

# Somatization in Survivors of Catastrophic Trauma: A Methodological Review

Carol S. North

Department of Psychiatry, School of Medicine, Washington University, St. Louis, Missouri, USA

The literature on mental health effects of catastrophic trauma such as community disasters focuses on posttraumatic stress disorder. Somatization disorder is not listed among the classic responses to disaster, nor have other somatoform disorders been described in this literature. Nondiagnostic "somatization," "somatization symptoms," and "somatic symptoms" form the basis of most information about somatization in the literature. However, these concepts have not been validated, and therefore this work suffers from multiple methodological problems of ascertainment and interpretation. Future research is encouraged to consider many methodological issues in obtaining adequate data to address questions about the association of somatization with traumatic events, including *a*) appropriate comparison groups, *b*) satisfactory definition and measurement of somatization, *c*) exclusion of medical explanations for the symptoms, *d*) recognition of somatizers' spurious attribution of symptoms to medical causes, *e*) collection of data from additional sources beyond single-subject interviews, *f*) validation of diagnosis-unrelated symptom reporting or reconsideration of symptoms within diagnostic frameworks, *g*) separation of somatization after an event into new (incident) and preexisting categories, *h*) development of research models that include sufficient variables to examine the broader scope of potential relationships, and *i*) novel consideration of alternative causal directionalities. **Key words:** causal directionality, disaster, epidemiology, psychiatric diagnosis, somatic, somatization, symptoms, traumatic events. *Environ Health Perspect* 110(suppl 4):637–640 (2002).

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## Introduction to Somatization Disorder

In healthcare settings, the term somatization is applied to symptoms without medically identifiable origins. Some patients with chronic prolific complaints of such symptoms receive a psychiatric diagnosis of somatization disorder. Somatization disorder is defined by chronic complaints of widespread medically unexplained symptoms across multiple organ systems. Diagnosis requires documentation at some time in the patient's history (not necessarily simultaneously) of clinically significant, medically unexplained symptoms in four symptom groups: pain complaints (four symptoms), gastrointestinal complaints (two symptoms), pseudoneurological complaints called conversion symptoms (one symptom), and complaints referent to sexual or reproductive functions (one symptom). By definition the onset must be documented to have occurred before the individual is 30 years of age to qualify for the diagnosis (1). The condition occurs predominantly in women (1,2), typically beginning during the decade following puberty (3) and persisting for life. One in every 50 to 100 women in the population suffers from this disorder (4). Its origins are partly genetic (1,5): 25% of the first-degree female relatives of these patients also have somatization disorder, and their male relatives have elevated rates of antisocial personality disorder, criminality, and alcohol problems, and often a history of physical abusiveness.

Somatization disorder is one of the oldest psychiatric and medical disorders known to

mankind. Ancient Egypt and classical Greek and Roman cultures acknowledged the condition, previously called hysteria, the Latin root for uterus. The uterus was thought to wander through the body, provoking an array of physical symptoms wherever it came to rest. Many of Freud's patients had hysteria. In therapy these patients described extensive histories of childhood trauma and sexual abuse. At first Freud assumed hysteria to result from these traumas, but with a later change of heart he revised his theories reflecting reinterpretation of his patients' allegations as fabricated. The disorder was renamed somatization disorder in 1980 to reflect a revised definition based on its characteristic multiple, medically unexplained symptoms. Of seven psychiatric diagnoses involving medically unexplained symptoms defined in the somatoform disorders category of the *Diagnostic and Statistical Manual of Mental Disorders-IV* (1), somatization disorder is the most severe.

Patients with somatization disorders complain not only of somatic symptoms of physical disorders they do not have but also of psychological symptoms of psychiatric disorders they do not have, demonstrating the phenomenon is not only somatoform but psychoform as well (3,6–8). A study in a university psychiatry clinic by Lenze and colleagues (9) found that patients with somatization disorder complained of more depressive symptoms than patients from the same clinic who had major depression, as many manic symptoms as clinic patients with bipolar disorder, as many anxiety symptoms as

anxiety disorder patients, and as many psychotic symptoms as those with schizophrenia. Thus, patients with somatization disorder can be described as complaining that everything is wrong, both physical and mental. The volumes of symptom complaints from these patients prompts diagnosis of an abundance of additional psychiatric diagnoses (8,10–12). Therefore, clinician vigilance is important to detect somatization disorder whose psychopathology mimics other psychiatric disorders. Somatization disorder is present in about 10% of psychiatric patients and hospitalized medical and surgical populations (13,14).

