Prevalence Rate and Psychological Correlates of Somatization Among Schizophrenic Patients in an Egyptian Sample

Thesis

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Introduction

Somatization is defined in many ways, the hallmark of each definition being the presentation of physical symptoms that are not sufficiently explained by a medical condition. ***Lipowski (1988)*** defines somatization as the tendency to experience and communicate medically unexplained physical symptoms (UPS) in response to psychological stress and to seek medical help for these symptoms. ***Kirmayer and Robbins (1991)***, define somatization as the somatic presentation of a psychiatric disorder. ***Bridges and Goldberg (1985)*** also define somatization as the somatic presentation of a psychiatric disorder but in their definition they include the patient's tendency to attribute these somatic symptoms to a physical problem and to search for a physical solution for them. Kirmayer and Robbins use the term true somatization for it while they use the term facultative somatization for patients who readily consider emotional problems for their somatic symptoms when invited to by their physician.

Epidemiological studies have demonstrated a high prevalence of such symptoms in the general population ***(Kroenke & Price, 1993)*** and in all medical settings ***(Escobar and Waitzkin, 1998)***. Somatization has been operationalized in distinct ways. It can be measured along a continuum in which case it is defined as the number of medically unexplained symptoms reported or as diagnostic categories grouped together under the heading of somatoform disorders in the Diagnostic and Statistical Manual of Mental Disorders (DSM) ***(APA, 1994)***. In addition a number of discrete somatoform disorders of functional somatic syndromes have been described in the literature ***(Manu, 1998)***.

A large number of factors have been studied to explain the phenomenon of somatization including a number of personality traits,

hypothesized as risk factors for the development or persistence of medically unexplained symptoms. Alexithymia is one of these factors ***(DeGucht & Heiser, 2003)***. ***Sifneos (1973)*** was the first to use the word alexithymia which literally means no words for feelings in order to capture what he considered to be a core characteristic of patients suffering from psychosomatic diseases. Although the alexithymia concept has originated within psychoanalysis the wider scientific arena has subsequently taken an interest in it. This has resulted in an accumulating body of research supporting the view that alexithymia is reflecting a deficit in the cognitive processing and regulation of emotions ***(Bagby et al., 1994)***.

Alexithymia which is defined as difficulties in describing and identifying feelings because it is supposed that alexithymic patients insufficiently realize that physical sensations may be the somatic manifestations of emotions, these patients are considered to be vulnerable to incorrectly attributing innocent physical symptoms to physical disease and to seeking medical care for symptoms for which no medical explanation can be found ***(Taylor et al., 1997)***.

Although alexithymia has been originally conceptualized as a dimensional construct distributed normally in the general population ***(Bagby & Taylor, 1997)***, cut off scores have been established empirically enabling researchers to distinguish alexithymic from non alexithymic subjects. While several previous studies have found evidence for an association between alexithymia and the presence of functional somatic symptoms ***(Acklin & Alexander, 1988)***, the generalizability of these results remains doubtful since most of these studies have used instruments for the assessment of alexithymia that have later been shown to lack reliability and validity ***(Taylor et al., 1990)***. However controlled treatment studies addressing the clinical course of alexithymia and somatization symptoms are still required to fully elucidate the potential role of alexithymia in predicting treatment outcome for somatizing patients ***(Bach et al., 1996)***.

Subjects and Method

Type of study:

This study is a cross-sectional, case-control study.

Site of study:

The study was carried out in Heliopolis Mental hospital. It is a governmental hospital including about 100 beds for inpatient service and an outpatient service covering all days of the working week. The hospital is one of the hospitals belonging to the General Secretariat of Mental health. It covers a catchment's area including the eastern part of Cairo.

(Districts like Heliopolis, Nasr City, Ain Shams, Zeitoun,….).

Subjects:

One hundred and forty cases participated in this study. They were organized into case group and control groups.

All cases and control groups gave an oral agreement to participate in the study after providing them with detailed information about the steps and aims of the study.

Case Selection:

The first 100 cases visiting the outpatient clinic were selected. The sample size was calculated using API/INFO program version 6 assuming: 59% confidence level, 80% power of the test, 60% prevalence of somatization in population ***(Simon and Vonkorff, 1992)***.

The work covers a period of about 2 years starting from June 2005 . The diagnosis depends on the criteria of DSM-IV (1994). The diagnosis is confirmed by one of the specialist attending the clinic. Another confirmation was given by the application of SCID-I. Cases were excluded if there was no agreement with the specialist or the SCID-I diagnosis.

Inclusion Criteria:

Age : 20-55 years

Sex : both sexes were included

Education: literate

Exclusion criteria:

- Those who refused to give consent.
- Patients with past or present history of other psychiatric disorder, substance abuse or serious medical disease.
- Discordance of diagnosis with the specialist.

Control Groups:

1- *Somatization group;*

20 patients presenting with the diagnoses of somatization according to the DSM-IV criteria. They were chosen from outpatient clinic of the same hospital and undergo the same method of selection like the case group.

2- Healthy control;

20 persons with no evidence of physical or psychological disorders were selected to match the other samples as much as possible .They were chosen from the workers in the hospital. The GHQ has been applied to them to rule out the presence of psychiatric illness.

Methodology:

1- All patients (schizophrenia and somatization) will undergo complete neuropsychiatric and medical history with emphasis on demographic and clinical data (age, sex, level of education, work, family history, number and presenting somatic symptoms).

2- The patients' groups were evaluated according to DSM-IV criteria for schizophrenia and other psychotic disorders in addition to somatoform disorders.

3- Cases were examined by the SCID-I to give further confirmation of the diagnosis before proceeding to other steps.

4- Every case will be subjected to the following test battery:

   1) The Symptom Check List (SCL-90R) is applied for the three groups to screen for somatization. SCL-90R is considered a trait marker for somatization disorder. Those with high scores tend to have amplification of cognitive, behavioral and emotional components. A case is defined by a global severity index score > to a T score of 60 or if any two primary dimensions scores are > to a T score of 60 (+ve diagnosis T GSI >60 or T2 DIM >60) ***(Derogatis, 1992)***.

   2) SAQ (Symptom Attribution Questionnaire) to classify.

      Them into somatizers, psychologizers, and externalizers according to their attribution of symptoms.

   3) TAS-20 (Toronto Alexithymia Scale) to relate somatic complaints to the ability of patients to express their feelings.

   4) EPQ (Eysenck Personality Questionnaire) to relate the somatic complaints to personality construct.

(1) Structured Clinical Interview for DSM-IV (SCID I): *(First et al., 1995).*

The SCID-I was developed in the early 1990s to provide a standardized DSM-III-R axis I diagnosis based on an efficient but thorough clinical evaluation. It has since been updated for DSM-IV. The semi-structured diagnostic interview begins with a section on demographic information and clinical background. Then there are 7 diagnostic modules, focused on different diagnostic groups: mood, psychotic, substance abuse, anxiety, somatoform, eating and adjustment disorders. Both required and optional probes are provided, and skip outs are subjected when no further questioning is warranted. All available information, including that from hospital records, informants, and patient's observation should be used to rate the SCID. The SCID-I is designed to be administered by experienced clinicians and is generally not recommended for use by lay interviewers. In addition formal training in the SCID is required, and training books and videos are available to facilitate this. In individuals without symptoms, the interview takes approximately 1 hour, but it may take up to 3 hours in individuals with extensive symptomatology. Although the primary focus is research with psychiatric patients, a non patient's version (with no reference to a chief complaint) and a more clinical version (without as much detailed subtyping) are also available. Reliability data on the SCID suggest that it performs better on more severe disorders (e.g. bipolar I disorder, alcohol dependence), than on milder one (e.g. dysthymia). Validity data are limited, as the SCID is more often used as the gold standard to judge other instruments. It is considered the standard interview to verify diagnosis in clinical trials and is extensively used in other forms of psychiatric research. It can be used to insure a systematic evaluation in psychiatric patients, for instance, on admission to an inpatient unit or at intake in an outpatient's clinic. It is also used in forensic practice to insure a formal and reproducible examination.

(2) The Symptom Checklist:

The Symptom Cheklist-90-R (SCL-90-R) instrument has been designed to evaluate a broad range of psychological problems and symptoms of psychopathology. The instrument is also useful in measuring patient progress or treatment outcomes.

The SCL-90-R is a measure of psychological distress, appropriate for both adolescents and adults. It is a revised from (two items were replaced and seven were modified) of the SCL-90. It consists of 90 items total, with 83 items representing nine subscales: somatization (SOM), obsessive-compulsive, interpersonal sensitivity (I-S), depression (DEP), anxiety (ANX), hostility (HOS), phobic anxiety (PHOB), paranoid ideation (PAR) and psychoticism (PSY). In addition to these nine symptoms, the SCL-90-R also contains seven items which relate to appetite and sleep disturbances.

Administration: 12-15 minutes.

Publication: 1975.

Scale Format: 5-point Likert, ranging from "not at all distressing" (0) to "extremely distressing" (4).

Administration: Self-administered.

Scoring and Interpretation: Raw scores are converted to T-scores and plotted on a profile showing the centile equivalent for each subscale. Subscale scores are found by summing item scores for the entire instrument and dividing by the total number of items on each subscale.

(3) Symptom Attribution Questionnaire (SAQ):

This 39-item questionnaire is a modified version of Robbins and Kirmayer's Symptom Interpretation Questionnaire (SIQ), which was demonstrated by these authors to have good validity and reliability ***(Robbins & Kirmayer, 1991)***. The SAQ lists 13 common somatic symptoms, each followed by an item addressing the likelihood of a

physical disease cause, an item addressing the likelihood of an emotional cause, and an item addressing the likelihood of an environmental cause for this symptom. The SAQ items are nearly identical to those of the SIQ. The main difference is that while the SIQ asks the participants to rate what they would probably think were the causes of various somatic symptoms if they had them (e.g., "If I had a prolonged headache, I would probably think it is because…."), the SAQ asks the participants to rate how common they think it is for the various symptoms to have each of three different kinds of causes. The participants were given the following instruction "Listed below are a number of different somatic symptoms that may have various kinds of explanations. Your task is to judge how common you think it is that these symptoms have these various kinds of causes. Please circle the figure next to each suggested cause that corresponds to how common you think it is for the symptoms to have this particular explanation."

Each item was rated on a 4 point Likert-type scale: 0= Seldom, 1= Sometimes, 2= Often, and 3= Most of the time. Summing the items with similar casual explanations gives three scale scores (each based on the same 13 symptoms) representing the extent to which physical disease (somatic attribution), emotional distress (psychological attribution), or environmental events (external attribution), were endorsed as possible causes of the symptoms.

It has been translated and validated by one of the Superior Professor Faisal Yunis.

Reliability [Symptoms Attribution Questionnaire (SAQ)].

Table (5)*: Shows Symptoms Attribution Questionnaire reliability.*

Table (6)*: Shows internal consistencies for the SAQ scale factors.*

All correlations were significant at (0.01) level, except items (p, t) in emotional (somatic) scale, items (c, f, w, aa, aj, ak) in environmental scale, and item (d) in psychological sale, all were significant at the (0.05) level.

(4) Toronto Alexithymia Scale (TAS):

The 20-Item Toronto Alexithymia Scale (TAS-20, German version). This recently developed 20-item self-report measure of alexithymia has been demonstrated to be a psychometrically sound measure of alexithymia. Written instructions were given asking the subjects to respond on a 5-point Likert scale the extent to which they agreed or disagreed with each statement. The results are expressed as TAS-20 global scores as well as factor scores, using the following item-factor distribution: Factor 1 (difficulty identifying feelings and distinguishing them from bodily sensations of emotion): Items 1, 3, 6, 7, 9, 13, and 14; Factor 2 (difficulty expressing feelings): Items 2, 4, 11, 12 and 17; and Factor 3 (externally oriented thinking): Items 5, 8, 10, 15, 16, 18, 19, and 20.

It has been translated and validated by one of the Superior Professor Faisal Yunis.

Reliability (Toronto Alexthymia Scale).

Table (7)*: Shows Toronto alexythmia scale reliability.*

Item total correlations of the Toronto Alexthemia Scale Factors.

Table (8): *Shows internal consistencies for Toronto alexthymia scale factors.*

All correlations are significant beyond the (0.01) level, except item (10) in factor 3 which was significant level at (0.05).

(5) Eysenck Personality Questionnaire (E.P.Q): *(Eysenck, 1975):*

This is simple self-report test. It has 101 questions (of which 11 questions were omitted) on the right side of the page the patient has to answer Yes of No to these questions, which are phrased in formal Arabic and can be understood by all those people who can read simple Arabic ***(Abdel Rahman, 1999)***. For illiterate people the test was read in colloquial formal Arabic.

The E.P.Q was designed to measure two major dimensions of personality, psychoticism (P) which is related to odd cruel, antisocial behavior, suspicion and a lack of feeling towards even those close to one, for females P= 2±2, neuroticism (N) is defined as emotional liability, over-responsiveness and liability to neurotic breakdown under stress, N=12±4, and extraversion – introversion (E) which implies the presence of an outgoing personality with uninhibited social tendencies, E=12±5, it includes also lie scale (L) which is regarded by Eysenck as a measure of "faking good".

(6) General Health Questionnaire (GHQ): *(Goldberg and Williams 1991) (Arabic version by Okasha, 1988):*

The GHQ has been applied to the healthy control group to rule out the presence of psychiatric illness.

It was initially developed as a first stage screening instrument for psychiatric illness in order to identify potential cases which could then be verified and the nature of which could be determined by using second stage instrument as clinical interview schedule as it shouldn't be used as a sole criterion for diagnosis, it is mainly used to detect caseness ***(Goldberg and Williams 1991)***.

It contains 60 items that refer to the severity of psychological complains during the past 4 weeks relative to the person's normal situation. Shorter versions of the GHQ have been developed. The GHQ 28 items is one of them. It includes 4 factors labeled scale "A" somatic symptom scale, "B" anxiety and insomnia scale, "C" social dysfunction scale and "D" severe depression scale, it takes approximately 5-10 minutes to be completed ***(Goldberg and Williams 1991)***.

The version used in this study is the Arabic version of a short 28-items scale ***(Okasha et al., 1988)***, with the sample scorer method which is (0-0-1-1). The cut off point of GHQ will be 7 according to similar previous national studies to minimize the associated fallacies with the original low threshold score ***(Abd El-Hakeem, 1988)***.

The instrument showed good test-retest reliability which was the best for the GHQ-28 demonstrating a coefficient of 0.90. The construct validity of the questionnaire is fairly supported by factor analysis and some studies provided evidence for minimal predictive validity ***(Goldberg and Williams 1991)***.

Statistical Methodology:

Analysis of data was done by IBM computer using SPSS (statistical program for social science version 12) as follows ***(Miller et al., 1992)***:

- Description of quantitative variables as mean, SD and range.
- Description of qualitative variables as number and percentage.
- Chi-square test was used to compare qualitative variables between groups.
- Unpaired t-test was used to compare two groups as regard quantitative variable in parametric data (SD<50% mean).
- Mann Whitney Willcoxon test was used instead of unpaired t-test in non parametric data (SD>50% mean).
- Spearman correlation co-efficient test was used to rank different variables versus each others either positively or inversely.

P-value >0.05 insignificant.

P<0.05 significant

P<0.01 highly significant

Results

Table (9)*: Differences in demographic data between the studied groups.*

This table shows no statistically significant difference that could be detected between the studied groups as regard age by using one way ANOVA-test and other data by using chi-square test yet statistical significance difference between studied groups as regard marital status and high statistical significance difference as regard social class.

Table (10)*: Distribution of the studied cases as regard prevalence of somatization in schizophrenia group.(1st aim of work).*

This table showed that 27% of the studied cases had positive somatization.

Table (11)*: Frequency distribution of physical symptoms of the SCL-90R somatization subscale in the studied groups.*

This table showed that the prevalent somatic symptoms in the schizophrenia cases in males were headache and GIT troubles while throat lumb and pain symptoms were more common in females. In somatization disorder the most common physical symptoms in males were pain symptoms and GIT troubles while chest pain, low back pain ,headache and fatigability were the most common in females. In healthy male group dyspnea and headache were more common while headache and low back pain were the more common in females.

Table (12)*: Socio-demographic data description.*

This table showed that the schizophrenic cases with somatization were middle aged, single, divorce was also frequent, university graduate, vocational occupation, and low social class.

Males out numbered females in schizophrenia sample, affecting results of gender prevalence being more in males.

Table (13)*:Comparison between gender as regard somatization among the studies cases.*

This table showed no significant statistical association between gender and somatization in schizophrenic cases

Table (14)*:Relation between family history and somatization among the studied cases (schizophrenic patients).*

This table showed no statistically significant association that could be detected between family history of schizophrenia and somatization by Z-Score test.

Table (15)*:Relation between type of schizophrenia and somatization in the studied cases.*

This table shows no statistical significant association between type of schizophrenia and somatization. Yet somatization is more prevalent among the undifferentiated type.

Table (16)*:Relation between social class and somatization among the studied cases (Schizophrenic patients).*

This table shows that somatization mainly was frequently present among low and very low social class with no statistically significant differences in between positive and negative cases by using Z-score test.

Table (17): ***Relation between marital status and somatization among the studied cases (schizophrenic patients).***

This table shows somatization is more frequent among singles with no statistically significant association that could be detected between marital status and somatization by using Z-Score test

Table (18): ***Relation between somatization and occupation.***

This table shows no statistically significant association that could be detected between occupation and somatization by using Z-score test (somatization is more freq in vocational occupations).

Table (19): ***Comparison between the studied cases and somatization disorder control group for the prevalence of other associated symptoms with somatization over SCL-90R subscales.***

This table shows a highly statistically significant difference between both groups as regard different psychological manifestation by using chi-square test.

Table (20): ***Differences on SCL-R90 subscales between high and low somatizers in the schizophrenic group.***

Schizophrenic patients scored significantly higher on somatization (T-score mean =63.9±2.9, T.N 24 Male & 3 Female, P<0.001) were considered to be high somatizers. This group also scored significantly higher on intersensitivity (mean=62.7 SD±3.6, P<0.01), on anxiety (mean=61.3±3.8, P<0.001), ph. anxiety (62.6±3.5, P<0.001), Paranoia (65.8±4.9NS), Psychotocism (64.5±3.2, P<0.001), and OCD (59.7±4) Subscales of the SCL-R90 than the schizophrenic group scored low somatization versus the schizophrenic group with low level of somatiztion. However the paranoid scale scored higher (65.8±4.9) in high som group that the difference is not significant with low somatization (65.4±7.8). In addition the schizophrenic patients with low level of somatization revealed higher statistical significance on global severity index (mean T-score GSI=59.7±2.4).

Positive symptom total (mean=56.9±1.3) and positive symptom distress index (mean=17.9±7.5) subscales of symptom checklist-R90 versus the low som schizophrenic group. On the other hand, female schizophrenic group with high level of somatization showed the same results as the male group yet showed no statistical significance on OCD-scale and positive symptom total versus the low female group

Table (21): ***Differences on SCL-R90 subscales between high and low somatizers in the somatization group.***

Only high somatizers exceeded the threshold of T-score of somatization subscale >/=60 in both male and female somatization disorder groups. The differences in all items of SCL-90R were not statistically significant between the low and high somatizers except for anxiety subscale; males with high level of somatization had higher t score of anxiety subscale more than males with low somatization subgroup.

Table (22): ***Differences between positive cases and somatization disorder and healthy group on TAS-20 items.***

This table showed that positive cases had high score over factor1; difficulty identifying feelings and with were more external oriented thinking more than other groups. Although somatization disorder group had highest score on factor 3.

Both positive cases and somatization disorder group scored higher than the normal group on TAS-20 global score indicating that both were alexithymic but positive cases had clinical significant alexithymia.

There was statistical highly significant difference between the score means of TAS-20 factors.

Table (23): ***Differences between the studied cases and control groups over TAS-20.***

There was statistical high significant differences between the studied cases and control groups on TAS-20 factors and global score by using ANOVA test for the high kevel of somatization subgroups and t-test for the low level of somatiztion subgroups.

This table showed that schizophrenic cases and somatization disorder group with low level of somatization had difficulty identifying feelings and were more externally oriented thinkers.

But the schizophrenic cases revealed clinical significant alexithymia.

Somatization disorder group with low level of somatization were more alexithymic than those with high level of somatization within the same group as they scored higher on the global score.

Table (24): ***Differences on TAS-20 items between high and low somatizers in male schizophrenic group.***

This table shows statistical significant differences and associated between high and low som over TAS-20 on factor 1 and factor 3 and total score global severity index.

Table (25): ***Differences on TAS-20 items between high and low somatizers in female schizophrenic group.***

This table shows statistical significant differences exist between high and low female somatizers in total global severity index. All other item of TAS-20 are not statistically significant.

Table (26): ***Differences in symptom attribution style between studied group.***

This table showed that positive cases tend to attribute symptoms specially to somatic causes (scored high on somatic attribution over other attribution dimensions) and were labeled as somatizers.

Somatization disorder group were more psychologizer and attributing their symptoms to psychological causes.

There was statistical significant association between the studied groups by using ANOVA test.

