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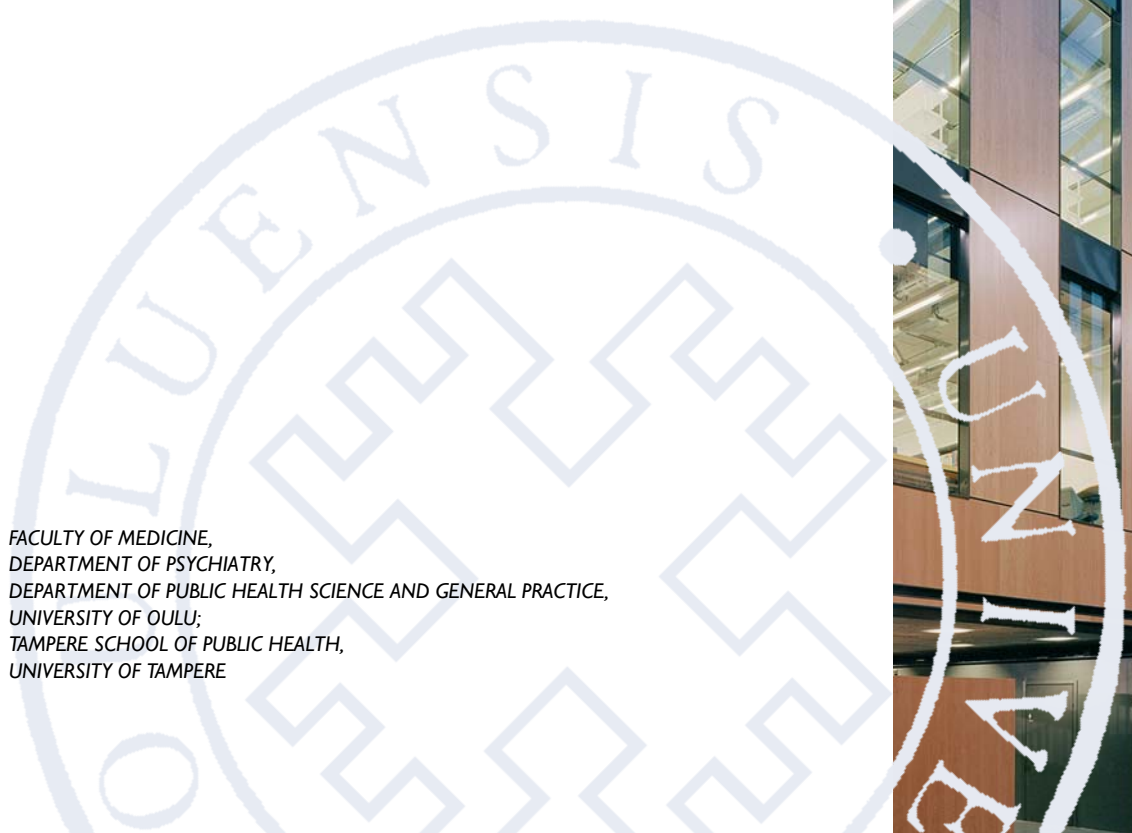
# SOMATIZATION IN YOUNG ADULTS

*THE NORTHERN FINLAND 1966  
BIRTH COHORT STUDY*

FACULTY OF MEDICINE,  
DEPARTMENT OF PSYCHIATRY,  
DEPARTMENT OF PUBLIC HEALTH SCIENCE AND GENERAL PRACTICE,  
UNIVERSITY OF OULU;  
TAMPERE SCHOOL OF PUBLIC HEALTH,  
UNIVERSITY OF TAMPERE

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*JUHA T. KARVONEN*

**SOMATIZATION IN YOUNG ADULTS**

The Northern Finland 1966 Birth Cohort Study

Academic Dissertation to be presented, with the assent  
of the Faculty of Medicine of the University of Oulu,  
for public defence in Auditorium I, Building PT1 of  
the Department of Psychiatry (Peltolantie 17),  
on September 28th, 2007, at 12 noon

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## **Karvonen, Juha T., Somatization in young adults. The Northern Finland 1966 Birth Cohort Study**

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### ***Abstract***

Somatization is a widespread phenomenon causing subjective suffering and disability.

The aim of the study was to assess somatization disorder (SD) and somatization symptoms among young adult population and their associations with sociodemographic factors, alexithymia and temperament as well as psychiatric comorbidity.

Various suggestions have been presented to operationalize somatization but none of them has been shown to be superior to others. In this study two definitions were used: SD by DSM-III-R classification diagnostic criteria and "somatization" meaning four or more symptoms of the 35 symptoms of DSM-III-R SD criteria.

The study population was a subsample of the Northern Finland Birth Cohort 1966 (NFBC 1966), consisting of cohort members living in Oulu (N = 1,609) on January 1<sup>st</sup> 1997. The NFBC 1966 is a general population birth cohort of 12,058 live-born children covering 96.3% of all deliveries in the catchment area.

The best-estimated procedure was used for assessment of psychiatric morbidity including SD and somatization. Data were collected from the Finnish Hospital Discharge Register and from all available outpatient and inpatient records. Data on education were gathered from Statistics Finland. Other sociodemographic variables, alexithymia and temperament scores were drawn from questionnaires of the field study conducted in 1997 and from earlier follow-up studies.

The prevalence of SD was 1.1% (N = 18). Of the subjects 6.1% (N = 97) had somatization. The female-to-male ratio was 5:1 and 6:1, respectively. SD was not recognized by any of the treating physicians, at least not documented in case notes. The observed occurrences of SD and somatization were at a level comparable with earlier international population studies. Somatization did not associate with depression or alexithymia, and neither could a characteristic temperament profile be recognized. Somatization was associated with psychological distress.

These results indicate a need for training physicians to recognize SD and somatization and its comorbidity. This will have implications both for psychiatry and other medical specialties regarding collaboration and underlines the importance of liaison-psychiatry at general hospitals. The results suggest a need for more studies about the etiology and development of SD and somatization.

*Keywords:* affective symptoms, alexithymia, cohort studies, DSM-III-R, Finland, general population study, liaison psychiatry, somatization, somatoform disorders, TAS-20, TCI, temperament



## **Karvonen, Juha T., Somatisaatio nuorilla aikuisilla. Tutkimus Pohjois-Suomen vuoden 1966 syntymäkohortista**

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### ***Tiivistelmä***

Somatisaatio on yleinen ilmiö, josta aiheutuu subjektiivista kärsimystä ja toimintakyvyn laskua.

Tämän tutkimuksen tarkoitus oli arvioida somatisaatiohäiriön ja somatisaatio-oireilun yleisyyttä nuorilla aikuisilla sekä näiden ilmiöiden yhteyttä sosiodemografisiin tekijöihin, aleksitymiaan, temperamenttiin ja psykiatriseen sairastavuuteen.

Somatisaation käsitteellistämiseksi on esitetty useita vaihtoehtoja mutta mikään niistä ei ole osoittautunut muita paremmaksi. Tässä tutkimuksessa käytettiin kahta määritelmää: DSM-III-R -diagnoosiluokituksen mukaista somatisaatiohäiriön diagnoosia tai somatisaatio-oireilua, jossa esiintyy neljä tai useampia DSM-III-R:n 35 somatisaatiohäiriön oireesta.

Tutkimusaineiston muodostivat Pohjois-Suomen vuoden 1966 syntymäkohortin ne jäsenet, jotka asuivat Oulussa 1. tammikuuta 1997 (N = 1,609). Alkuperäinen kohortti koostuu 12,058 elävänä syntyneestä tutkittavasta, mikä kattaa 96.3 % kaikista synnytyksistä Pohjois-Suomessa.

Niin kutsutun best-estimated -menettelyn avulla arvioitiin tutkittavien psykiatrista sairastavuutta mukaan lukien somatisaatiohäiriö ja -oireilu. Tietoa kerättiin sairaaloiden poistoilmoitusrekistereistä. Avohoidon sairauskertomustieto koottiin kattavasti. Koulutusasteesta saatiin tieto Tilastokeskukselta. Muita sosiodemografisia tekijöitä, aleksitymiaa ja temperamenttia arvioitiin vuoden 1997 kenttätutkimuksen ja aiempien seurantatutkimusten tietojen avulla.

Somatisaatiohäiriön esiintyvyys oli 1.1 % (N = 18). Somatisaatio-oireita todettiin 6.1 % (N = 97) tutkittavista. Naisten osuus oli somatisaatiohäiriössä 5:1 ja somatisaatio-oireilussa 6:1. Osoitettiin, että lääkärit eivät tunnistaneet somatisaatiohäiriötä, ainakaan sitä ei oltu kirjattu sairauskertomuksiin. Havaitut somatisaatiohäiriön ja -oireilun esiintyvyydet ovat sopusuhteissa aiempien kansainvälisten tutkimusten kanssa. Somatisaatio-oireilu ei liittynyt masennukseen tai aleksitymiaan eikä somatisaatio-oireilusta kärsiville tutkittavilla todettu tyypillistä temperamenttiprofiilia. Somatisaatio liittyy psyykkiseen stressiin.

Johtopäätöksenä voidaan todeta, että lääkäreille tulisi tarjota koulutusta somatisaatiohäiriön ja -oireilun tunnistamisessa. On tärkeää tunnistaa somatisaatio ja siihen liittyvä oheissairastavuus. Havainnot korostavat yleissairaaloiden yhteistyopsykiatrian ja muiden erikoisalojen yhteistyön merkitystä somatisaatiosta kärsivien potilaiden tutkimuksessa ja hoidossa. Somatisaatiohäiriön ja -oireilun etiologian ja kehittymisen selvittämiseksi tarvitaan uusia tutkimuksia.

*Asiasanat:* aleksitymia, DSM-III-R, kohorttitutkimus, somatisaatio, somatoformiset häiriöt, TAS-20, TCI, temperamentti, väestötutkimus, yhteistyopsykiatria





*To Jouko*



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Oulu August 12<sup>th</sup>, 2007

Juha T. Karvonen



## Abbreviations

ANOVA	Analysis of Variance
B.C.	Before Christ
CI	Confidence Interval
CIDI	Composite International Diagnostic Interview
CNS	Central Nervous System
DCPR	Diagnostic Criteria for Psychosomatic Research
DDF	Difficulty in Describing Feelings
DIF	Difficulty in Identifying Feelings
DIS	Diagnostic Interview Schedule
DSM	Diagnostic and Statistical Manual of Mental Disorders
ECA	Epidemiologic Catchment Area study
EOT	Externally Oriented Thinking
FHDR	Finnish Hospital Discharge Register
HA	Harm Avoidance
HSCCL	Hopkins Symptom Checklist
ICD	Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death
MANOVA	Multivariate Analysis of Variance
MSD	Multisomatoform Disorder
MUS	Medically unexplained symptom
NA	Not Available
NS	Novelty Seeking
NFBC	Northern Finland Birth Cohort
PS	Persistence
PSD	Polysymptomatic Somatoform Disorder
RD	Reward Dependence
SCID	Structured Clinical Interview
SD	Somatization Disorder
SOMS	Screening for somatoform symptoms
SSI	Somatic Symptom Index
TAS	Toronto Alexithymia Scale
TCI	Temperament and Character Inventory
TSIA	Toronto Structured Interview for Alexithymia
WHO	World Health Organization





## List of original publications

This thesis is based on the following original publications, which are referred to in the text by the Roman numerals I-IV.

- I Karvonen JT, Veijola J, Jokelainen J, Läksy K, Järvelin MR & Joukamaa M (2004) Somatization Disorder in young adult population. *General Hospital Psychiatry* 26: 9-12.
- II Karvonen JT, Joukamaa M, Herva A, Jokelainen J, Läksy K & Veijola J (2007) Somatization symptoms in young adult Finnish population - associations with sex, educational level and mental health. *Nordic Journal of Psychiatry* 61: 219-224.
- III Karvonen JT, Veijola J, Kokkonen P, Läksy K, Miettunen J & Joukamaa M (2005) Somatization and alexithymia in young adult Finnish population. *General Hospital Psychiatry* 27(4): 244-9.
- IV Karvonen JT, Veijola J, Kantojärvi L, Läksy K, Miettunen J & Joukamaa J (2006) Temperament profiles and somatization – an epidemiological study of young adult people. *Journal of Psychosomatic Research* 61(6): 841-6.



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# 1 Introduction

The word “somatization” stems from the word *soma* which refers to the body. *Soma* is used in the terms somatic (of the body), somatoform (bodylike) and psychosomatic (the union of the mind and the body) (Chaturvedi & Desai 2006). Somatization is a widespread phenomenon throughout different cultures (Isaac & Janca 1996, Kirmayer & Young 1998) and health-care systems (Lipowski 1988). Subjects suffering from somatization perceive themselves as sick and physically disabled. The help-seeking behavior of persons with somatization symptoms may be difficult for physicians to understand. This may easily lead to problems in the patient-physician relationship (Lin *et al.* 1991, Hahn *et al.* 1996, Jackson & Kroenke 1999, Kroenke 2000, Hartz *et al.* 2000).

Initially, somatization was thought to be similar to hysterical conversion (Fink 1996). Nowadays somatization has two meanings: firstly, the expression of psychological illness through physical symptoms (Bridges & Goldberg 1985), as in the term “presenting somatization” (Kirmayer & Robbins 1991, Craig *et al.* 1993), and secondly, repeated medical help-seeking for multiple medical symptoms without organic disease (Escobar *et al.* 1998a), as for example in “somatization disorder” (Kirmayer & Robbins 1991).

The first definition was influenced by psychoanalytic theories. This definition suggested that there existed a causal relation between the experience of psychological distress and the presentation of somatic symptoms (Bridges *et al.* 1991, Craig *et al.* 1993). In contrast to this, Lipowski defined somatization as a tendency to experience and communicate somatic distress and symptoms. Absence of emotional experience is a central descriptive feature in this definition of somatization (Lipowski 1988).

Various suggestions have been proposed for methods to operationalize somatization and its severity, but none of them has been shown to be superior to others (Reid *et al.* 2002, De Gucht & Fischler 2002, Creed 2006). There are five widely used definitions of somatization in the literature: somatization, somatoform disorders, Somatization Disorder (SD), functional syndromes and medically unexplained symptom (MUS). The epidemiology of somatization disorder and somatization symptoms have not been studied previously in Finland.