In both clinical and research settings, the problem with trying to establish a diagnosis of somatization disorder is that the syndrome does not present in a straightforward manner. Somatization disorder is usually overlooked in both clinical and research settings (15–17), partly because of failure to factor in the differential, but also because even when it is considered, data gathered from the patient interview alone are often insufficient to establish the diagnosis (3,18,19). This is because these patients *a*) routinely deny symptoms, even symptoms that were chief complaints documented in their medical records during previous visits to healthcare specialists, and *b*) offer spurious medical explanations for their symptoms (18,20). Typically, it is necessary to obtain the information historically from various medical records and/or to observe the patient over time to collect the range of symptom complaints needed to meet criteria for the diagnosis (1,18–20). Identification of somatization disorder requires the ability to rule out medically based explanations of symptoms. Only when medically based etiologies can be confidently dismissed can symptoms be considered somatoform, and assessment cannot proceed without it.

Given these problems with identifying somatization disorder, it is understandable that studies relying solely on patient interview data grossly underestimate the prevalence (18,19,21,22). In clinical settings, patient interview studies may underestimate rates of

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Address correspondence to C.S. North, School of Medicine, Washington University, 660 S. Euclid Ave., Campus Box 8134, St. Louis, MO 63110 USA. Telephone: (314) 747-2013. Fax: (314) 747-2140. E-mail: northc@psychiatry.wustl.edu

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somatization disorder by a factor of 20 or more (23). Even the widely acclaimed Epidemiologic Catchment Area study identified only about one-tenth the prevalence of the disorder estimated for the population by more comprehensive assessment methods (24). Patients with identified somatization disorder therefore represent only the tip of the iceberg of the disorder in these populations. Collecting the extensive clinical documentation necessary to rule out the diagnosis is excessively laborious for most clinical and research endeavors. In ordinary practice the diagnosis often relies solely on patient report. Because nondetection of somatization disorder by patient interview techniques yields apparently low case rates in clinical and research settings, the disorder as formally defined has been assumed to be rare and non-applicable for use in various populations (23). A large reservoir of patients who somatize yet do not meet criteria for a somatoform disorder diagnosis is assumed to describe the widely recognized presence of medically unexplained symptoms in the absence of a detectable somatoform disorder in various patient populations.

To capture this hypothesized broader population, alternative methods have been used to identify somatization. One alternative involves application of screening tools or abridged indices of somatization to describe lesser degrees of the phenomenon, using a lower threshold or relaxed diagnostic criteria (22,23,25–28). Studies of patients identified by less stringent criteria found them to exhibit the typical associated features of the full somatization disorder, which researchers interpreted as indicating the validity of abridged methods of ascertainment (22,27,29–31). However, the characteristic features of somatization disorder in patients with apparent sub-syndromal somatoform presentations may simply reflect the inclusion of many unrecognized somatization disorder patients in a mixed group of somatizing patients.

More commonly, somatization has been conceptualized as a behavior outside the context of diagnosable syndromes (32,33), such as on a continuum from mild to severe and chronic (34). It is not clear, however, what the dimensional construct of somatization represents in the absence of delineated disorders that have been validated by conventional methods established for diagnostic entities. Ascertainment of symptoms outside the context of diagnosis creates the same difficulties encountered with consideration of medical symptoms in general outside the context of medical diagnosis. The problem is illustrated in medical settings with the symptom of chest pain and attempts to characterize it and recommend treatment without consideration of whether the symptom is due to a heart attack,

pneumonia, a pulmonary embolus, esophageal erosion or spasm, musculoskeletal injury, or even a panic attack. Although studying somatization as a dimensional construct rather than as dichotomous diagnosis requires far less effort and may improve statistical power, any potential benefits can actually provide little real advantage considering problems of validity of symptom-based data outside the context of established diagnostic nosology (35).

### Somatization in Disaster Literature

The literature on mental health effects of catastrophic trauma such as community disasters focuses on posttraumatic stress disorder. Somatization disorder is not listed among the classic responses to disaster, nor have other somatoform disorders been described in this literature. However, nondiagnostic “somatization,” “somatization symptoms,” and “somatic symptoms” have been abundantly described in the literature. Failure to differentiate “somatic” symptoms (referring to any physical symptom complaint without regard to medical basis) from “somatoform” symptoms (limited to physical symptoms without medical basis) is a significant shortcoming of many studies that use symptom scales such as the Symptom Checklist-90 (SCL-90). The SCL-90 has been the most popular symptom scale in postdisaster psychiatric assessment (36). Illustrating this instrument’s problem with validity, a study that used the SCL-90-R to assess patients with brain tumors found that one-half of the brain tumor patients endorsed somatic symptoms, leading them to be classified with a group of somatizers (37,38).

