Assessing Beliefs about 'Environmental Illness/Multiple Chemical Sensitivity'

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Abstract

Knowledge representation was used to characterize beliefs in patients with Environmental Illness/Multiple Chemical Sensitivity (EI/MCS). EI/MCS patients, allergy and asthma patients, doctors and controls made relatedness judgments on concepts relevant to EI/MCS. Associative networks showed that EI/MCS patients viewed these concepts differently from others. *Multiple chemical exposure* was central in EI/MCS networks, with many links to every other concept, but was only peripherally connected in the other subject networks. Similarity comparisons to an EI/MCS prototype network discriminated EI/MCS patients from the other control populations, as did an index based on critical concept pairs. This approach shows promise for distinguishing patient groups using belief structure.

Keywords

assessing beliefs, chemical sensitivity, patient beliefs

THAT EXPOSURE to or ingestion of chemicals can produce or exacerbate disease is a well-documented and accepted tenet of modern medicine. There is no question that environmentally related illness can result from immunologic sensitization, various physical factors, infection or direct toxic or pharmacological effects of natural and synthetic substances (Upton, 1990). Specialists in fields such as allergy and clinical immunology, clinical toxicology, neurotoxicology and occupational medicine clearly demonstrate adverse effects associated with exposure to chemicals in well-documented, critically reviewed, scientific publications (Frank et al., 1985; Gammage & Kaye, 1985; Hathaway, Proctor, Hughes, & Fischman, 1991; National Research Council, 1992; National Institutes of Health [NIH], 1984).

These diseases generally present with characteristic symptoms and pathology, but in recent years, physicians of various specialties noted a rising number of patients presenting with vague, multisystem somatic complaints characteristic of general malaise (see Table 1). Despite a comprehensive medical history, physical examination, laboratory tests and careful clinical observation, many of these cases fail to show concrete signs of organic disease (Terr, 1986, 1989). There are increasing incidents of patients, physicians and other health-care practitioners attributing such subjective symptoms to exposure to environmental chemicals, but without concrete evidence to support the assertion. In many cases, infinitesimal quantities of environmental chemicals emanating from domestic, industrial workplaces or 'sick buildings' are implicated. Symptoms originally induced by a single chemical are often subsequently reported following exposure to an ever-increasing number of diverse chemicals.

For lack of a diagnostic label, these patients and their illness have been given various names including 'universal reactors', 'total allergy syndrome', 'allergic to the 20th century', 'chemically hypersensitive', 'having environmental illness' or 'multiple chemical sensitivities'. Because some of the attributions of environmental agents go beyond chemicals, we refer to this phenomenon as 'Environmental Illness/Multiple Chemical Sensitivity', EI/MCS (Sparks et al., 1993). The polysomatic symptoms of these patients are strikingly similar to the gen-

Table 1: Pool of common presenting symptoms of EI/MCS patients

Head, Eye, Ear, Nose and Throat	Eye burning, phoria, tinnitus, vertigo, rhinorrhea, nasal obstruction, nasal burning, glossodynia, pharyngeal irritation, dysphonia
Pulmonary	Dyspnea, cough, chest pain
Cardiovascular	Palpitations, irregular heartbeat
Gastrointestinal	Dyspepsia, eructation, flatus, recurrent diarrhea, constipation, cramping pain
Genitourinary	Dysmenorrhea, vaginitis, dysuria, urinary frequency, urinary retention
Musculoskeletal	Myalgia, weakness, muscle tension, arthralgia, dyskinesia, skin irritation, pruritus without rash
Lymphatic	Fluctuation in size of lymph nodes
Neurological, Psychological	Headaches, neurasthenia, numbness, anesthesia, paresthesia, hyperesthesia, fatigue, irritability, hyperactivity, sleep dysfunction, cognitive dysfunction, decreased attention span, loss of concentration, memory loss, anxiety, depression, mood swings

eral malaise complaints characteristic of somatoform disorders (Barsky & Klerman, 1983; Quill, 1985; e.g., see Shorter's [1992] discussion on neurasthenia). Much of the evidence suggests that psychological mechanisms are at the root of some patients' beliefs in environmental illness and the accompanying symptoms (Black, Rathe, & Goldstein, 1990; Brodsky, 1983; Selner, J. C., & Staudenmayer, 1986, 1992a, 1992b; Selner, J. C., Staudenmayer, Koepke, Harvey, & Christopher, 1987; Simon, Daniell, Stockbridge, Claypoole, & Rosenstock, 1993; Simon, Katon, & Sparks, 1990; Staudenmayer & Selner, J. C., 1990; Staudenmayer, Selner, J. C., & Buhr, 1993; Staudenmayer, Selner, M. E., & Selner, J. C., 1993; Stewart, 1987; Stewart & Raskin, 1985).