This thesis is part of the psychiatric follow-up project of the ongoing Northern Finland 1966 Birth Cohort (NFBC 1966). A subsample within the population-based birth cohort NFBC 1966, the “Oulu Study”, provided the

possibility to evaluate the role of SD and somatization among young adult cohort members.

## **2 Review of the literature**

### **2.1 History of the somatization concept**

#### ***2.1.1 Ancient concept of somatization (i.e. hysteria)***

The first known description of hysteria dates back to the Egyptian papyrus rolls from about 1900 B.C.. Those rolls assumed that hysteria was caused by a wandering womb – that is, a displacement of the uterus (hystera in Greek) (Millon 2004).

Hippocrates' (460-370 B.C.) concept of hysteria was much like the one described in the early Egyptian papyrus rolls (Howells 1975). Although Hippocrates located mental disorders to the brain, he thought that hysteria was a disorder of uterine origin. The uterus could be displaced due to drying up and weight loss caused by celibacy. It searched for moisture and rose towards the hypochondrium and abdomen. Respiratory difficulty and globus hystericus (a feeling of lump in one's throat) were believed to be due to the pressure caused by the uterus that had wandered to the abdominal cavity or to the throat. If the uterus came to rest at the heart, the patient would feel anxiety and obstipation (Fink 1996).

Galen (129-199) believed that hysteria was related to the uterus and that women produced a secretion in the uterus analogous to the male sperm. The retention of this secretion in the uterus due to, for example, celibacy caused hysteria. Such a "seminal retention" could result in poisoning of the blood or cooling of the body that would give rise to hysterical attacks. As male sperm could also be retained, men could get hysteria as well. Both Hippocrates and Galen considered hysteria a concrete logical reaction to a temporary organic imbalance of the body which was predominantly caused by sexual factors (Fink 1996).

St. Augustine (354-430) altered the attitude towards hysteria. He considered a hysteric person to be a human being who is more or less willfully possessed by or in league with the devil. Consequently, the cure of hysteria was the responsibility of the theologians. In this period and in the following centuries the impression that a person with hysteria was willfully joined with the devil resulted in severe suffering and, at times, torture for the hysterics (Fink 1996).

### **2.1.2 Modern Time concept of somatization (i.e. hysteria)**

The 1600s brought understanding of the central nervous system (CNS) and ideas that unexplained somatic symptoms were a product of the brain. In 1603 Jordan stated that the brain was the primary seat of hysteria. Furthermore, he said that perturbations of the mind were responsible for the disease (Fink 1996).

Willis (1621-1675) regarded hysteria in women as a nervous disorder of the brain (Howells 1975). Sydenham (1624-1689) considered hysteria a psychological disease of the mind and not of the body (Howells 1975). He assumed that hysteria was among the most common of chronic diseases and proposed that emotions can generate and simulate physical diseases. Whytt (1714-1766) categorized less severe mental conditions into three groups: hysteria, hypochondriasis (i.e. persistent belief in and preoccupation of having a serious physical disease) and nervous exhaustion (Shorter 1992). Beard (1839-1883) later referred to nervous exhaustion as neurasthenia (feeling of exhaustion after minor effort) (Millon 2004).

Charcot (1825-1893) considered hysteria in women as an inherited organic functional disorder of the nervous system. Hysteria produced symptoms that emerged throughout the body. Charcot found the symptom pattern in hysteria to be rather uniform from one patient to another and described three characteristic symptom types of hysteria: 1) sensory disturbances, such as hemianesthesias; 2) disturbances of the special senses, such as a narrowing of the field of vision and deafness; 3) motor disturbances, such as aphonia, paralysis, and general convulsive fits with *arc-de-cerle* (so called opisthotonus; only the back of head and heels of the patient lying on the ground) (Shorter 1992, Fink 1996). Briquet (1796-1881) advocated also the notion that the cause of hysteria was organic brain damage located in “that portion of the encephalon where affective functions are located”. He thought that developmental factors and life experiences may play a pathogenic role contributing to the symptoms of the disorder (Shorter 1992, Hollifield 2004, Millon 2004).

Spinal irritation theory and reflex theory were the leading theories in medical science, especially in association with hysteria in the last half of the nineteenth century. According to the spinal irritation theory the disease manifested if the tissue was either too little excited (asthenic) or overexcited (sthenic). Local irritation of the spinal marrow (spinal irritation) would cause symptoms from the reflex arcs (Howells 1975). To diagnose a patient, physicians looked for signs of irritation – so called “tender points” in the musculature, tenderness between the



shoulders, and so forth. In 1857 Romberg (1795-1873) wrote that hysteria is a “reflex neurosis” caused by irritation of the genital organs. The attacks spread themselves via sympathetic ganglia of the abdomen and not the spine. Female genitals were in a condition of permanent irritation, in contrast to male genitals, which were only occasionally irritated. Consequently, women had more hysterical attacks (Shorter 1992).

### **2.1.3 The 20<sup>th</sup> century concept of somatization**

The early 20<sup>th</sup> century brought about a change in the somatization theory: unexplained physical symptoms were thought to be primarily psychological. Babinski (1857-1932) defined hysteria in 1901 as any symptoms that could be induced by suggestion and abolished by persuasion. Unexplained medical symptoms moved to the field of psychiatry. Hysteria, hypochondria and neurasthenia were considered the three most frequently occurring mental illnesses. Hypochondria was considered the male counterpart to hysteria in females, whereas neurasthenia occurred equally in both sexes (Howells 1975, Shorter 1992, Fink 1996).

After Charcot’s death around the turn of 19<sup>th</sup> and 20<sup>th</sup> centuries hysterical pseudoepileptic symptoms disappeared. The reason for this change may partly be the improvements in medical ability to diagnose and rule out organic brain diseases, especially epilepsy. The development in psychiatric nosology and classification systems also helped to classify former hysteria diagnoses as schizophrenia or other psychiatric disorders. The introduction of the diagnoses of psychoneuroses and the appearance of psychoanalysis might imply that hysteria lost ground to anxiety neurosis and other neuroses (Shorter 1992, Fink 1996).

Psychoanalysis is based on Freud’s (1856-1939) interest in hysteria (Millon 2004). Freud distinguished between two different phenomena characterized by the presence of somatic symptoms, namely conversion hysteria and neurasthenia. Conversion hysteria (psychoneurosis) was defined as psychic in origin (i.e., the symptoms were the symbolic expression of infantile sexual conflicts), and neurasthenia (an actual neurosis) was defined as somatic in origin (i.e., the symptoms were a direct consequence of unresolved sexual tension in the present) (Freud 1981).

The term somatization was first used by Stekel in 1924 (Stekel 1943, Shorter 1992), who defined it as a bodily disorder that arises as the expression of a deep-seated neurosis, especially of a “disease of the conscious”. The word "hysterical"

was replaced with synonyms such as functional, nonorganic, psychogenic or medically unexplained (Fink 1996).

Alexander (1891-1964) considered that psychosomatic patients have specific forms of unconscious conflicts rather than symbolic conversion (Alexander 1950). Thus, emotional states of these patients activated certain physiological reactions; for example, rage was specifically associated with cardiovascular responses, dependency needs characteristically stimulated gastrointestinal activity, and difficulties in social communication were associated with respiratory functions. To Alexander, psychosomatic disorders in adults reflected the reactivation of childhood emotional reactions (Shorter 1992, Millon 2004).

It was not socially accepted to have a psychogenic disorder or to be called “hysteric”. Therefore the symptoms were renamed, using names such as the chronic fatigue syndrome, myalgic encephalomyelitis, the Gulf War syndrome or burn-out syndrome (Shorter 1992). In the last few decades major changes have occurred in psychosomatic medicine. The paradigm has moved to a version of global holistic theory. This theory postulates that any human disease is in some sense both biologically grounded and psycho-socially conditioned, as suggested by Engel’s bio-psycho-social model, for example (Engel 1962, Brown 1993).

#### ***2.1.4 Somatization disorder in the DSM and ICD classification systems***

Currently, there are two diagnostic classification systems in psychiatry. The Diagnostic and Statistical Manual of Mental Disorders (DSM) system has been developed by the American Psychiatric Association. Nowadays the fourth edition (DSM-IV, (American Psychiatric Association 1994)) is in use. In Finland the DSM system is mostly used for research purposes in psychiatry. The official diagnostic classification system in Finland is the International Classification of Diseases (ICD) system of the World Health Organization (WHO). The tenth version ICD-10 came into use in Finland in 1996 (World Health Organization 1992).

##### ***The DSM classification***

The second edition of the DSM (DSM-II, published in 1968) divided unexplained physical symptoms into the diagnostic categories of neuroses, psychophysiological disorders (ten types), and special symptoms. Neurotic disturbances of this kind

were further divided into hysterical neuroses (conversion and dissociative types), neurasthenia, depersonalization, hypochondriasis and other neuroses (Hollifield 2004).

Briquet's syndrome was also included in the DSM-II. It is a chronic syndrome of recurrent symptoms in many different organ systems (Guze 1967). It begins before the age 30 years and is associated with psychological distress but without any demonstrable physical etiology. Characteristic symptoms include frequently bodily pains, gastrointestinal symptoms, pseudoneurological conversion symptoms, sexual and menstrual problems, anxiety and depressive symptoms, and patients believing that they have been sick for most of their lives. Perley and Guze described 59 different potential symptoms in 10 categories. They required at least 20 medically unexplained symptoms in nine categories for a diagnosis of Briquet's syndrome (Perley & Guze 1962, Cloninger *et al.* 1986a).

The third edition of the DSM (DSM-III, published in 1980) separated mental disorders with physical symptoms (subtyped organic mental disorders or somatoform disorders) from dissociative disorders, a new category. Conversion disorder, somatization, psychogenic pain, and hypochondriasis were classified as somatoform disorders (Hollifield 2004). To avoid overlapping, the criteria of Briquet's syndrome were modified in such a way that all psychic symptoms were eliminated, and only 37 somatic symptoms remained in the definition. The requirements for the sexes were differentiated in such a way that men had to fulfill 12 symptoms and women 14 symptoms. The syndrome was renamed Somatization Disorder (Swartz *et al.* 1986a, Swartz *et al.* 1986b, Kellner 1990, Swartz *et al.* 1991, Fink 1996).

In the DSM-III-R, disorders labeled as somatization were largely classified in the categories of Somatization Disorder (SD), Undifferentiated Somatoform Disorder, Adjustment Disorders with physical complaints, and Somatoform Disorder, not otherwise specified. SD was the presentation of multiple physical complaints in multiple organ systems for which no organic etiology can be found. SD was a chronic, undulating, and relapsing disorder that rarely remits completely (American Psychiatric Association 1987, Hollifield 2004).

DSM-IV requires eight symptoms from four clearly defined symptom groups to fulfill the diagnosis of SD. Symptoms should start before the age of 30 years. Because of these symptoms a patient has to seek treatment or to have a significant impairment in social, occupational or other important areas of functioning over a period of several years (American Psychiatric Association 1994).

### *The ICD classification*

In the early 1960s, the Mental Health Program of the WHO became actively engaged in a task aiming to improve the diagnosing and classification of mental disorders in the ICD classification system. The result of the program was imbedded in the Eighth Revision of the International Classification of Diseases (ICD-8) published in 1968 (Millon 2004).

In the Finnish translation of the ICD-8 (in 1969) the section of neurosis had various subcategories such as neurosis hysterica, hypochondrica and neurasthenia. The section psychosomatic diseases (morbi psychosomatici) was defined as physical disorders of presumably psychogenic origin and was divided by organ systems, for example the skin (cutanei), muscles and bones (musculorum et ossium) and the respiratory system (systematis respirationis). There were also sections for symptoms that associated with certain organs such as convulsions (symptomata systematis nervosi et organorum sensuum) and palpitation (symptomata organorum cardiovascularium et lymphaticorum). In the section of incompletely defined cases (casus male definiti) for example nervousness (nervosismus) and asthenia were represented.

The ICD-9 was published in 1977 (Millon 2004). The Finnish version of the ICD-9 was used from 1987 onwards. The fifth main chapter “Mental health disorders” of the Finnish version was based on DSM-III-R classification. It was used until 1996 when the Finnish version of ICD-10 was published. The section of neurosis included among others conversion syndrome, syndrome hypochondrica and SD. In the section “not in another sections defined syndromes” included chronic pain syndrome and psychogenic vomiting.

For a diagnosis of SD ICD-10 requires six medically unexplained symptoms in two separate symptom groups. The duration of symptoms has to be at least two years but there is no limitation concerning the age of onset. The symptoms should both cause persistent distress and repeated consumption of medical services. The ICD-10 emphasizes “persistent request for medical investigations in spite of repeated negative findings and reassurance by doctors that the symptoms have no physical basis” and exclusion of the possibility of “psychological causation” (World Health Organization 1992).

Fink *et al.* found a significant discrepancy in the rates of SD between the ICD-10 and DSM-IV diagnostic systems (Fink *et al.* 2004). The prevalence of SD was higher in the ICD-10 (5%) than in the DSM-IV (1.5%). The contrary was found in the prevalence of undifferentiated somatoform disorder, which was

lower for ICD-10 (0.7%) and higher (10%) for DSM-IV categories. The fact that the DSM-IV and ICD-10 classifications differ substantially gives rise to the currently existing confusion (Yutzy *et al.* 1995, Lipsanen *et al.* 2004).

## **2.2 Theories of somatization**

### **2.2.1 Psychodynamic theory**

Freud defined hysterical neurosis as a conversion mechanism against knowledge of an internal conflict. In his early work with Breuer, in “Studies in Hysteria” from 1895 (Breuer & Freud 1981) it was suggested that the person does not react in the actual situation to the psychological trauma but represses it. This trauma will later on manifest itself in the form of physical symptoms in defense of knowledge of the repressed memories. Freud suggested that hysteria was caused by sexual trauma or abuse during childhood, especially if it occurred at the time of the so-called phallic phase in the child’s sexual development. He believed that the somatic complaints of hysteria represented unconscious manifestations of the effects of that abuse or trauma. Later on he changed his view, believing instead that reported histories of sexual abuse or trauma represented childhood fantasy (Freud 1981, Pribor *et al.* 1993).

Stekel expressed the view that the body was translating the mental troubles of the individual into physiological language. Somatization was a method of communication often of symbolic significance, an “organ speech of the mind” (Stekel 1943).

