industrial and university town in Germany. The natural spontaneous course of these disorders in a population sample over a long period remains largely unknown. *Method*: Beginning in 1979 ($n_{t1} = 600$) a random population sample was investigated three times over a mean period of approximately 11 years. The last follow-up study ended in 1994 ($n_{t3} = 301$). The follow-up sample was representative of the t1 sample. Psychodynamically trained and clinically experienced interviewers used a semi-structured interview and standardized clinical and psychometric instruments. Psychogenic impairment was assessed using a standardized expert rating (Impairment Score, IS). *Results*: The mean sum-score of psychogenic impairment after 11 years exceeded the value at t1. The case rate (point prevalence, ICD diagnosis + clinical cut-off/IS) increased from 21.6% at t1 to 26.2% at t3 in the investigated follow-up sample. Intra-individual correlation of psychogenic impairment between t1 and t3 was high ($r = 0.55$). We found strong evidence for an unfavorable long-term course of psychogenic impairment and only a weak tendency (23.1%) for spontaneous remission of clinically relevant psychogenic impairment. Within a regression model clinical variables, childhood development conditions and personality traits at t1 predicted psychogenic impairment at t3. *Conclusion*: All clinical variables conclusively indicate an unfavorable spontaneous long-term course of psychogenic impairment. Together with the well-known high prevalence of psychogenic disorders in the normal population, this underlines the need for early therapeutic and preventive intervention.

Key words Psychogenic disorders · Epidemiology · Spontaneous long-term course · Neurotic spectrum disorders · Somatoform disorders · Psychogenic impairment · Natural history

Introduction

From the beginning of the Mannheim Cohort Study on the Epidemiology of Psychogenic Disorders (Schepank 1987, 1990; Franz et al. 1994, 1998a), the term ''psychogenic'' disorders covered the group of mainly psychosocially influenced disorders: psychoneuroses (neurotic spectrum disorders, personality disorders, stress reactions and somatoform disorders). Beyond the common subjection of these disorders to the psychosocial biography of an individual, it seemed appropriate to focus on this group of disorders as whole, because there is a well-known and considerable symptom shift and comorbidity among these subgroups.

Discussion about the spontaneous course of these disorders started after the first studies on the effect of psychotherapy (Fenichel 1930; Jones 1936; Knight 1943) were challenged by Eysenck's hypothesis of a high spontaneous remission rate. He claimed that nearly two-thirds of psychogenically impaired patients improve within 2 years whether they are treated with psychotherapy or not (Eysenck 1952). Later, these presumptions were heavily criticized, since they referred to studies that had serious methodological shortcomings

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(Luborsky 1954; Dührssen and Jorswieck 1962; Strupp 1963; Bergin 1971). In a re-analysis of Eysenck's data, McNeilly and Howard (1991) showed that about 50% of treated patients and only 2% of untreated patients improved within 8 weeks. In his review of 14 studies on the spontaneous remission of neurotic disorders, Bergin (1971) reported a median remission rate of 30%, while Lambert (1976) reported a wide range of remission rates, from 18% up to 66% (median remission rate of 43%). This variance is caused by differences in clinical conditions, catamnestic intervals, sampling effects (clinical or institutional selection), and noncomparable outcome variables in the studies investigated.

In contrast to the investigation of those highly selected clinical samples, epidemiological surveys allow a more appropriate estimation of the true course characteristics. In a representative random sample, psychogenically impaired individuals are also included. These individuals are not considered in clinical studies due to their inadequate help-seeking behavior or a lack of contact with clinical institutions. In this paper, we present data of the spontaneous course of psychogenic impairment within a community population sample, which was investigated between 1979 and 1994 in the German industrial and university city of Mannheim.

In a previous paper (Franz et al. 1998a), we investigated the long-term course of psychogenic impairment for two extreme groups. Subjects with the most positive and the most negative spontaneous long-term course were compared with respect to personality characteristics, sociodemographic variables, network structure, and critical life events. Both groups were selected from a risk population of the initial sample and differed by several personality variables. Although this approach identified etiologically relevant predictors that discriminated between the two extreme groups, it did not assess the core conditions of the spontaneous long-term course of psychogenic impairment for the whole sample. Therefore, the aim of the present study was to explore the long-term course for the whole sample rather than to differentiate between extreme patterns. The present study was a development of the first, in that different (but overlapping) samples and instruments were used and a different analysis was employed.