Table (27):***Differences between the studied cases and control groups in symptom attribution.***

This table showed that:

1. Low level of somatization schizophrenic subgroup were using the somatic attribution style and those in somatization subgroup were using the psychologizing attribution .
2. The somatization subgroup with high level of somatization were labeled as psychologizers / externalizers as they scored higher on psychological attribution more than other attributional styles and high score on externalizing attribution with low somatizing score.

Table (28):***Shows differences on symptom attribution questionnaire between high and low levels of somatization in male schizophrenic group.***

This table shows non-statistical significant differences between studied group and regard attribution of symptoms in male high and low schizophrenics by using T test.

Table (29):***Shows differences on symptom attribution questionnaire between high som and low som in female schizophrenic group.***

This table shows non-statistical significant differences between high som and low som in female schizophrenic group as regards symptom attribution.

Table (30)*:* ***Differences between the studied cases and control groups over the EPI.***

This table shows high statistical significant differences between subgroups with the highest score for the somatization subgroup, by using t-test for low subgroups and ANOVA for high groups.

The table reveals:

1- The schizophrenia group:

low som subgroup ……N>E

High som subgroup……E>N

This could be explained by TAS-20 factors as there is -ve pearson correlation between factor 1(difficulty identifying feelings) and N

Also by somatic symptom attribution –ve correlation with N

2- The somatization group;

low som subgroup……..N = E with no statistical significant.

High som subgroups……..E>N

This could be explained by externalizing attribution –ve correlation with E.

And by factor 3 (externally oriented thinking) +ve correlation with E.

Discussion

The term somatization was used differently by various authors. In reviewing both the term and the concept, ***Lipowski (1987)*** considered somatization to be a process as well as a disorder. He suggested that the most useful definition was from ***Kleinman (1982),*** in which somatization was defined as a "somatic idiom of psychological distress". In research studies, somatization had been operationalized in three ways:

1) as medically unexplained somatic symptoms.
2) as hypochondriacal worry or somatic preoccupation.
3) as somatic clinical presentations of affective, anxiety or other; the expression psychiatric disorders ***(Janca et al., 1995).***

The current study was designed to achieve the following aims:

1) Predict the prevalence of somatization among the schizophrenic patients.
2) Describe the psychological correlates of somatization in schizophrenic patients.

The studied groups consisted of a group of schizophrenic cases (n=100), a somatization disorder control group (n=20) and a healthy control group (n=20).The comparison of the three studied groups showed no statistically significant differences in all socio-demographic variables except in the social class and the marital status. The results revealed that half of the schizophrenic cases studied (representing 50%) were included in the very low social class and 47% (approaching half the cases) were unmarried .whilst 60% of the somatization disorder group were representative in the low social class and 100% of cases were married .Furthermore, 90% of healthy group were included in the low social class and 75% were married.

This can be explained by the following:

The majority of schizophrenics were from low and very low social class. A finding that could be explained by the known drifting hypothesis that affect schizophrenics during the course of illness. In addition this was in line with what ***Wool & Barsky (1994)*** hypothesized that somatization was more prevalent in lower social class. The higher rates of unmarried cases in the schizophrenic group could be related to the instability of social life that schizophrenics suffer from being affected by this disorder.

The Symptom Check List (SCL-90R) was applied for the three groups to screen for somatization. The raw scores on the somatization subscale were linearly transformed to t-scores across a 0-100 range with higher scores indicating higher levels of somatization. The somatization subscale was not a criterion measure of the DSM-IV somatization disorder ,but was instead a continuous measure of somatization trait- a marker of increased cognitive, affective ,and behavioral responses to physical sensations ***(Derogatis, 1983).*** Although somatization trait did not reflect a unique diagnosis, the typical symptoms of somatization trait compromise a measurable psychological dimension marked by a heightened perception of bodily function. The items of the SCL-90R somatization subscale were identified by factor analysis to jointly capture a definable psychological dimension that was distinct from the psychiatric diagnosis of somatization disorder. Therefore although somatization trait might be the end product of other underlying medical, psychological, or social conditions i.e, epiphnomenon, it nonetheless behaved as a reproducible and definable construct ***(Brennan et al., 2005)***. A case was defined by a global severity index score >or= to a T score of 60 or if any two primary dimensions scores were >or= to a T score of 60 (+ve diagnosis T GSI >or=60 or T2 DIM >or=60) ***(Derogatis, 1992).***

Using the SCL-90R as a screening tool for somatization had revealed the following findings in the schizophrenic group:

1) Regarding the first aim, the results of the current study pointed out that the prevalence of somatization in the schizophrenic group is 27%.

2) The schizophrenics who had a T-score for somatization less than 60 were still showing manifestations of somatic complaints that could be considered a subthreshold or subsyndromal form of somatization

3) The schizophrenics scored higher than the somatization on other items of the scale: obsessive compulsive - intersensitivity anxiety-phobic anxiety-paranoid ideation-psychoticism and the additional items.

Many patients with somatization did not meet the full diagnostic criteria for the disorder and could be considered a subsyndromal variant of the condition ***(Margo & Margo, 1994).*** Researchers had noted that when criteria were used, the prevalence of somatization disorder in community studies was extremely low (0.7%) ***(Swartz et al., 1991).***

An additional limitation was that somatization trait was measure (using the SCL-90) instead of somatization disorder (using the DSM-IV criteria). Because somatization trait represented a less extreme pathology than somatization disorder, measuring the trait might well underestimate the impact of true somatoform disorder on resource utilization. Nonetheless, there might be several advantages to focusing on somatization trait instead of somatization disorder. First, somatization trait was much more prevalent than somatization disorder ***(Whitehead et al., 2002)*** and was therefore of higher everyday clinical relevance. Second, somatization trait represented an earlier stage along the psychological spectrum, and therefore represented a more modifiable stage than somatization disorder (i.e., early identification of somatization trait might lead to early appropriate therapy). Third, as an early stage along a spectrum of severity, somatization trait was potentially more likely to go unrecognized-in contrast to the more explicit psychopathology of somatization disorder ***(Spiegel et al., 2005).***

There are several problems with the current definition of somatization disorder. These include the restrictive diagnostic criteria, the focus on symptom counting, and the failure to include aspects of this disorder such as behavior, cognitive attribution and personality ***(Phillips, 2001).***

In addition the clinical status of individuals whose symptoms do not meet DSM-IV criteria for somatization disorder, but who are troubled by their medically unexplained complaints is unclear ***(Philips, 2001).***

Several studies had suggested included an abridged or subsyndromal form of somatization in the official psychiatric nosology DSM. ***Rief and Hiller (1999)*** proposed the term polysymptomatic somatoform disorder to refer the presence of at least seven unexplained physical symptoms eg; sustained focused attention on bodily process or a general tendency to misinterpret bodily sensations as evidence of physical illness.

Escobar et al. (1989) also proposed a less severe form of somatization disorder. This form required the presence of four or more physical symptoms for men and six or more symptoms for women of the forty specific somatization symptoms included in the composite international diagnostic interview. These symptoms had to reach certain severity levels and be medically unexplained.

Swartz et al. (1986) also defined a subsyndromal form of somatization disorder associated with higher rates of health care seeking – behavior than in patients with DSM-defined somatization disorder; 11.6% of the general population met criteria for this category.

Kroenke and Spitzer (1998) also introduced a subsyndromal form of somatization disorder called “Multisomatoform Disorder”. This concept stressed the presence of 3 or more current somatoform symptoms from a 15-symptom checklist along with at least a 2 year history of somatoform symptoms.

Ethnographic research by ***Kirmayer and Young (1998)*** urged the inclusion of cultural meanings of symptoms in the development of somatization classification criteria. These and other researchers proposed the potential utility of viewing somatization as a continuum in which increasing degree of somatic symptoms indicating distress, disability of maladaptive illness behavior ***(Lipowski, 1987).***

The clinical utility of this broader concept was significant in that it might better identify treatable somatizing patients with comorbid psychiatric disorders ***(Lipowski, 1990).***

Subsequently, the distinction between high and low somatizations might be more valid than viewing them as merely present or absent. Accordingly, the three groups are divided into high (with T score > 60) and low somatizers (with T score < 60). Thus, 27 from the schizophrenic group and 8 from the somatization disorder control group were high somatizers. On the other hand, 73 from the schizophrenic group, 12 from the somatization disorder control group and all the healthy control group were low somatizers.

Regarding gender differences that usually explain that women outnumber men in somatization disorder, the current study showed the following results:

The high somatizers from the schizophrenic group were 24 out of 87 males (27.5%) and 4 out of 13 females (30.7%). Whereas the high somatizers from the somatization control group constitute 2 out of 8 males (25%) and 4 out of 12 females (33.3%).

This result pointed out to the increase of number of male low somatizers in both the schizophrenic group and the somatization control group. Also, the percentage of females to the total number of females was higher than the percentage of males to the total number of males. However, it was noteworthy that the total number of males exceeded that of females and this might have a direct effect on this result.

The results in the current study revealed that the schizophrenic cases with high level of somatization were middle aged males, singles, university graduates, more frequent in those with vocational occupation and mostly included in the very low social class. However somatization disorder were more prevalent in middle aged females, married, house wife and more frequent in the low social class.

The epidemiologic catchement area study revealed that the most common medically-unexplained somatic symptoms in the United States were gynecological complaints followed by gastro-intestinal and cardiovascular symptoms ***(Escobar et al., 1987).***

In the current study, the most prevalent symptoms in the schizophrenic cases in males were headache and GIT troubles while neurasthenia, feeling a lump in the throat and pain symptoms (headache, backache and muscle pains)were more common in females. However in the somatization disorder group the most common symptoms in males were pain symptoms and GIT troubles whilst chest pain ,low back pain, headache and fatigability were the most common in females .yet in healthy group dyspnea and headache were more common in males but headache and low back pain were the more frequent in females.

The results revealed that there was neither association between family history nor the type of schizophrenia and somatization, however somatization was more frequent in undifferentiated type representing 48%.

It was often assumed that somatization was a characteristic of mental disorders in non-western societies. The reason behind that was the less acceptance of psychological symptoms and mental illness in these societies. However, it was acknowledged that somatic presentations were characteristic of all cultures. Furthermore, the psychological symptoms of common mental disorders could often be elicited on inquiry ***(Araya et al., 2001).***

A large number of factors had been studied to explain the phenomenon of somatization including a number of personality traits, hypothesized as risk factors for the development or persistence of medically unexplained symptoms. Alexithymia was found to be one of these factors ***(DeGucht & Heiser, 2003).***

Alexithymia was defined as a personality trait characterized by difficulties in describing and identifying feelings, a limited capacity for imagination, and externally oriented thinking rather than reflection on inner experience.

Because it was supposed that alexithymic patients insufficiently realize that physical sensations may be the somatic manifestations of emotions, these would be considered to be vulnerable to incorrectly attributing innocent physical symptoms to physical disease and to be seeking medical care for symptoms for which no medical explanation could be found. Alexithymia was therefore believed to be a predisposing and sustaining factor for the development of unexplained physical symptoms ***(Taylor et al., 1997).*** Thus it had also been called “blind feel” analogous to blind sight ***(Lane et al., 1997).***

However little was known about the brain mechanisms by which this feature predisposes individuals to somatic symptoms and illness. There was also evidence that the neural processing of emotions was different between men and women ***(George et al., 1996).*** The motor and somatosensory areas were activated to a greater extent and the anterior cingulated cortex to a lesser extent in the alexithymic group. This could be related to the tendency of people with alexithymia to experience bodily sensations in an emotion provoking situation. These findings also corresponded to the areas involved in introception, that was the feeling of the body state ***(Karlsson et al., 2008).***

According to ***Criag (2003),*** this afferent path represented the physiological condition of all tissues in the body. This included both distinct

sensations engendered in the interoceptive and anterior insular cortex as well as affective motivations engendered in the anterior cingulated cortex. It seemed that there was a disruption of these functions in alexithymia and this phenomenon might in the long run lead to the tendency of individuals with this condition to develop psychosomatic symptoms.

According to a hypothesis by ***Lane et al. (1997),*** environmental events trigger an emotional response that was associated with an impoverished conscious experience of emotion in individuals with alexithymia. Instead these individuals manifested behavioral and autonomic responses, including a heightened awareness of bodily sensations. This theory of alexithymia suggested that people with this condition exhibit less activation in the anterior cingulated cortex, an area known to have a role in the capacity to experience emotion in a differentiated and complex way ***(Lane et al., 1998),*** and this assumption had been supported by two previous imaging studies ***(Kano et al., 2003 and Berthoz et al., 2002).***

According to the older theories of emotion, the left hemisphere was specialized in cognitive processing an the right hemisphere in the processing of emotions. However, in a meta-analysis of healthy people ***(Wager et al., 2003),*** the authors found no support for the hypothesis of overall right lateralization of emotion processing. It could be that the normal balance between the hemispheres was lost in alexithymia, and that individuals with this condition thus exhibited relatively more left sided activations.

Some evidence in favour of this view exists. Several studies had suggested left hemisphere dominance in participants with alexithymia ***(Parker et al., 1992 and Jessimer & Markhan, 1997).*** Deficits in the perception of emotion had also been found in people with right hemisphere damage ***(Mandal et al., 1999 and Cicero et al., 1999).***

Finally ***Karlson et al (2008)*** hypothesized that alexithymia was related both to impairment in the processing of emotions(less activation in the anterior cingulated) and to a tendency to activate brain areas relating to bodily sensations in emotion evoking situations.

The Toronto Alexithymia Scale 20 (TAS-20) is generally accepted as the standard instrument for determining the degree of alexithymia. It is a self-report questionnaire with 20 items on a 5-point Likert scale. The items in the TAS-20 relate to three alexithymic dimensions: difficulties in identifying feelings, difficulties in describing feelings, and externally oriented thinking. The theoretical score range is from 20 to 100 and a total score >or=53 is considered to indicate alexithymia ***(Parker et al., 1993).***

The application of TAS-20 in the current study revealed that schizophrenic cases scored higher than the control groups over TAS-20 factor 1,factor 2 and factor 3 and there was statistically high significant differences between the studied cases and the control groups . The schizophrenic cases with high level of somatization had difficulty in identifying feelings while the schizophrenic cases with low level of somatization had difficulty in identifying feelings with externally oriented thinking. There was statistical significant differences between the high and low level of somatization in male schizophrenic cases on factor 1 and factor 3. On the other hand there was no statistical significant difference between the low and high levels of somatization in female schizophrenia cases. however the schizophrenic male cases had difficulty in identifying and describing feelings more than the female cases. while the female cases were more externally oriented thinking than the male cases.

The global severity index of the schizophrenic cases was higher than that of the control groups and the differences were statistically significant, indicating significant alexithymia. The high level of somatization in the male schizophrenic cases had higher global severity index than those with lower

level of somatization and the difference was statistically significant. On the other hand the female schizophrenic cases with low level of somatization had higher global severity index than those with high level of somatization.

Similarly the total score of the global severity index of the somatization disorder group was higher than that of the healthy control group and the difference is statistically significant. On the other hand, high and low somatizers of the somatization disorder group revealed no statistically significant differences in all items between male and female high versus low somatizers. The only exception was difficulty identifying feelings between high versus low male somatizers. The somatization disorder group with low level of somatization had more difficulty in identifying feelings than the group with low level of somatization.

To sum up the schizophrenic cases revealed significant clinical alexithymia, whilst somatization disorder group with low level of somatization revealed more alexithymia than somatization disorder with high level of somatization.

Patients with unexplained physical symptoms did not have all alexithymic characteristics to the same degree; one could therefore hypothesize that the prognosis of more alexithymic patients would be less positive than that of patients who are alexithymic to a lesser degree ***(Kooiman et al., 2004).***

Moreover the factors of the TAS showed different impact on health related behavior, eg., difficulty identying feelings was related to higher use of out patient treatment ***(Lumley & Norman, 1996)*** and increased pain sensitivity ***(Niklicek & Vingerhoets, 2000***), whereas externally oriented thinking correlated with a decreased use of medical and psychotherapeutic treatment ***(Lumley & Norman, 1996).***

Personality dimensions that influence the reporting of symptoms and the pursuit of health care might contribute to somatization ***(Kirmayer et al., 1994).*** For example, neuroticism, harm avoidance and negative affectivity had all been shown to increase the experience of somatic as well as psychological symptoms ***(Russo et al., 1994).*** Likewise, traits that heighten bodily perception or increase self-absorption might also influence symptom reporting ***(Kirmayer et al., 1994).*** Certain other dimensions might contribute to the seeking of health care and to conflictual or unsatisfactory interactions with health care providers. Somatizing patients had been described as angry, resentful, and mistrustful – characteristics that suggest the negative pole of agreeableness ***(Starcevic, 1990).***

The current study showed that by using the EPI, somatization group had the highest score compared to schizophrenic cases with high significant difference in between and healthy controls with highly significant difference in between by using one way ANOVA test. The schizophrenic cases with high level of somatization revealed less neuroticism than the schizophrenic cases with low level of somatization. This could be explained by negative pearson correlation between factor 1 (difficulty identifying feelings) with neuroticism, furthermore by negative pearson correlation between somatic attribution with neuroticism.

Similarly somatization disorder group with high level of somatization were more extraversion than the somatization disorder with low level of somatization. This could be rationalized by the negative pearson correlation with extraversion, and by the positive pearson correlation between factor 3(externally oriented thinking)with extraversion.

Other studies have found that difficulty in factor 1 relates most highly to neuroticism (and secondary to introversion), where as externally oriented thinking relates quite high and uniquely to low openness ***(Taylor et al., 1997 & Wise et al., 1992).***

In the same line, ***Noyes et al. (2001)*** associated more neuroticism to somatizers but contrary to the current results, less extraversion was associated. Moreover, other personality traits have been described. They observed an increase in obsessive-compulsive, self-defeating and negativistic traits. Difficult and frustrating patients have characteristics suggestive of very similar personality disturbances, including vague complaints, excessive demands, chronic complaining, lack of cooperation, lack of treatment response and high utilization of services.

The last point that the present study tried to cover was the attribution styles of patients to explain their somatic complaints.

Robbins and Kirmayer (1991) identified three dimensions of casual explanations for common physical complaints:

- Somatizers who attributed somatic complaints to somatic causes like physical illness. They scored high on somatic attribution and low on the other attribution dimensions;

- Psychologizers who attribute somatic complaints to psychological causes like emotional distress. They scored high on psychological attribution and low on the other attribution dimensions;

- Externalizers (normalizers) who attributed somatic complaints to environmental causes or behaviors like eating drinking, exercise. They scored high on external attribution and low on the somatic attribution dimensions.

In this attribution model, when unable to find normalizing (external or environmental) attributions to his unexplained somatic complaint, the person might use pathological explanations – either somatic or psychological ***(Gulec, 2008).***

In the current study, the results revealed that the schizophrenic group was generally high attributers especially when compared to the healthy control. The higher attribution was for somatic more than psychological and external causes. However the highest attribution was for somatic causes more than other attributional styles. Meanwhile, there was no statically significant differences between the high and low somatizers.

This was in concordance with ***Dumen et al. (2004)*** who had reported in their study that the patients with medically unexplained syndromes for whom psychiatric consultations were performed used somatic attribution more than the healthy control group. However ***Kirmayer (1991, Study 2)*** reported that somatic symptoms was significantly associated with psychological attribution but not with the somatic attribution

Similarly, the somatization disorder group was also high attributers when compared to the healthy group. The high somatizers were using psychological and external attribution; while the low somatizers were using the somatic and the psychological attribution styles. The differences in attributional styles between the high and low somatizers were not statistically significant except for the external attribution as the somatization disorder group with low level of somatization were using the external attribution style more than the group with low level of somatization.

Robbins and Kirmayer (1991, Study 3) found that the somatic attribution, but not the psychological attribution, was predictive of the number of somatic symptoms and that the psychological attribution ,on the other hand, was predictive of the number of psychological problems on the other hand psychological attribution was predictive of the number of psychosocial problems in the same study.

Lundh and Wangby (2002) compared psychologizers/externalizers with the somatizers who had the opposite attributional style ie; scoring high on

somatic attribution but not on psychological and external attribution; the former scored substantially higher on the trait measures of somatic complaints and negative affect.

They also suggested that individuals with a preponderance of beliefs that implicate emotional and behavioral processes as causes of somatic symptoms, tend to overestimate their somatic symptoms and negative emotional experiences in retrospect, as compared with their aggregated daily observations of somatic symptoms and negative affect. Paradoxically, this could suggest that somatization in the sense of a retrospective overestimation of somatic symptoms was not primarily due to somatizing in the sense of an elevated attribution of somatic symptoms to medical disease, but to a peculiar combination of nonsomatizing ie; emotional and external/behavioral attributions ***(Lundh & Wangby, 2002).***

One possible hypothesis was that psychological and external attribution of somatic symptoms involved more of cognitive elaboration than somatic attribution, as the latter generally represented less of a conceptual leap(attribution of somatic symptoms to somatic causes). If so psychological and external symptom attribution might be expected to lead to better memory for somatic symptoms that had been experienced and thereby to higher scores on trait measures of somatic symptoms ***(Lundh & Wangby, 2002)***

Thus causal attributions might influence help-seeking pathways, communications with clinicians, treatment compliance, and hence the course of illness ***(Gulec, 2008).***

Finally the current study estimated the prevalence of somatization in schizophrenia about 27%, the frequency of somatization defined as the presentation of 12 medically unexplained somatic symptoms (SCL-90R somatization subscale t score >or= 60). Somatization score was not associated

with gender, age, education, occupation, family history or type of schizophrenia.