Since the 1970s there has been a breaking up of psychoanalytic theories. Especially Kohut’s self-psychology and further development of it seem to help the understanding of the somatization phenomenon, at least in its most severe forms. Kohut’s basic hypothesis is that anxiety with the threat of defragmentation or disintegration of the self forms the most severe type of anxiety that a person can experience. Therefore he will defend himself against it with all his might. According to this model the physical symptoms can be regarded as an unspecific reaction pattern without symbolic meaning (Kohut 1988).

Somatization has been referred to as a defense mechanism. Defense mechanisms of this kind include denial, displacement or rationalization, or an attempt at conflict resolution (Kellner 1990). These defenses involve the turning away from unacceptable thoughts and situations towards a focus on physical

problems. For example, the patient may blame bodily symptoms of causing her/his failings. It seems likely that when the patient is concerned about physical symptoms, she/he manages to diminish attention from current psychic problems. The patient may become preoccupied with bodily sensations instead of dealing with an intolerable conflict. The patient may thus avoid blaming herself/himself for such problems, and in doing so avoid depression. Seen in this way, somatization may have an adaptive function in protecting the patient from a more severe mental disorder. Somatizing is therefore not always pathological (Pilowsky 1978, Bridges *et al.* 1991, Taylor & Mann 1999).

### **2.2.2 Cognitive theory**

According to the model of emotional awareness (Lane & Schwartz 1987) the capacity to consciously experience feelings is a cognitive skill. In this model somatization is linked to a deficit in emotion processing. Niemi *et al.* suggested that somatization is associated with cognitive dysfunctioning, particularly impaired control of attention and memory (Niemi *et al.* 2002).

The “cognitive representation of illness” model (Horne 1997) describes how an individual constructs an internal representation of what is happening when one experiences physical or psychological symptoms. It suggests that, no matter what the nature of the symptoms, most people organize their thinking around the five key themes of identity (What is it?), cause (Why has it happen?), timeline (How long will it last and will it recur?), consequences (What effects will it have?) and cure/control (What can I do to make it go away?). The key elements of a psychological approach are recapitulation of the problem using the patients’ explanatory model and acknowledging to the patient that the symptoms, distress and disability are genuine. The nature of somatic complaints will be explained to the patient, concentrating on the patient’s own explanatory models (Patel & Sumathipala 2006).

People with somatization may have a lower threshold for maladapted reaction to benign normal body sensations. They experience minor physical complaints as catastrophic physical events and misinterpret these complaints as being caused by a physical disease (Fink *et al.* 2005). Martin and Pihl have suggested that failure to regulate and modulate stress-related emotions at the cognitive level may result in exaggerated physiological and behavioral responses to stressful situations and increased vulnerability to disease (Martin & Pihl 1985).

Barsky used the term “somatosensory amplification” to refer to the cognitive tendency to experience somatic sensations as pathological rather than normal. The amplification of benign bodily sensations may be related to more general processes of somatic symptom reporting and health appraisal (Barsky 1992).

Attribution is another cognitive process whereby somatic sensations are interpreted in the context of the body and its physical and social environment. Using the example of fatigue, attributions can either be normalizing (“I’m tired because I’m overworking and unfit”), somatic (“I’m tired because my muscles have been weakened by a virus”), or psychological (“I’m tired because I have depression”) (Burton 2003). The attribution styles are also determined by the culture and organization of the health service (Duddu *et al.* 2006). While medically unexplained symptoms tend to change over time, attributional style appears to be much more consistent (Garcia-Campayo *et al.* 1997, Simon & Gureje 1999).

### **2.2.3 Illness behavior in somatization**

Patients with persistent somatization are often observed to demonstrate abnormal illness behavior, a concept introduced in 1969 by Pilowsky (Pilowsky 1969). Illness behavior includes such features as patterns of health care utilization, urging physicians to do investigations, requesting medications or treatments, being disabled at work, avoidance of physical activity, and expression of symptoms to family members and significant others (Barsky & Borus 1999, Duddu *et al.* 2006). The patient’s abnormal illness behavior is disproportionate to the clinician’s assessment of objective pathology and the patient persists in the sick role (Chaturvedi *et al.* 2006).

Somatization can be seen as a search for aid, an attention-seeking device. On the other hand, most somatizing patients appear to suffer because of their symptoms rather than to profit from them (Kellner 1990). Waddell suggested that the behavioral signs and symptoms may be a pattern of communication between the patient and the doctor (Waddell *et al.* 1984). Somatization is closely related to illness behavior and frequent use of health services (Lipowski 1988, Katon *et al.* 1991, Portegijs *et al.* 1996, Karlsson *et al.* 1997, Jyväsjarvi *et al.* 2001).

### **2.2.4 Biological aspects**

There is no known tissue pathology in SD. Somatizing patients may be more somatically sensitive and often more accurate in distinguishing between small differences in stimuli compared to other people. The increased intensity and duration of pain sensation may be related to altered peripheral neuromuscular processing and/or to not suppressed ascending sensory input in the CNS (Rief *et al.* 1998, Rief & Auer 2000, Rief & Auer 2001, Fink *et al.* 2005, Dantzer 2005, Rief & Barsky 2005, Kirmayer & Looper 2006).

There may be a complex dysregulation of the hypothalamic-pituitary-adrenal axis in patients with somatization. Hypoactive biological stress response is a risk factor for the development of functional symptoms and syndromes. This activity level may be reduced by illness or stress (Kirmayer & Looper 2006). Rief and colleagues found a different immune alteration in somatizing syndrome compared to major depression (Rief *et al.* 2002).

The serum amino acids may contribute in somatization process by the serotonergic and noradrenergic systems that modulate pain perception and the perception of physical symptoms. This modulation is independent from their effects on major depression. Physical weakness, bodily exhaustion and fatigue may not only be triggered by the CNS, but also by energy metabolism in the muscles. An imbalance of energetic processes in the peripheral muscle may contribute to muscle pain. The substantial association between tryptophan availability to the brain and somatoform symptoms persisted even after controlling for other influences. Patients with somatization exhibit a decrease in blood levels of serotonergic amino acids compared with control individuals. These results suggest that the serotonergic system is involved in the development of functional somatic symptoms in general (Rief *et al.* 2004). Regional cerebral glucose hypometabolism and enlargement of the caudate nuclei might be associated with the pathophysiology of somatization (Hakala *et al.* 2002, Hakala *et al.* 2004, Hakala *et al.* 2006).

## **2.3 Alexithymia theory**

Alexithymia (from the Greek words *a* for lack and *lexis* for word, and *thymos* for emotion) refers to a specific disturbance in emotional processing. Alexithymia is manifested clinically by difficulties in identifying and verbalizing feelings, in elaborating fantasies. Nowadays, alexithymia refers to a multifaceted construct of



personality features including the following core traits: difficulty in recognizing and verbalizing subjective feelings, a cognitive style characterized by absence of fantasy life and a tendency to focus on factual aspects and to recount minute details of external events. Alexithymia is assumed to involve an impaired capacity to distinguish one's feelings from the somatic sensations that accompany emotional arousal. This leads to somatosensory amplification and misinterpretation of somatic sensations as signs of physical illness (i.e., somatization) and to exacerbated responses in the autonomic nervous system and neuroendocrine system (Taylor 1984, Taylor *et al.* 1997, Gundel *et al.* 2004, Lipsanen *et al.* 2004).

Initially, it was thought that alexithymia predisposed the patient particularly to physical diseases. More recently, it has been suggested that alexithymia may be a predisposing factor for various psychiatric problems such as medically unexplained symptom (MUS), eating disorders, and substance dependence (Taylor *et al.* 1997, Kooiman *et al.* 2002). When the concept of alexithymia and its association to the predisposition of somatoform disorders was created, a link between hysteria and alexithymia was suggested (Yutzy *et al.* 1995, Lipsanen *et al.* 2004).

The alexithymia construct deals with psychological characteristics originally thought to be common among patients with classical psychosomatic illnesses. It was argued that alexithymic individuals had never learned to express emotions verbally because of a severe disturbance in their early psychosocial development or even because of some pathology in brain functioning, such as perhaps a defect in interhemispheric communication (Salminen *et al.* 1995).

There has been controversy and debate as to whether alexithymia is a stable personality trait, a transient state secondary to a stressful situation or a coping response to chronic illness (Taylor *et al.* 1997, Honkalampi *et al.* 2001). The theory of stable alexithymia traits postulates that alexithymia plays a central role in the pathogenesis of somatization (Taylor *et al.* 1997, Kooiman *et al.* 2000). A recent review concluded that there is an association between different self-reporting measures of somatization and alexithymia, but the evidence is not very strong (De Gucht & Heiser 2003). The existing studies have yielded generally consistent evidence of increased levels of alexithymia in somatoform disorders. Patients with somatoform disorders were also found to show elevated alexithymia scores when compared to medically ill patients (Kooiman *et al.* 2000).

An essential assumption underlying alexithymia theory is that the failure to experience complex emotional states is associated with exaggerated or

dysregulated autonomic activation. Lumley has suggested that alexithymia is associated with tonic physiological hyperarousal (Lumley *et al.* 1996). However, empirical findings of experimental studies exploring this hypothesis have been contradictory. Instead, alexithymia may contribute to somatic symptoms by affecting illness behavior through cognitive and social mechanism (Lumley *et al.* 1996, Taylor & Bagby 2004, Waller & Scheidt 2006).

## **2.4 Temperament and somatization**

Somatization can also be regarded as a personality trait, akin to temperament (Bass & Murphy 1995). Personality is a complex pattern of thoughts, emotions, and behaviors that is thought to be stable across time and different situations (American Psychiatric Association 1994). Several models have been proposed for classifying personality and temperament. The model of Cloninger is one recently adopted model for classifying temperament in psychiatric research. This model includes four genetically homogeneous and largely independent profiles of temperament: novelty seeking (NS), harm avoidance (HA), reward dependence (RD) and persistence (PS) (Cloninger 1986, Cloninger 1987, Cloninger *et al.* 1993, Cloninger *et al.* 1994).

Other commonly used personality models include Cattell's model of sixteen personality factors measured with the 16PF Questionnaire (Cattell 1949), Eysenck's three-factor model of personality measured with the Eysenck Personality Questionnaire (Eysenck & Eysenck 2007), and the five-factor model of personality measured with the Neuroticism, Extraversion, Openness (NEO) Personality Inventory (Costa & McCrae 1985) and the NEO Five Factor Inventory (Costa & McCrae 1992). The Minnesota Multiphasic Personality Inventory (MMPI) also includes scales related to personality and psychopathology (Dahlstrom *et al.* 1982).

Only few studies have dealt with somatization and temperament. Battaglia *et al.* reported a positive association between somatization and high NS in a female psychiatric outpatient sample (Battaglia *et al.* 1998). Russo *et al.* found an association between NS and somatization in three medical outpatient samples (Russo *et al.* 1994). Recently Hakala *et al.* reported low NS and high HA in female SD outpatients (Hakala *et al.* 2006). According to Cloninger's theory of personality, a temperamental pattern of high NS and low HA may lead to chronic somatic anxiety and can be more specifically identified by clinical presentation of somatization (Guze *et al.* 1986, Cloninger 1986).

Somatic anxiety is characteristic of patients with Briquet's syndrome (or SD) and of some individuals with histrionic or antisocial personality. Cloninger and colleagues found in studies with adopted Swedish men and women that probably the same etiologic factors lead to male-limited alcoholism but are expressed as somatization in women (Bohman *et al.* 1984, Cloninger *et al.* 1984, Sigvardsson *et al.* 1984, Sigvardsson *et al.* 1986, Cloninger *et al.* 1986b).

Garyfallos *et al.* found that histrionic personality disorder and dependent personality disorder discriminated best the somatoform disorders from the control group. Frick *et al.* concluded that behavioral disinhibition may be a common predisposition that underlies both antisocial behavior and somatization (Frick *et al.* 1995). However, findings from a community sample (Simon & von Korff 1991), primary care patients (Rost *et al.* 1992), and psychiatric outpatient services (Stern *et al.* 1993) did not show significant association between the two disorders. Somatoform patients with a concomitant personality disorder also manifested more severe overall psychopathology and a worse level of functioning than those without it (Garyfallos *et al.* 1999).

According to the DSM-III-R criteria (American Psychiatric Association 1987) the PDs are classified into three clusters: Cluster A (paranoid, schizoid and schizotypal PD), cluster B (antisocial, borderline, histrionic and narcissistic PD) and cluster C (avoidant, dependent, obsessive-compulsive and passive-aggressive PD). Persons with SD are more likely to have an underlying personality disorder or traits, particularly those belonging to cluster B (Stern *et al.* 1993). Passive-dependent, histrionic, and sensitive-aggressive traits have been shown to be two times more prevalent among SD patients than among patients with anxiety and depression. Conversely, there was a high prevalence of SD in patients diagnosed with borderline personality disorder. This association may reflect a similar pattern of social interactions between SD patients and those with cluster B personality disorders (Mai 2004).

Bass and Murphy have argued persuasively that the early onset and enduring nature of somatoform disorders indicate that they can be understood as an abnormality of personality development rather than a psychiatric disorder. Between two thirds and three quarters of patients with chronic somatoform disorders in secondary health care have personality disorders (Bass & Murphy 1995, Taylor & Mann 1999, Noyes *et al.* 2001).

## 2.5 Epidemiology of somatization

### 2.5.1 Occurrence of somatization

In population studies with standardized and structured interviews by laymen (Swartz *et al.* 1986a, Bland *et al.* 1988, Wells *et al.* 1989, Lee *et al.* 1990a, Lee *et al.* 1990b, Swartz *et al.* 1991) or professional interviewers (Wittchen *et al.* 1992, Faravelli *et al.* 1997, Lieb *et al.* 2000) using DSM-III and DSM-III-R criteria the prevalence of SD has been found to be 0.1 to 0.8%. The estimated prevalence of SD presented in Table 1 is based on wider definitions starting from Briquet's syndrome to DSM-IV and ICD-10 criteria (Weissman *et al.* 1978, Roca *et al.* 1999, Rief *et al.* 2001, Ladwig *et al.* 2001, Grabe *et al.* 2003). The occurrence of SD has varied between 0.02 and 1.84% (Table 1).