Subjects and methods

In 1979, Schepank's group started a long-term investigation of the prevalence, natural history, and determinants of psychogenic disorders. The first survey, carried out between 1979 and 1983, studied the prevalence of psychogenic disorders (t1, Schepank 1987). In this cohort study, a representative sample of 600 German adults born in 1935, 1945, and 1955 was randomly selected from the urban population of Mannheim. Three years later, we re-evaluated 528 (88%) of the original probands (t2, Schepank 1990). Between 1991 and 1994, further follow-up interviews were carried out on 333 of the probands. The mean time interval between t1 and t3 was 11.1 years (SD 0.9) with a minimum of 9 and a maximum of 13 years. Thirty-two probands had accepted our offer of psychodynamic individual or group psychotherapy within a study on psychotherapy motivation (Franz 1997), thus the sample regarding the spontaneous course was reduced to 301 at t3 (50.2% of t1). After the subjects were provided with a complete description of the study, written informed consent was obtained for the first study and each follow-up.

Personality characteristics and clinical and sociodemographic variables were evaluated in each of the surveys. The degree of psychogenic impairment was assessed by psychodynamically trained and clinically experienced interviewers using the Impairment Score (IS, German: BSS, Schepank 1995) (Franz et al. 1998a). In addition to this expert rating, the number of psychogenic symptoms was specified by a symptom checklist filled out by the probands. In a second step, a symptom was assessed as psychogenic if there was no physical cause to be found. This was assured by anamnesis, with special attention given to earlier medical diagnostic procedures. Furthermore, a symptom-triggering psychosocial conflict had to be identified within the psychodynamic interview.

The follow-up sample t1 $\rightarrow$ t3 ($n = 301$) was representative of the t1 sample ($n = 600$). With regard to social core variables, there were no significant differences between the t1 and the follow-up samples at t1. In addition, the IS ratings did not differ between the t1 sample and the follow-up sample at t1.

The social and clinical profiles of both samples at t1 showed no differences, indicating that the follow-up sample was highly representative. Therefore, we were able to compare t1 and t3 for the follow-up sample without selection bias. The study methods have been reported in detail elsewhere (Franz et al. 1998a).

Statistical tests of the differences between the t1 and t3 values of the t3 sample were performed two-tailed using the χ^2-test or the *t*-test for dependent samples. Intra-individual correlation coefficients (Spearman) between t1 and t3 were also performed two-tailed. Stepwise regression analysis was used to examine the contribution of gender, age, early childhood development, clinical impairment and personality traits to the clinical impairment at t3.

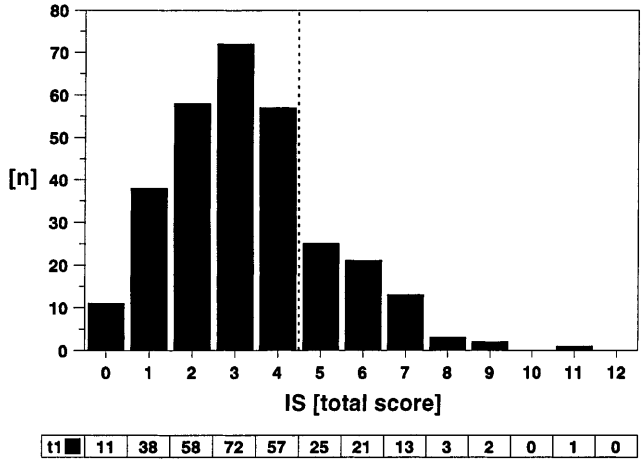
Early childhood development conditions (reported at t1) were assessed by the interviewers using a reference example with regard to the absence and emotional distance of mother/father (Schepank 1987). Similarly, the global burden during childhood development was assessed for the ages 1–6 and 6–12 years. Reference examples of traumatic influences (such as loss of primary binding, broken home constellations, early experiences of aggressive assaults, emotional abuse or psychopathologically impaired parents) were used in this rating (Schepank 1987). The clinical impairment of the probands at t1 was operationalized by the Impairment Score (BSS, Schepank 1995), the sum-score of the Goldberg-Cooper interview (Goldberg et al. 1970) and the number of symptoms according to a symptom check list with predominantly psychogenic/psychosocial-triggered complaints (Schepank 1987). Personality traits (t1) were integrated in the model using the Freiburger Persönlichkeitsinventar (FPI, Fahrenberg et al. 1978), a factor analytic, well-standardized personality inventory. Data were analyzed using SPSS (Statistical Package for the Social Science) for Windows, version 7.5.