The current study also revealed that there was positive association between high somatization score in female schizophrenic cases with difficulty in identifying feelings and with difficulty describing feelings (correlation & S=0.01, 0.999 & S=0.5 respectively), moreover a positive association with externally oriented thinking and anxiety score (correlation 0.997& S=0.5).

There was a positive association between high somatization score in male schizophrenic cases and intersenstivity subscale score (correlation 0.473 & S=0.5), and a positive association between high somatization score in male schizophrenic cases with exteraversion versus hostility subscale T-score (correlation 0.532 & S=0.01) and negative association on neuroticisim (correlation 0.626 & S=0.01). In addition to a positive association between low somatization score in schizophrenia with ocd, intersenstivity depression, phobic anxiety, paranoid and psychoticism subscales.

OCD (pearson correlation 0.480 & S=0.01), intersenstivity (correlation 0.371 & S=0.01), depression (correlation 0.420 & S=0.1), anxiety (correlation 0.602 & S=0.01), phobicanxiety (correlation 0.489 & S=0.01), paranoid (correlation 0.271 & S=0.01), and psychoticim subscales (correlation 0.397 & S=0.01). There was also a positive association between these subscales and factor (difficulty identifying feelings). Moreover a positive association between factor 3 (externally oriented thinking and the score of phobic anxiety and psychoticism subscales.

Although somatization was a prevalent problem among schizophrenia patients, knowledge was limited concerning somatic symptoms that cannot be accounted for by detectable somatic illness among schizophrenia patients ***(Ritsner, 2003).***

Among the very few research having a similar path ***Brown et al. (1990)*** estimated the prevalence rates of unexplained somatic complaints who met DSM III-R for somatization disorder for nine comorbid conditions, the prevalence rates were as follows: 54.6% in major depression, 33.6% in generalized anxiety disorder, 31.1% in phobic disorders, 4.2% in mania, 4.9% in drug abuse. Risk ratios were highest for panic disorder (16.25), major depression (9.41), schizophrenia (7.77) and OCD (7.04).

However somatization score in schizophrenia was associated with emotional distress attributed to psychopathology, side effects of antipsychotic agents, and family member's attitudes toward schizophrenia patients. Studies suggested that insight, self esteem and social support might protect against somatization in schizophrenia patient ***(Ritsner, 2003).***

The paucity of comparative studies was an indicator of the insufficient research in this domain. Yet from the experience derived from clinical observations it can be accepted that about one out of four schizophrenics may present with somatization disorder. Practically, clinicians are more inclined when they examine schizophrenic cases to focus on complaints that lead to more disturbance of the patients' or their families like: delusions, hallucinations, disorganized thought or behavior.

Another point that may explain the scarcity of research in this area was the inability of patients to explain their internal feelings. Patients were often unable to communicate their symptoms and give a coherent account because of the internal chaos associated with their psychiatric illness ***(Friedrich & Florry, 1999).*** Moreover, patients were more alexithymic than the normal subjects ***(Bach et al., 1996).***

A major challenge to future somatization diagnosis is the substantial overlap between existing individual functional syndromes, with similarities

outweighing the differences between them ***(Wessely et al., 1999).*** Another challenge is the comorbidity issue ***(Fink et al., 2003).***

The importance of probing for or ruling out concomitant physical disease is yet another issue. In order not to miss any coexistent physical

To conclude, in addition to ruling out medical causes for the patient's symptoms, a doctor who is evaluating a patient for a somatization disorder will consider the possibility of other psychiatric diagnosis or of overlapping psychiatric disorders. Whether they are a separate diagnosis or a clinical presentation of other diagnosis, they represent a significant problem for the health care system because patients with these disturbances overuse medical services and resources.

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Whether they are a separate diagnosis or a clinical presentation of other diagnosis, they represent a significant problem for the health care system because patients with these disturbances overuse medical services and resources.

Conclusion and Recommendations:

It becomes clear that somatization is not merely a separate diagnostic entity. Moreover, it may be prominent in many other psychiatric disorders or occur during their course. It is not restricted to non-western societies or the minorities of western countries as was conceptualized .It may affect men and women. It has tremendous effects on the psycho-socio-biological aspects of individuals as well as an economic burden. It is not well understood but it affects important areas of the clinical practice like the decision of medical consultation, help-seeking pathways, communication with clinicians, course of illness and quality of life. Therefore, it is an area where light should be thrown to solve its enigma.

The current study reveals the following results:

- 27 % of the schizophrenic group score high on somatization subscale of Symptom Check List-90;

- The comparison of the three studied groups revealed no statistically significant differences in all socio-demographic data except the social class and marital status;

- The distinction between high and low somatizers is probably more useful and deliver more information since cases from the three studied groups even the healthy control group present with somatic complaints. Therefore the low somatizers are considered a subsyndrome of the somatization disorder;

- The most common prevalent somatic symptoms in the schizophrenic group were GIT troubles, neurasthenia, feeling a lump in the throat, headache, backache and muscle pains;

- The application of Toronto Alexithymia scale revealed statistically significant differences between high and low somatizers in difficulty in

identifying feelings, externally oriented thinking and the total score of global severity index;

The global severity index of schizophrenics is higher than that of both somatization and the control healthy groups and the differences are statistically significant.

the total score of the global severity index of the somatization group is higher than that of the healthy control group and the difference is highly statistically significant. The global severity index of schizophrenics is higher than that of both somatization and the control healthy groups and the differences are statistically significant.

The current study showed that by using the EPI, somatization group had the highest score compared to schizophrenic cases with high significant difference in between and healthy controls with highly significant difference in between by using one way ANOVA test.

The schizophrenic group was generally high attributers especially when compared to the healthy control. The highest attribution was for somatic attribution. Meanwhile, there was no statistically significant differences between the high and low somatizers.

Similarly, the somatization group was also high attributers when compared to the healthy group. The high somatizers were using psychological and external attribution; while the low somatizers were using psychological attribution styles. The differences in attribution styles between the high and low somatizers were not statistically significant except for the external attribution.

Limitations of the study:

(1) The number of females in the three studied groups was limited.

(2) The psychological battery of testing was very exhausting.

(3) A list of side effects of psychotropic drugs needs to be used to exclude any confusion with medical complaints.

Research Recommendations:

Research in the area of comorbidity of somatization is still scarce; therefore further research efforts are still needed:

(1) to replicate the study on larger sample.

(2) to use neuro-physiological battery of investigations to confirm or rule out the existence of the somatic complaints.

(3) to replicate the study with other psychiatric diagnosis.

(4) to compare the somatic complaints on patients taking or not taking medications, general, and schizophrenics, in particular, to explore the presence of somatic complaints and to manage them properly if present; as well as to avoid the overuse of medical services and resources.

(5) to take seriously the somatic complaints of psychiatric patients to exclude any possible organic findings.

(6) to help patients how to identify and describe their feelings to avoid the sequelae of alexithymia.

(7) to try to understand the attribution styles of patients and to help them to use normalizing attribution style.

Summary

Somatization is considered the expression of psychological distress in the form of physical symptoms. Although, these symptoms have no biomedical explainable origin, somatizing patients suffer greatly and frequently seek symptomatic relief from family physicians.

Epidemiological studies have demonstrated a high prevalence of such symptoms in the general population and in all medical settings.

A large number of factors have been studied to explain the phenomenon of somatization including a number of personality traits. Alexithymia is one of these traits.

It has been postulated that functional somatic symptoms are more in women than men and in non-western cultures more than western. Patients are continuing to seek medical care, take several medications and submit to needless diagnostic and surgical procedures.

The aim of this study is to estimate the prevalence of somatization in schizophrenic patients and to describe their psychological correlates.

The study is a case-control study:

The studied groups consisted of a group of schizophrenic cases (n=100), a somatization control group (n=20) and a healthy control group (n =20).

The Symptom Check List (SCL-90R) was applied for the three groups to screen for somatization. SCL-90R is considered a trait marker for somatization disorder.

General health questionnaire had only been applied the healthy control to rule out the presence of any psychiatric disorder.

A number of questionnaires taping personality constructs found to be related to somatization ere applied:

- Toronto alexithymia scale.

- EPI.

- Symptom attribution scale.

The current study revealed the following results:

- 27 % of the schizophrenic group scored high on somatization subscale of Symptom Check List-90-R;

- The comparison of the three studied groups revealed no statistically significant differences in all socio-demographic data except the social class and marital status;

- The distinction between high and low somatizers is probably more useful and deliver more information since cases from the three studied groups even the healthy control group present with somatic complaints. Therefore the low somatizers are considered a subsyndrome of the somatisation disorder;

- The most common prevalent somatic symptoms in the schizophrenic group were GIT troubles, neurasthenia, feeling a lump in the throat, headache, backache and muscle pains;

- The application of Toronto Alexihymia scale revealed statistically significant differences between high and low somatizers in difficulty in identifying feelings, externally oriented thinking and the total score of global severity index.

The global severity index of schizophrenics is higher than that of both somatization and the control healthy groups and the differences are statistically significant.

Despite the total score of the global severity index of the somatization group is higher than that of the healthy control group, the difference is not statistically significant.

The current study showed that by using the EPI, somatization group had the highest score in extraversion and neuroticism compared to schizophrenic cases with high significant difference in between and healthy controls with highly significant difference in between by using one way ANOVA test.

The schizophrenic group was generally high attributers especially when compared to the healthy control. The highest attribution was for somatic and psychological causes more than external causes. Meanwhile, there was no statistically significant differences between the high and low somatizers.

Similarly, the somatization group was also high attributers when compared to the healthy group. The high somatizers were using psychological and external attribution; while the low somatizers were using somatic and psychological.

It becomes clear that somatization is not merely a separate diagnostic entity. Moreover, it may be prominent in many other psychiatric disorders or at least occurs during their course. It is not restricted to non-western societies or the minorities of western countries as was conceptualized. It may affect men and women. It has tremendous effects on the psycho-socio-biological aspects of individuals as well as an economic burden. It is not well understood but it affects important areas of the clinical practice like the decision of medical consultation, help-seeking pathways, communication with clinicians, course of illness and quality of life. Therefore, it is an area where light should be thrown to solve its enigma attribution styles. The differences in attribution

styles between the high and low somatizers were not statistically significant except for the external attribution.

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References

Acklin MW, Alexander G (1998): Alexithymia and somatization. A Rorschach study of four psychosomatic groups. J Nerv Ment Dis; 176:343-350.

American Psychiatric Association (1994): Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV). Washington, DC: American Psychiatric Association.

Andreski P, Chilcoat H, Breslau N (1998): Post-traumatic stress disorder and somatiztaion symptoms: a prospective study. Psychiatry Res; 79(2):131-138.

Araya, R; Rojas, G and Fritsch, R (2001): Common mental disorders in Santiago, Chile. Prevalence and socio-demographic correlates. British Journal Psychiatry, 178, 288-233.

Bach, M; Bach, D and De Zween, M (1996): Indepdences of alexithymia and somatization. A factor analysis study. Psychosomatics; 37:451-458.

Barsky AJ (1992): Amplification, somatization, and the somatoform disorders. Psychosomatics; 33:28-34.

Barsky AJ (1998): A comprehensive approach to the chronically somatizing patient. Journal of Psychosomatic Research; Vol. 45, No. 4, pp 301-306.

Barsky AJ, Borus JF (1995): Somatization and medicalization in the era of managed care. JAMA; 274:1931-1934.

Barsky AJ, Wyshak G, Latham KS, et al (1990): The prevalence of hypochondriasis in medical outpatients. Soc Psychiatry Psychiatr Epidemiol; 25:89-94.

Bass C, Murphy M (1995): Somatoform and personality disorders: syndromal comorbidity and overlapping developmental pathways. J Psychosom Res.; 39:403-427.

Bass C, Peveler R (2001): House A. Somatoform disorders: severe psychiatric illness neglected by psychiatrists. British Journal of Psychiatry; 179:1-14.

Bell IR, Miller CS, Schwartz GE (1992): An olfactory-limbic model of multiple chemical sensitivity syndrome: possible relationships to kindly and affective spectrum disorders. Biol Psychiatry; 32:218-42.

Bell IR, Szarek MJ, Dicenso DR, Baldwin CM, Schwartz GE, Bootzin RR (1999): Patterns of waking EEG spectral power in chemically intolerant individuals during repeated chemical exposures. Int J Neurosci; 97:41-59.

Benjamin S, Eminson DM (1992): Abnormal illness behaviour : Childhood experience and long-term consequences. Int Rev Psychiatry; 4:55-70.

Berry JM, Storandt M (1984): Coyne A: Age and sex differences in somatic complaints associated with depression. J Gerontol; 39:465-467.

Berthoz S, Artiges E, Van de Moortele PF, Poline J-B, Rouquette S, Consoli SM, and Martinot J-L (2002): Effect of impaired recognition and expression of emotions an frontocinulate cortices:an FMRI study of men with alexithymia. Am J Psychiatry; 159:961-7.

Bridges KW, Goldberg D, Evans B, Sharpe T (1991): Determinants of somatization in primary care. Psychol Med; 21:473-483.

Bridges KW, Goldberg DP (1985): Somatic presentation of DSM-III psychiatric disorders in primary care. J Psychosom Res; 29:563-569.

Brosschot JF (2002): Cognitive-emotional sensitization and somatic health complaints. Scand J Psychol; 43(2):113-21.

Brown, FW, Golding, JM and Smith, GR (1990): Psychiatric comorbidity in primary care somatization disorder. Psychosomatic Medicine, 52(4):445-51.

Carol A Wool, Arthur J Barsky (1994): Do woman somatize more than men? Gender Differences in somatization. Psychosomatics; 35:445-452.

Cassem NH, Barsky AJ (1991): Functional somatic symptoms and somatoform disorder. In Handbook of General Hospital Psychiatry. Cassem NH, ed. St. Louis: Mosby-Year Book, 1991:131-157.

Chen CAN (1993): Human brain measures of clinical pain: a review. II. Tomographic imagings. Pain; 54:133-144.

Cicero BA, Borod JC, Santschi C, Erhan HM, Obler LK, Agosti RM, Welkowitz J, and Grunwald IS (1999): Emotional versus nonemotional lexical perception in patients with right and left brain damage.Neuropstchiatry Neuropsychol Behav Neurol; 12:255-64.

Cloninger CR, Martin RL, Guze SB, et al (1986): A prospective follow-up and family study of somatiztion in men and women. Am J Psychiatry; 143:873-878.

Cloninger CR, Reich T, Guze S (1995): The multifactorial model of disease transmission: III. Familial relations between sociopathy and hysteria (Briquet's syndrome). Br J Psychiatry; 127:23-32.

Cohen K, Auld F, Brooker H (1994): Is alexithymia related to psychosomatic disorder and somatizing? J psychosom Res; 38:119- .

Craig AD (2003): Interoception:the sense of the physiological condition of the body. Curr opin Neurobiol; 13:500-5.

Craig TKJ, Boardman AP, Mills K, et al (1993): The South London Somatization Study: I. Longitudinal course and the influence of early life experiences. Br J Psychiatry; 163:579-588.

Craig TKJ, Drake H, Mills K, et al (1994): The South London Somatization Study II. Influence of stressful life events and secondary gain. Br J Psychiatry; 165:248-258.

De Waal MW, Amold IA, Eekhof JA, van Hemen AM (2004): Somatoform disorders in general practice. Prevalence, functional impairment and comorbidity with anxiety and depressive disorders. British Journal of Psychiatry; 184:470-476.

DeGacht, V and Heiser, W (2003): Alexithymia and somatization, A quantitative review of the literature. Journal of Psychosomatic Research: 59:425-434.

DeGrug F, Columbia L, Dickinson P (1987): Somatization disorder in a family practice. J Fam Pract; 25:45-51.

Derogatis (1992): SCL-90-R; Administration, scoring a procedure manual-II, Baltimore, MD: Clinical psychometric research.

Drossman, DA; Talley, MJ and Leserman, J (1995): Sexual and physical abuse and gastrointestinal illness. Review and recommendations. Ann Intern Med, 123:782-794.

Dumen, OY; Usubutun, S and Goxa, E (2004): The reliability and validity of the symptom interpretation questionnaire scale. Turkish J Psychiatry; 15-26.

Ebigbo PO (1982): Development of a culture specific (Nigeria) screening scale of somatic complaints indicating psychiatric disturbance. Cult Med Psychiatry; 6:29-43.

Elliott RL (1987): The masochistic patient in consultation-liaison psychiatry. Gen Hosp Psychiatry; 9:241-250.

El-Rufaie OEF, Al-Sabosy MMA, Bener A, et al (1999): Somatized mental disorders among primary care Arab patients: I. Prevalence and clinical and sociodemographic characteristics. J Psychosom Res; 46:549-555.

Emerson J, Pankratz L, Joos S, et al (1994): Personality disorders in problematic medical patients. Psychosomatics; 35:469-473.

Eriksen HR, Ursin H (2002): Sensitization and subjective health complaints. Scand J Psychol; 43:189-96.

Eriksen HR, Ursin H (2004): Subjective health complaints, sensitization, and sustained cognitive activation (stress). Department of Biological and Medical Psychology. Journal of Psychosomatic Research; 56:445-448.

Escobar JI (1995): Transcultural aspects of dissociative and somatoform disorders. Psychiatr Clin North Am; 18(3):555-569.

Escobar JI (1998): Immigration and mental health: why are immigrants better off? Arch Gen Psychiatry; 55(9):781-782.

Escobar JI, Burnham A, Karno M, et al (1987): Somatization in the community. Arch Gen Psychiatry; 44:713-718.

Escobar JI, Gara M, Silver RC et al (1998): Somatisation disorder in primary care. Br J Psychiatry; 173:262-266.

Escobar JI, Gomez J, Tuason VB (1983): Depressive phenomenology in North and South American patients. Am J Psychiatry; 140(1):47-51.

Escobar JI, Waitzkin H, Cohen Silver R, Gara M, Holman A (1998): Abridged somatisation: a study in primary care. Psychosom Med; 60:466-72.

Escobar JL, Rubio-Stipec M, Canino G, et al (1989): Somatic symptom Index (SSI): A new and abridged somatization construct. J Nerv Ment Dis; 177:140-146.

Escobar, JL; Burnam, A and Karno, M (1987): Somatization in the community. Arch Gen Psychiatry, 44:713-718.

Escolar JI, Waitzkin HN, Silver RC, et al (1998): Abridged somatization: a study in primary care. Psychosom Med; 60:466-472.

Fahrenberg J (1995): Somatic complaints in the German population. J Psychosom Res; 39:809-17.

Fink P (1990): Physical disorders associated with mental illness. A register investigation. Psychol Med; 20(4):829-34.

Fink P (1992): Surgery and medical treatment in persistent somatizing patients. Journal of Psychosomatic Research; 36:439-447.

Fink P (1995): Psychiatric illness in patients with persistent somatisation. British Journal of Psychiatry; 166:93-99.

Fink P (1996): Somatization – beyond symptom count [editorial]. Journal of Psychosomatic Research; 40:7-10.

Fink P (1997): Persistent somatization. Thesis, Faculty of Health Siciences, University of Aarbus, Denmark.

Fink P (2002): Somatization disorder and related disorders. In: Gelder MG, Lopez-Ibor JJ, Andreasen N, eds. New Oxford textbook of psychiatry. Oxford: Oxford University Press.

Fink P, Rosendal M, and Olesen F (2005): Australian and New Zealand Classification of somatization and functional so.matic symptoms in primary care. Journal of psychiatry; 39:772-781.

Fink P, Sorensen L Engberg M, Holm M, Munk-Jorgensen P (1999): Somatization in primary care. Psychosomatics; 40:330-8.

Fink P; Rosendal M; and Toft T (2002): Assessment and treatment of functional disorders in general practice. The extended reattribution and management model – an advanced educational program for nonpsychiatric doctors. Psychosomatics; 43:93-131.

Fink, P; Hansen, MS; and Sondergaard, L (2003): Mental illness in new neurological patients. J Neurol Neurosurg Psychiatry, 74:817-819.

Fischler B, D'Haenen H, Cluydts R et al. (1996): Comparison of 99m Tc HMPAO SPECT scan between chronic fatigue syndrome, major depression and healthy controls: an exploratory study of clinical correlates of regional cerebral blood flow. Neuropsychology; 34:175-183.

Francis Creed, Arthur Barsky (2004): A systematic review of the epidemiology of somatisation disorder and hypochondriasis. Journal of Psychosomatic Research; 56:391-408.

Francois Mai (2004): Somatization Disorder: A practical review. Can J Psychiatry, Vol 49, No 10.

Friedrich, RM and Florry, CB (1999): Half a million liberated from institutions to community setting without provision for long term care. Copyright, the treatment advocacy center originally appeared in catalyst, Vol 1, No 2.

Gelder M, Mayou R, Geddes J (1999): Psychiatry. 2nd ed. Oxford (UK): Oxford University Press; 1999. p 217-9.

George MS, Ketter TA, Parekh PI, Herscovitch P, and Post RM.(1996): Gender differences in regional cerebral blood flow during transient self induced sadness or happiness. Biol Psychiatry; 40:859-71.