The criteria for somatoform disorders in the ICD and DSM classifications have been considered to be too broad and the criteria for SD too stringent and complex to use (Rief & Sharpe 2004, Mayou *et al.* 2005). Therefore some researchers have suggested modified symptom thresholds stating that sub-threshold variants of somatoform disorders have comparable morbidity to the prototypical SD. Examples of such approaches include the Somatic Symptom Index (SSI4,6; requiring four somatization symptoms for males and six for females; SSI3,5 has also been used: three somatization symptoms for males and five for females and SSI5,5: five for both sexes, respectively) (Escobar *et al.* 1989), Multisomatoform Disorder (MSD) (Kroenke *et al.* 1997), polysymptomatic somatoform disorder (PSD) (Rief & Hiller 1999) and chronic somatization in the Diagnostic Criteria for Psychosomatic Research (DCPR) (Fava *et al.* 1995).

The estimated prevalence of somatization symptoms is based on the occurrence of abridged definition of somatization only in population studies and has been observed to vary between 0.7%-20.1% when using SSI4,6. In two studies SSI5,5 gave the occurrences 12.9% and 14.9%. One study with SSI3,5 showed the occurrence of somatization to be 23.6%, but in this study Screening for somatoform symptoms (SOMS) was used (Table 2).

Swartz and colleagues reported the occurrence of somatization symptoms in the Epidemiologic Catchment Area study (ECA) to be 11.6% assessed with the SSI4/6 (Swartz *et al.* 1991). Escobar and colleagues found in Puerto Rico the occurrence to be 19.9% assessed with SSI4,6 (Escobar *et al.* 1989). Among a population sample of adolescents and young adults Lieb and colleagues found the

lifetime prevalence of somatization to be 1.7% measured with SSI4/6 (Lieb *et al.* 2000, Lieb *et al.* 2002). Ladwig and colleagues defined somatization by ICD-10 criteria (six or more symptoms from at least two different body sites without an identifiable organic cause) and found a prevalence of 1.8% in a population-based survey (Ladwig *et al.* 2001).

Rief and colleagues reported in a population sample the prevalence of somatization to be 23.6% measured with SSI3/5, 16.0% measured with SSI4/6 and 12.5% with PSD (Rief *et al.* 2001). In an immigrant population sample in Israel, Ritsner and colleagues found that the 6-month prevalence of somatization measured was 13.8% with SSI4/6 and 14.9% with SSI5/5 (Ritsner *et al.* 2000). In a community sample DCPR criteria gave the prevalence of chronic somatization as 2% (Mangelli *et al.* 2006). Somatization is even more prevalent at different levels of health care: 10-35% in GP surgeries, and 30% or more in hospital-based studies (Fink 1992, Gureje *et al.* 1997, Karlsson *et al.* 1997, Garcia-Campayo *et al.* 1997, Escobar *et al.* 1998b, Schilte *et al.* 2000).

Some population studies have only reported the prevalence of somatoform disorders. Jacobi and colleagues found in the general adult population in Germany the lifetime prevalence of any somatoform disorders or syndromes (including SD, Undifferentiated SD, SSI4,6, Hypochondriasis and Pain Disorder) using DSM-IV criteria and CIDI interview among 4,181 subjects to be 16.2% (Jacobi *et al.* 2004). In a Norwegian study (N=2,066) using CIDI and DSM-III-R criteria the lifetime prevalence for somatoform disorders was 3.7% in the capital area Oslo (Kringlen *et al.* 2001). The same group interviewed 1,080 subjects in a rural area of Norway using CIDI and DSM-III-R criteria. The lifetime prevalence of somatoform disorder was 3.4% (Kringlen *et al.* 2006). In another Norwegian population study with 617 subjects the two-week prevalence of ICD-10 somatoform disorders was 5.9% (Sandanger *et al.* 1999).

**Table 1. Somatization disorder—studies in population-based samples using full diagnostic criteria.**

Author(s)	Name of the study and sample size (N)	Definition of Somatization Disorder (SD)	Prevalence of SD (%) and [number of subjects with SD (N)]	Female:Male Ratio * [Female(%):Male(%)] prevalence]
Weissman <i>et al.</i> (1978)	New Haven, Conn., urban (938)	Schedule for Affective Disorders and Schizophrenia (SADS) based Research Diagnostic Criteria (RDC) for Briquet's syndrome	SD = 0.4 [N = 2] current	Not Available (NA)
Lee <i>et al.</i> (1990a)	Seoul, Korea, urban (3,134)	Diagnostic Interview Schedule (DIS) for Diagnostic and Statistical Manual of Mental Disorders (DSM-II) DIS/DSM-III	SD = 0.03 [N = 1] lifetime	NA [0.06:0.00]
Lee <i>et al.</i> (1990b)	Korea, rural (1,966)	DIS/DSM-III	SD = 0.18 [N = NA] lifetime	0.73:1 [0.16:0.22]
Roca <i>et al.</i> (1999)	Island of Formentera, Spain (Stage I: 697) (Stage II: 242)	Stage I = General Health Questionnaire (GHQ-28) SD = 0.3 [N = NA] weighted Stage II = Schedules for Clinical Assessment in Neuropsychiatry (SCAN) for International Classification of Diseases (ICD) SCAN/ICD-10 DIS/DSM-III		NA [NA]
Canino <i>et al.</i> (1987)	Puerto Rico (1,503)	DIS/DSM-III	SD = 0.7 [N = 12] weighted lifetime	1:1 [0.7:0.7]
Swartz <i>et al.</i> (1991)	Epidemiologic Catchment Area (ECA) five sites: New Haven (5,106) Baltimore (3,584) St. Louis (3,228) Durham (4,141) Los Angeles (3,508) Piedmont (ECA) (3,798)	DIS/DSM-III	SD = 0.13 [N = NA] weighted lifetime All sites	11:1 [0.23:0.02] All sites
Swartz <i>et al.</i> (1986a)		DIS/DSM-III	SD = 0.16 [N = NA] weighted lifetime SD = 0.09 [N = NA] weighted lifetime SD = 0.13 [N = NA] weighted lifetime SD = 0.44 [N = NA] weighted lifetime SD = 0.03 [N = NA] weighted lifetime SD = 0.38 [N = 15] weighted lifetime	29:1 [0.29:0.01] NA [0.16:0.00] 14:1 [0.28:0.02] 5:1 [0.71:0.13] NA [0.05:0.00] 6.5:1 [NA]

**Table 1. Continued.**

Author(s)	Name of the study and sample size (N)	Definition of Somatization Disorder (SD)	Prevalence of SD (%) and [number of subjects with SD (N)]	Female:Male Ratio * [Female(%):Male(% prevalence)]
Hwu <i>et al.</i> (1989)	Taiwan Psychiatric Epidemiological Project Taipei, urban (5,005) two towns (3,004) six villages, rural (2,995)	DIS (Chinese modification (CM)) DIS-CM/DSM-III	SD = 0.4 [N = NA] lifetime SD = 1.3 [N = NA] lifetime SD = 1.0 [N = NA] lifetime SD = 0.7 [N = 5] one-year	NA [0.8:0.0] 1.1:1 [1.4:1.3] 2.5:1 [1.5:0.6] 5:0 [NA]
Faravelli <i>et al.</i> (1997)	Florence, urban (673)	SADS/DSM-III-R		
Rief <i>et al.</i> (2001)	German (2,050)	Screening for somatoform symptoms (SOMS)/DSM-IV and ICD-10	SD (DSM-IV) = 0.3 [N = 7] SD (ICD-10) = 0.3 [N = 6] SD = 0.84 [N = 5] weighted lifetime	6:1 [0.5:0.1] DSM-IV 5:1 [0.4:0.1] ICD-10 NA [1.60:0.00]
Wittchen <i>et al.</i> (1992)	Munich follow-up study (4839)	DIS/DSM-III		
Wells <i>et al.</i> (1989)	Christchurch psychiatric epidemiology study (1,498)	DIS/DSM-III	SD < 0.1 [N = 1] lifetime	NA [0.1:0.0]
Bland <i>et al.</i> (1988)	Edmonton (3,258)	DIS/DSM-III	SD = 0.0 [N = 2] weighted lifetime	NA [0.1:0.0]
Ladwig <i>et al.</i> (2001)	National health examination survey, German (7,460)	ICD-10 criteria for SD, six symptoms not explained by organic disease or anxiety/panic	SD=1.84 [N = 137]	0.96:1
Grabe <i>et al.</i> (2003)	Transitions in Alcohol Consumption and Smoking (4,075)	Munich-Composite international diagnostic interview (CIDI) M-CIDI/DSM-IV	SD = 0.02 [N = 1]	NA [NA]
Lieb <i>et al.</i> (2000) Lieb <i>et al.</i> (2002)	Early development Stages of psychopathology (3,021)	M-CIDI/DSM-IV	SD < 0.0 [N = 0]	NA [NA]

\* calculated from absolute numbers, when available, if not, calculated from female:male prevalence

**Table 2. Population-based studies of somatization.**

Author(s)	Name of the study and sample size (N)	Measure	Prevalence of somatization (%) and [number of subjects with somatization (N)]	Female:Male Ratio * [Female(%):Male(%) prevalence]
Swartz <i>et al.</i> (1991)	Epidemiologic Catchment Area (ECA) four sites: Baltimore (3,587) St. Louis (3,231) Durham (4,141) Los Angeles (3,511) Puerto Rico (1,513)	Diagnostic Interview Schedule (DIS) for Diagnostic and Statistical Manual of Mental Disorders (DSM-III); Somatic Symptom Index (SSI4,6) DIS/DSM-III/SSI4,6	SSI4,6 = 11.62 [N = Not Available (NA)] weighted lifetime All sites SSI4,6 = 9.48 [N = NA] weighted lifetime SSI4,6 = 10.41 [N = NA] weighted lifetime SSI4,6 = 20.06 [N = NA] weighted lifetime SSI4,6 = 9.32 [N = NA] weighted lifetime SSI4,6 = 19.9 [N = 301]	1.65:1 [14.30:8.68] All sites 1.74:1 [11.69:6.82] 1.63:1 [12.71:7.79] 1.43:1 [23.28:16.25] 2.18:1 [12.63:5.80] 1.1:1 [20:18]
Escobar <i>et al.</i> (1989)		DIS/DSM-III/SSI4,6		
Ritsner <i>et al.</i> (2000)	Immigrant Psychological Distress Project (966)	Brief Symptom Inventory (BSI)/SSI4,6/SSI5,5	BSI raw score=21.9 [N = 212] SSI4/6=13.8 [N = 133] SSI5/5=14.9 [N = 144]	NA 0.61:1 [18.5:30.1] 1.49:1 [14.6:9.8]
Rief <i>et al.</i> (2001)	German (2,050)	Screening for somatoform symptoms (SOMS)/DSM-IV/ICD-10/SSI3,5/Polysymptomatic SD (PSD)	SSI3/5 = 23.6 [N=484] two-years PSD = 12.5 [N=255] two-years	
Lieb <i>et al.</i> (2000)	Early development Stages of psychopathology (3,021)	Munich-Composite international diagnostic interview (M-CIDI)/DSM-IV/SSI 4,6	SSI4/6= 1.7 [N = 40] lifetime weighted prevalence	2.1:1 [2.3:1.1]
Lieb <i>et al.</i> (2002)				
Jacobi <i>et al.</i> (2004)	German Health Interview and Examination (4,181)	M-CIDI/DSM-IV	SSI4,6 = 5.6 [N = 235] weighted lifetime	1.9:1 [7.3:3.9]
Mak <i>et al.</i> (2004)	Los Angeles (1,747)	University of Michigan version of CIDI (UM-CIDI)/Symptom Checklist (SCL-90R)/SSI5,5	SSI5,5 = 12.9 [N = 225] past week	1.59:1 [16.0:9.9]
Grabe <i>et al.</i> (2003)	Transitions in Alcohol Consumption and Smoking (4,075)	M-CIDI/ DSM-IV/SSI4,6	SSI4/6 = 0.69 [N = 28]	NA

\* calculated from absolute numbers, when available, if not, calculated from female:male prevalence



### 2.5.2 Sex difference

Studies reporting a sex difference among subjects with somatization have constantly found a clear female dominance (Swartz *et al.* 1991, Fink 1992, Terre & Ghiselli 1997, Kolk *et al.* 2002). Women have a significantly higher somatization score and higher number of somatic symptoms than men (Escobar *et al.* 1987a, Swartz *et al.* 1989, Garcia-Campayo *et al.* 1998, Mak & Zane 2004, Huurre *et al.* 2005, Hiller *et al.* 2006).

Similarly, in the case of SD, a significant preponderance of females has been observed (Robins *et al.* 1984, Swartz *et al.* 1986a, Swartz *et al.* 1986b, Swartz *et al.* 1989, Swartz *et al.* 1991, Garcia-Campayo *et al.* 1998, Garyfallos *et al.* 1999). A 10-to-1 female-to-male ratio in the lifetime prevalence of SD has been found in the ECA (Swartz *et al.* 1991). Briquet's syndrome has predominantly been diagnosed in women (Cloninger *et al.* 1986a). In population-based studies with SD diagnosis the female-male-ratio has been reported to be between 0.73:1 and 29:1 (Table 1). The lowest ratio was reported from rural Korea (Lee *et al.* 1990b).

In some general population studies only female SD subjects have been identified (Wells *et al.* 1989, Wittchen *et al.* 1992, Faravelli *et al.* 1997). On the other hand, in studies conducted in Puerto Rico (Canino *et al.* 1987), rural Taiwan (Hwu *et al.* 1989), and Korea (Lee *et al.* 1990b), identical lifetime prevalences have been reported for males and females. Terre and Ghiselli also found a female predominance among somatizers (Terre & Ghiselli 1997), while Creed and Barsky did not (Creed & Barsky 2004). In population-based studies with abridged SD diagnosis the female-male-ratio has been reported to be between 0.8:1 and 2.2:1. The only study which reported male predominance used the SOMS and SSI3,5 criteria (Rief *et al.* 2001), while the other eleven studies that reported female-male-ratio found a female predominance, even the study of Rief *et al.* when used the PSD (Table 2).

In a large general population study by Hiller and colleagues the SOMS was used (Hiller *et al.* 2006). Somatization of any degree was found to be associated with female gender, also when only gender-neutral symptoms were analyzed. Women had an odds ratio (OR) of 1.49 to develop severe somatization symptoms and of 1.95 to develop somatization complaints of any degree when compared with men. In other population-based studies with somatoform disorder diagnosis the female-male-ratio has been reported to be between 1.6:1 and 3.4:1 (Sandanger *et al.* 1999, Kringlen *et al.* 2001, Jacobi *et al.* 2004, Kringlen *et al.* 2006).