Results

We found the mean IS score (total score and subscales) of the follow-up sample was nearly the same at t3 as at t1. Group statistically, psychogenic impairment showed no improvement after 11 years.

In addition, the distribution of psychogenic impairment at t1 was replicated 11 years later (Fig. 1), with only a little alteration.

Of the 301 probands of the follow-up sample, 54.2 % had an IS total score (last year) within the clinically not relevant range of 0–4 at both t1 and t3. Subject to a measurement error of $+/-$ 1 point of the IS total score,



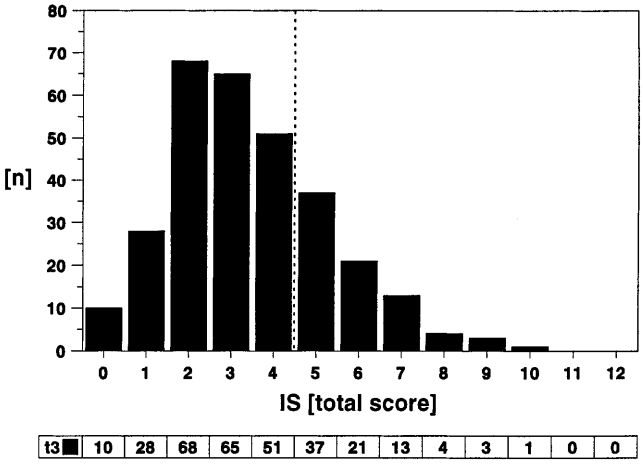


Fig. 1 Distribution of the probands ([*n*] = absolute counts) of the follow-up sample ($n = 301$) with regard to Impairment Score (IS) total score (last 7 days) at t1 and t3 (IS case threshold ≥5 beyond the *dotted line*.)

20.9% of the follow-up sample showed the same level of impairment at t3 as at t1, 14.3% showed a higher impairment than at t1, and only 10.6% showed an improvement. Excluding the 54.2% sub-clinical probands and focusing on the probands who at t1 and/or t3 showed a clinically relevant impairment, we found 45.7% with a similar, 31.2% with a higher and only 23.1% with decreased impairment at t3. Accordingly, the case rate (ICD diagnosis of a psychogenic disorder + IS total score ≥5 in the last 7 days) within the follow-up sample increased from 21.6% at t1 to 26.2% at t3. A total of 97 (65.4%) of the 301 probands failed the case criteria at both t1 and t3. Forty probands (13.3%) were cases at both t1 and t3. Of the 65 cases at t1, 40 (61.5%) remained cases at t3 and only 39 (16.5%) of the 236 non-cases at t1 met the case criteria at t3. The odds ratio for a proband to remain as a case from t1 to t3 was 8.1 (CI 95%: 4.4–14.8). As expected the IS total score at t1 was higher in cases (6.08, SD 1.20) than in non-cases (IS total score 2.53, SD 1.15). Eleven years later, the former t1 cases showed a mean IS total score of 5.05 (SD 1.80) and the former non-cases were assessed to have a mean score of 2.99 (SD 1.66).

At t1, the ICD diagnoses of the cases were assessed according to ICD-8 and at t3 according to ICD-9. To compare the distribution of diagnostic categories at t1 and t3, we divided them into four main groups: neurotic spectrum disorders (neuroses), personality disorders (including addictions and sexual deviations), somato-

Table 1 Social and clinical variables of the t1 and the follow-up samples at the time of t1 (*IS* impairment score)