Goddard GV (1987): Development of epileptic seizures through brain stimulation of low intensity. Nature; 214:1020.

Goldberg D, Huxley P (1992): Common Mental Disorders: A Bio-Social Model. London, Tavistock / Routledge.

Goldberg DP, Bridges K (1998): Somatic presentations of psychiatric illness in primary care setting. J Psychosom Res; 32:137-144.

Guez SB (1993): Genetics of Briquet's syndrome and somatization disorder. A review of family, adoption, and twin studies. Annals of Clinical Psychiatry; 5:225-230.

Gulec, H (2008): Normalizing attributions may contribute to non-help seeking behavior in people with fibromyalgia syndrome. Psychosomatics 49:212-217.

Gureje O, Simon GE, Ustun TB, and Goldberg DP (1997): Somatization in cross-cultural perspective; a World Health Organization study in primary care. Am J Psychiatry; 154(7):989-995.

Guze SB (1993): Genetics of Briquet's syndrome and somatization disorder. A review of family, adoption, and twin studies. Annals of Clinical Psychiatry; 5:225-230.

Hahn SR, Thompson KS, Wills TA, et al. (1994): The difficult doctor-patient relationship: somatization, personality and psychopathology. J Clin Epidemiol; 47:647-657.

Hathaway SR, McKinley JC, and Minneapolis, MN (1989): University of Minnesota Press.

Holman EA, Waitzkin H, and Cohen Silver R et al. (1996): Interethnic differences in somatization: a study in primary care. Paper presented a X World Congress of Psychiatry. Madrid, Spain; Aug, 26.

Hsu LKG and Folstein MF (1997): Somatoform disorders in Caucasian and Chinese Americans. J Nerv Ment Dis; 185:382-387.

James L, Gordon E, Kraiuhin C, Howson A, Meares R (1990): Augmentation of auditory evoked potentials in somatization disorder. Journal of Psychiatric Research; 24:155-163.

James L, Gordon E, Kraiuhin C, Meares R (1989): Selective attention and auditory event-related potentials in somatization disorder. Comprehensive Psychiatry; 30:84-89.

Janca, A; Burke, JD; Isaac, M (1995): Helath organization somatoform disorders schedule. A preliminary report on design and reliability. Eur J Psychiatry; 10:373-378.

Javier Garcia, Conception Sanz-Carrillo, Teresa Baringo, and Concepcion Ceballos (2001): SPECT scan in somatization disorder patients: an exploratory study of eleven cases. Australian and New Zealand Journal of Psychiatry; 35:359-363.

Javier J, Escobar (2004): Transcultural Aspects of Dissociative and Somatoform Disorders. Psychiatric Times; Vol XXI-5.

Jessimer M and Markham R (1997): Alexithymia: the interpretation of emotion relevant information. Cogn Emot; 8:231-44.

Kano M, Fukudo S, Gyoba J, Tagawa M, Mochizuki H, Itoh M, Hongo M and Yanai K (2003): Specific brain processing of facial expressions in people with alexithynia : An H2-150-PET study. Brain; 126:1474-84.

Karlson H, Naatanen P, and Stenman H (2008): Cortical Activation in Alexithymia As a Response to Emotional Stimuli, The Britich Journal of Psychiatry, 192, 32-38.doi:10.1192/bjp.106.034728.

Katon W, Ries RK, and Kleinman A (1984): The prevalence of somatization in primary care. Comprehensive Psychiatry; 25:208-215.

Katon WJ, and Walker EA (1993): The relationship of chronic fatigue to psychiatric illness in community, primary care and tertiary care samples. Cliba Foundation Symptosium; 173:193-204.

Kellner R (1990): Somatization theories and research. J Nerv Ment Dis; 178:150-60.

Kirmayer LJ (1987):. Languages of suffering and healing: Alexithymia as a social and cultural process. Transcult Psychiatr Res; 24:119-136.

Kirmayer LJ (1988): Mind and body as metaphors: Hidden values in biomedicine. In Lock M, Gordon D (eds), Biomedicine Examined. Dordrecht, Kluwer; 57-92.

Kirmayer LJ (1992): The body's insistence on meaning: Metaphor presentation and representation in illness experience. Med Anthropol Q; 6:323-346.

Kirmayer LJ (1994): Improvisation and authority in illness meaning. Cult Med Psychiatry; 18:183-214.

Kirmayer LJ, and Robbins JM (1991): Three forms of somatization in primary care. Prevalence, co-occurrence, and sociodemorgraphic characteristics. Journal of Nervous and Mental Disease; 179:647-655.

Kirmayer LJ, and Taillefer S (1997): Somatoform disorders. In: Adult psychopathology and diagnosis. Turner S, Hersen M, eds. Chickester: Wiley; 333-383.

Kirmayer, L; and Young, A (1998): Culture and somatization: Clinical, epidemiological and ethnographic perspectives. Psychosom Med; 60:420-430.

Kirmayer, LJ; Robbins, JM and Paris, J (1994): Somatoform disorders: Personality and the social matrix of somatic distress. J Abnormal Psychology, 103:125-136.

Kleinman A (1982): Neurasthenia and depression: a study of somatization and culture in China. Cult Med Psychiatry; 6(2):117-190.

Kleinman A (1987): Anthropology and psychiatry. The role of culture in cross-cultural research on illness. Br J Psychiatry; 151:447-454.

Komaroff AL (1990): "Minor" illness symptoms: the magnitude of their burden and of our ignorance. Arch Intern Med; 150:1586-1587.

Kooiman CG, Bolk JH, Brand R, and Harry GM (2000): Rooizmans: Psychosomatic Medicine; 62:768-778.

Kooiman, CG, Bolk, JH, Rooijmans, HG and Trijsburg, BW (2004): Alexithymia does not predict the persistence of medically unexplained physical symptoms. Psychosomatic Medicine; 66:224-232.

Kristal-Bonch E, Mclamed S, Kushnir T, Froom P, Harari G, and Ribak J (1998): Association between somatic symptoms and 24-hour ambulatory blood pressure levels. Psychosom Med; 60:616-9.

Kroenke K (2003): Patient presenting with somatic complaints: epidemiology, psychiatric comorbidity and management. Int J Methods Psychiatr Res; 12:34-43.

Kroenke K, and Price RK (1993): Symptoms in the community. Prevalence, classification, and psychiatric comorbidity. Arch Intern Med; 153:2474-80.

Kroenke K, and Swindle R (2000): Cognitive-behavioural therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. Psychotherapy and Psychosomatics; 69:205-215.

Kroenke K, Spitzer RL, deGruy FV, Hahn SR, Linzer M, Williams JBW, Brody D, and Davies M (1997): Multisomatoform disorder. An alternative to undifferentiated somatoform disorder for the somatizating patient in primary care. Arch Gen Psychiatry; 54:352-8.

Kroenke, K; Robert L and Spitzer R (1998): Gender differences in the reporting of physical and somatoform symptoms. Psychosomatic Medicine; 60:150-155.

Lane RD, Ahern GL, Shwartz GE and Kaszniak AW (1997): Is alexithymia the emotional equivalent of blindsight? Biol Psychiatry; 42:834-44.

Lane RD, Reiman EM, Ahern GL, Schwartz GE, Davidson RJ, Friston KJ, Yun LS, and Chen K (1997): Neuroanatomical correlates of externally and internally generated human emotions . Am J Psychiatry; 154:918-25.

Lane RD, Reiman EM, Axelord B, Yun L-S, Holmes A, and Schwartz GE (1998): Neural correlates of levels of emotional awareness: evidence of an interaction between emotion and attention in the anterior cingulated cortex. J Cogn Neurosci; 10:525-35.

Laurence J Kirmayer, James M Robbins, Michael Dworkind, and Mark J Yaffe (1993): Somatization and the recognition of depression and anxiety in primary care. Aj J Psychiatry; 150:734-741.

Laurence J, Kirmayer, and Allan Young (1998): Culture and somatization: Clinical, epidemiological, and ethnographic perspective. Psychosomatic Medicine; 60:420-430.

Lennartsson J, Bengtsson C, and Hallberg L et al. (1979): Characteristics of anaemic women. The population study of women in Goteborg 1968-1969. Scandinavian Journal of Haematology; 22:17-24.

Lieb R, Zimmermann P, Friis RH, Hofler M, Tholen S, and Wittchen HU (2002): The natural course of DSM-IV somatoform disorders and syndromes among adolescents and young adults: a prospective-longitudinal community study. Eur Psychiatry; 17:321-31.

Lilienfeld SO (1992): The association between antisocial personality and somatization disorder: a review and integration of theoretical models. Clin Psychol Rev; 12:641-662.

Lilienfeld SO, Van Valkenburg C, and Larntz K. et al. (1986): The relationship of histrionic personality disorder to antisocial personality and so-matization disorder. Am J Psychiatry; 143:718-722.

Lin EHB, Katon W, and Von Korff M, et al. (1991): Frustrating patients: physician and patient perspectives among distressed high users of medical services. J Gen Intern Med; 6:241-246.

Linzer, M; Spitzer, M and Kroenke, K (1996): Gender, Quality of life and mental disorders in primary care: Results from the Prime-MD 1000 study. Am J Med, 101:526-533.

Lipowski ZJ (1988): Somatization: The concept and its clinical application. Am J Psychiatry; 145:1358-1368.

Lipowski, ZJ (1987): Somatization: the experience and communication of psychological distress as somatic symptoms. Psychother Psychom, 47:160-167.

Lipowski, ZJ (1990): Somatization and depression. Psychosomatics; 31:13-21.

Lobo A, Garcia-Campayo S, Campos R, et al. (1996): Somatization in primary care in Spain: I. Estimates of prevalence and clinical characteristics. Br J Psychiatry; 168:344-348.

Lock M (1993): Cultivating the body: Anthropology and epistemology of bodily practice and knowledge. Annu Rev Anthropol; 22:130-135.

Lumley MA, and Norman S (1996): Alexithymia and health care utilization. Psychosom Med; 58:197-202.

Lundh, L and Wangby, M (2002): Causal thinking about somatic symptoms – how is it related to the experience of symptoms and negative affect cognitive therapy and research, Vol 26, No 6, 701-717.

Mandal MK, Borod JC, Asthana HS, Mohanty A, Mohanty S, and Koff E (1999): ffecs of lesion variables and emotion type on the perception of facial emotion.

Margo, LL and Margo, GM (1999): The problem of somatization in familty practice. American Family Physician, 1873-1879.

Marin C, and Carron R (2002): The origin of the concept of somatization. Psychosomatics 2002; 43:249-50.

Markus HR, and Kitayama S (1991): Culture and the self: Implications for cognition, emotion, and motivation. Psychol Rev; 98:224-253.

Mayou R (1976): The nature of bodily symptoms. British Journal of Psychiatry; 129:55-60.

Mayou R (2000): Somatoform disorders and medically unexplained symptoms. In: Gelder MG. Lopez-Ibor JJ, Andreasen N, eds. New Oxford textbook of psychiatry. Oxford: Oxford University Press.

Mayou R, and Hawton K (1986): Psychiatric disorder in the general hospital. Br J Psychiatry; 149:172-90.

McCahill ME (1995): Focus on the somatoform disorders. Hospital Practice; 30(2):59-66.

McCauley J, Kern DE, Kolodner K, Dill L, Schroeder AF, and Dechant HK; et al. (1997): Clinical characteristics of women with a history of childhood abuse. JAMA; 1362-1368.

McGaphie WC, and Whitenack DC (1982): A scale for measurement of the problem patient labeling process. J Nerv Ment Dis; 170:598-604.

Meares R, Stevenson J, and Gordon E (1999): A Jacksonian and biopsychosocial hypothesis concerning borderline and related phenomena. Australian and New Zealand Journal of Psychiatry; 33:831-840.

Mechanic D (1972): The concept of illness behavior. J Chronic Dis; 15:189-94.

Meyer BU, Britton TC, Beneeke R, Bischoff C, Machetanz J. and Conrad B (1992): Motor responses evoked by magnetic brain stimulation in psychogenic limb weakness. J Neurol; 239:251-5.

Michael Bach, Doris Bach, Martina De Zwaan (1996): Independency of alexithymia and somatization: A factor analytic study. Psychosomatics; 37:451-458.

Miller L (1984): Neuropsychological concepts of somatoform disorders. International Journal of Psychiatry in Medicine; 14:31-46.

Miller, MC and Knapp, RG (1992): Clinical epid emiology and biostatistics, published by Williams & Wilkins, Maryland, 3rd, ed.

Moore DP, and Jefferson JW (1996): Handbook of Medical Psychiatry. St. Louis: Mosby-Year Book, 198-200.

Morrison J (1989): Histrionic personality disorders in women with so-matization disorder. Psychosomatics; 30:433-437.

Mumford DB (1989): Somatic sensations and psychological distress among students in Britain and Pakistan. Soc Psychiatry Psychiatr Epidemiol; 24:321-326.

Mumford DB, Bavington JT, and Bhatnagar KS, et al. (1991): The Bradford Somatic Inventory: A multi-ethnic inventory of somatic symptoms reported by anxious and depressed patients in Britain and the Indo-Pakistan subcontinent. Br J Psychiatry; 158:379-386.

Mumford DB, Taren IAK, and Bajwa MAZ, et al. (1991): An investigation of "functional" somatic symptoms among patients attending medical clinicals in Pakistan. I. Characteristics of "non-organic" patients. J Psychosom Res; 35:245-255.

Murphy MR (1990): Classification of the somatoform disorders. In: Bass C, ed. Somatization. Physical symptoms and psychological illness. Oxford: Blackwell Scientific Publications.

Nichter M (1982): Idioms of distress: Alternatives in the expression of psychosocial distress. Cult Med Psychiatry; 5:379-408.

Niemi PM, Portin R, Aalto S, Hakala M, and Karlsson H (2002): Cognitive functioning in severe somatization a pilot study. Acta Psychiatr Scand; 106:461-3.

Nimnuan C, Rabe-Hesketh S, Wessely S, and Hotopf M (2001): How many functional somatic syndromes? J Psychosom Res; 51:549-57.

Noyes, R; Langbehn, Dr; Happel, RL; Stout, LR; Muller, BA and Longley, SL (2001): Personality dysfunction among somatizing patients. Psychosomatics; 42:320-329.

Ohaeri JU and Odejide OA (1994): Somatization symptoms among patients using primary health care facilities in a rural community in Nigeria. Am J Psychiatry; 151:728-731.

Omer EF El-Rufaie, Moza AA Al Sabosy, Abdulbari Bener and Mohamed SO Abuzeid (1999): Somatized mental disorder among primary care Arab patients: I. Prevalence and clinical and sociodemographic characteristics. Journal of Psychosomatic Research; Vol. 46, No. 6, pp 549-555.

Othmer E and DeSouza C (1985): A screening test for somatization disorder (hysteria). Am J Psychiatry; 142:1146-1149.

Paker JDA, Taylor GJ, and Bagby RM (1992): Relationship between conjugate lateral eye movements and alexithymia . Psychother Pychosom; 7:94-101.

Parker, J; Taylor, G and Bagby, M (1993): Alexithymia and the processing of emotional stimuli an experimental study. New Trends Exp Clin Psychiatry, 9; 9-14.

Patel V, Kirkwood BR, and Weiss H et al. (2005): Chronic fatigue in developing countries: population based survey of women in India; 330:1190-1193.

Payer L (1990): Borderline cases: how medical practice reflects national culture. The Sciences; 30(4):38-42.

Pennebaker JW and Watson D (1991): The psychology of somatic symptoms, in Current Concepts of Somatization: Research and Clinical Perspectives, edited by Kirmayer LJ, Robbins JM. Washington, DC, American Psychiatric Press, pp. 21-35.

Petrie K and Weinman J (1997): Perceptions of health and illness. Amsterdam: Hawrood Academic Publishers.

Phan KL, Taylor SF, and Liberzon I (2002): Functional neuroanatomy of emotion: a meta-analysis of emotion activation studies in PET and FMRI. Neuroimage; 16:331-48.

Phillips, K (2001): Somatoform and factitious disorder, review of psychiatry. Vol 20, No 3, American Psychiatry Publishing, Inc-Washington-DC, London, England.

Pilowsky I and Barsky AJ (1986): Abnormal illness behavior (dysnosognosia). Psychother Psychosom; 46:76-84.

Rief W and Hiller W (1999):. Toward empirically based criteria for somatoform disorders. J Psychosom Res; 46:507-18.

Rief W, Heuser J, Mayrhuber E, Stelzer I, Hiller W, and Fichter MM (1996): The classification of multiple somatoform symptoms. Journal of Nervous and Mental Disease; 184:680-687.

Rief W, Shaw R and Fichter M (1998):. Elevated Levels of physiologic arousal and cortisol in patients with somatization syndrome. Psychosom Med; 60:198-203.

Robbins, JM and Kirmayer, LJ (1991): Attributions of common somatic symptoms. Psychol med, 1; 21:1029-45.

Roberts AD, Wessely S, Chalder T, Papadopoulos A, and Cleare AJ (2004): Salivary cortisol response to awakening in chronic fatigue syndrome. British Journal of Psychiatry; 184:136-141.

Robins E, Purtell JJ, and Cohen ME (1952): "Hysteria" in men. N Engl J Med; 246:677-685.

Robins LN and Regier D (1991): Psychiatric Disorders ind America: The Epidemiologic Catchment Area Study. New York, Free Press.

Rosendal M, Bro F, Fink P, Christensen KS, and Olesen F (2003): Diagnosis of somatisation: effect of an educational intervention in a cluster randomized controlled trial. British Journal of General Practice; 53:917-922.

Rosendal M, Olesen F, and Pink P (2005): Management of medically unexplained symptoms (editorial). British Medical Journal; 330:4-5.

Rost KM, Akins RN, and Brown FN, et al. (1992): The comorbidity of DSM-III-R personality disorders in somatization disorders. Gen Hosp Psychiatry; 14-322-326.

Russo, J; Katon, W and Sullivan, M (1994): Severity of somatiztion and its relationship to psychiatric disorders and personality. Psychosomatics, 35:546-556.

Rygh LJ, Tjolsen A, Hole K, and Svendsen F (2002): Cellular memory in spinal nociceptive circuitry. Scand J Psychol; 43(2):153-9.

Saxe GN, Chinman G, and Berkowitz R et al. (1994): Somatization in patients with dissociative disorders. Am J Psychiatry; 151(9):1329-1334.

Schappert SM (1992): National Ambulatory Medical Care Survey: 1989 summary. Vital Health Stat 13; 110:1-80.

Schmidt AJ, Wolfs Takens DJ, Oosterlaan J, and van den Hout MA (1994): Psychological mechanisms in hypochondriasis: attention-induced physical symptoms without sensory simulation. Psychotherapy and Psychosomatics; 16:117-120.

Schulberg HC and Bruns BJ (1988): Mental disorders in primary care: epidemiologic, diagnostic, and treatment research directions. Gen Hosp Psychiatry; 10:79-87.

Schwartz RB, Garada BM, and Komaroff AL et al. (1994): Detection of intracranial abnormalities in patients with chronic fatigue syndrome: comparison of MR imaging and SPECT. American Journal of Roentgenology; 162:935-941.

Sethi B (1973): Depression in India. J Soc Psychol; 91:3-13.

Sharpe M and Mayou R (2004): Somatoform disorders: a help or hindrance to good patient care? British Journal of Psychiatry: 184:465-467.

Shelder J, Mayman M and Manis M (1993): The illusion of mental health. Am Psychologist; 48:1117-1131.

Shields J (1982): Genetic studies of hysterical disorders. In: Roy A, editor. Hysteria. New York: John Wiley; P 41-56.

Shorter E (1992): From paralysis to fatigue. A history of psychosomatic illness in the modern era. New York: Fee/Macmillan.

Shorter E. From the mind into the body: The cultural Origins of Psychosomatic Symptoms. 1994; New York: Free Press.

Sigvardsson S, Von Knorring AL, Bohman M, and Cloninger CR (1984): An adoption study of somatoform disorders. I. The relationship of somatization to psychiatric disability. Arch Gen Psychiatry; 41:853-9.

Simon G and Von Korff M (1991): Somatization and psychiatric disorders in the NIHM Epidemiological Catchment Area (ECA) Study. Am J Psychiatry; 48:1494-500.

Smith, GR (1995): Somatization disorder and undifferentiated somatoform disorder. In Gabbard GO (ed), Treatment, of psychiatric disorders, 2nd Ed., Washington DC, American Psychiatric Press, 1718-1733.

Spiegel, B; Kanwal, F, Naliboff, B and Mayer, E (2005): The impact of somatization on the use of gastrointestinal health care resources in patients with irritable bowel syndrome. Am J Gastroenterol; 100(10):2262-2273.

Starcevic (1990): Relationship between hypochondriasis and obsessive compulsive personality disorder: Close relatives separated by nosological schemes? Am J Psychother; 44:340-347.

Staud R and Smitherman ML (2002): Peripheral and central sensitization in fibromyalgia: pathogenic role. Curr Pain Headache; Rep 6:259-66.

Stern J, Murphy M and Bass C (1993):. Attitudes of British psychiatrists to the diagnosis of somatization disorder: a questionnaire survey. Br J Psychiatry; 162:463-466.