### **2.5.3 Other sociodemographic correlates**

Somatization symptoms have been reported to be more prevalent among lower socioeconomic groups, rural residents, lower household income, lower education level and unmarried subjects (Robins *et al.* 1984, Swartz *et al.* 1986a, Swartz *et al.* 1986b, Escobar *et al.* 1987a, Swartz *et al.* 1989, Garcia-Campayo *et al.* 1998, Creed & Barsky 2004, Mak & Zane 2004, Huurre *et al.* 2005, Hiller *et al.* 2006).

Subjects with somatization symptoms tend to be older than average people (Escobar *et al.* 1987a, Swartz *et al.* 1989, Hiller *et al.* 2006). Only one study found somatization associated with younger age (Garcia-Campayo *et al.* 1998). A Norwegian study reported an equal prevalence of somatoform disorders in a rural and urban region (Kringlen *et al.* 2006). Swartz *et al.* reported that a higher mean number of somatic symptoms was significantly associated with urban residence (Swartz *et al.* 1989).

### **2.5.4 Association with psychiatric disorders**

Somatization has been associated with an increased probability of a psychiatric comorbidity such as mood, anxiety, and personality disorders (Smith *et al.* 1986, Escobar *et al.* 1987a, Katon *et al.* 1991, Simon & von Korff 1991, Swartz *et al.* 1991, Fink 1992, Terre & Ghiselli 1997, Garcia-Campayo *et al.* 1997, Rief & Hiller 1998, Escobar *et al.* 1998b, Garyfallos *et al.* 1999, Kolk *et al.* 2002, Creed & Barsky 2004, de Waal *et al.* 2004, Mak & Zane 2004). In the ECA study only 9.7% of the respondents without SD had more than one diagnosis, while 77.9% of those with SD had more than one other diagnosis (Swartz *et al.* 1986a).

Smith *et al.* found mood disorder, personality disorder and psychoactive substance use disorder to be common psychiatric comorbid disorders among somatoform disorder patients (Smith *et al.* 2000). Antisocial behavior, drug and alcohol abuse were often reported among SD patients in the ECA study (Swartz *et al.* 1986a).

MUS increase the likelihood of psychiatric comorbidity, and studies suggested that the number of MUS, rather than the specific symptom type, can indicate the presence of a depressive or an anxiety disorder (Kirmayer & Robbins 1991, Simon & von Korff 1991, Lieb *et al.* 2002).

### **3 Aims of the present study**

The purpose of the present study was to investigate somatization in young adult population. The numbers I-IV hereafter refer to the original publications. The detailed aims of the present study were:

1. To investigate epidemiology of somatization disorder and somatization symptoms in a population-based birth cohort (I).
2. To assess the extent of somatization in young adults and its associations with sex, educational level and psychiatric morbidity (II).
3. To assess alexithymia and alexithymic features among young adults with and without somatization symptoms (III).
4. To test the hypothesis that somatization is a stable trait that might be associated with the temperament profile assessed with the TCI (IV).



## **4 Material and methods**

### **4.1 Material**

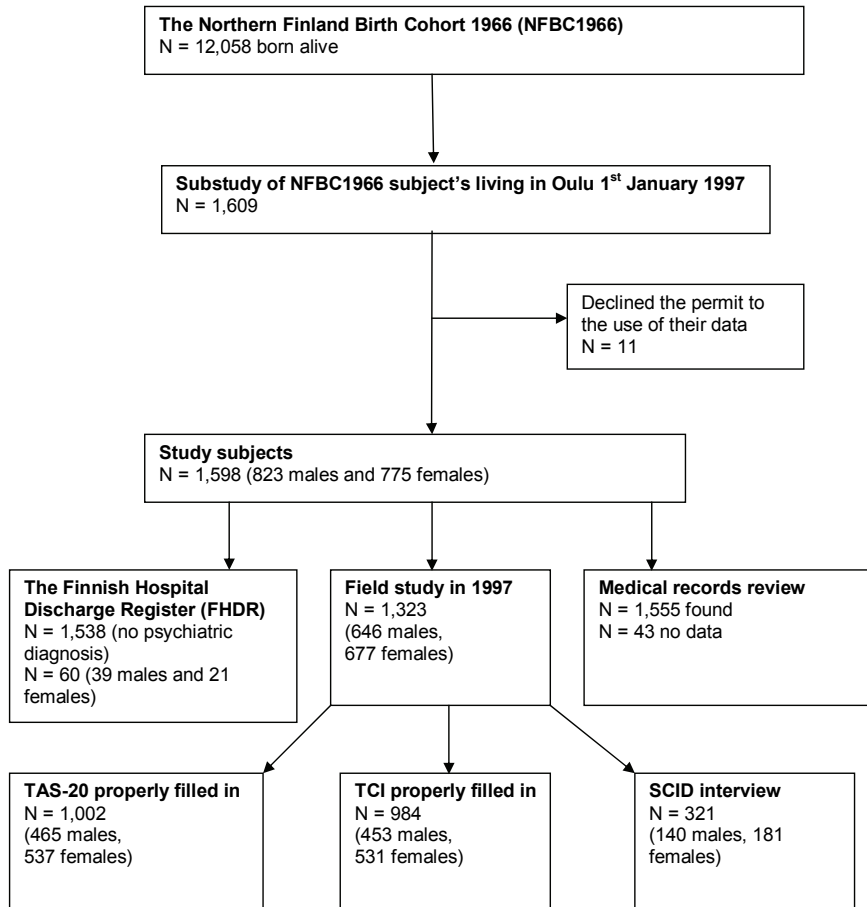
#### ***4.1.1 The Northern Finland 1966 Birth Cohort***

The Northern Finland 1966 Birth Cohort (NFBC 1966) is an unselected, representative, general population birth cohort ascertained during mid-pregnancy and based upon 12,068 pregnant women and their 12,058 live-born children in the two northernmost Finnish provinces of Lapland and Oulu with an expected delivery date during 1966 (Rantakallio 1969, Rantakallio 1988). The NFBC 1966 was originally assembled by Professor (emerita) Paula Rantakallio with the purpose of describing and analyzing the risk factors for perinatal deaths and low birth weight. Thus far, three follow-ups have been completed at the cohort members' age of 1, 14 and 31 years.

In 1997 through 1998, a 31-year follow-up field study was conducted by means of various interviews and questionnaires and clinical examination of the cohort members. The questionnaires included the Toronto Alexithymia Scale, twenty-item version (TAS-20), the Hopkins Symptom Checklist 25 (HSCL-25), and the Temperament and Character Inventory (TCI). Of the cohort members living in Northern Finland or in the capital area of Helsinki 8,465 were invited to participate in the field study and 5,999 of them (70.9%) participated in the examination.

#### ***4.1.2 Oulu Study; a subsample of the NFBC 1966***

The sample of the present study consisted of a subsample of the NFBC 1966. All 1,609 subjects who lived in the town of Oulu on 1st January 1997 were included. Eleven individuals did not consent to the use of their data and were excluded, leaving 1,598 subjects (823 male and 775 female). Of them 793 (49.6%) were born in Oulu. All the participants were 31 years old and of white Caucasian ethnicity (Figure 1).



**Fig. 1.** The flowchart of the data collection procedure in the Oulu Study, subsample of the Northern Finland 1966 Birth Cohort Study and diagnostic data used in the original publications I-IV.

## 4.2 Methods

### 4.2.1 Assessment of somatization

The assessment of somatic symptoms was based on the subjects' medical records in public sector health care. The author of this thesis reviewed all patient records of the subjects (N=1,598) from the public outpatient clinics in Oulu. Data were

gathered from the period 1982-1996. Forty-three of the 1,598 subjects had not used any of these health services until the end of 1996 (Figure 1).

The reviewer was blinded to the possible psychiatric diagnoses gathered from the Finnish Hospital Discharge Register (FHDR) and hospital case notes (Isohanni *et al.* 1997, Moilanen *et al.* 2003). The reviewer was also blind to the TAS-20, HSCL-25 and TCI scores of the subjects. The patient records were available at the municipal primary and mental health care of the City of Oulu, the A-Clinic Foundation clinic (for substance abuse treatment), the Finnish Student Health Service, the Child and Family Guidance Center and outpatient clinics at Oulu University Hospital. Any of the 35 symptoms of the DSM-III-R criteria of SD (American Psychiatric Association 1987) (Table 3) noted by the physician in the patient records were entered into the study data.

Symptoms not sufficiently explained by medical diseases, injury or the use of medication, drugs or alcohol were assessed as somatization symptoms. No patient had been diagnosed as suffering from SD by attending physicians.

Thirteen of the 35 symptoms listed in DSM-III-R were required for a diagnosis of SD in Study I. Subjects who found to suffer from four or more symptoms of the 35 symptoms in the DSM-III-R SD criteria were accepted to the somatizer group. Somatization symptoms were dichotomized (four or more somatization symptoms vs. less than four) and used in Studies II-IV.

**Table 3. Diagnostic criteria for Somatization Disorder based on the DSM-III-R.**

Criteria	Definition
A	A history of many physical complaints or a belief that one is sickly, beginning before age 30 and persisting for several years.
B	At least 13 symptoms from the list below (Table 4). To count a symptom as significant, the following criteria must be met: <ol style="list-style-type: none"> <li>1. no organic pathology or pathophysiologic mechanism (e.g., a physical disorder or the effects of injury, medication, drugs, or alcohol) to account for the symptom or, when there is related organic pathology, the complaint or resulting social or occupational impairment is grossly in excess of what would be expected from the physical findings</li> <li>2. has not occurred only during panic attack</li> <li>3. has caused the person to take medicine (other than over-the-counter pain medication), see a doctor, or alter his/her life-style</li> </ol>

**Table 4. Symptom List for Somatization Disorder based on the DSM-III-R.**

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Symptom List

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Gastrointestinal symptoms:

- vomiting (other than during pregnancy)
- abdominal pain (other than when menstruating)
- nausea (other than motion sickness)
- bloating (gassy)
- diarrhea
- intolerance to (gets sick from) several different foods

Pain symptoms:

- pain in extremities
- back pain
- joint pain
- pain during urination
- other pain (excluding headaches)

Cardiopulmonary symptoms:

- shortness of breath when not exerting oneself
- palpitations
- chest pain
- dizziness

Conversion or pseudoneurological symptoms:

- amnesia
- difficulty swallowing
- loss of voice
- deafness
- double vision
- blurred vision
- blindness
- fainting or loss of consciousness
- seizure or convulsion
- trouble walking
- paralysis or muscle weakness
- urinary retention or difficulty urinating

Sexual symptoms for the major part of a person's life after opportunities for sexual activity:

- burning sensation in sexual organs or rectum (other than during intercourse)
- sexual indifference
- pain during intercourse
- impotence

Female reproductive symptoms judged by the person to occur more frequently or severely than in most women:

- painful menstruation
  - irregular menstrual periods
  - excessive menstrual bleeding
  - vomiting throughout pregnancy
-



#### **4.2.2 Assessment of psychiatric disorders**

Data on psychiatric morbidity were gathered from the hospital discharge register, from public outpatient records review and from the 31-year field survey, where Structured Clinical Interview for DSM-III-R (SCID) was carried out. The following three case finding procedures were carried out independently blinded to each other.

1. All hospital treatment periods during 1982 through 1997 due to psychiatric disorder were identified from the Finnish Hospital Discharge Register (FHDR). FHDR covers all mental, general, private and prison hospitals, military wards as well as beds at health centers nationwide. Hospital notes were scrutinized and diagnoses were re-checked against the DSM-III-R criteria and validated (Isohanni *et al.* 1997, Moilanen *et al.* 2003). Sixty of the subjects (39 males and 21 females) had been in hospital care due to a psychiatric disorder.

2. The author of this thesis reviewed all the patient records in public outpatient care as described in 4.2.1 and also collected information about psychiatric morbidity. The diagnostic assessments were made according to the DSM-III-R criteria in light of the patient records.

3. A two-phase sampling design was adopted for the field study in 1997 to 1998 (Veijola *et al.* 2003). The Hopkins Symptom Checklist 25 (HSCL-25) was used as a screening instrument. The HSCL-25, a twenty-five-item version of the original scale Symptom Check List (SCL-90) (Derogatis *et al.* 1973) has been used in the Nordic countries e.g. for screening purposes (Sandanger *et al.* 1998). The HSCL-25 included questions on the presence and intensity of anxiety and depression symptoms over the previous week. The answers are scored on a scale from 1 (not bothered) to 4 (extremely bothered). The HSCL-25 score is the sum of items divided by the number of items answered. Psychiatric distress according to the HSCL-25 was dichotomized using the cut-off point 1.55 (Joukamaa *et al.* 1994, Veijola *et al.* 2003). The SCID was used as a diagnostic instrument (Spizer *et al.* 1989a, Spizer *et al.* 1989b). A satisfactorily completed HSCL-25 was obtained from 1,311 subjects (642 men and 669 women). All 235 cases screened positive were invited for a psychiatric interview. Of them 209 (87 men and 122 women) participated. Every tenth subject screened negative was interviewed as well. If a screen-negative person refused to participate, the next consecutive screen-negative subject was recruited. Altogether 112 (53 men and 59 women) screen-negative subjects were interviewed. The psychiatric part of the 31-year

field survey has been described in detail by Veijola and colleagues (Veijola *et al.* 2003) (Figure 1.).

Psychiatric morbidity was assessed according to the best-estimate procedure (Leckman *et al.* 1982, Kosten & Rounsaville 1992). All data were integrated and discussed in an expert consensus panel of the investigators to formulate the best-estimate lifetime psychiatric diagnoses. In Study I the categories “definite” and “probable” were used to describe the certainty of the diagnosis for a psychiatric disorder. For a diagnosis to be “definite,” all the required minimum criteria of DSM-III-R to make a diagnosis had to be met. For “probable,” the required minimum criteria were not fully met, but the diagnosis seemed likely on a clinical basis. Lifetime prevalence was defined as occurrence of the disorder any time during the life course.

All investigators participating in data collection and the best-estimate procedure underwent DSM-III-R diagnostic training. In addition to this, all interviewers participating in the psychiatric field survey underwent comprehensive training in the use of the SCID instrument (Veijola *et al.* 2003). An acceptable level of agreement with diagnoses (mean kappa 0.92), inter-rater reliability (mean kappa 0.68) and validity (mean kappa 0.68) was found.