Variables	t1 sample ($n = 600$) *n* (%)	Follow-up sample ($n = 301$) at t1 *n* (%)
Sex		
Women	287 (47.8%)	141 (46.8%)
Men	313 (52.2%)	160 (53.2%)
Birth cohort		
1935	199 (33.2%)	126 (41.9%)
1945	199 (33.2%)	99 (32.9%)
1955	202 (33.7%)	76 (25.2%)
SES[a]		
Lower class	196 (32.6%)	93 (30.9%)
Middle class	247 (41.2%)	125 (41.5%)
Upper class	150 (25.0%)	79 (26.3%)
Misc.	7 (1.2%)	4 (1.3%)
Education[b]		
Elementary school	343 (57.1%)	186 (61.7%)
Work status		
Employed	467 (77.8%)	238 (79.1%)
	Mean (SD)	Mean (SD)
IS last 7 days		
Total score	3.39 (1.87)	3.30 (1.87)
Physical	1.21 (0.84)	1.22 (0.82)
Psychic	1.09 (0.80)	1.03 (0.80)
Behavioral	1.10 (0.83)	1.04 (0.83)
IS last year		
Total score	3.94 (1.92)	3.86 (1.96)
Physical	1.50 (0.82)	1.53 (0.81)
Psychic	1.27 (0.82)	1.21 (0.82)
Behavioral	1.18 (0.85)	1.12 (0.86)

[a] Socioeconomic status (SES) according to Kleining and Moore (1968)
[b] With regard to education, there were also no differences in other categories of the German school system

Table 2 Spontaneous course of psychogenic impairment (Impairment Score) in a population sample ($n = 301$); there is approximately 11 years between t1 and t3

	Follow-up sample ($n = 301$) at t1 Mean (SD)	Follow-up sample ($n = 301$) at t3 Mean (SD)
IS last 7 days		
Total score	**3.30** (1.87)	**3.44** (1.89)
Physical	1.22 (0.82)	1.12 (0.80)
Psychic	1.03 (0.80)	1.02 (0.79)
Behavioral	1.04 (0.83)	1.30 (0.87)
IS last year		
Total score	**3.86** (1.96)	**4.00** (1.93)
Physical	1.53 (0.81)	1.40 (0.80)
Psychic	1.21 (0.82)	1.26 (0.82)
Behavioral	1.12 (0.86)	1.35 (0.86)

Table 3 Diagnostic categories of the cases ($n = 65$) of the t3 sample ($n = 301$) at t1 and 11 years later at t3. Psychogenic disorders according to ICD; also displayed is the total Impairment Score (last 7 days) for each diagnostic category at t1 and t3

ICD-8/last 7 days			ICD-9/last 7 days		
	Cases at t1 *n* (%)	IS Total (SD)	Same subjects at t3 *n* (%)	IS Total (SD)	
No ICD diagnosis	0 (0.0)	–	23 (35.9)	3.82 (1.80)	No ICD diagnosis
Neurotic disorders (ICD-8, 300)	16 (25.0)	6.06 (1.18)	6 (9.4)	5.50 (1.38)	Neurotic disorders (ICD-9, 300)
Personality disorders (ICD-8, 301–304)	16 (25.0)	6.53 (1.46)	22 (34.4)	5.81 (1.59)	Personality disorders (ICD-9, 301–305)
Somatoform/psychosom. disord. (ICD-8, 305, 306)	32 (50.0)	5.84 (1.02)	10 (15.6)	5.90 (1.10)	Somatoform/psychosom. disord. (ICD-9, 306, 307)
Reactions (ICD-8, 307)	0 (0.0)	–	3 (4.7)	5.00 (1.73)	Reactions (ICD-9, 308, 309)
Total	65 (100.0)	6.08 (1.20)	64 (100.0)	5.04 (1.82)	Total[b]
Missing	0		1[a]		Missing

[a] The missing proband was diagnosed as a case of ICD 316+ (physical disorder with psychogenic background)
[b] One of the 64 probands at t3 received an ICD diagnosis but did not exceed the case threshold of the IS

form/psychosomatic disorders, and reactions. Table 3 shows the distribution of diagnostic categories among the 65 probands who met the case criteria at t1.

To investigate the intra-individual shift of impairment, we correlated the IS total scores of the follow-up sample at t1 with those at t3. Initial sub-scale scores and, in particular, total scores correlated very highly and significantly with the corresponding scores after 11 years. The correlation coefficient of the physical subscale was low compared to the other subscales (IS last 7 days t1/t3: total score 0.531, physical 0.356, psychic 0.386, behavioral 0.437; IS last year t1/t3: total score 0.551, physical 0.272, psychic 0.421, behavioral 0.432, Spearman correlation coefficients; P for all correlations two tailed <0.001; $n = 301$).