Swartz, M; Haghes, D and Georges, L et al. (1986): Developing a screening index for community studies of somatization disorder. J Psychiatry Res; 20:335-343.

Swartz, M; Landerman, R and George LK (1991): Somatization disorder in psychiatric disorders in America, Edited by Robins LN, Ragier, DA. New York, Free Press, 220-257.

Taylor G (1984): Alexithymia: Concept, measurement, and implicat for treatment. Am J Psychiatry; 141:725-732.

Taylor GJ, Bagby RM, Parker JDA (1997): Disorders of affect regulation: alexithymia in medical and psychiatric illness. Cambridge: Cambridge University Press.

Taylor GJ, Bagby RM, Ryan DP, et al. (1990): Validation of the alexithymia construct: a measurement-based approach. Can J Psychiatry; 35:290-296.

Torgersen S (1986): Genetics of somatoform disorders. Arch Gen Psychiatry; 43:502-5.

Trull TJ (1992): DSM-III-R personality disorders and the five-factor model of personality: an empirical comparison. J Abnorm Psychol; 101:553-560.

Tyrer P, Gunderson J and Lyons M, et al. (1997): Extent of comorbidity between mental state and personality disorders. J Personal Disord; 11:242-259.

Ursin H and Eriksen HR (2001): Sensitization, subjective health complaints, and sustained arousal. In: Sorg B, Bell I, editors. The role of neural plasticity in chemical intolerance. Ann NY Acad Sci; 933:119-29.

Ustun TB and Sartorius N (1995): Mental Illness in General Health Care: An International Study. Chichester, John Wiley.

Van den Bergh O, Winters W, Devriese S, and Van Diest I (2002): Learning subjective health complaints. Scand J Psychol; 43(2):147-52.

Van Dulmen AM, Fennis JF, Mokkink HC, and Blejenberg C (1996): The relationship between complaint-related cognitions in referred patients with irritable bowel syndrome and subsequent health care seeking behaviour in primary care. Fam Pract; 13:12-17.

Wager TD, Phan KL, Liberzon I and Taylor SF (2003): Valence, gender, and lateralization of functional brain anatomy in emotion: a meta-analysis of findings from neuroimaging. Neuroimage; 19:513-31

Walker EA, Katon WJ, Keegan D, et al. (1997): Predictors of physician frustration in the care of patients with rheumatological complaints. Gen Hosp Psychiatry; 9:315-323.

Weich S, Lewis G, Mann A (1996): Effect of early life experiences and personality on the reporting of psychosocial distress in general practice: a preliminary investigation. Br J Psychiatry; 168:116-120.

Wessely S, Nimnuan C, Sharpe M (1999): Functional somatic syndromes: one or many? Lancet; 354:936-939.

Whitehead WE, Crowell MD, Heller BR, Robinson JC, Schuster MM, Horn S (1994): Modeling and reinforcement of the sick role during childhood predicts adult illness behavior. Psychosom Med; 56:541-550.

Whitehead, WE; Palsson, O, and Jones, KR (2002): Systematic review of the comorbidity of irritable bowel syndrome with other disorders. What are the causes and implications. Gastroenterology, 122:1140-56.

WHO (1992): The ICD-10 classification of Mental and Behavioral Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva World Health Organization.

Wilhelmsen I (2002): Somatization and functional dyspepsia. Scand J Psychol; 43:177-80.

Winfried Rief, Aike Hessel, Elmar Braehler (2001): Somatization Symptoms and Hypochondriacal Features in the General Population. Psychosomatic Medicine; 63:595-602.

Wool, CA and Barsky, AJ (1994): Do woman somatize more than men? Gender differences in somatization. Psychosomatics; 35:445-452.

World Health Organization (1992): The ICD-10 Classification of Mental and Behavioral Disorders: Clinical Descriptions and Diagnostic Guidelines, Genera: World Health Organization; P 161.

Yazici KM, Kostakoglu L (1998): Cerebral blood flow changes in patients with conversion disorder. Psychiatry Research; 83:163-168.

Internet References:

Bridges KW, Goldberg DP, Evans B, et al (1991): Determinants of somatization in primary care. Psychol Med; 21:473-483.[Medline]

Escobar JI, Rubio-Stipec M, Canino G, et al (1989): Somatic Symptom Index (SSI): a new and abridged somatization construct. Prevalence and epidemiological correlates in two large community samples. J Nerv Ment Dis; 177:140-146.[Medline]

Helzer JE, Robins LN, McEvoy LT, et al (1985): A comparison of clinical and diagnostic interview schedule diagnoses. Arch Gen Psychiatry; 42:657-666.[Abstract]

Kellner R (1990): Somatization: theories and research. J Nerv Ment Dis; 178:150-160.[Medline]

Kirmayer LJ, Robbins JM (1991): Three forms of somatization in primary care: prevalence, co-occurrence, and sociodemographic characteristics. J Nerv Ment Dis; 179:647-655.[Medline]

Lipowski ZJ (1968): Review of consultation psychiatry and psychosomatic medicine. Psychosom Med; 30:395-422.[Medline]

Lipowski ZJ (1987): Somatization: medicine's unsolved problem. Psychosomatics; 28:294-297.[Medline]

Lipowski ZJ (1988): Somatization: the concept and its clinical application. Am J Psychiatry; 145:1358-1368.[Abstract]

Mayou R (1993): Somatization. Psychother Psychosom; 59:69-83.[Medline]

الملخص العربى

تمثل الجسدنة التعبير عن التوتر النفسى عن طريق أعراض جسدية. وعلى الرغم من أن هذه الأعراض ليس لها مصدر طبى بيولوجى يفسرها إلا أن ذلك لا يعنى أن مرضى اضطراب الجسدنة لا يعانون شدة وأنهم لا يلجئون إلى الأطباء بإلحاح للبحث عن التخلص من هذه الأعراض.

وقد أثبتت الدراست الانتشارية كثرة انتشار هذه الأعراض بين عموم الناس وفى كل الأوساط الطبية. ولا شك أن هناك العديد من العوامل التى تمت دراستها لفهم ظاهرة الجسدنة، ويشمل ذلك عدداً من سمات الشخصية منها عدم القدرة عن التعبير عن العواطف.

وهناك ميل لاعتبار الجسدنة أكثر حدوثا بين النساء عن الرجال. وفى الثقافات الغير غربية عن الثقافات الغربية. وعموما فإن المرضى يحاولون بصورة حثيثة الحصول على رعاية طبية ويأخذون العديد من العلاجات الدوائية ويخضعون للعديد من الفحوصات أو العمليات الجراحية بدون أى داعى.

ويهدف هذا البحث لمعرفة نسبة حدوث الجسدنة بين مرضى الفصام ودراسة المتعلقات النفسية للجسدنة.

ويعد هذا البحث من نوعية مقارنة الحالات بعينة ضابطة، ولهذا الغرض تم اختيار مائة مريض يعانون من اضطراب الفصام وعشرين مريضا باضطراب الجسدنة وعشرون شخصا من الأصحاء لا يعانون من أى مرض عضوى أو نفسى. وبعد إجراء فحص كامل للمرضى والتشخيص تم تطبيق قائمة الأعراض المعدلة حيث أنها تبين الأعراض الجسمانية كسمة من سمات الشخصية.

كما تم تطبيق عدد آخر من الاختبارات توضح تركيبات الشخصية المختلفة، وهذه الاختبارات هى:-
- اختبار تورنتو لقياس عدم القدرة على التعبير الوظيفى عن العواطف.
- اختبار ايزنك للشخصية.
- اختبار تفسير ظهور الأعراض.

وقد أظهرت الدراسة النتائج التالية:-
- نسبة إصابة مجموعة الفصام بالجسدنة 27% وذلك على مقياس الأعراض الجسمية لقائمة الأعراض المعدلة.
- لم تظهر مقارنة المجموعات الثلاثة أى فروق ذات دلالة إحصائية فى المعطيات الاجتماعية والديموجرافية باستثناء المستوى الاجتماعى والحالة الزواجية.

- يعد تقسيم الحالات إلى جسدنة مرتفعة وأخرى منخفضة أفضل من تقسيمها إلى جسدنة موجودة أو غير موجودة حيث أن المعلومات المتاحة أكثر، وحيث أن كل المجموعات الثلاثة تعانى من أعراض جسمانية حتى الأصحاء، وهؤلاء المفترض أن تكون أعراض الجسدنة غير موجودة بها.
ولذلك ربما يكون من الأفضل اعتبار ذوى الجسدنة المنخفضة حالات جسدنة، ولكن بدرجات منخفضة عن عتبة المتلازمة الأصلية.
- أهم الأعراض الجسمانية فى مجموعة الفصام: اضطرابات الجهاز الهضمى – الإعياء – الإحساس بوجود ورم فى الحلق – الصداع – آلام الظهر والعضلات.
- تطبيق اختبار تورنتو لقياس عدم القدرة على التعبير اللفظى عن العواطف ظهرت فروقات ذات دلالة إحصائية بين المجموعة ذات الجسدنة المرتفعة والأخرى ذات الجسدنة المنخفضة وذلك فى صعوبة التعرف على العواطف والتفكير الموجه إلى الخارج والنتيجة الإجمالية لمعيار الشدة الكلية.

كما كانت النتيجة الإجمالية لمعيار الشدة لمجموعة الفصام أعلى من كل من مجموعة الجسدنة ومجموعة الأصحاء وبدلالة إحصائية.

ورغم أن هذا المعيار كان أعلى فى مجموعة الجسدنة عن مجموعة الأصحاء إلا أن الفرق ليس له دلالة إحصائية.

- أظهرت نتائج اختبار إيزنك للشخصية أن مجموعة الجسدنة درجاتها أعلى فى الانبساطية والعصائية من كل من مجموعة الفصام ومجموعة الأصحاء.
- وضح أن مجموعة الفصام تميل إلى إيجاد تفسيرات للأعراض الجسدية خاصة عند مقارنتها بالأصحاء. وكان الفصاميين ذوى الجسدنة المرتفعة أميل للتفسير السيكولوجى والخارجى بينما كان ذوى الجسدنة المنخفضة أميل للتفسير الجسدى والسيكولوجى.

ولاشك أنه قد أصبح واضحاً أن الجسدنة ليست تشخيصاً قائماً بذاته فقط، وإنما هى موجودة بصورة واضحة فى اضطرابات نفسية أخرى أو على الأقل تظهر خلال مسار المرض. كما أنها ليست موجودة فقط فى المجتمعات غير الغربية أو فى الأقليات فى البلدان الغربية كما كان يُعتقد. وهى تصيب الرجال والنساء على السواء. ولا شك أن لها تأثيرات واضحة على الجوانب النفسية والاجتماعية والبيولوجية للأشخاص بالإضافة إلى العبء الاقتصادى وهى ظاهرة غير مفهومة بوضوح، لكن المؤكد أنها تؤثر على جوانب هامة من الممارسة الإكلينيكية كطلب المشورة الطبية ومسارات طلب المساعدة والعلاقات العلاجية مع الأطباء ومسار المرض ونوعية الحياة. ولذلك فإنها تستحق منا الاهتمام حتى يتم حل ألغازها.

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Review of Literature

Concept of Somatization:

The concept of somatization was first used by Stekel, who defined it as a bodily disorder arising as the expression of a deep-seated neurosis.

The definition put forward by ***Lipowski (1968)*** described somatization as "the tendency to experience, conceptualize and/or communicate psychological states or contents as bodily sensations, functional changes or somatic metaphors." Nineteen years later, he changed this definition into "a tendency to experience and communicate psychological distress in the form of somatic symptoms and to seek medical help for them." ***(Lipowski, 1987)***. This definition suggested the existence of a causal relation between the experience of psychological distress and the presentation of somatic symptoms.

The operational criteria for somatization developed by Bridges and ***Goldberg (1985)*** conceptualized somatization as a somatic expression of an Axis I psychiatric disorder.

Somatization as Somatic Distress:

A whole range of definitions of somatization are characterized by the assumption that the presentation of somatic symptoms is the hallmark of somatization, to the exclusion of assumptions about causality ***(Fishler et al., 2002)***.

Lipowski (1988) adopted a more descriptive definition of somatization. In particular, he defined somatization as "the tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help for

them." In contrast to his earlier definitions, he proceeded carefully, stating that "it is usually assumed that this tendency (to somatize) becomes manifest in response to psychosocial stress."

Within this conceptualization of somatization, three progressively more restrictive orientations can be distinguished ***(Fishler et al., 2002)***.

The first of these, equating somatization with simple symptom counting, can be defined as somatization in the broadest sense ***(Kellner, 1990)***. Because of the lack of clinical specificity of this concept, ***Mayou (1993)*** proposed to use a more general term, such as nonorganic physical symptoms or medically unexplained symptoms, for this phenomenon.

The second orientation, introducing a criterion of severity (e.g., the chronic or recurrent character of the symptoms, the number of symptom groups involved, the degree of associated disability, and medical consumption), can be illustrated by the definition of the DSM-III, DSM-III-R, DSM-IV, ICD-10, the Somatic Symptom Index (SSI), and the multisomatoform disorder ***(De Gucht and Fischler, 2002)***.

Finally, the third orientation reintroduces a theoretical dimension because it considers specific cognitive and/or behavioral characteristics as a necessary part of somatization. The definitions of Lipowski, are representative of this orientation.

Presenting and Functional Somatization:

Within primary care, ***Kirmayer and Robbins (1991)*** studied the aforementioned somatization concepts as two separate clinical phenomena, termed " *presenting somatization* " and "*functional somatization".*

Whereas presenting somatization was defined as "the predominantly or exclusively somatic presentation of psychiatric disorder, most commonly

depression and anxiety," functional somatization referred to "high levels of medically unexplained symptom reporting in multiple physiological systems."

Kirmayer and Robbins (1991), have demonstrated that presenting and functional somatization tend to identify two distinct groups of patients, with some degree of overlap between them.

Presenting and Functional Somatization: Conceptual And Methodological Issues:

Presenting Somatization:

Conceptual Developments:

In the primary care studies of ***Bridges and Goldberg (1985)*** and ***Bridges et al. (1991)***, patients were identified as somatizers if:

1) They presented only with somatic symptoms to their primary care physician,

2) They attributed these symptoms to a physical problem, and

3) They were subsequently diagnosed by a research psychiatrist with a psychiatric disorder.

From a conceptual point of view, it is important to emphasize that a causal relation was assumed between the somatic symptoms the patients presented with and the psychiatric disorder subsequently diagnosed.

In addition, treatment of the psychiatric disorder was expected to reduce or eliminate the somatic symptoms ***(Fischler et al., 2002)***.

When defining presenting somatization, ***Kirmayer and Robbins (1991)*** introduced two important modifications to their operational criteria.

First of all, they considered the connection between the somatic symptoms reported by the patient and the psychiatric disorder subsequently diagnosed as a research question, not an a priori fact. In other words, the existence of a causal relation was not considered self-evident.

Second, they emphasized the difficulty of putting into practice the assumption that in treating the underlying psychiatric disorder, the somatic symptoms would disappear. Leaving out these two characteristics, the criteria of Kirmayer and Robbins for presenting somatization required that patients ***(De Gucht and Fischler, 2002)***.

1) reach the diagnostic threshold for major depression or an anxiety disorder,
2) present to their physician only with non-organic physical symptoms,
3) make only somatic attributions, and
4) reject psychosocial attributions for their symptoms.

These criteria were intended to cover three increasingly persistent forms ***(Fishler et al., 2002)***.

1- presenting somatization, with "true somatizers" satisfying all four criteria,
2- "facultative somatizers" satisfying the first three criteria, and
3- "initial somatizers" satisfying only the first and second criteria.

Methodological Issues:

Two methodological inadequacies are clearly identified by ***De Gucht and Fischler (2002)*** in the studies by Bridges and ***Goldberg (1985) and Bridges et al. (1991)*** namely:

1) no objective medical information was consulted to exclude organic causes for presenting symptoms, and

2) the assumption of a causal relation between somatic symptoms and psychiatric disorders was based exclusively on the subjective judgment of the research psychiatrist, without putting this assumption to the test.

On the basis of the South London Somatisation Study, a 2-year follow-up study in primary care, ***Craig et al. (1993)*** succeeded in solving these methodological flaws. First of all, the judgment of whether a symptom was medically explained or not was based on objective medical information.

Second, to avoid the pitfall of subjective judgment, two independent raters measured the presence of psychiatric disorder and the nature of the somatic symptoms (medically unexplained versus medically explained).

In addition, ***Craig et al. (1993)*** tried to test the assumption that the somatic symptoms the patient presented with were indeed a manifestation of the psychiatric disorder that was diagnosed. They demonstrated, both in the index period and at follow-up, that there was a temporal association between changes in the severity of the psychiatric disorder and the severity of somatic symptoms. No relation was found between specific symptoms and particular psychiatric disorders.

Functional Somatization:

Conceptual Developments:

The major conceptualizations of functional somatization are SD as defined in DSM-III, DSM-III-R, DSM-IV, and ICD-10.

Table (1) presents a description of the defining characteristics of each of these concepts. To highlight the differences and similarities between them, each of the concepts is described according to the same dimensions, namely:

1) the number of medically unexplained somatic symptoms required (symptom cutoff);

2) the duration of the symptoms, representing a continuum from current to lifetime;

3) the presence or absence of criteria of severity, such as interference with life and medical consumption;

4) the number of symptom groups that are required; and

5) the presence or absence of specific psychological characteristics. (DeGucht and Fischler, 2002).

One of the two characteristics indicated by "+" is required.

Methodological Issues *(De Gucht and Fischler, 2002)*

(1) Validity of Symptom Cutoffs:

In introducing the diagnostic entity of SD in the research criteria of the ICD-10, WHO has followed the American lead. It remains unclear, however, on what basis the symptom cutoff for a diagnosis of SD has been selected. The DSM-IV field trial revealed a poor agreement between ICD-10 and the different DSM criteria

The cutoff of three symptoms to define multisomatoform disorder was selected primarily because of its predictive validity with respect to disability. Accordingly, a cutoff of seven symptoms has been selected to define the polysymptomatic

Somatoform disorder because of its discriminative power with respect to disability.

(2) Assessment:

Escobar (1987) studied the phenomenon of somatization primarily within the framework of the Epidemiological Catchment Area (ECA) study. The major problem with the ECA study, as with most epidemiologic studies on SD, is that the structured psychiatric interviews used to assess SD and the SSI were conducted by lay people. These lay interviewers could rely only on the subjective report of the study subjects and may have had difficulty distinguishing between symptoms that were due to organic causes and those that were not.

Comparing diagnoses based on lay interviews with diagnoses made by psychiatrists, both using the same structured diagnostic interview. ***Helzer et al. (1985)*** found only moderate concordance between the approaches. In the same line, a psychiatrist additionally checked each of the medically explained

symptoms to decide whether the medical explanation given by the patient was a plausible one. He did not have access to the medical records of the study subjects, however, and was thus unable to check the symptoms reported by the subject against objective medical information ***(Escolar et al., 1989)***.

Somatization disorder: Clinical presentation.

Somatization disorder also called Briquet's syndrome is a distinct clinical and epidemiological condition that lies in the borderland between clinical medicine and psychiatry ***(Escobar et al., 1998)***. Patients with somatization disorder usually present with numerous symptoms such as headaches, back pain , persistent lack of sleep, stomach upset and chronic tiredness, all without demonstrable medical causes ***(McCahill, 1995)***.

Patients with somatization disorder have a persistent conviction of being ill, despite repeated negative results on laboratory tests, diagnostic tests, consultations with specialists and recurrent hospitalizations.

Moreover they continue to seek medical care, take several medications and submit to needless diagnostic and surgical procedures. By the time these patients reach middle age ,they may have undergone an average of 10 operations and acquired several volumes of medical records ***(Escobar et al., 1998)***.

Patients with somatization disorder have the tendency to react to psychological distress and environmental stressors with physical bodily symptoms ***(Moore & Jefferson, 1996)***. Their complaints are usually centered on cardiovascular, gastrointestinal, respiratory, skin and other organ systems that have a strong autonomic nervous system medication ***(Barsky & Borus, 1995)***.

The most common complaints are musculoskeletal pain, gastrointestinal complaints (tiredness, sleep problems, fatigue, and mood changes). These complaints are common in the general population, but for some these complaints reach a level that requires care and assistance .

These complaints are based on sensations from what in most people are normal physiological processes. In some individuals these sensations become intolerable. In some cases it may signal somatic disease, in most cases not. Cases without somatic disease, or with minimal somatic findings, occur under diagnoses like burnout, epidemic fatigue, multiple chemical sensitivity, chronic musculoskeletal pain, chronic back pain, chronic fatigue syndrome, and fibromyalgia. These complaints are particularly common in individuals with low coping and high levels of helplessness and hopelessness. The psychobiological mechanisms by sustained attention and arousal ***(Eriksen & Ursin, 2002)***.

Eriksen and Ursin (2002) suggested that Sensitization is the psychobiological mechanism explaining the individual differences in tolerance and acceptance of common health complaints. The simplest form of plasticity in nervous systems is that repeated stimulation may lead to habituation (decreased response) or sensitization (increased response). Sensitization process may be present at multiple levels in the organism; at the cellular level ***(Rygh et al., 2002)***, at the psychological level (Van den Bergh et al., 2002), and at the interpersonal level ***(Brosschot, 2002)***. Limbic structures show a decreased threshold for epileptic seizures upon repeated subconvulsive stimulation “Kindling”, ***(Goddard, 1987)***. Finally at the higher levels sensitization covers increased attention and cognitive bias ***(Brosschot, 2002)***.