The four diagnostic categories used in this study were separately classified as follows: mood disorders (DSM-III-R codes 296.20 – 296.70, 300.40, 301.13, 311.00), anxiety disorders (300.00 - 300.02, 300.21 - 300.30, 309.89), substance use disorders (303.90 - 305.90), and personality disorders (301.00, 301.20, 301.22, 301.40, 301.50, 301.60- 301.90). Cases fulfilling the diagnostic criteria of at least one of the major mental disorders were classified as “any mental disorder”.

#### **4.2.3 Assessment of alexithymia**

The assessments (in paragraphs 4.2.3 - 4.2.5) were obtained during the 31-year follow-up study of the cohort, except for educational level.

Of the different methods for measuring alexithymia, the TAS-20 questionnaire is the most widely used and carefully validated (Bagby *et al.* 1994a, Bagby *et al.* 1994b, Taylor *et al.* 2003, Parker *et al.* 2003). The psychometric properties of the Finnish version of the TAS-20 have been shown to be good (Joukamaa *et al.* 2001). The TAS-20 consists of 20 statements, each rated on a Likert-type scale from 1 (strongly disagree) to 5 (strongly agree) for a maximum score of 100; five items are negatively keyed. The TAS-20 has a three-factor

structure: difficulty in identifying feelings (DIF); difficulty in describing feelings (DDF); and externally oriented thinking (EOT).

The total score and the scores of TAS-20 Factors DIF, DDF and EOT were calculated. According to the developers' recommendation (Taylor *et al.* 1997), subjects with a total TAS-20 score over 60 were considered as being alexithymic, and those with a score under 52 were considered non-alexithymic. Subjects with total TAS-20 scores from 52 to 60 were considered as having alexithymic features. The TAS-20 questionnaire was returned properly filled in (Kokkonen *et al.* 2001) by 1,002 (465 male and 537 female) of the subjects, and they were included in the present study (Figure 1.).

#### **4.2.4 Assessment of temperament**

Cloninger's actual temperament model consists of four largely independent profiles of temperament: novelty seeking (NS), harm avoidance (HA), reward dependence (RD) and persistence (PS) (Svrakic *et al.* 1993, Cloninger *et al.* 1994). NS is a tendency to respond with intense excitement to novel stimuli, cues for potential rewards or potential relief of punishment, thereby activating or initiating behavior. HA is defined as a tendency to respond intensively to signals of aversive stimuli, thereby inhibiting or stopping behavior. RD is a tendency to respond intensely to signals of reward, especially social rewards, thereby maintaining and continuing particular behaviors. PS includes a tendency to persevere with behaviors that have been associated with reward or relief from punishment (Cloninger *et al.* 1993, Cloninger *et al.* 1994, Miettunen *et al.* 2004).

The assessment of temperament profiles was made by the TCI (Cloninger 1986, Cloninger 1987, Cloninger *et al.* 1993, Cloninger *et al.* 1994), which is a 240-item personality questionnaire for the assessment of the seven basic profiles of personality including four temperament profiles and three character profiles (Self-Directedness, Co-operativeness and Self-Transcendence). The character items of the TCI were not collected in this study. The psychometric properties of the Finnish version of the scale have been shown to be satisfactory (Miettunen *et al.* 2004).

The questionnaires including the TCI were given to the participants in the 31-year clinical study. The participants were asked to fill it in at home and to mail it back to the study office. Only TCIs with six or less missing items were included. Of the participants 984 subjects (453 males and 531 females; 61.6% of subjects) filled it in properly (Figure 1).

#### **4.2.5 Other variables**

Sex, marital status, educational level and psychological distress were considered as covariates in the study. Marital status (dichotomized here as unmarried, divorced, widowed/cohabiting, married) was obtained with a questionnaire in the field study. Psychological distress was measured with the HSCL-25 questionnaire (Veijola *et al.* 2003) in the field study as well (See 4.2.2). Data on educational level of all subjects were obtained from Statistics Finland up to the year 1997. Educational level was divided into three classes based on duration: under 9 years of education, 9 to 12 years, and over 12 years of education. Data on educational level were lacking for two male subjects.

#### **4.2.6 Statistical analysis**

Kappa values were used to assess the diagnostic aptness among field investigators who interviewed the study subjects. The categorized data were analyzed with cross-tabulation and statistical significance was tested with Chi-square test or Fisher's exact test when appropriate. All significance levels were two-tailed and the significance threshold was  $p < 0.05$ . The mean scores of the TAS-20 and its three factors were calculated in subjects with and without somatization symptoms and also stratified by sex. The mean scores of the four TCI temperament profiles were calculated in subjects with and without somatization symptoms for the whole sample and separately for males and females. The statistical significance of differences between the means was evaluated using independent-samples Student's t-test and analysis of variance (ANOVA).

Logistic regression analysis was used in modeling the relative significance of somatization as a predictive variable for categorized alexithymia. It was also used in modeling the relative significance of temperament profiles as predictive variables for categorized somatization. In these analyses sex, marital status, educational level and psychiatric distress were used as confounding factors. Multivariate logistic regression analyses were used to explore the association between somatization symptoms and sex, educational level and psychiatric morbidity. Adjusted odds ratios (OR) were calculated for the variables sex, educational level, any mental disorder, and separately for all four major mental health categories. Multivariate analysis of variance (MANOVA) was used to assess the association of somatization and the continuous alexithymia variables (the TAS-20 total score and the TAS-20 factors) using sex, marital status,

educational level and psychiatric distress as covariates. The Odds Ratios (OR)s were calculated, and 95% Confidence Intervals (95% CI) were calculated for the ORs and proportions.

#### **4.2.7 Ethical considerations**

Permission for gathering data for the entire NFBC 1966 was obtained from the Ministry of Social Welfare and Health Affairs in 1993. The research plan for the NFBC 1966 31-year follow-up study was approved by the Ethical Committee of Oulu University, Faculty of Medicine. All the participants gave a written informed consent for the study.

#### **4.2.8 Personal involvement**

The author of this thesis has participated in the NFBC 1966 as a researcher since 1996. The author has been accorded permission to use the data, and has made a contribution towards the conception and design of this study and interpretation of the data and statistical analysis. As the first author, the author has drafted the main content of original studies number I-IV, as well as reviewed and revised them for intellectual content. The author reviewed all the patient records in public outpatient care of the study population (N=1,609).



## **5 Results**

### **5.1 Somatization disorder in young adult population (I)**

Eighteen SD cases (1.1%, 95% CI 0.7–1.8%) were identified. Fifteen of them were classified as definite and three as probable cases (Table 1 in Study I). There were 15 females and three males, giving a female-to-male ratio of 5:1.

All SD cases were found from public outpatient care records review. Two males and one female with SD had been treated in hospital because of a psychiatric disorder. One had suffered from adjustment disorder with depressive mood and two from alcohol dependence. One of the alcohol dependence patients also had borderline personality disorder. Six of the SD subjects (1 male and 5 females) were interviewed with SCID in the field study. According to the interview all of them had at least one lifetime psychiatric disorder. Three had major depression, one conversion disorder, one alcohol dependence, and one had obsessive-compulsive personality disorder. SD subjects with depression had other comorbid psychiatric disorders as well, such as anxiety disorders, eating disorders and avoidant personality disorder.

### **5.2 Associations of somatization symptoms (II)**

Of the subjects 6.1% (N=97) fulfilled the criteria of somatization symptoms. There were 14 males (1.7%; 95% CI 1.0-2.8) and 83 (10.7%; 95% CI 8.7-13.1) females, giving a female-to-male ratio of 6:1 (Table 1 in Study II). The mean number of somatization symptoms was 7.6 for male and 7.7 for female somatizers.

Somatization was more common among subjects with less education (Table 2 in Study II). Among males those with only basic-level education had somatization five times more commonly than males with tertiary educational level.

One in five of the subjects (142 males and 177 females) had at least one lifetime psychiatric disorder. The occurrence of somatization was 16.6% among subjects with any mental disorder and 3.4% among subjects without any mental disorder ( $p<0.001$ ). About half of male and female somatizers suffered from some mental disorder. In logistic regression analysis sex, educational level and separately the four diagnostic categories were analyzed (Table 3 in Study II). Female sex, low educational level and all major psychiatric disorders except

substance use disorders were associated with somatization in univariate analyses. After adjusting, sex, anxiety and personality disorders had an independent association with somatization. As mood disorders were not significantly associated with somatization the subjects with mood disorders and without other psychiatric disorders were analyzed separately. In a model with sex, educational level and the other three diagnostic categories the adjusted OR for subjects with mood disorders without other psychiatric disorders was 2.0 (95% CI 0.8-4.6).

### **5.3 Somatization and alexithymia (III)**

Alexithymia was associated with sex, education, marital status and psychological distress (Table 1 in Study III). Somatization associated with sex and psychological distress (Table 2 in Study III).

The prevalence of alexithymia was 6.0% among somatizers and 4.8% among subjects without somatization symptoms, and the prevalence of alexithymic features was 7.5% and 12.6%, respectively (Table 3 in Study III). No significant difference was found. Somatization did not associate with alexithymia in logistic regression analysis when adjusted for sex, marital status, educational level and psychiatric distress. Males visited a physician on average 8 times and females 12 times during the follow-up period.

The total score of alexithymia did not associate with somatization when all subjects were analyzed together or when stratified by sex (Table 4 in Study III). Somatizers and nonsomatizers did not differ in terms of their ability to describe feelings (DDF) or to identify feelings (DIF) in all subjects or separately in males and females. Externally oriented thinking (EOT) associated significantly with somatization. In the group of somatizers the mean EOT was lower than in the group of nonsomatizers. In the MANOVA using sex, marital status, educational level and psychiatric distress as covariates for the EOT, the association disappeared.

### **5.4 Temperament profiles and somatization (IV)**

Six males and 61 females (1.3% vs. 11.5%,  $p < 0.001$ ) met the criteria for somatization. Somatizers suffered more commonly from psychological distress than nonsomatizers (43.3% vs. 15.9%,  $p < 0.001$ ). Educational level or marital status did not associate with somatization (Table 1 in Study IV).



All temperament profiles were associated with sex. Among females the means of NS, HA, and RD were higher, but PS lower than among males. HA and PS associated with education, PS being higher among those who had the highest (over 12 years) education and HA being lowest among them. HA and RD associated with marital status, HA being higher among unmarried and RD among married subjects. HA associated with psychological distress, being higher among subjects with psychological distress (Table 1 in Study IV).

In the whole sample the means of HA and RD were higher among somatizers than among other subjects. When analyzed separately in males and females, the association was not statistically significant (Table 2 in Study IV).

Pair-wise logistic regression analysis revealed that somatization associated with sex (OR 9.7; 95% CI 4.1-22.6), psychological distress (OR 4.0; 95% CI 2.4-6.8), and of the TCI temperament profiles with HA (OR 1.045; 95% CI 1.002-1.089) and RD (OR 1.079; 95% CI 1.006-1.157). In further modeling with sex, psychological distress, HA and RD as covariates, somatization associated only with sex (OR 9.8; 95% CI 4.0-23.8) and psychological distress (OR 3.9; 95% CI 2.3-6.8). We found no significant interactions in modeling.



## 6 Discussion

### 6.1 Discussion of the results

#### 6.1.1 Somatization disorder in young adult population (I)

SD cases could only be identified through the review of the public outpatient records. Methodologically, information from outpatient records may be more sensitive in detecting SD than FHDR or even psychiatric interview. The one percent lifetime prevalence of SD was comparable with previous population studies (Swartz *et al.* 1991, Rief *et al.* 2001, Grabe *et al.* 2003).

This was the first Finnish population study assessing the prevalence of SD. General population studies on this topic are sparse internationally as well. Our material covered the study population sufficiently. SD patients are mostly treated in somatic health services. Public health care is widely accepted by Finnish people. The services were free of charge during the period covered by the study, except for the minimal fee charged by the Finnish Student Health Service.

The lifetime prevalence of SD (1.1%) included definite and probable cases. The prevalence was within the limits of previous population studies, where the prevalence rates have varied from 0.02 to 1.84% (Table 1). The “true” prevalence of SD (1%) according to Bass *et al.* is almost identical to our result (Bass *et al.* 2001). In previous studies the only case-finding method has been personal interview by trained laymen or professionals (Canino *et al.* 1987, Bland *et al.* 1988, Hwu *et al.* 1989, Wells *et al.* 1989, Lee *et al.* 1990a, Lee *et al.* 1990b, Swartz *et al.* 1991, Wittchen *et al.* 1992, Faravelli *et al.* 1997, Roca *et al.* 1999, Lieb *et al.* 2000). Measurement error due to faulty recall is doubtlessly one reason why the reported prevalence rates of SDs in epidemiological studies have varied greatly (Creed & Barsky 2004, Leiknes *et al.* 2006). We were only able to find SDs with the aid of patient record data.

The female-to-male ratio of the SD cases was 5:1 in our study. This is in line with results from previous population studies even though there has been large variation in the ratios (Table 1). In two studies, however, the prevalences were almost equal for both sexes (Hwu *et al.* 1989, Ladwig *et al.* 2001) and in one study there was male predominance (Lee *et al.* 1990b). The small number of subjects in the studies should be taken into account. In Taiwan Hwu *et al.* found an almost equal prevalence in small towns but not in the capital area or rural

villages. Lee and colleagues reported male predominance in a rural area of Korea but not in the capital area. Cultural factors may influence the way a somatic complaint is described or the types of illness models that might predispose individuals to or precipitate these complaints (Janca *et al.* 2006, Patel & Sumathipala 2006). Ladwig used ICD-10 criteria, which might have some effect on their results. A marked discrepancy has been found in the rates of SD between the ICD-10 and DSM-IV diagnostic systems (Yutzy *et al.* 1995, Fink *et al.* 2004).

### **6.1.2 Somatization symptoms in young adult population (II)**

The occurrence of somatization in this study was found to be the same as reported by Escobar *et al.* from Puerto Rico and by Jacobi from Germany, but higher than reported by Lieb *et al.* and Grabe *et al.* (Table 2). The reported occurrence has varied depending on the definition of somatization (Lieb *et al.* 2000, Ritsner *et al.* 2000, Rief *et al.* 2001, Ladwig *et al.* 2001).

The female-to-male ratio among somatizers was 6:1 in the present study. This is similar to the ratio reported from the ECA (Swartz *et al.* 1991) and comparable to the ratio of 'persistent somatizer' reported by Fink (Fink 1992). Terre and Ghiselli also found a female predominance among somatizers (Terre & Ghiselli 1997), but Creed and Barsky did not (Creed & Barsky 2004). In population studies all the reported female-to-male ratios had a female predominance, with the exception of Rief *et al.* (Table 2).