At t1, probands of the follow-up sample showed 4.6 psychogenic symptoms (mean, last 7 days; SD 2.9). Eleven years later, the mean number was 4.0 (SD 3.0). For the last 12 months the mean was 5.9 (SD 2.9) at t1 and 5.7 (SD 3.20) as t3. These differences are not statistically significant. The number of symptoms at t1 correlated significantly ($P < 0.001$, two-tailed) with t3. Spearman correlation coefficients were 0.41 (last 7 days) and 0.35 (last year).

Linear regression analysis was performed using ten variables of childhood development conditions (separation from mother/father during childhood development, global burden assessment during childhood development, expert ratings of mother/father deficit scores), three variables concerning clinical impairment [impairment score (IS), sum score of the Goldberg-Cooper interview (GCI), and number of symptoms according to a symptom check list with predominantly psychogenic/psychosocial-triggered complaints], 12 personality variables (FPI) and gender and age as independent variables. The IS total score (for the last year) at t3 was defined as the dependent variable.

Stepwise regression analysis identified five t1 variables as associated with clinical impairment (IS) at t3: IS total score, GCI total score, separation from father between 1st and 6th year for more than 6 months, global burden assessment during childhood development between 6th and 12th year and FPI subscale "openness". These significant independent variables explained 41.0% of the variance in the t3 impairment score.

Discussion

The most important finding of our long-term investigation of the spontaneous course of psychogenic disorders is a very time-stable clinical manifestation of illness-related impairment over 11 years in a representative random sample of the normal adult urban population.

Table 4 Multiple regression analysis of the long-term course of psychogenic impairment between t1 and t3. The dependent variable was defined as the impairment score at t3. Goodness of fit: $r^2 = 0.412$, adjusted $r^2 = 0.397$, β-coefficients non-standardized (*GCI* Goldberg-Cooper interview FPI Freiburger Persönlichkeitsinventar)

Variable at t1	β-coefficient	SD	*t*-value	*P*-value
IS sum score	0.362	0.088	4.14	0.000
GCI sum score	0.045	0.021	2.14	0.034
Separation from father between 1 and 6 years for >6 months	0.653	0.245	2.66	0.008
Global burden assessment between 6^{th} and 12^{th} year	0.878	0.320	2.75	0.007
FPI subscale "openness"	0.124	0.040	3.11	0.002

Using the IS (BSS, Schepank 1995), a standardized and reliable instrument, to assess the degree of psychogenic impairment, in the investigated follow-up sample the means of the total score and the three subscales were nearly the same at t3 as they were at t1. Furthermore, the distributions of the IS total score values, the case rate, and the mean number of psychogenic symptoms in the follow-up sample were very similar at t1 and t3. Cases of psychogenic disorders tended towards a certain decrease of impairment from t1 to t3, but this effect was compensated by the increasing impairment of the non-cases. Even the t1 cases who were not diagnosed according to the ICD ($n = 23$) at t3 showed a total impairment (3.82) close to the case threshold of the IS. In the group statistics, we found no hint of a mostly favorable course of psychogenic disorders in the long term. Only 23.1% of the clinically relevant impaired probands (at t1 and/or t3 beyond the threshold of ≥5 IS total score) showed a decreased impairment after 11 years spontaneous course. Under natural conditions, only 5.2% of the whole t1 sample ($n = 600$) reported contact with psychotherapeutic professionals in a broad sense (including counselling or single supportive contacts with public health service units). Thus, the impact of psychotherapeutic support on the psychogenic impairment in a normal population sample seems to be low.

Beyond these group statistics results, the probands who met the case criteria at t1 ($n = 65$) showed a high risk (61.5%) of remaining psychogenic disorder cases for more than a decade. This is also indicated by an odds ratio of 8.1 for t1 cases being diagnosed as a case again at t3. In contrast to these probands, the portion of the non-cases at t1 ($n = 197$) becoming cases at t3 was only 16.5%. The probands who were cases both at t1 and 11 years later at t3 showed a significantly stronger total impairment than the non-cases.

Furthermore, intra-individual correlation within the subscales and the IS total score for the entire sample indicated a surprising time stability between t1 and t3. The weakest inter-correlation was in impairment due to physical psychogenic (e.g. somatoform) symptoms. Since the IS total score was more stable over time ($r = 0.55$), physical/somatoform symptoms probably changed into psychic symptoms or into behavioral impairment. This result is in line with other investigations on the course of somatoform symptoms (Franz et al. 1998b). According to this, the ICD diagnoses and the clinical diagnoses of the probands changed within 11 years. However, the intra-individual variance in clinical manifestations (physical, psychic, and behavioral dimensions of psychogenic impairment), in the sense of changing co-morbidity or symptom shift, supports our approach of investigating the predominantly psychosocially influenced (psychogenic) disorders as a whole.