Sensitization has also been suggested as the underlying mechanism for other subjective health complaints reaching a level not tolerated by the patient. Irritable bowel and functional dyspepsia patients have been reported

to have a lower threshold for sensations from the gut ***(Wilhelmsen, 2002)***. Chemical intolerance has been attributed to limbic and mesolimbic sensitization ***(Bell et al., 1999)***. Sensitization of central nervous loops has also been suggested as an explanatory concept for comorbid psychiatric disorders in somatization patients like major depression, panic disorder, mania, phobic disorder, and anxiety ***(Ursin & Eriksen, 2001)***. All these conditions may depend on kindling of limbic structures ***(Bell et al., 1992)*** may explain why some subjects get more sensitive than others do to a variety of stimuli. This could explain the high comorbidity of these subjective states, and would offer a model for cross sensitization from one source of stimuli to another ***(Eriksen & Ursin, 2002)***.

At the higher cognitive level sensitization is remarkably similar to attentional bias or cognitive bias ***(Brosschot, 2002)***. ***Brosschot (2002)*** refers to this as cognitive emotional sensitization. There is evidence that anxious persons have a cognitive processing priority for information that is related to their fears .Anxious persons will detect fear related information than other persons. Their normal cognitive performance is interrupted and their cognitive capacity is absorbed in enhanced processing of information that is related to their concerns. The focus on one or several dominant complaints in somatization patients suggests an information bias or selection of complaints that may be driven by the suggestibility of somatizing patients, or the preoccupation with physical symptoms of the clinician and the researcher. The only limitation on the types of physical symptoms presented by the patient may be the patient's imagination and knowledge about physical illnesses ***(Fink, 1996)***.

Cognitive emotional sensitization ***(Brosschot, 2002)*** may also be used to explain why psychiatric patients are more frequently admitted to medical facilities and stay longer in hospitals with medical admissions than patients treated for the same disorder but without psychiatric illness ***(Fink, 1990)***. This may be due to a general unspecific increased vulnerability to all kinds of

somatic illness. It may also indicate that psychiatric patients are more sensitive to physical sensations ***(Mayou & Hawton, 1986)***.

Table (2) summarizes the common symptoms associated with somatization disorder ***(Moore & Jefferson, 1996)***:

Generalized symptoms:

1- Abdominal pain that is vague and nonfocal

2- Arthralgia

3- Backache

4- Chest pain that is nonspecific

5- Chronic tiredness

6- Headache

Gastrointestinal symptoms:

1- Chronic bloating

2- Constipation

3- Diarrhea

4- Food intolerance to multiple foods

5- Nausea

6- Rectal pain

7- Vomiting

Genitourinary symptoms:

1- Erectile dysfunction, ejaculatory disturbance, and impotence.
2- Decreased libido.
3- Dyspareunia
4- Dysuria
5- Menses that is painful, irregular ,and heavy
6- Vomiting that is prolonged or frequent during pregnancy.

Neurologic symptoms:

1- Fainting,
2- Fits,
3- Paralysis,
4- Lump in throat,
5- Deafness,
6- Blindness,
7- Paresthesias

Patients with somatization disorder can be vague dramatic in reporting their medical history ***(Moore & Jefferson, 1996)***. These patients frequently move abruptly from complaining of one symptom to another symptom and subsequently complicate the task of isolating one medical problem at a time rendering the office visit an arduous and frustrating task for the physician ***(Othmer & DeSouza, 1985)***. Often the only reliable conclusion that is reached during the initial assessment of a patient with somatization disorder is that objectively the review of systems is grossly and diffusely normal

(Escobar et al., 1998). Physical examination may reveal some skin lesions or scares that resulted from previously performed surgeries; however, these minor abnormalities do not account for the magnitude of the patient's complaints ***(Barsky & Borus, 1995)***.

In addition these patients tend to obtain care from multiple providers ,to fail to keep scheduled appointments, and to use medial services in maladaptive and inefficient ways ***(Barsky & Borus, 1995)***. Convinced that their illnesses are medically based, patients with somatization disorder characteristically deny influences of psychosocial distress in producing the symptoms of their disorder and resist psychiatric referral ***(Escobar et al., 1987)***. These patients are often refractory to conservative, palliative and supportive management ***(van Dulmen et al., 1996)***.

Thus these patients do not feel relieved by hearing a physician's statements such as "Nothing is wrong". To the contrary, these patients may become resentful, disappointed and frustrated when told by physicians that they are not clinically ill. Some patients may even express anger and dissatisfaction with the physician's medical assessment and may demand further diagnostic procedures, ranging from routine laboratory tests, radiolographic studies and electrocardiograhic studies to computed tomography, magnetic resonance imaging, endoscopy and exploratory surgery ***(Cassem & Barsky, 1991)***. Unconvinced by the negative findings of these tests, patients with somatization disorder eventually fire the exasperated physician and then move on to another physician ***(McCahill, 1995)***. The difficult and challenging clinical features of somatization disorder require appropriate diagnosis and management if the patient, the primary care provider and the health care delivery system are to benefit ***(van Dulmen et al., 1996)***.

The diagnosis of somatization disorder can be established using the ***Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV)*** diagnostic criteria ***(APA, 1994)***.

Table (3): **Diagnostic criteria for somatization disorder.**

A- History of physical complaints lasting for several years and beginning before age of 30 years, resulting in the request for treatment or leading to a significant impairment in social, occupational and other types of functioning.

B- *The following four criteria must be met , with individual symptoms occurring at any time*,

1- History of pain related to at least four sites or functions.

2- History of at least two gastrointestinal symptoms other than pain.

3- History of at least one sexual or reproductive symptom other than pain.

4- History of at least one symptom or deficit suggesting a neurologic condition not related to pain.

The diagnosis of somatization disorder may influence the response of primary care providers to patients with the disorder; therefore, it is important for physicians to exclude other medical and psychiatric conditions when the patient presents with a new complaint ***(Barsky & Borus, 1995)***. The features suggest the diagnosis of somatization disorder than a general medical condition include the involvement of multiple organ systems, the early onset of disease with a chronic course of illness in the absence of physical signs or structural abnormalities and the absence of laboratory abnormalities that characterize the suggested general medical condition ***(APA, 1994)***.

Many mental disorders are considered in the differential diagnosis which is complicated by the observation ht at least 50% of patients with somatization disorder have a co-existing mental disorder. Patients with major depressive disorder. Generalized anxiety disorder and schizophrenia may all have an initial complaint that focuses on somatic symptoms. In all these disorders, however, the symptoms of depression, anxiety, or psychosis eventually predominate over the somatic complaints. Although patients with panic disorder may complain of many somatic symptoms related to their panic attacks, they are not bothered by somatic symptoms between panic attacks ***(Sadock and Sadock, 2004)***.

Somatization and Gender:

It has long been held that women report more functional somatic symptoms than do men. In 1900 B.C., the ancient Egyptians made note in the Khun Papyrus of "a woman aching in all her limbs with pain to the sockets of her eyes", a complaint often echoed today. Both the Egyptians and Greeks ascribed the etiology of mysterious female pains to hysteria, which is Greek for wandering uterus. In a therapeutic effort to attract the uterus into proper alignment, sweet smelling balms and herbs were placed at the vagina and noxious potions were positioned at the nostrils. The assumption that only women could display the syndrome has continued into modern times. Despite reports by Sydenham in the seventeenth century and Charcot in the nineteenth that men displayed the same symptoms. The concept of hysteria has been inextricably bound to gender ***(Wool & Barsky, 1994)***.

Several observations have consistently emerged that women endorse more somatic symptoms than men and report more disability due to illness than men, even after adjusting for the presence of gynaecological disease and symptoms ***(Verbrugge, 1985)***.

Purtell et al. (1951) conceptualized hysteria as multiple somatic complaints rather than as intrapsychic drive and defense. The researchers described referred for psychiatric consultation with unexplained somatic complaints. These subjects were diagnosed with hysteria when no other medical, neurological or psychiatric diagnosis could account for their symptoms. It was noteworthy that these patients were all female.

In another study, ***Rubins et al. (1952)*** examined a group of men with somewhat similar symptoms, but considered them to have a different condition, compensation neurosis, because their symptoms were connected with efforts to gain recompense.

Also in the 1950s large community surveys using the Minnesota Multiphasic Personality Inventory reported that women more commonly than men endorse somatic symptoms generally considered functional ***(Wool & Barsky, 1994)***. ***Green, in 1980*** found that women's scores on hypochondriasis scale exceeded men's, but this difference was not seen among college age respondents. MMPI hypochondriasis scale scores are also known to vary with ethnicity and educationlevel. The 1989 revised scale shows some decrease in this difference between men and women ***(Hathaway & McKinley, 1989)***.

Recently community surveys with other self report instruments to measure somatization have had somewhat contradictory results.

Men with somatization disorder were of low socioeconomic status, and there was often a dramatic precipitating event as a harbinger of their illness. Most had other comorbid psychiatric disorders such as alcohol abuse and major depression. One possible interpretation of these findings is that men do not present with somatic complaints until there is greater disruption in their lives, which is compatible with work showing that men referred for

psychiatric treatment have more comorbid psychopathology than women ***(Smith et al., 1985)***.

Data on gender and somatization have also come from the Epidemiologic Catchment Area (ECA). This multicommunity survey of psychiatric disorders employed the Diagnostic Interview Schedule (DIS) and included the 40 somatic complaints that are the component symptoms of somatization disorder. The prevelance of these symptoms as reported by Escobar was greater in women than in men and in those psychiatric comorbidity particularly dythymia and major depression ***(Escobar et al., 1987)***. Escobar also noted the importance of ethnicity in this relationship: MexicanAmerican women were found to somatize more than non Hispanic women. ***Swartz et al. (1989)*** reported that somatization was more common in urban residents than in rural dwellers. The differences in residence were greatest among women and high school graduates. Somatization was associated with being older 45-64 as well as being separated, widowed or divorced. There was an inverse relationship with educational level and no relationship with ethnicity.

Cloninger and Colleagues (1986) have focused not on gender difference in prevalence, but on differences in phenomenology between men and women somatizers ***(Cloninger et al., 1986)***. The researchers found that somatizers fell into two distinct categories. One group, *the diversiform somatizers* were marked by a high number of brief sicknesses with diverse complaints especially pain. *Asthenic somatizers*, in contrast, have less diverse complaints and are more chronically disabled by fatigue, weakness or common minor illnesses. Contrary to popular belief asthenic somatizers were more likely to be male whereas the reverse was true in the diversiform category. The possible gender linked difference in phenomenology raises the question of inconsistencies in gender predominance across somatoform disorders. It is interesting that although the somatoform disorders as a group are diagnosed more often in women, hypochondriasis may be an exception

because it is thought to be more equally distributed between sexes ***(Barsky et al., 1990)***.

Although men have been diagnosed with somatization disorder and identified as somatizers, women still predominate. However this perceived difference is much influenced by social variables, referral patterns and psychiatric comorbidity, changing cultural factors and advances in research technique ***(Escobar et al., 1987)***. Thus the notion of somatization as an illness linked with hysteria exclusively affecting women has changed since 1950s. The influence of gender may reflect more the pattern of somatization than its prevalence.

The relation ship between gender and somatization may differ in medical patients and in community samples. If, for example, women have a lower threshold for visiting a physician with a somatic symptom then it would be expected the gender difference to be greater in patient samples than in community samples. Indeed, in patients aged 17 to 44 years women visit the doctor and are hospitalized twice as often as men. After 45 years of age women visit the doctor and are hospitalized twice as often as men. After 45 years of age, women have 20-40% more doctor visits than men ***(Verbrugge, 1985)***.

Numerous theories supported by varying degrees of evidence have been proposed to account for increased symptom reporting in women ***(Wool & Barsky, 1994)***. One difference may be at the physiological level: laboratory and field studies have shown that women are more sensitive to external environment cues including stress and men to internal physiological stimuli in noticing, defining and reacting to physical symptoms ***(Kirmayer & Robbins, 1991)***. Secondly, certain psychosocial factors more prevalent in women are strongly associated with symptom reporting, particularly depressive and anxiety disorders ***(Linzer et al., 1996)***, as well as a history of sexual or physical abuse ***(McCauley et al., 1995)***. Other psychosocial antecedents that

have been postulated include cultural factors permitting less stoicism and greater expressiveness among women; amplification of somatic symptoms; a lower threshold for seeking health care; and gender differences in social roles and responsibilities ***(Wool & Barsky, 1994)***.

Depressive and anxiety disorders clearly had the strongest association with symptom reporting, and adjusting for these disorders did reduce the effect of gender. Even after adjustment, however, gender effect remained significant and stronger than the effect of other demographic variables. Because depressive and anxiety disorders are both more prevalent in women and also powerful correlates of symptom reporting, the independent effect of gender is of particular interest ***(Kroenke & Spitzer, 1998)***.

The fact that similar proportions of women and men acknowledged serious illness worry suggests that gender related differences in symptom reporting is not a consequence of excess illness worry among women. Previous studies have similarly shown a higher prevelance of somatization, but not hypochondrical concerns in women ***(Kirmayer & Robbins, 1991)***.

Women also had a higher prevalence of somatoform symptoms. Although some of this could be simply because of increased symptom reporting in general, the proportion of symptoms considered unexplained in women was also greater for most symptoms. Certainly, this is consistent with the extensive literature on gender differences in somatization and somatoform disorders ***(Smith, 1995)***.

However the greater reporting of most symptoms as well as unexplained symptoms may account for the increased prevalence of functional syndromes among women, including irritable bowel syndrome, fibromyalgia, chronic fatigue syndrome, and migraine headache ***(Kellner, 1991)***. These disorders are defined predominantly by symptoms for which the precise etiology and pathophysiology is yet to be established.

Possible explanations or causes of gender differences:

At least six possible mechanisms may contribute to this phenomenon ***(Wool & Brasky, 1994)***. ***These include:***

- *gender differences in the willingness to admit to discomfort;*
- *the readiness to seek medical attention;*
- *the prevalence of psychiatric disorders with prominent somatic features;*
- *innate differences between men and women in their threshold and tolerance;*
- *sensitivity to minor bodily sensation; and differences in relational patterns.*

1- <u>Admitting discomfort:</u>

The predominant cultural ethos in the United States has impressed men more than women with the importance of not crying or acting like a baby, of maintaining a stiff upper lip and of not acknowledging weakness or distress ***(Lipsitt, 1982)***.

This may make men more reluctant to admit to an interviewer or investigator somatic distress and feelings of illness. Thus, some of the differences in somatization between men and women may actually be different in reportorial style, not differences in bodily experience. In addition, this style may have changed over the last 40 years; changes in the MMPI, as noted earlier, support this point.

2- <u>Readiness to seek medical attention:</u>

Women apparently have a lower threshold for visiting the doctor; ***(Verbrugge, 1985)*** thus they seek attention for relatively more benign

illnesses, trivial symptoms, and self limited dysfunction. The apparent gender difference in somatization might actually therefore result from gender differences in sick role behavior rather than from differences in bodily sensation. As ***Mechanic (1983)*** notes women not only report more symptoms more frequently than men, but they also use internists and psychiatrists more frequently. Psychologically distressed people who have no support may and often do turn to the medical care system for help. Differing role responsibilities, role strains ***(Gove, 1981)*** and vulnerabilities, as well as the accessibility of health care might ,explain why women turn to physicians more than men.

3- Psychiatric disorders with prominent somatic features:

Women might somatize more because they more frequently have psychiatric disorder with prominent somatic features. Depression may be the most important of these disorders because it is more common in women ***(Barrett et al., 1988)*** and frequently includes and even presents with somatic symptoms ***(Kandel DB, 1985)***. Anxiety disorders especially panic, can also present with somatic complaints ***(Orenstein H, 1989)***. These disorders are also more prevalent in women54. Panic disorder is reported in 1.6-2.9% of women and 4-1.7% of men ***(Crowe et al., 1983)***.

4- Etiologic factors:

Another possible explanation for differences in somatization among men and women is a difference in one predisposing factor, trauma. If childhood physical and/or sexual trauma predispose one to somatize latter in life ***(Lowenstein, 1990)***. Then a higher incidence of such trauma in girls might explain a higher prevalence of somatization in women. In addition, women who are abused as adults often present to their physician with somatic complaints as way of getting help because of fear of asking for help directly ***(Carmen, 1982)***.

5- Innate differences in bodily perception:

It is also possible that women are simply more sensitive to bodily stimuli and experience more somatic distress than men. Some work suggests that male and female college students process visceral and somatic sensation differently ***(Pennebaker & Watson, 1991)***. Women apparently use more situational and circumstantial cues and more external information than men do. Men, on the other hand, focus more on the somatic sensation itself and rely less on gathering information about the sensation.

Pennebaker and Roberts (1991) concluded that men's visceral sensation tends to be more consistent over time and more independent of the circumstances or situations they are in at the moment they experience the sensation.

6- Relational pattern:

Women use external cues and information sources more than men because they would be far more attuned to the input from their around them. They might also use others as sounding boards for their fears and suspicions about symptoms. This could lower their threshold for bringing these complaints to medical attention or endorsing them in community surveys. In these ways social interaction might serve to increase women's presentation with somatic complaints while dampening this behavior in men ***(Wool & Barsky, 1994)***.

Finally the research in gender differences in somatization disorders suggests however inconclusively that women report more functional symptoms than men. Although this has changed in the recent literature, fundamental questions about symptom appraisal and reporting, sociocultural influences, and developmental factors are all raised by a review of the literature. This is an important area with implications not only for psychiatric

disorder, but also for the different presentation of medical disease as well as such, it deserves further intensive investigation ***(Wool & Barsky, 1994)***.

Somatization and Personality Dysfunction:

The relationship of somatization and somatoform disorders to personality appears to be a close one, although it has received little study. Early descriptions of hysteria, for instance, linked the disorder to histrionic traits, but in the course of developing verifiable criteria, personality features were eliminated from the axis I condition ***(Lilienfeld et al., 1986)***. However, observers of the derivative somatization disorder continued to regard personality dysfunction as an important, even essential, feature, and when British psychiatrists were surveyed, over half viewed the disorder as a disturbance of both personality and mental state ***(Stern et al., 1993)***. Also, based on their review, ***Bass and Murphy (1995)*** concluded that most patients with somatoform disorders have personality pathology and that these disturbances are best conceptualized as disorders of personality.

Research based largely on psychiatric populations has demonstrated a link between personality and somatoform disorders, especially somatization disorder. Early interest was focused on histrionic and antisocial personality disorders that were found in substantial proportions of these patients ***(Morrison, 1989)***. On the basis of such findings, investigators proposed that somatization disorder and antisocial personality might be gender-linked expressions of a common hereditary diathesis ***(Lilienfeld, 1992)***. However, studies leading to this hypothesis looked at the disorders of interest rather than the entire spectrum of personality pathology. Also, treatment-seeking and referral biases may have influenced results. When the full range of disorders was examined, patients with somatization disorder were found to have high rates of personality disorders, but the increase was nonspecific ***(Emerson et al., 1994)***. For example, Stern et al. to found an excess of

personality disorders among these patients compared to control patients, the most common being passive-dependent, histrionic, and sensitive-aggressive.

Personality dimensions that influence the reporting of symptoms and the pursuit of health care may contribute to somatization and somatoform disorders ***(Kirmayer et al., 1994)***. For example, neuroticism, harm avoidance, and negative affectivity have all been shown to increase the experience of somatic as well as psychological symptoms ***(Russo et al., 1994)***. Likewise, traits that heighten bodily perception or increase self-absorption may also influence symptom reporting ***(Kirmayer et al., 1994)***. Certain other dimensions may contribute to the seeking of health care and to conflictual or unsatisfactory interactions with health care providers. Somatizing patients have been described as angry, resentful, and mistrustful-characteristics that suggest the negative pole of agreeableness ***(Starcevic, 1990)***. Also, research on difficult and frustrating patients has revealed antagonism related to personality disturbances ***(Hahn et al., 1994)***.

Although the somatizing patients showed more axis II pathology than non somatizing control patients, the increase appeared strongest for certain disorders and traits. Among the DSM-lV disorders, obsessive-compulsive personality was significantly more common among the somatizers, and its frequency was more than twice that of any of the remaining disorders. Previous authors have observed obsessive-compulsive personality traits and disorder among patients with somatization disorder ***(Rost et al., 1992)*** and hypochondriasis ***(Starcevic, 1990)***. The obsessive-compulsive pattern is a likely contributor to somatization. Patients with this disorder are concerned about maintaining control over their physical and mental functioning, and consequently, are threatened by unexplained symptoms. Also, such patients commonly engage in power struggles with physicians, and out of perceived inferiority, challenge their diagnoses and treatments. Such efforts to take control make for difficult doctor-patient relationships ***(Walker et al., 1997)***. On the other hand, the obsessive-compulsive personality pattern may

contribute to treatment compliance, responsible patient hood, and a more favorable outcome in some instances ***(Tyrer et al., 1997)***.