The higher prevalence of somatization symptoms in women could be explained by various factors (van Wijk & Kolk 1997, Kroenke & Spitzer 1998). The increased rate among women could be due to greater willingness to admit discomfort. Women are more eager to seek medical attention. Women are suggested to be more focused on their bodies because of an innate difference in bodily perception (Wool & Barsky 1994, Janca *et al.* 2006). Some investigators have suggested that the sex difference in somatization symptoms might be artificial. Warner found that male and female professionals tend to label men as antisocial and women as hysterical even when they have identical clinical features (Warner 1978). Kaminsky and Slavney suggested that the criteria for Briquet's syndrome are biased against men because some of the characteristic symptoms are inapplicable to men (e.g., pregnancy and menstrual complaints) (Kaminsky & Slavney 1976). They also noted that men generally report fewer symptoms than women (Cloninger *et al.* 1986a).

In the present study somatizers tended to belong to the lower education group more commonly than nonsomatizers. The difference reached statistical significance among males. In the ECA study somatization was associated with lower educational level (Swartz *et al.* 1991). Creed and Barsky also found a consistent association with lower educational level (Creed & Barsky 2004). The present findings were in harmony with the results of the ECA. On the other hand, educational level was not independently associated with somatization symptoms in the analysis that took into account sex and psychiatric disorders. Additionally, the present sample had a higher educational level than the whole NFBC 1966 Cohort. As educational level associated with somatization symptoms, we cannot directly generalize our results to the NFBC 1966 Cohort as a whole.

Anxiety and personality disorders were independently associated with somatization symptoms whereas mood disorders were not, which is an opposite finding to most other studies in this area (Smith *et al.* 1986, Escobar *et al.* 1987a, Katon *et al.* 1991, Simon & von Korff 1991, Garcia-Campayo *et al.* 1997, Rief & Hiller 1998, Escobar *et al.* 1998b, Creed & Barsky 2004). In line with the present results, a Danish study showed somatoform disorders to associate with anxiety disorders OR 2.6 (95% CI 1.4–4.7) but not with alcohol/drug abuse 1.7 (0.8–3.6) or depression 1.5 (0.9–2.3) (Toft *et al.* 2005). Henningsen *et al.* also found that in patients with medically unexplained gastrointestinal symptoms, help-seeking correlated with increased levels of anxiety, but not with depression symptoms (Henningsen *et al.* 2003).

Garyfallos and colleagues have summarized possible causes for comorbidity in the case of somatoform disorders. Firstly, somatoform disorders may be masked expressions of depression or anxiety. Secondly, patients with somatoform disorders suffer chronically and are disabled, and because of that they develop depression and anxiety. Thirdly, these disorders share common underlying processes such as neurophysiologic dysfunction or dysregulation of serotonin metabolism, or there is a common familial origin, whether genetic, social or both. Fourthly, an underlying diathesis between depressive, anxiety and somatoform disorders in conjunction with developmental experiences and life events leads to the manifestation of these clinical syndromes simultaneously or in different phases of the patient's life (Garyfallos *et al.* 1999).

### **6.1.3 Somatization and alexithymia (III)**

In accordance with the theory of alexithymia, it was hypothesized that alexithymia is common among subjects with somatization symptoms. However, no evidence was found for an association between alexithymia and somatization. One of the subscales of TAS-20 (EOT) was even negatively associated with somatization. These findings do not support the hypothesis that alexithymia is a common feature among subjects with somatization symptoms.

Alexithymic persons find it hard to recognize that physical sensations are sometimes the somatic concomitants of affect. This has led to the suggestion that alexithymia may play a central role in the pathogenesis of somatization. In several studies, alexithymia has indeed been shown to associate with reporting of physical symptoms (Taylor *et al.* 1997, Kooiman *et al.* 2000). However, in a recent review the association between somatization and alexithymia was not found to be strong (De Gucht & Heiser 2003). The finding questions the earlier theory of an association between alexithymia and somatization.

It has been assumed that somatization and alexithymia develop from the same psychosomatic context and construct. Alexithymia has its origins in clinical observations of psychosomatic patients who had difficulties in talking about feelings and fantasies when assessed in psychodynamically oriented interviews (Sifneos 1973). Somatizing and alexithymic persons have both been suggested to have difficulties in expressing their emotions verbally. In a recent formulation of the theory (Taylor *et al.* 1997), alexithymia is assumed to involve an impaired capacity to construct mental representations of emotions. Alexithymic individuals are suggested to focus on somatic sensations. These sensations accompany emotional arousal, which leads to somatosensory amplification and misinterpretation of somatic sensations as signs of physical illness (i.e., somatization). This recent theory predicts that alexithymia is associated with a tendency to experience and communicate psychological distress in somatic rather than emotional terms (Taylor *et al.* 1997, Taylor 2000).

However, according to our results somatization and alexithymia seem to be independent and to express different aspects of subjects. There are basically two different kinds of possible explanations for the lack of association between TAS-20 and somatization: either alexithymia is not essentially related to somatization, or there is an association which the TAS-20 is not able to capture because it does not possess sufficient validity as a measure of alexithymia (Lundh & Simonsson-Sarnecki 2001).

Similarly to Kooiman *et al.*, the present study found a higher prevalence of psychiatric pathology among somatizers than among other subjects (Kooiman *et al.* 2000). It seems that somatization is associated with other psychiatric pathology. In particular, somatization has been found to be associated with anxiety and depression (Escobar *et al.* 1987a, Katon *et al.* 1991, Simon & von Korff 1991). Some studies have shown that high percentages of patients with somatoform disorders have personality disorders (Kooiman *et al.* 2000). Alexithymia has been associated with mood disorders, anxiety disorders and substance dependence (Kooiman *et al.* 2002). In the present study, it was found that both alexithymia and somatization associated with actual psychological distress. However, with and without adjustment for psychiatric distress there was no association between somatization and alexithymia.

#### **6.1.4 Temperament profiles and somatization (IV)**

The present study did not identify any characteristic temperament profile for somatizers. HA and RD were associated with somatization in the whole sample, but in separate analyses for males and females these associations disappeared. In addition to sex, psychological distress associated with somatization. The hypothesis of a characteristic temperament profile for somatizers could not be confirmed.

Adoption studies demonstrate a weak but persistent familial relationship for the somatization phenomenon (Cloninger *et al.* 1986b, Guze 1993, Hollifield 2004). The reported familial transmission may be rooted in sociogenetic variables. Both environmental and genetic factors may be operative in the development of somatization (Guze 1993, Campo & Fritsch 1994, Hotopf *et al.* 1999, Craig *et al.* 2002, Craig *et al.* 2004, Fink *et al.* 2005). Certain somatoform syndromes are associated with psychopathology in the biological parent, whereas others are associated with psychopathology in the adoptive parent. Thus, genetic factors play a substantial role in some syndromes of somatization, whereas others are determined predominantly by other factors including environmental stresses (Arkonac & Guze 1963, Woerner & Guze 1968, Kellner 1990).

Battaglia *et al.* found an association between SD and high NS in a female psychiatric outpatient population with comorbid panic disorder (Battaglia *et al.* 1998). Russo *et al.* found that high HA associates with an increased number of MUS in three medical outpatient settings (Russo *et al.* 1994). In the present general population sample of young male and female adults no support for such

an association was found. This may be because of the limited number of somatization subjects in the sample or selection bias in earlier studies with outpatient settings. Both Battaglia *et al.* and Russo *et al.* used the Tridimensional Personality Questionnaire, an earlier version of the TCI. However, in the present study an association was found between high psychiatric distress and somatization. This might support the cognitive theory, with the idea of health anxiety and subjects reactions to distress as a process to explain somatization behavior. Russo and colleagues reported an association between the number of earlier and present anxiety and depressive diagnoses and an increased number of medically unexplained symptoms (Russo *et al.* 1994). Battaglia and colleagues reported more somatization among panic patients compared to controls (Battaglia *et al.* 1998).

Recently Hakala *et al.* found low NS and high HA as temperament traits among female SD outpatients. This was also one hypothesis for the present study. Hakala *et al.* concluded that both observed metabolic changes and temperament traits converged with the clinical picture of the patients: low energy level, fatigability, tendency to avoid new stimuli, need of rest and restriction of additional activities (Hakala *et al.* 2002, Hakala *et al.* 2006). Our results of temperament character of subjects with somatization symptoms differ significantly from those studies. The subjects in the studies of Hakala *et al.* were older than in our study, and they suffered only from SD or undifferentiated somatization disorder without mental health comorbidity. The subjects in the present study had many psychiatric comorbidities and were drawn from a population-based cohort of the same age, which may partly explain the difference between the findings in these studies.

## **6.2 Methodological discussion**

### **6.2.1 Definition and assessment of somatization**

In Studies II-IV somatization was defined by a count of somatization symptoms found during data collection from all public health care facilities and in best-estimated diagnosis process. In Study I, SD was defined by DSM-III-R criteria (Table 3). Most of the earlier studies have used questionnaires that can only check for symptoms or whether these symptoms are medically explained (De Gucht & Heiser 2003). We based our data on a review of outpatient medical records.



Kroenke suggested that, in general, medical records may result in an underestimation of symptom frequency, whereas interviews and questionnaires tend to overestimate the frequency of symptoms (Kroenke 2001). Therefore, our results may be an underestimation rather than overestimation of the real situation (Portegijs *et al.* 1996). This might explain why the occurrence of somatization symptoms was lower than in most of the previous reports (Table 2).

According to the diagnostic criteria of the DSM-III-R and ICD-10 the identification of SD cases is based on case history (anamnesis). A comprehensive review of patient records may be able to reduce the patients' recall bias regarding earlier use of services. Reid *et al.* have considered that case note examination may be a reliable method of determining whether presenting symptoms are medically unexplained or not (Reid *et al.* 1999). Opposite to our finding Kolk and colleagues (Kolk *et al.* 2002) suggested that in general, medical records might result in an underestimation of all symptom frequency. This problem may diminish when studying patient records from a longer time period (here 1982–1996) and from various treatment units. The recognition of occurring symptoms is also highly dependent on the quality of patient records.

The lifetime psychiatric diagnoses and somatization symptoms of the subjects were assessed according to the best-estimate procedure in an expert panel using information from all data sources available (Leckman *et al.* 1982, Taiminen *et al.* 2001). It has been suggested that the use of the best-estimate procedure represents a clear improvement in diagnostic accuracy. It may be particularly useful to include data from medical records to formulate the best-estimate diagnoses (Kosten & Rounsaville 1992).

Somatization interviews performed by nonprofessionals seem to have little advantage over self-ratings. Both methods refer primarily to the subjective patient report rather than to objective tests or medical examination (which would be too expensive to be used in large epidemiological surveys). Data from self-ratings and data from interview-based studies are comparable, although it is sometimes assumed that self-report methods yield higher rates (Peveler *et al.* 1997). A tendency of false negative diagnoses for SD has been found both in lay interviews and with the use of questionnaire data (Hiller *et al.* 2006). The present findings support the view that the use of review of all patient records as a method of data collection, even though rather laborious, might overcome some of the above-mentioned limitations such as subjective patient report and recall bias of symptoms.

Medically unexplained symptoms (MUS) have been conceptualized in various ways in the literature. Some authors have questioned the validity of physical symptom counts in defining somatization, and have even suggested that the Somatoform disorder category should be revised in future classifications (Fink 1996, Rief & Hiller 1999, Rief & Sharpe 2004, Sharpe & Mayou 2004, Mayou *et al.* 2005, Janca *et al.* 2006, Chaturvedi *et al.* 2006).

In introducing the diagnostic entity of SD to the research criteria of the ICD-10, the 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 for SD as well as the Feighner criteria. Excellent concordance was found between DSM-III, DSM-III-R, DSM-IV and earlier Feighner criteria for Briquet's syndrome (Feighner *et al.* 1972, Yutzy *et al.* 1995). For clinical or research use, the DSM and ICD diagnostic criteria for SD are too restrictive, while the criteria for undifferentiated somatoform disorder are overly inclusive (Escobar *et al.* 1987b). An abridged somatization construct (the Somatic Symptom Index; SSI4,6) derived from the Diagnostic Interview Schedule's (DIS) SD items (Escobar *et al.* 1989). SSI4,6 has a cutoff score of four or more symptoms for males and six for females. These cutoff scores have been empirically derived on the basis of their ability to discriminate Hispanic from non-Hispanic white schizophrenia patients (Escobar *et al.* 1986).

The "Primary care evaluation of mental disorders" study developed criteria for Multisomatoform disorder (MSD) (Kroenke *et al.* 1997). MSD was defined as three or more medically unexplained, currently bothersome physical symptoms along with at least two years' history of somatization. Polysymptomatic somatoform disorder (PSD) was developed based on the selection of the 32 items from the original 53 SOMS items, which covered both DSM-IV and ICD-10 criteria for SD and showed a satisfactory psychometric performance. A cutoff of seven or more symptoms for PSD yielded the best discrimination between low and high disability. PSD accounted for the strong association of somatization and abnormal illness behavior (Rief & Hiller 1999). The Diagnostic Criteria for Psychosomatic Research (DCPR) have been developed with the aim of translating psychosocial variables issued from a wide body of psychosomatic literature into working categories whereby individual patients could be identified (Fava *et al.* 1995, Porcelli *et al.* 2004). The DCPR included 12 psychosomatic syndromes, among them alexithymia and chronic somatization (Fava *et al.* 1995).

Comparison between studies is difficult because the measures of the somatization phenomenon are so distinct.

Various functional somatic symptom clusters are widely recognized, with almost every medical specialty having its own syndrome clusters (Kroenke & Swindle 2000, Nimnuan *et al.* 2001). One of the major problems is that there is no general consensus on how to diagnose and classify a patient presenting with functional somatic symptoms (Fink *et al.* 2005). Studies of patients with these conditions have found striking similarities between them (Wessely *et al.* 1999, Nimnuan *et al.* 2001, Wessely & White 2004), with a substantial proportion of patients showing evidence of psychological distress that is either not expressed or unrecognized in the general practice consultation. The diagnosis of functional somatic symptom clusters was not included in this study because of remarkable overlapping between the clusters, and because they are not included in the DSM-III-R psychiatric classification system that was used in the study.