Symptom scales have numerous additional methodologic shortcomings when used in trauma research. Even though the originator of the SCL-90 clearly stated the scale does not provide diagnostic information (36), it and other symptom scales have been used incorrectly to identify cases of somatization through establishment of cutoff points for caseness. In purely dimensional use, scores are compared with other variables to show associations with somatization behavior, a construct lacking validity outside established nosologic frameworks. Many instruments in trauma research measure global distress and fail to differentiate somatization from depression and anxiety (39). Somatization symptom scales are unable to detect and correct for response sets and social desirability (40–42), significant features of the presentation of symptoms among patients who somatize extensively. Finally, measurement of somatization in studies of mental health effects of trauma encounters all the problems identified earlier in this chapter related to studying somatization in other settings, including selective reporting and medicalization of symptoms characteristic of

somatizing populations and lack of provisions to exclude a medical basis for physical symptom complaints.

In most traumatized populations, risk for traumatic events is confounded with preexisting psychosocial factors. Somatization disorder patients, for example, report higher rates of traumatic events than other people, and after traumatic events they report more problems and symptoms than others. To assume that the problems (including somatization) observed after the traumatic event are caused by the traumatic event itself invokes the classic logical error of *post hoc ergo propter hoc* (after the fact, therefore because of the fact). Uncertainty about attribution of symptoms is a particular problem in somatizing populations due to the well-known suggestibility characteristic of patients with somatoform disorders, which further complicates the vagaries of normal recollection. Normal memory links occurrences with other events as temporal signposts of memory (e.g., “I remember it well, because it was the week after my mother had died, I had a house full of company, and I went to the emergency room thinking I was having a heart attack.”). Somatization disorder patients find events to anchor their many somatoform symptoms in time.

Somatizing patients are well known for their suggestibility and unreliability in reporting, and attribution of symptoms is subject to redirection. Causal assumptions may be generated without basis, for example, after a traumatic event such as a disaster, when symptoms may be reattributed to the event. Careful examination of pre-event history to document the occurrence of the same symptoms and patterns of multiple complaints prior to the event is necessary to consider causal associations. Therefore, prospective data are superior to retrospective data to sort out causality and directionalities of apparent associations, particularly when they involve somatoform complaints. In disaster research, these deliberations demand two simultaneous comparisons: *a*) between exposed and unexposed (or less exposed) populations, and *b*) pre-event versus postevent status, preferably in a prospective design. Disaster research, however, does not lend itself to a prospective, randomized experimental design because of the general lack of announcements preceding such impending events. Thus, the best proxy is to study populations prior to a disaster and then compare the postdisaster status of the exposed to that of the unexposed.

One of the best sources of material for studying mental health effects of traumatic events is the setting of major disasters, which tend to be equal-opportunity events selecting people randomly. (A notable exception is flooding, which tends to affect lower-income populations that settle on flood plains because

the land is less expensive. These populations have endemically high rates of psychopathology independent of disasters). Disaster studies can therefore sidestep much of the confounding of preexisting psychopathology with risk for traumatic events in other populations of trauma survivors such as victims of assault, rape, and motor vehicle accidents.

Selection of treatment populations further compounds the methodological issues in studies of mental health effects of trauma. People who seek out treatment are nonrepresentative of all people with psychiatric disorders; sampling from treatment settings selects for individuals with severe psychopathology (those with the most frequent treatment contacts) and constitutional personality factors, thus adding further bias to the sample selection.

The above methodological issues are critical for the interpretation of the literature on somatization after disasters. Numerous studies have reported that somatization and somatic symptoms are associated with increased risk for exposure to traumatic events and with occurrence and severity of posttraumatic stress symptoms and disorders. Many such studies are so hindered by the methodological limitations described above, however, that their conclusions may not be justified from the data collected.

Two remarkable studies have surmounted many of the methodological problems identified in this research and in prospective research designs, diagnostically assess disaster-exposed compared with unexposed populations before and after disaster. In the first study, population data on psychiatric disorders obtained a year before the occurrence of torrential rains and mudslides in Puerto Rico (43–45) were used in a prospective examination of predisaster and postdisaster somatoform symptoms in the unexposed population compared with those of the disaster-exposed group. Disaster exposure was significantly associated with development of new, medically unexplained physical symptoms not present before the disaster, especially gastrointestinal and pseudoneurological symptoms. The authors concluded that somatic symptoms are “an important component of the disaster-reactive psychopathological repertoire” [(45) p. 966] and represent “true outcomes” [(44) p. 674] of the disaster. They cautioned, however, that the magnitude of the findings was very small (indicated by regression slopes on the order of 0.05 to 0.07), allowing for contribution of nonspecific effects such as unsanitary conditions after the disaster (44).