Among the abnormal personality traits that distinguished somatizers from nonsomatizing control patients, we found a number to have self-defeating, depressive, and negativistic personalities. These traits belong to provisional disorders for which diagnostic criteria have been proposed but not included in DSM-IV Within this group of individual traits, we found the self-defeating or masochistic personality pattern strongly represented. Depressive and negativistic traits were also prominent, but the former may be difficult to distinguish from chronic depressive disorders in this population. In fact, as indicated, current depression was identified in a substantial proportion of our somatizing patients, and among those with depression, we observed a much higher rate of personality disorders. Of course, depression is likely to increase masochistic tendencies; and guilt, pessimism, and self-deprecation are common to both ***(Walker et al., 1997)***. Even so, the traits of these three personalities-self-defeating, depressive and negativistic appeared to be inter-correlated and to correspond roughly to a dimension of covert or internally directed. According to ***Elliot (1987)*** self-defeating or masochistic traits are often found among somatizing patients, including those with pain and hypochondriasis. Such traits no doubt contribute to care-seeking and to difficult doctor-patient relationships. Patients with this pattern seek relationships in which they feel mistreated and, at the same time, thwart the attempts of others to be helpful. Physicians, whose role it is to help and heal, would seem to provide a ready source of such relationships.

Psychologizers-patients who present with psychological symptoms--differ from somatizers with respect not only to psychological symptoms but also attitudes and attribution of symptoms. Typically psychologizers have more severe depressive symptoms than do somatizers ***(EL-Rufaie et al., 1999)***. Somatizers, on the other hand, have less positive attitudes toward mental illness, are less introspective, and are more suspicious ***(Kirmayer &***

Robbins, 1999). Somatizers also report more secure interpersonal relationships and score lower on neuroticism ***(Weich et al,, 1996)***. One interpretation of these findings is that, by presenting somatic rather than psychological symptoms, true somatizers avoid blame ***(Craig et al., 1994)***. According to this explanation, true somatizers may have as much personality dysfunction as initial somatizers yet not report it. An alternative explanation is that true somatizers have less psychopathology than initial somatizers and, consistent with that interpretation, have less in the way of personality disturbance. Among true somatizers, physiologic mechanisms, such as autonomic hyperactivity, may make more of a contribution to somatic symptoms.

Neuroticism is a major dimension of personality that reflects the tendency of an individual to experience unpleasant and disturbing emotions. Extensive research has shown that this and the similar, mood-based disposition, negative affectivity, are strongly related to symptom reporting ***(Pennebaker & Watson, 1991)***.

Low agreeableness in somatizing patients may be related to help-seeking and to problematic doctor-patient relationships. The negative pole of the agreeableness dimension represents mistrust and antagonism. Individuals with such traits may repeatedly seek relationships with health care providers only to find them conflictual or unsatisfactory. Such a pattern has been described among somatizing patients and has been thought to reflect low agreeableness ***(Kirmayer et al., 1994)***. This finding compliments our observation of an increase in obsessive-compulsive, self-defeating, and negativistic traits, all of which are negatively correlated with agreeableness ***(Trull, 1992)***. Difficult and frustrating patients have characteristics suggestive of very similar personality disturbances, including Vague complaints, excessive demands, chronic complaining, lack of cooperation, lack of treatment response, and high utilization of services ***(McGaghie & Whitenack, 1982)***. ***Both Lin et al.***

(1991) and ***Hahn et al. (1994)*** have found personality disorders overrepresented among such patients.

Somatization and Culture:

The repertoire of tradition, belief systems, expectations and attitudes called culture profoundly influences the formation, presentation and management of somatization disorder ***(Escobar, 2004)***.

Kleinman (1987) has articulated with eloquence, culture not only shapes illness, but also determines the ways one convinces of illness. Even in similar countries such as those of western hemisphere (Europe and North America), dramatic differences can be documented in areas such as the way patients present, how medical tests are interpreted, and type and formulation of treatments provided ***(Payer, 1990)***. It is therefore expected that even deeper differences may exist for Americans, Asians and Latin Americans that persist following migration.

Because symptoms of somatization often follow exposure to traumatic events and may coexist in the same individual ***(Saxe et al., 1994)***, it is likely that these syndromes share similar origins and pathophysiologyical mechanisms. Somatization are perhaps the most commonly described sequelae of psychological trauma, and their presence augurs posttraumatic stress disorder and other major psychiatric disorders ***(Andreski et al., 1998)***.

A well accepted tent in cross cultural psychiatry is that transformation of personal or social distress into somatic complaints is the norm in most cultures ***(Kleinman, 1987)***. According to ***Shorter (1994)***, the cultural influx on symptom presentation follows socially correct models of proper behavior in the various societies and the prevailing medical paradigms.

Thus patients tend to develop symptoms that are medically correct that is symptoms that physicians expect and understand. Because somatizing individuals are relatively easy to characterize, they provide a useful construct for international comparison. For example, somatic symptoms are easier to recognize and their scrutiny proves less intrusive than that of psychological constructs. Thus they can be reliably elicited with little resistance offered by the subject because they tend to be less stigmatizing than psychological symptoms ***(Escobar & Javier, 2004)***.

Some of the large scale epidemiological surveys, such as those using the Diagnostic Interview Schedule (DIS) or the Composite International Diagnostic Interview (CIDI), have included detailed lists of somatic symptoms. Worldwide, the most common medically unexplained symptoms appear to be gastrointestinal complaints and abnormal skin sensations ***(WHO, 1992)***. The epidemiologic catchment area study revealed that the most common medically unexplained somatic symptoms in the United States were gynecological complaints, followed by gastrointestinal and cardiovascular symptoms ***(Escobar et al., 1987)***. A number of U.S studies have documented higher levels of somatization among Lations, particularly Puerto Rican respondents both in Puerto Rica and on the mainland ***(Escobar, 1995)***.

An international study reported frequent somatic presentations in primary care worldwide, with patients at the Latin American sites reporting the highest number of somatization symptoms ***(Gureje et al., 1997)***. In a primary care study of a multiethnic sample in the United States, ***Escobar et al. (1998)*** found that Central American immigrants had significantly higher rates of somatization than other ethnic groups and that war exposure was the single most predictor of medically unexplained somatic symptoms in these patients ***(Holman et al., 1996)***. Interstingly in that study it was found that recent immigrants had lower rates of PTSD than U.S. born patients despite the immigrant patients' higher trauma exposure. This resilience of recent Latino immigrants has been well documented for Mexican-Americans and extends to

several other health and mental health dimensions ***(Escobar, 1998)***. Cross cultural studies of depressed patients have documented higher levels of somatic complaints among depressed psychiatric patients in Asia ***(Kleinman, 1982)*** and Latin America ***(Escobar et al., 1983)*** compared to depressed patients in the United States.

There are also anecdotal reports suggesting that the type of symptoms presented by somatizing patients may differ in various settings. For example, in Nigeria and India common somatic symptoms are feeling of heat, peppery and crawling sensations, numbness, burning hands and feet, and hot peppery sensations in head symptoms that seem extremely rare in Western countries ***(Escobar, 2004)***.

Fabrega (1991) argues that in the case of traditional Chinese medicine, symptom clusters or syndromes that reflected imbalances in bodily systems that were also aligned with aspects of larger social and ecological systems. The holism of Chinese medicine did not develop psychology as a separate realm of discourse. Instead in conformity with the Confucian ethos, there was attention to disharmony in relationships as a potent cause of illness; but this disharmony was to be understood, not in terms of conflictual relationships and struggles for independence, but in terms of contraventions of a social order ruled by Confucian ideals of hierarchical order.

Somatic complaints are more common in Chinese Americans than white patients, so much so that 29% of Chinese Americans meet criteria for somatoform disorder, compared with 9% of whites, and both had concurrent psychiatric disorders such as major depressive disorder [71% and 63%, respectively] ***(Folstein, 1997)***. There are other culture-bound syndromes or local specific patterns of symptoms that we should be aware of, such as neurasthenia, a syndrome of physical and emotional weakness, shin-byung, a syndrome of anxiety and weakness, and hwa-byung, or "anger sickness **(see Table 3).**

Table (4): Asian Culture Bound Syndromes.

Somatization in Elementary Societies:

Among people of more elementary societies, the boy and its disorders constitute a medium that is richly elaborated from a symbolic stand point.

In a broad frame of reference, one could say that somatic referents constitute important elements for the idioms of distress of a people ***(Nichter, 1982)***. The people have no options but to offer help and treatment thereby socially certifying the individual's own presentations and enactment of illness. The concept of somatization when loosely can imply, in a moral sense, at worth a feigning of illness and at best an exaggeration, deviation and/or idiosyncratic mode of showing distress. None of these modes of qualifying phenomena of a medical nature operate in elementary societies.

Somatization in developing countries:

The impotence of somatic complaints in developing countries is underscored by the fact that the epidemiological pattern of the burden of disease is very different. for example the complaint of fatigue is typically attributed to nutritional deficiencies and, in particular, anaemia, which is common n women on account of menstrual blood loss and poorer nutrition, child-bearing and multiple pregnancies ***(Lennartsson et al., 1979)***. As a consequence, physicians are likely to prescribe irons, vitamins, tonics and nutritional supplements to treat the symptom presumptively. However ,this response has been challenged by a recent community study in India that sheds light on the prevalence and risk factors for the complaint of chronic fatigue in women ***(Patel et al., 2005)***. More than 1 in 10 women reported fatigue of at least 6 months' duration.

The strongest risk factors for these complaints were:

1- Socio economic deprivation, poor education, being in debt and having experienced hunger in the recent past.
2- Gender disadvantage, widowhood, oppressive restrictions on women's lives, lack of a trusting relationship with a spouse, and marital violence.
3- Poor mental health, comorbidity with other physical complaints, and symptoms of depression and anxiety.

It is noteworthy to mention that high rates of medically unexplained symptoms have been reported in non-Western cultures and in ethnic minorities of developed countries ***(Sethi, 1973)***. However, there is evidence that such symptoms are also a common problem in Western societies ***(Bridges & Goldberg, 1985)***. It seems that the clinical presentation for such medically unexplained symptoms is influenced by linguistic habits, such as using body language for distress and illness-related beliefs related to social class and cultural background ***(Lipowski, 1988)***. For instance, it has been reported that symptoms that typically manifest themselves in Africans include heat sensation from inside the head or body, peppery and crawling sensations in various parts of the body, numbness and poorly localized aches and pains ***(Ohaeri and Odejide, 1994)***. on the other hand, backache was the most common presentation of medically unexplained symptoms in a Spanish primary care sample ***(Lobo et al,, 1996)***. A Saudi study demonstrated a correlation between functional abdominal pain and higher frequency of anxiety and depressive disorders ***(El-Rufaie et al., 1990)***. however, a recent WHO somatization study in 14 countries claimed that the frequency of unexplained somatic symptoms did not clearly vary according to geography, level of economic development, or culture ***(Gureje et al., 1997)***.

Causes of Somatization:

The aetioloy of functional disorders is unknown ,but is probably multifaceted. Different unspecific predisposing factors commonly seen in a wide array of mental disorders assume importance depending on the person's general vulnerability and coping strategies ***(Fink et al., 2005)***. The reported familial transmission may be rooted in sociocultral variables but there is also some support for a genetic transmission ***(Guze, 1993)***.

However certain enduring trait like characteristics put an individual at risk for somatization. This vulnerability may remain latent until an acute precipitant such as the onset of a psychiatric disorder or a major life stress causes it to become clinically manifest. This clinical state tends to be self limited and to abate with time, palliative treatment and reassurance but it can become chronic if prolonged by several perpetuating and maintaining factors ***(Barsky, 1998)***.

Predisposing factors:

1- Childhood experience: Three types of childhood experience have been found to predispose to subsequent somatization in adulthood: First, an exaggerated parental interest in and attention to health or a tendency to selectively reinforce the child's somatic complaints by rewarding them ***(Benjamin & Eminson, 1992)***; conversely, physical and sexual abuse or trauma in child hood can predispose one to subsequently develop medically unexplained complaints, especially chronic idiopathic pain ***(McCauley et al., 1992)***. Finally, childhood exposure to a "figure of identity" to someone who obtained substantial secondary gain from either organic or medically unexplained symptoms places one at risk for subsequent somatization ***(Whitehead et al., 1994)***.

Personality traits: Certain personality characteristics or types are thought to predispose one to somatization ***(Kiramayer & Taillefer, 1997)***. These include neuroticism or negative affectivity because individuals high on negative affectivity are more likely to report somatic symptoms than individuals lower in neuroticim ***(Larsen, 1992)***. Some personality disorders in particular the personality disorders comprising Cluster B in the DSM-IV (borderline personality, antisocial personality, histrionic personality, and narcissistic personality) are also more likely to report medically unexplained complaints ***(Kirmayer & Taillefer, 1997)***. Alexithymia may also be related to somatization but this relationship remains controversial at present.

Personality factors as expressed in the concept of alexithymia may be relevant to the etiology of SD in another way ***(Mai, 2004)***. The word coined by Sifneos, is derived from Greek and literally means no words for feelings. The term is based on the psychoanalytic concept that patients somatize because they are unable to express their feelings in words. There is an extensive literature on the relation between alexithymia and somatization ***(DE Gucht & Heiser, 2003)***. Much cross-sectional research has been carried out and most studies show a positive correlation between alexethymia and somatization. However there is a need for verification of longitudinal studies using techniques that distinguish somatization from organic illness ***(Mai, 2004)***.

2- Cognitive style: Certain enduring cognitive characteristics or styles appear to make one more likely to somatize ***(Kirmayer & Taillefer, 1997)***. These include: catastrophizing in which the individual endorses the most serious, ominous and alarming possible interpretations for bodily sensations; the tendency to attribute ambiguous bodily discomfort to disease rather than to emotional states or to normalize it ***(Barsky et al., 1993)***; and external locus of control.

3- Perceptual style: Certain characteristic and enduring styles of bodily perception predispose one to experience somatic distress ***(Kirmayer & Taillefer, 1997)***. Individuals vary in somatosensory amplification i.e.; in the tendency to experience mild, benign, and ambiguous bodily sensation as noxious, intense, disturbing, and uncomfortable and this characteristic appears to be associated with symptom reporting and to be relatively enduring and stable over time ***(Barsky & Wyshak, 1990)***. Individuals characterized as having high levels of private self consciousness are also more likely to somatize. These individuals' attentional focus tends to be inward, on their own bodily experience, and they exhibit a heightened degree of bodily vigilance ***(Kirmayer & Taillefer, 1997)***.

Precipitating factors:

Unspecific precipitating factors are stress and strain for example bereavement, loss of job ,physical disease or other significant events .It is not known whether specific precipitating factors may exist ***(Fink et al., 2005)***.

1- Life stress: Somatization often follows stressful life events ***(Kellner, 1986)***. In many instances, the symptom appears to bear a clear relationship to the stressor which precipitated it. Some sorts of stress are especially likely to prompt somatization. These include threats to health (eg, myocardial infarction) and the deaths of significant other people.

2- Psychiatric disorder: Many psychiatric disorders in particular affective disorders ***(Katon et al., 1984)*** (major depressive disorder, dysthymia, and minor depression) and anxiety disorders ***(Katon, 1991)*** (panic disorder, generalized anxiety disorder, and obsessive compulsive disorder), frequently present with somatic symptoms. Indeed three quarters of the patients in general medical practice who are found to have an anxiety or depressive disorder on a structured, diagnostic interview presented initially with somatic complaints ***(Kirmayer et al., 1993)***.

3- Activation of health related cognitive schema: Critical events that are personally meaningful eg, prominent celebrity falling ill, or reaching the age of a loved one who died suddenly or unexpectedly can activate previously latent, health related cognitive schema which are faulty unduly pessimistic or alarming. Examples of such a schema include “Because everyone in my family has a weak heart. I am going to die of heart disease.” Once activated, such schema then powerfully shape subsequent perceptual experience of emotional reactions and behavioral responses to the symptom ***(Barsky, 1998)***.

Maintaining and Perpetuating Factors:

Most instances of somatization are self limited and subside spontaneously. But some patients exhibit a more prolonged and chronic course in which the presenting symptom persists or subsides only to be replaced by another symptom. Several factors are known to maintain somatization and prevent spontaneous resolution.

1- Secondary gains*:** Although the presence of secondary gain does not differentially distinguish psychogenic from organically based symptoms, it fosters and perpetuates all symptoms, regardless of their etiology ***(Kellner, 1991). Symptoms allow us to avoid facing seemingly insurmountable obstacles in our lives, postpone challenges, excuse ourselves from duties and responsibilities ,control and manipulate others and sanction failure. And they do so without any amplification of fault failure, or blame because we are not held responsible for being sick. Thus there may be powerful secondary gains that tend to perpetuate the symptoms of illness.

2- Ongoing and continuing stress*:** Stressful events as noted above precipitate somatization. In addition if stressors become prolonged, they can sustain and maintain the initial somatization response ***(Kellner, 1990). Thus the persistence of the precipitating stressor may result in chronic somtization.

Illness behavior*:** The illness behaviors which symptoms prompt such as medical help seeking, altering leisure activities, and curtailing work can in themselves perpetuate somatization ***(Salkovskis, 1989).

Thus it needs to be emphasized that there is a practical value in establishing a probable psychopathology in each individual patient ***(Mai, 2004)***. The predisposing - precipitating - perpetuating - protective framework is a useful format for this psychopathological construct. Not only does this framework provide an understanding of the individual and the illness ,it also has therapeutic implications.

Moreover there are four relevant domains to characterize the patient's current clinical status: *perceptual, cognitive, behavioral and affective*.

In assessing each of these domains ,the clinician arrives at a description or profile of the patient's somatic style, characterizing his/her tendency to amplify bodily discomfort, the cognitive elaboration of this somatic discomfort, the degree to which the patient has acted upon his/her concerns with illness behavior and medical help seeking and the degree to which he/she is affectively distressed by it ***(Barsky, 1998)***.

Bodily sensation: Somatization by definition involves a somatic or visceral sensation and entails an element of bodily distress. But the character, nature, and degree of that physical discomfort varies. At one end of the spectrum lie patients with somatization disorder for whom somatic symptoms are the predominant or even sole source of distress, who may deny any emotional or cognitive disturbance.

Thoughts and beliefs: Patients have ideas about the meaning, cause, implications and significance of their somatic symptoms and these beliefs can be major sources of distress and disability in themselves. The authenticity and legitimacy of medically unexplained complaints is always an issue because the absence of a demonstrable medical basis for a symptom casts the shadow of skepticism, disbelief, and illegitimacy over the suffer, implying that he is somehow malingering or imagining his distress. But beyond this universal concern, some patients are more preoccupied with the meaning themselves. Their thoughts and presumptions about the cause and mechanism of their symptoms and their suspicious about their state of health may be a major preoccupation and source of worry ***(Barsky, 1998)***.

Behavior and actions: Somatic symptoms and illness beliefs prompt people to take specific actions to further appraise and ameliorate them. These illness behaviors may themselves cause additional distress, invalidism, disability, and role impairment. Some somatizing patients become completely disabled and fully adopt sick role. Others are far more stoical and less demonstrative ***(Barsky, 1998)***.

Affect and emotions: Finally the nature and extent of the patient's emotional dysphoria must be appraised. Health and anxiety, fear, helplessness, frustration and depressed mood may be relatively modest in some patients but may be the most salient and distressing clinical features in other cases. For some of the latter, the primary, underlying problem may be anxiety or depressive disorder and the somatization is best viewed as a secondary manifestation of this pervasive underlying disorder ***(Barsky, 1998)***.

The cognitive behavioral model suggests that functional illness is a result of maladaptive reaction to benign normal bodily sensation and banal ailments that are interpreted as caused by a physical disease. The model focuses on the patient's symptom perception and the resulting illness behavior ***(Petrie & Weinmam, 1997)***.

The model assumes that everybody has a personal sensation or symptom panorama with which the person is familiar. This knowledge accumulates throughout life in a continuous process when girls reach puberty they become familiar with new symptoms in connection with menstruation which they add to their personal symptom panorama. Experiences with symptoms from various diseases throughout life will also form part of the individual's inner frame of reference ***(Fink et al., 2005)***.

People may also seek information from external sources that is family members, colleagues, medical encyclopedias, weekly magazines, as well as physicians. External information can initiate the process of the individual changing the interpretation of personally well-known sensations as outlined in Fig. (A) that is they suddenly interpret symptoms as signs of illness. The new information will cause focused attention on the body part that is suspected to harbour the disease. This may cause new sensations or symptoms to surface and a cycle of worry and expectation of symptoms will ensue ***(Fink et al., 2005)***.

Fig A *; symptom perception and illness understanding (Fink et al., 2005):*

In addition studies have indicated that biological mechanisms and pathophysiological changes may be of importance in functional disorders ***(Miller, 1984)***. The multisymptomatic illness pattern found in functional disorders may be caused by patient's intensification of all bodily sensations because of afferent stimuli to the brain not being suppressed or because of an increased sensitivity of the central nervous system structures ***(Meares et al., 1999)***. Also the stress response mediated by the hypothalamic - pituitary - adrenocortical (HPA) axis may be involved ***(Roberts et al., 2004)***.