Somatization seems to be relatively stable over time, especially in females, and supports the DSM criteria of chronicity (Cloninger *et al.* 1986a, Liu *et al.* 1997, Gureje & Simon 1999, Simon & Gureje 1999, Lieb *et al.* 2002). A few previous follow-up studies, ranging from 1 to 5 years in length, have investigated the stability of SDs (Kent *et al.* 1995, Simon & Gureje 1999, Lieb *et al.* 2002). It is unusual for an individual with SD to be free of symptoms or help-seeking for longer than one year. A person diagnosed with SD has an approximately 80 percent change of being diagnosed with this disorder after six to eight years (Arkonac & Guze 1963). From one third to about half of the patients were re-diagnosed with SDs according to the DSM-IV criteria after one to three years of follow-up (Simon & Gureje 1999, Lieb *et al.* 2002). The stability of DSM-III SD has been rated higher, with reports of about two thirds of patients meeting the same diagnostic criteria after 4–5 years (Kent *et al.* 1995). About one half of patients with MUS were experiencing at least some symptomatic improvement after a follow-up period of one to six years (Crimlisk *et al.* 1998, Simon & Gureje 1999, Engel *et al.* 2002, Carson *et al.* 2003).

### **6.2.2 Assessment of alexithymia**

Most studies comparing the TAS-20 scores in somatoform disorder patients and psychiatric patients have shown no significant differences. This gives rise to a critical question: how specific are the findings regarding alexithymia in patients with somatoform disorders? Lane has questioned whether self-report assessments

such as TAS-20 are accurate in subjects with severe impairments in emotional self-awareness (Lane *et al.* 1998). The TAS-20 is the most commonly used and best validated measure of alexithymia (Joukamaa *et al.* 2001). However, self-rating measures such as the TAS-20 have recently been seriously criticized (Lane *et al.* 1998, Suslow *et al.* 2000, Kooiman *et al.* 2002, Muller *et al.* 2003). It may be possible that the TAS-20 cannot detect the most severe cases of alexithymia (Lane *et al.* 1996).

Lane *et al.* have speculated that for certain subjects self-reported ratings on DIF and DDF may be less accurate than those on the EOT (Lane *et al.* 1998). They suggested that highly alexithymic subjects rate themselves unreliably because of their own lack of awareness of the deficits. New measures to assess alexithymia have been developed (Bermond *et al.* 1999, Haviland *et al.* 2000, Haviland *et al.* 2001), but knowledge of their psychometric properties is still limited (De Gucht & Heiser 2003). A new structured interview method, the Toronto Structured Interview for Alexithymia (TSIA), has recently been developed for the assessment of alexithymia (Bagby *et al.* 2006).

Consistency of the factor structure of TAS-20 has been demonstrated with different English-speaking samples and with versions translated into about twenty languages (Taylor *et al.* 2003). Recently some criticism of this factor solution has been presented, and other factorial structures have been studied as well (Kooiman *et al.* 2002, Muller *et al.* 2003). The factor analytic studies suggest that alexithymia and somatization reflect separate constructs that may occur simultaneously, but can be independently measured (Bach *et al.* 1996). Lipsanen *et al.* suggested that somatization, dissociation, depression and alexithymia correlate noticeably, while at the same time being distinct constructs (Lipsanen *et al.* 2004).

### **6.2.3 Assessment of temperament**

According to Cloninger's theory of personality, a temperamental pattern of high NS and low HA that is reflected at behavioral level by impulsiveness and a disposition toward excitability, can ultimately lead to chronic somatic anxiety (Cloninger 1986). In this context, chronic somatic anxiety is defined broadly as the presence of frequent autonomic disturbances, bodily aches, and a global feeling of alarm in the absence of specific premonitory cues and can be more specifically identified by a clinical presentation of somatization. However, the

specific prediction made by Cloninger about the basic temperamental features related to somatization has not received direct support so far (Cloninger 1986).

The four TCI temperament profiles are hypothesized to be independently heritable and manifesting early in life (Cloninger *et al.* 1993, Cloninger *et al.* 1994). The heritability of each of these four temperament factors has been estimated to be between 50% and 65%. The factors have been found to be genetically homogenous and independent of each other (Heath *et al.* 1994, Stallings *et al.* 1996, Ando *et al.* 2002, Gillespie *et al.* 2003, Ando *et al.* 2004), but contradictory results have also been recently reported (Herbst *et al.* 2000).

#### **6.2.4 Study population**

The original NFBC 1966 included 12,058 live births and covered 96.3% of all children born in the two northernmost Finnish provinces, i.e., Oulu and Lapland, in the year 1966 (Rantakallio 1988). The majority of the cohort members are Finns (white Caucasians), less than 1% of the subjects being Lapps and Gypsies.

With respect to representativeness of our study subjects compared with the rest of NFBC 1966, we found in Study II that there were no differences in gender distribution. Subjects in the Oulu Study subsample had more commonly over 12 years of education than the whole NFBC 1966 sample (36.0% vs. 20.5%,  $p < 0.001$ ). When comparing the drop-outs of the field study to the participants, the drop-outs were more commonly males (nonparticipant males vs. nonparticipant females  $p < 0.001$ ) and had less education ( $p < 0.001$ ). No difference was found in the prevalence of somatization.

### **6.3 Limitations of the study**

The focus in the literature review and in Studies I-IV was more on the development of the somatization concept, its epidemiology and some theories to explain somatization than on treatment or its effects on health care resources.

There could be some limitations in supplying SD symptoms emerging during doctor-patient discussion to patient records. Portegijs and colleagues suggested that a problem in using general practitioners' records in somatization research is the fact that the concept (somatization) as such does not seem to get written down in the patient records (Portegijs *et al.* 1996). Findings in Study I support this opinion.

The use of the SCID may have limitations in detecting SD as compared to more in-depth sections on SD in the Composite International Diagnostic Interview (CIDI) (Robins *et al.* 1988) and Diagnostic Interview Schedule (DIS) (Robins *et al.* 1981).

One problem in the present study, as in every other study of SD, is that we cannot be absolutely sure that none of the symptoms rated as medically unexplained had a medical explanation (Gureje & Simon 1999). Additionally, the definition of somatization was not validated. The use of only a single chart abstractor is a study limitation, since information in the medical records of many patients is quite varying and often inadequate to be completely certain. Using multiple raters would have allowed estimating the reliability of the determination of somatization symptoms (Khan *et al.* 2003).

Half of the subjects had moved to Oulu from other areas. We supposed that, as is common in Finnish health care, the essential health information had followed the subjects to their new public health care units in Oulu. It is possible that the information about their health care visits was limited. It was not possible to obtain occupational or private medical consultation data about the subjects.

The study sample was drawn from an urban population with no subjects from rural areas. The prevalence of alexithymia was lower in the study than previously reported from a large population study from Finland with a sample of working-age subjects (Honkalampi *et al.* 2000). This may be explained by age, our subjects being young adults, and by urban dwelling and the higher educational level of our subjects. Without a relatively large proportion of nonparticipants the prevalence of alexithymia might have been higher, as the dropouts were more commonly males and less educated. In previous population studies SD has been associated with older age (Canino *et al.* 1987, Bland *et al.* 1988, Wells *et al.* 1989, Swartz *et al.* 1991, Wittchen *et al.* 1992). The prevalence of SD in the study is probably an underestimation of the prevalence in the entire adult population.

There were only six men with somatization, even though the relatively low cutoff point of four somatization symptoms was used. Because of the small number of male somatizers it is difficult to conclude the significance of the results for males. We applied regression analysis to minimize the effect of skewed distribution of sex in the statistical analysis. This was done to make the findings comparable to earlier studies. However, the result of absence of an association between alexithymia and somatization applies in a strict sense only to females. Young adult males probably tend to have fewer physician visits to the health

center than women and are therefore less likely to have four or more symptoms recorded in their medical records, even though they may have similar symptoms.

Power calculations were not made before statistical analysis. It is possible that the TCI is not sensitive enough to detect characteristic temperament profiles among somatizers. The character items of the TCI were, unfortunately, not collected in this study (Miettunen *et al.* 2004).

In the present study it was not analyzed whether the development of somatization depends on social background, heredity, or interaction between the two. Subjects with and without somatization symptoms could not be differentiated with the temperament profiles of the TCI. Temperament profiles are considered heritable to a substantial degree (Heath *et al.* 1994, Stallings *et al.* 1996, Ando *et al.* 2002, Gillespie *et al.* 2003, Ando *et al.* 2004). The results of the present study are thus not disproportionate to the social environment influence on the development of somatization. Unfortunately, there were no data on the parents or other relatives of the study subjects.

#### **6.4 Strengths of study**

The retrospective use of medical records in the present study for data collection, as opposed to interview or self-reporting questionnaire, can be seen as an advantage. In Finland the public outpatient records are as comprehensive as inpatient records. There were only a few subjects for whom no patient records were available at any of these services. In general, the patient records were extensive, with details of investigations well recorded. Details of investigations and final diagnosis are generally well recorded in case notes, as is attendance for outpatient appointments (Reid *et al.* 2002). The coverage of the information gathered seems to be acceptable. In Finland the use of public health services is four to five times more common than that of private or occupational health services (Statistical yearbook of the social insurance institution 1998, Health care visits 1985-2002 2004). We may have overcome the recall bias that happens when asking patients how they had used health services before (Peveler *et al.* 1997, Crimlisk *et al.* 1998, Reid *et al.* 1999, Kroenke 2001).

The present study dealt with a sample of subjects of the same age and ethnicity. As we had a relatively large sample, we were able to avoid type I error, which may be one cause for the earlier positive findings regarding the association of somatization and alexithymia. Other causes may be methodological differences in different studies, such as different populations, different measures of

somatization, alexithymia or temperament. The findings of Miettunen *et al.* showed that the TCI is applicable to young Finnish people (Miettunen *et al.* 2004), and the results are probably not biased due to cross-cultural problems in measuring temperament profiles. TAS-20 has been shown to be applicable to young Finnish people (Joukamaa *et al.* 2001).



## **7 Conclusions**

### **7.1 Main results**

Our study shows that it is difficult to recognize somatizing patients in standard clinical work at either primary or secondary level of services. This may imply that general practitioners or hospital physicians present these patients on consultation as difficult cases or frequent attenders without medically explained reasons. The patient records review method that we used in our study was a laborious one. However, we would like to stress the importance of the review of somatic patient records during psychiatric consultation as well. Combined with a personal interview it may help to make an accurate SD diagnosis.

The prevalence of SD in this study was 1.1% (N=18) and that of somatization 6.1% (N=97). There was a female predominance of 5:1 in SD and 6:1 in somatization. SD was recognized only with the review of case notes. Somatization did not associate with alexithymia, neither could a characteristic temperament profile be found for subjects with somatization. Somatization was associated with psychiatric disorders.

Regarding the current theories for somatization we were unable to confirm our hypothesis of a certain temperament construction of subjects with somatizing symptoms in Study IV. In Study III we found no evidence for the association between alexithymia and somatization symptoms. The findings do not support the hypothesis that alexithymia is a common feature among subjects with somatization symptoms. In our data, only anxiety disorders of the four main psychiatric disorder groups correlated significantly with somatization symptoms.

### **7.2 Clinical implications**

Patients with somatization symptoms believe they have a physical illness and do not seek psychiatric assistance. Psychiatrists and primary care physicians need better training and education about the recognition, differential diagnosis and treatment of both individual somatoform disorders and those that are comorbid with other psychiatric and medical illnesses. Psychiatrists and primary care physicians need to forge further multidisciplinary relationships and care models appropriate for the care of the somatoform-disordered patient. These patients need

a place to be seen and to be taken seriously rather than being shunted back and forth between clinics.

The findings of the present study may have implications for liaison and other consulting psychiatrists. Various reports have been published concerning the patient-doctor relationship of somatizers that seemed to be associated with problems and difficulties experienced by the physician. These patient contacts seem to be particularly dissatisfying for physicians to manage (Lin *et al.* 1991, Hahn *et al.* 1996, Jackson & Kroenke 1999, Kroenke 2000, Hartz *et al.* 2000). We agree with Bass *et al.* that a joint business case between the medical and psychiatric providers is needed (Bass *et al.* 2001).

It is important to recognize psychiatric disorders behind somatization symptoms. Roughly half of the somatizers have a comorbid psychiatric disorder. Somatizers with anxiety disorders may worry and seek help from health care probably more frequently than somatizers with depression, who may be passive and tend to become isolated.

Somatization can be considered to be an important clinical phenomenon and socioeconomic problem. There is evidence that if primary health care physicians, rather than psychiatrists, treat the patients with somatic complaints, this leads not only to improved clinical outcomes, but is also associated with improved physical functioning and reduced health care costs.

### **7.3 Implications for future research**

There is a need for studies assessing the impact of heritability and social environment in the development process of somatization. A better understanding of the etiology and inter-generational transmission of chronic somatization symptoms would make prevention efforts more feasible. A search for a natural course, associated predisposing and precipitating factors, and a characteristic family history should be conducted. Searching for the most appropriate method for measuring somatization calls for more study. Future research should also explore the possibility that somatization might associate with increased mortality.

Thus far, the longitudinal database of the NFBC 1966 has provided unique opportunities to investigate various outcomes up to early adulthood. In the future there will be a possibility to study the occupational and social performance as well as various psychological and somatic outcomes among middle-aged cohort members with and without somatization symptoms in a longitudinal approach. Obviously not all possibilities have been used to study somatization symptoms

among these subjects using longitudinal follow-up data and register data, for example updated FHDR data. Future follow-up studies might offer a possibility to evaluate other theories presented in the literature review.



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## Original Publications

- I Karvonen JT, Veijola J, Jokelainen J, Läksy K, Järvelin MR & Joukamaa M (2004) Somatization Disorder in young adult population. *General Hospital Psychiatry* 26: 9-12.
- II Karvonen JT, Joukamaa M, Herva A, Jokelainen J, Läksy K & Veijola J (2007) Somatization symptoms in young adult Finnish population - associations with sex, educational level and mental health. *Nordic Journal of Psychiatry* 61: 219-224.
- III Karvonen JT, Veijola J, Kokkonen P, Läksy K, Miettunen J & Joukamaa M (2005) Somatization and alexithymia in young adult Finnish population. *General Hospital Psychiatry* 27(4): 244-9.
- IV Karvonen JT, Veijola J, Kantojärvi L, Läksy K, Miettunen J & Joukamaa J (2006) Temperament profiles and somatization – an epidemiological study of young adult people. *Journal of Psychosomatic Research* 61(6): 841-6.

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