In the second study, Robins, Smith, Solomon and colleagues (46–48) conducted a prospective analysis of somatoform symptoms in disaster survivors relative to an unexposed comparison group. The study was conducted in association with a series of disasters (radioactivity in well water, dioxin

contamination, floods, and tornadoes) in Times Beach, Missouri, near St. Louis. Serendipitously, predisaster psychiatric diagnosis data had been collected in the same area just 1 year before the disasters as part of the large Epidemiologic Catchment Area study (49) of psychiatric disorders in the general population. After the disasters, Robins and colleagues (47) identified only one case of somatization disorder—in the unexposed comparison group, with onset before the events of interest—and the rates of somatization symptoms were not unusually high in this population. Somatization symptoms were not more prevalent after the disasters, and new symptoms had not developed. The study determined that the disaster-exposed group differed from the nonexposed group before the disaster and had a higher prevalence of phobias, interpersonal problems, and poorer health, illustrating selection bias for disaster exposure based on preexisting personal vulnerabilities. The authors concluded that somatization was not an observed outcome of the disasters because the disaster-exposed group had only small and clinically inconsequential changes.

## Discussion and Conclusions

As discussed above, disasters in general represent the most pure form of traumatic events for research on mental health effects of traumatic events because of less confounding from selection bias for predisposing characteristics that increase the odds for exposure to traumatic events. Even this research, however, has serious potential methodological pitfalls, including

- failure to exclude medical explanations for the symptoms (contaminating the desired somatization data with somatic symptoms of medically based conditions, thereby overestimating somatization)
- spurious attribution of medically unexplained symptoms to medical causes by somatizers, thus overlooking somatization
- failure to detect the majority of somatization disorder cases in single-subject interviews
- lack of established validity of diagnostically unrelated symptom report data
- failure to classify somatization following specific events into new (incident) and pre-existing categories
- inappropriate assumption of causal directionality from data merely identifying association.

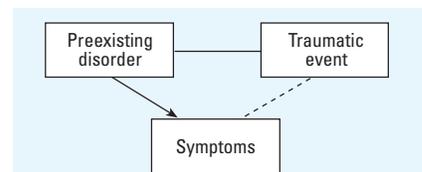
Somatoform symptoms or disorders present after a disaster must be interpreted carefully to determine that they were either not present before the disaster or, if they were, that they were worse after the disaster. Studies have generally failed to do this. The two studies exercising the most methodological care in

examining these issues found little evidence of clinically important new somatization after disasters.

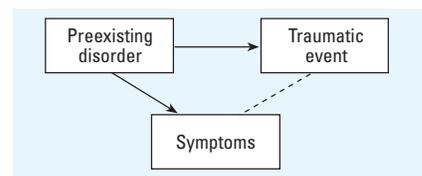
Robins (50) pointed out the importance of searching for possible common antecedents in stress research, allowing other potential interpretations in more sophisticated models. The simplest model involves straight-line causal effects of a specific stressor leading directly to a subsequent disorder. Robins encouraged researchers to develop more complex models with greater numbers of variables to compete with other plausible explanations for the association. In these alternative models, consideration of directionality may lead to novel conclusions about causation. For example, the occurrence of symptoms following exposure to traumatic events might be assumed to reflect causal effects of trauma. Alternatively, other possible interpretations might be that individuals with preexisting psychiatric disorders will continue to exhibit symptoms after the event as well, thus accounting for the apparent association of traumatic events and symptoms (illustrated by the dotted line in Figure 1).

The complexity of the issues may be magnified in studies of traumatic events other than disaster, with confounding that may occur between preexisting symptoms and traumatic events. Other variables associated with psychiatric symptoms may increase vulnerability for traumatic events, creating indirect associations between symptoms and traumatic events (illustrated in Figure 2).

Assumption of causal directionality from the traumatic event to those symptoms may be an incorrect interpretation of the data. For example, one needs to consider that preexisting somatoform disorders could increase the actual risk for traumatic events or be associated through biased reporting of traumatic event history, thus explaining the apparent



**Figure 1.** First causal model of trauma and psychopathology.



**Figure 2.** Second causal model of trauma and psychopathology.

association of traumatic events and somatization symptoms indirectly based on the preexisting characteristics of these individuals. Widom (51) thus recommended that future studies control for confounding factors and additional factors (e.g., dispositional attributes, environmental conditions, biological predispositions, and positive events) that may mediate mental health effects of traumatic experiences.

Future research is encouraged to consider the many methodological issues discussed above in obtaining adequate data to address questions about the association of psychopathology such as somatization with traumatic events. These specific methodological issues include

- appropriate choice of comparison groups
- satisfactory definition and measurement of somatization
- exclusion of medical explanations for the symptoms
- recognition of somatizers' spurious attribution of symptoms to medical causes
- collection of data from additional sources beyond single interviews of subjects
- validation of nondiagnostic symptom reporting or reconsideration of symptoms within diagnostic frameworks
- classification of somatization after an event into new (incident) and preexisting categories
- development of research models that include sufficient variables to examine the broader scope of potential relationships
- novel consideration of alternative causal directionalities.

These modifications of research may dramatically affect conclusions reached in research studies investigating mental health effects of exposure to trauma.

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