Fig (B)*: Etiology of unexplained medical symptoms (From Gelder & Others, 1999).*

Current theories about causation of somatization are grouped into three categories; genetic, organic and psychosocial ***(Mai, 2004)***:

I- Genetic theory:

Some twin studies have found evidence of a genetic component ***(Torgersen, 1986)*** but others have a negative conclusions ***(Shields, 1982)***. It may therefore be concluded that although genetic factors may play a role in SD, the effect is a limited one ***(Mai, 2004)***. Another study of women (n=859) who had been adopted at an early age by non relatives found that the adoptees had a significant excess of somatizers compared with a non adopted control group. The parents of the adoptees were known to have excess numbers who showed criminal or psychotic behavior ***(Sigvardsson et al., 1984)***.

II- <u>Organic theory:</u>

Patients with SD compared with control subjects have been found to have deficiencies on neurobiological testing ***(Niemi, 2002)***. In a study comparing magnetic potentials in patients with psychogenic and organic limb weakness, Meyer found that the former were associated with normal values ***(Meyer et al., 1992)***.

An association between somatization and elevated 24-hr cortisol levels (physiological arousal) has been found ***(Rief et al., 1998)***, as has an association between somatization and systolic blood pressure ***(Kristal et al., 1998)***. Taken together, these studies strongly suggest a non specific association between somatization and organic cerebral disease. The mechanism by which this effect is mediated is not clear ***(Mai, 2004)***.

SPECT research in diseases strongly related to SD such as chronic fatigue syndrome (CFS) demonstrates abnormalities that seem to correlate with clinical status ***(Fischler et al., 1996)***. Concretely an asymmetry right more than left of tracer uptake at the parieto temporal level is demonstrated in CFS compared with major depression ***(Schwartz et al., 1994)***.

As a consequence, a pathological role of frontal blood flow in the cognitive impairment and physical activity limitations in the CFS has been hypothesized ***(Schwartz et al., 1994)***. In addition it seems that SPECT is more useful than magnetic resonance imaging (MRI) in following the clinical progress of CFS patients for two reasons ***(Fischler et al., 1996)***;

i- SPECT detects significantly more abnormalities than MRI.

ii- SPECT abnormalities appeared to correlate with clinical status where as anomalies detected with MRI did not reverse with improved clinical status ***(Yazici & Kostakoglu, 1998)***.

In a study aiming to assess SPECT imaging abnormalities in SD patients and study any relation to laterality ***(Gracia-Campayo et al., 2001)***, eleven of SD patients from the somatization disorder unit of Miguelservrt university hospital, Zaragoza, spain, not fulfilling criteria for any psychiatric disorder and showing normal CT and MRI images were studied with SPECT patients with DSMIV axisI comorbidity were ruled out because it has been demonstrated ***(Yazici & Kostakoglu, 1998)***.

However there are several limitation in this research ***(Gracia-camppayo et al., 2001)***;

First; the small number of patients.

Second; this is not a controlled study with a matched sample of healthy controls.

Third; a profile of neuropsychological tests would be advisable to correlate functional abnormalities with neuropsychological impairment.

Forth; a follow up would be recommended to confirm that the SPECT abnormalities endure.

Despite these limitations, the findings point to possible brain function of SD patients and confirm studies ***(James et al., 1990)*** that suggest subtle neuropsychological disturbance .

III- Psychosocial theory:

"Illness behaviour" is a concept relevant to somatization. It was developed by Mechanic in 1970s and refers to the behaviours that an individual adopts when ill, such as going to bed, taking medicines, going to see the doctor ,or going to the emergency department; that is becoming a patient ***(Mechanic, 1972)***. The medical care process with its consultations,

laboratory tests and diagnostic procedures can itself foster somatization. The steps patients take to learn about and diagnose their own condition ranging from asking others for their opinions to reading up on the subject or consulting the internet often elicits alarming and distressing information that only exacerbates the patient's distress. Self treatment which may include the avoidance or alteration of activities suspect of causing the symptom or illness may in fact perpetuate the symptoms. For example, decreasing a physical activity to be causing one's pain or fatigue leads to deconditioning which ultimately exacerbates the original symptom ***(Barsky, 1998)***.

Learning theory presupposes that behaviour is learned through experience. Behaviour that is rewarded is thereby promoted and reinforced and that which is not rewarded is inhibited. Somatization may therefore be regarded as a maladaptive way of obtaining social needs that substitutes for deficits in a patient's adaptive behavioural repertoire ***(Kellner, 1990)***.

A child observing a sick parent or sibling may learn illness behaviour, previous experience of illness is an important factor in the pathogenesis of somatization. Through a process of identification, a child who observes its parent during an illness of any type, particularly if severe or chronic may develop a somatoform when older. It also has been shown that the fathers of women with SD are more likely to have an antisocial personality disorder ***(Cloninger et al., 1995)***. Whether this effect is mediated by genetic or psychosocial factors or a combination of both is not known .

Traditional psychoanalytic factors including conflicts with mother or father or difficulties in the control and regulation of aggression and frustration may also play a part in individual cases. However no experimental surveys have tested the validity of these hypotheses on larger groups of patients ***(Mai, 2004)***.

Ethnicity, education and sex are other social factors relevant to somatization. A high correlation has been found between somatization and ethnicity ***(Escobar & Canino, 1989)***, low social class with less education ***(Fink et al., 1999)***, and female sex ***(Lieb et al., 2002)***.

Doctors also play a part in the pathogenesis of SD. If patients are poorly managed and subjected to unnecessary specialist referral or expensive, invasive, diagnostic and therapeutic procedures this will ensure that complaints become imprinted and medicalized. In suchpatients, therefore, iatrogenic factors must be regarded as part of the etiology ***(Mai, 2004)***.

Psychopathology of Somatization:

Depending on the clinician's interpretive stance, somatic symptoms have been conceptualized and handled as ***(Kirmayer & Young, 1998)***:

a- an index of disease or disorder

b- a symbolic expression of intrapsychic conflict

c- an indication of specific psychopathology

d- an idiomatic expression of distress

e- a mataphore for experience

f- an act of positioning with a local world

g- a form of social commentary or protest.

(1) Somatic Symptoms as Indices of Disease:

As an index of disease or disorder, somatic symptoms follow automatically from disturbed physiology. Just as a thermometer provides a numerical index of fever, so symptom reports may be read as indices of

underlying disease. For many medically unexplained symptoms or functional somatic syndromes, the physiological perturbation may be too subtle or intermittent to be reliably measured with current technology ***(Kirmayer, 1994)***.

(2) <u>Somatic Symptoms as Expression of Psychological Conflict</u>:

Emotional distress due to intrapsychic or interpersonal conflict can give rise to physiological disturbances with a wide range of somatic symptoms. Physical symptoms then may simply be viewed as an index of psychosocial problems ***(Shedler et al., 1993)***. This theory allows the clinician to identify the links between potential stressful events and illness even where the patient does not see or actively denies any such link ***(Kirmayer & Young, 1998)***.

(3) <u>Somatic Symptoms as Manifestation of Specific Psychopathology:</u>

Somatic symptoms may also be interpreted as evidence of a specific type of psychopathology. There is some evidence that certain individuals have an amplifying somatic style that leads them to experience and report more distressing somatic symptoms in the context of recognized psychopathology ***(Barsky, 1992)***. Certain personality traits may lead some individuals to experience and focus on physical distress ***(Kirmayer et al., 1994)***. Childhood events that reinforce somatic illness as a legitimate means of seeking help or assistance from others and of resolving conflict may also lead to increased reporting of somatic symptoms and help seeking ***(Craig et al., 1993)***.

Current psychodynamic theory argues that many physical symptoms may occur as a result of deficits in the capacity to symbolically resolve and express emotional conflict and distress, a defect referred to as alexithymia ***(Taylor, 1984)***. On this view, high levels of help seeking for somatic symptoms may reflect a specific psychological deficit ***(Lumley & Norman, 1996)***. In recent studies, however, alexithymia seems to be more strongly related to the depression and dysphoria that often accompany somatic distress and that may

make it difficult to discriminate nuances of feeling and elaborate symbolism and fantasy ***(Cohen & Auld, 1994)***.

***(4) Somatic Symptoms as Cultural Idioms of Distress*:**

An individual's report of bodily symptoms can be understood as encoding cultural models of sickness or idioms of distress. These cultural models supply individuals with a vocabulary of symptoms more than this they also provide explanations for these symptoms and the associated suffering. As a culturally available idiom, somatic symptoms express discomfort and distress in ways that are intelligible within the individual's social milieu but may have different meanings to outsiders.

(5) Somatic Symptoms as Metaphors for Experience:

As extension of the nation idioms of distress considers the sense in which somatic symptoms provide metaphors for experience. When the patient speaks of heart distress, for example, he may simultaneously be speaking of physical sensations attributed to the heart and using an evocative metaphor that conveys specific affective meaning. At the same, metaphors allow the generation of new meaning some of which may be unintended by the patient, but which is nevertheless, taken up and used by others for their own purposes. As a result of this social embedding, the meaning of symptoms can be understood in terms of presentation rather than representation; that is, symptoms come to have meaning when they are used in a specific social context ***(Kirmayer, 1992)***.

***(6) Somatic Symptoms asocial Positioning*:**

The process of help seeking, adaptation, and disablement initiated in response to somatic symptoms can serve to reconfigure family relationships and other social roles. Some of this is captured in the clinical notion of secondary gain. The extent to which it is international varies with the

individual's own awareness of his or her social position and the degree to which is acceptable and safe for talk about social problems. In a larger sense then, symptoms can be understood as having meaning as moves within a local system of power.

Somatic symptoms may function as social moves or positioning whether or not the individual is aware of this process. The clearest examples of this are reported among minorities: the victims of exploration and humiliation, based on gender, race, ethinicity, and economic disadvantage ***(Lock, 1993)***. In this case, certain symptoms have been interpreted as being forms of resistance or weapons of the weak, used to evade or attenuate injustices or to undermine otherwise unassailable power holders.

(7) <u>Somatic Symptoms as Social Commentary or Contestation</u>:

Somatic symptoms function as a medium of communication whether or not they are so intended. The social origins of somatic distress are apparent to most people and so the simple declaration of ill health raises questions about the adequacy and legitimacy of existing social structures and arrangements. Whether used consciously and strategically or inadvertently, somatic symptoms then may present a commentary on social circumstances. At times, they may serve as a form of protest, challenge, or contestation of social conditions ***(Lock, 1993)***. Compared with frank complaints about one's psychological state or social situation, however, somatic symptoms are oblique or indirect and, hence, may protect the powerless from the counter attack that might be elicited by more direct criticism ***(Kirmayer & Young, 1998)***.

<u>Reconciling Multiple Interpretive Frameworks</u>:

The conflicting agendas represented by these different modes of interpretation means that the correct interpretation is a rhetorical achievement rather than an empirical discovery ***(Kirmayer, 1994)***. The choice of

interpretation is not simply a matter of truth in as much as there are many partial truths available but a strategic choice of what will be of the most benefit to the patient. Of course, the clinician's interpretation is only half of the story; it meets its match in the patient's own constructions of illness meaning. Patients themselves make more or less conscious and strategic choices of aspects of meaning in their interactions with health care providers to emphasize an effort to avoid psychiatric stigmatization and to receive the forms of help they deem acceptable and appropriate.

Theories of somatization:

The concept of somatization is based on a dualistic conception that views somatic symptoms as occurring in place of emotional expression. The therapeutic implication is that patients ought to replace their somatic expressions of distress with explicit talk about emotion, both because this psychological idiom is ,in some sense, a more accurate representation of their experience and situation ,and because such emotion talk will lead to a better resolution of intrapsychic and interpersonal problems ***(Kirmayer and Young, 1998)***.

Theories of somatization can be grouped under the following:

1) Psychodynamic theories:

Influenced by psychodynamic ideas and by more pervasive western attitudes toward the body, many theorists have viewed the expression of distress in somatic symptoms as somehow more "primitive, infantile or regressed ***(Kirmayer, 1988)***.

Equating somatization with primitive thinking or communication is based on several doubtful premises that:

- greater psychological insight necessarily results in fewer somatic symptoms
- psychological idioms are inherently more ‘advanced” than somatic idioms
- somatic idioms are” less differentiated” and finally
- psychological development moves along a one-dimensional continuum from primitive to advanced ***(Kirmayer, 1987)***.

Current psychodynamic theory argues that many physical symptoms may occur as a result of deficits in the capacity to symbolically resolve and express emotional conflict and distress, a defect referred to as ‘alexithymia”.

2) Interpersonal theories:

From an interpersonal perspective, it could be argued that explicit talk about emotions can serve as a more effective way of drawing attention to personal needs. Whereas emotion conveys information about interpersonal conflicts, it may serve to address problems in relationships and motivate others to change. Although emotions often are associated with psychological processes, they are also social constructions ***(Markus & Kitayama, 1991)***. Psychological talk about emotions tends to situate problems entirely within the individual and may distract the patient. his or her family, and the clinician from the social and situational problems and inequities that are signaled by the emotion.

3) Socio-cultural theories

It seems clear that western psychological notions-although they may at times be the cause of certain forms of suffering, for example, narcissism or alienation symptomatic of excessive individualism ***(Cushman, 1990)*** offer

powerful analytic tools for understanding and opening up new possibilities for way of understanding a problem is also introducing a culture-specific concept of the person, which may conflict with the values and perspectives of the patients' culture of origin and so create new dilemmas for them. Psychiatric diagnosis and treatment-even the prescription of medication-must then be understood not simply as technical interventions, but as interpretive actions aimed to improve the psychological and social status of individuals and families that also, inevitably, contribute to wider social and cultural change.

Whereas there is evidence for differences in the rates and form of somatization among different cultural groups, the clinical significance of these differences remain unclear. Differences among groups may reflect cultural styles of expressing distress that are influenced not only by cultural beliefs and practices but also by familiarity with health care systems and pathway to care ***(Kirmayer and Young, 1998)***.

In conclusion, these theories are better seen as complementary rather than competitive. Also, it is not clear whether features of somatization disorder form discrete diagnostic categories, or whether they are better conceptualized as personality characteristics, maladaptive coping styles, nonspecific symptoms of other more pervasive psychiatric disorders, or simply normal variants ***(Creed and Barsky, 2004)***.

Somatization Disorder in Primary Care:

Most people with psychiatric disorders are seen first by primary care physicians and for many this remains their only opportunity to receive diagnosis and treatment of their psychological problems or psychiatric disorders ***(Schulberg & Burns, 1988)***. Given the importance of the primary care system in mental health care, it is discouraging to find that in most settings physicians' rates of recognition of psychiatric disorders in their

patients are very low ***(Goldberg & Huxley, 1992)***. Even when psychological distress or social problems are recognized, imprecise diagnosis contributes to what is generally inappropriate or inadequate treatment.

It is possible that the lower rate of recognition of psychiatric conditions among somatizers was due to milder psychiatric conditions or psychiatric conditions or psychological problems that were simply elapsed by concomitant but unrelated somatic illness ***(Krimayer et al., 1993)***.

Somatic symptoms account for more than half of all outpatient encounters or an estimated 400 million clinic visits in the United States each year ***(Schappert, 1992)***. Although the prognosis for the majority of common symptoms as they present in primary care is favorable in terms of mortality or serious morbidity, these symptoms do account for substantial health care utilization and costs ***(Komaroff, 1990)***.

Somatic symptoms are prevalent in primary care practice and appear to be explained by a physical disorder in only 50% to 60% of cases. At least one in four symptoms documented in the medical record may persist at 1-year follow up. Ways of improving our approach to somatic symptoms in primary care might include the use of diagnostic algorithms for unexplained symptoms; better detection and management of depressive and anxiety disorders including collaboration with mental health professionals; attention to symptom related expectations and concerns; and development of new management strategies for unexplained or persistent symptoms ***(Kroenke, 2003)***.

The Life time prevalence of somatization disorder ranges from 0.2% to 2% among women and is less than 0.2% in men ***(APA, 1994)***. The disorder usually in the teenage and young adulthood years. Onset after the age of 30 years is extremely rare ***(Cassem & Barsky, 1991)***. Somatization disorder seems to be more common in less educated and lower socioeconomic groups

(Othmer & DeSouza, 1985). The disorder is observed in 10% to 20% of female first degree relatives of women with the disorder ***(APA, 1995)***.

The male relatives of women with somatization disorder have an increased risk of antisocial personality, substance abuse disorders, and somatization disorder ***(APA, 1995)***.

An estimated 25% to 75% of patients presenting with somatization disorder to primary care providers may have this disorder resulting from psychological distress ***(Katon, 1984)***. Compared with patients without the disorder, patients are found to have a six times higher rate of hospital expenses a 14-times higher rate of ambulatory care visits, and a nine times higher rate of total health care costs ***(de Grug et al., 1987)***. Atypical patient with somatization disorder spends an average of 7 days per month sick in bed compared to 0.48 day for a patient without the disorder ***(de Grug et al., 1987)***.

It had been demonstrated that persons with multiple unexplained physical symptoms are very common in the general population. ***Kroenke et al. (1997)*** found that somatoform symptoms were frequent and that about 8% of primary care patients fulfill the criteria for multisomatoform disorder but most of these patients did not fulfill the criteria of the diagnosis of somatization disorder.

Fahrenberg (1995) pointed out that the accumulation of physical symptoms to form somatization scores as proposed from the classification systems DSM and ICD-10 is questionable. Such a procedure assumes that the single symptoms have comparable frequency and little influence due to age and gender. However, the single symptoms of the symptom lists differ greatly in terms of base rates, age and gender of the subjects. Even in clinical high risk samples, the base rates of some symptoms relevant to the classification were extremely low ***(Rief & Hiller, 1999)***.

The so-defined somatization features are associated with aspects of illness behavior. Eighty one percent of all persons reporting at least one unexplained physical symptom also confirmed that they visited doctors because of these complaints during the previous 2 years. Therefore the health care relevance may be better reflected by somatization syndromes than from people fulfilling all criteria for somatization disorder ***(Rief et al., 2001)***.

In epidemiological research, somatization has been studied through somatic symptom checklists that are presumed to be non specific indicators of psychiatric illness. Epidemiological research on somatization in the United States has been dominated by the contruct of somatization disorder and a form fruste termed subsyndromal somatization disorder based on a life count of four medically unexplained symptoms for men and six for women on the somatization section of the NIMH Diagnostic Interview Schedule termed the Somatic Symptom Index and denoted as SSI 4,6 ***(Escobar et al., 1989)***.

In the general population, somatization disorder is quite rare. In ECA studies, somatization disorder was found in 0.01% of the population and was most prevalent among African-American women 0.8% followed by African-American men 0.4% ***(Robins & Regier, 1991)***. This difference in prevalence may be accounted for by differences in educational status. It seems that relevant reports are contradictory in relation to educational level. Where as some results suggest a lower educational level among SMD patients ***(Omer et al., 1998)***. ***Bridges et al. (1991)*** claimed that psychologized mental disorder tended to be less educated, although this finding fell short of statistical significance. On the other hand, ***Kirmayer and Robbins (1991)*** reported higher educational levels among somatized mental disorder patients but again there was no statistical difference. On the other hand the WHO's comprehensive study ***(Gureje et al., 1997)*** noted that somatizing patients had fewer years of formal education.

Epidemiological studies have used measures of somatization that are insensitive to culture specific symptoms and modes of expressing distress.

Although some efforts have been made to develop expanded inventories of somatic symptoms for example in Nigeria ***(Ebigbo, 1982)***, Pkistan, India, and England ***(Mumford et al., 1991)***, the scales resulting from these studies have not yet been widely applied ***(Mumford et al., 1991)***. Several studies with symptom checklists have not found much higher rates among non western groups ***(Mumford, 1989)***. However lack of attention to culture specific symptoms limits the ability to detect cultural differences.

Studies in primary care:

Wherever primary care samples have been studied somatization has been found to be extremely common. For example in a study of 700 patients attending family medicine clinics in Montreal on a self initiated visit for a new symptom or problem ,more than 75% of the patients with more depression, panic disorder, or milder forms of depression-anxiety made somatic clinical presentations;17%of the patients had a life time history of multiple unexplained symptoms (subsyndromal somatization disorder denoted SSI 4,6 ***(Escobar, 1989)***. Across ethenocultral groups, fully 26% of patients met study criteria for one or more forms of criteria for one or more forms of somatization. This high prevalence in a north American city challenges the notion that Asians or other non western groups are more prone to somatize than Europeans or Americans. Similar findings have been made in primary care in Britain ***(Goldberg & Bridges, 1988)***, Spain ***(Lobo et al., 1996)***, Nigeria ***(Ohaeri & Odejide, 1994)*** and elsewhere ***(Ustun & Sartorius, 1995)***.

The WHO cross national study of mental disorders in primary care studied 5438 patients from 15 centers in 14 countries (Ankara, Athens, Berlin

and Mainz, Bangalore, Groningen, Ibadan, Manchester, Nagasaki, Paris, Rio de Janerio, Santiago, Seattle, Shanghai, Verona) ***(Ustun & Sartorius, 1995)***.

Although a high prevalence of somatization was found across all centers the rates varied markedly ***(Gureje et al., 1997)***. Especially high rates of somatization were found at the two south American sites.

The finding of substantial differences in this WHO study is even more interesting given the fact that comparing primary care settings would be expected to minimize differences between patient groups in as much as the expectations for illness behaviour are likely to be similar across sites ***(Kirmayer & Young, 1998)***.