Within a regression model, we found a strong relation between the impairment score at t3 (dependent variable) and clinical, childhood and personality variables at t1. As expected, there is a strong association between the psychogenic impairment at t1 and at t3. In addition, a long separation from father in early childhood (the first 6 years) predisposed to a higher psychogenic impairment in later life. This is in line with psychodynamic concepts of early childhood development (Abelin 1971, 1975), other epidemiological findings (Werner 1989) and clinical studies (Dührssen 1984; Lieberz and Schwarz 1987). Due to World War II, in our follow-up sample ($n = 301$) a high rate of fathers were absent ($n = 122$; birth cohort 1935: 58.4%, 1945: 41.2%, 1955: 11.8%), while only a small number of mothers ($n = 26$) were absent during early childhood. So we were able to detect the effect of the missing father on an epidemiological basis. The "biographical echo" of a deficit of fathers' care and the missing support for the mother in establishing adequate mothering for the baby and the infant may be underestimated, and should be of interest especially for the assessment of long-term impact of wars on the mental health of a population. Our data also underline the importance of development conditions in later childhood for psychogenic impairments and complaints in adulthood.

FPI "openess" was associated weakly with an unfavorable course of psychogenic impairment. The interpretation is difficult. Probably the higher scoring probands find it easier to disclose themselves in a diagnostic interview situation. This could induce the tendency to assess the psychogenic impairment of those probands higher. How openess per se as a personality trait, standing for a lower orientation regarding social desirability, may contribute to a higher psychogenic impairment in the long term should be investigated in further studies.

However, the strong association of clinical variables at t1 (IS sum score, GCI sum score) with psychogenic impairment at t3 underlines the relevance of current for future psychogenic impairment in the sense of a bad spontaneous course. This does not, however, mean that personality traits (Ormel and Wohlfarth 1991; Costa and Widinger 1994) or childhood development (Tress 1986; Schepank 1987; Werner 1989; Pribor et al. 1993; Craig et al. 1993; Kubicka et al. 1995; Egle et al. 1997) have no effects on psychogenic impairment in later life.

In our previous study (Franz et al. 1998a), we used the IS for identifying several specific clusters and evaluated differences between two extreme groups using an extensive number of specific personality inventories. Subjects in a defined risk population (part of the entire sample) with a negative spontaneous long-term course (i.e. a time-stable bad state) differed from subjects with a constant positive spontaneous long-term course in several personality traits (e.g. depression) and showed higher pathology in all three surveys (stable over time). The present paper in contrast focuses on the entire sample and examines the relationship between the impairment score in the third survey and personality characteristics, clinical and sociodemographic variables in the first survey. We found only a weak association between one FPI subscale and psychogenic impairment.

Therefore, we were able to discriminate between positive and negative course in a highly selected sample using personality characteristics. On the other hand, personality characteristics were only weak predictors for psychogenic impairment a decade later for the entire sample.

Yet our methodological approach raises some critical issues. Our sample size of 301 is limited, and a simple generalization of our findings is therefore not possible. On the other hand, our follow-up sample is also representative of the t1 sample, derived from a random sample of the normal adult population of Mannheim. This means that we avoided the bias of individual or institutional selection effects that occur in patient-based studies. The IS we used for the assessment of the degree of psychogenic impairment is standardized and well-established in German samples only (Schepank 1995). Consequently, a careful and conservative interpretation of the results is indicated. However, due to the high co-morbidity and symptom shift in, for example, somatoform or psychoneurotically ill patients, a study of the natural history of psychogenic disorders needs a quantitative assessment of impairment in addition to a qualitative diagnostic classification (Schepank 1990; Nelson and Rice 1997).

All variables investigated (impairment, case rate, number of symptoms) indicate a predominantly unfavorable spontaneous long-term course of psychogenic impairment. Together with the well-known high prevalence of psychogenic/neurotic spectrum disorders in the normal population (26%; Schepank 1987, 1990) it underlines the need for early therapeutic and preventive intervention to avoid cases becoming chronic and developing secondary complications. In addition, further research is necessary to characterize the course-predicting variables of psychogenic disorders (Franz et al. 1998a).

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