Advocates of this unsubstantiated theory, who refer to themselves as *clinical ecologists* (and, more recently, *environmental medicine physicians*), can be identified by their approach to the evaluation and management of EI/MCS patients. These clinicians may employ some of the

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terminology and procedures of traditional allergy and clinical immunology but often use methods of provocation testing and treatment which lack a firm scientific foundation (American Academy of Allergy and Immunology, 1986; American College of Physicians, 1989; California Medical Association Scientific Task Force on Clinical Ecology, 1986; Council of Scientific Affairs, American Medical Association, 1992; Kahn & Letz, 1989; Terr, 1987; Thompson, 1985) and which also incur great financial, occupational and psychological cost to the patient (Black, 1993).

Learned sensitivity, symptoms and beliefs

The somatoform disorders are characterized by the awareness and reporting of physical symptoms which lack a clear physical or biological basis, but are nevertheless subjectively real (Barsky, Goodson, Lane, & Cleary, 1988; Lipowski, 1988; Pennebaker & Watson, 1991; Robbins & Kirmayer, 1986). Proponents of the EI/MCS phenomenon have advanced hypotheses about biological individual differences which could explain the subjective reality of such disorders (Ashford & Miller, 1991). For example, the heightened sensation hypothesis suggests that certain individuals may have sensory thresholds lower than others. In the context of the EI/MCS phenomenon, such individuals are assumed to suffer from chemical hypersensitivity (Morrow, Ryan, Goldstein, & Hodgson, 1989; Ryan, Morrow, & Hodgson, 1988). In controlled studies, however, EI/MCS patients do not show olfactory thresholds lower than other subject populations (Doty, Deems, Frye, Pelberg, & Shapiro, 1988). In fact, food imagery in the absence of odors had the same effect on the electroencephalogram (EEG) as when food odors were physically present (Lorig, 1989), suggesting that odor perception is cognitively mediated.

Another explanation for environmental illness focuses on limbic kindling in the central nervous system (Ashford & Miller, 1991). Limbic kindling is based on an animal model for seizure disorders induced by stimulation with electrical current or high doses of a pharmacologic agent (Gilbert, 1992a, 1992b). However, no evidence for the phenomenon has been presented in humans. In fact, the limbic kindling model has been revised to require only 'partial' kindling (Bell, Miller, & Schwartz, 1992), such that outcome measures are behavioral and subjective, without the standard criterion of observable seizure. As applied to the EI/MCS phenomenon, kindling or partial kindling proposes that odorous chemicals inhaled through the nose are transported via the olfactory bulb into the limbic system, either directly or by proximal excitation of neurons. From the limbic system, excitation then spreads (the kindling effect) to other regions of the brain, triggering a variety of cognitive and emotional symptoms. Interestingly, proponents of limbic- (or partial-limbic) kindling theory concede that there is no evidence to support the theory as it applies to the EI/MCS phenomenon (Bell et al., 1992).

Classic application of Pavlovian reflex conditioning has also been used to explain adverse reactions to environmental stimuli (Djuric & Bienenstock, 1993; MacQueen, Marshall, Perdue, Siefel, & Bienenstock, 1989; Russell et al., 1984). The popular concept of a conditioned response in environmental illness is that of a simple stimulus-response pairing. It is assumed that an exposure to a toxic chemical leads to a physiological response which then becomes associated with the odor of that chemical (Bolla-Wilson, Wilson, & Bleeker, 1988). Subsequent detection of the odor will lead to symptoms which were initially brought on by the toxic chemical.

Proponents of more recent conceptualizations of classical conditioning argue that cognitive mediation is essential to conditioning (Brewin, 1989; Dickinson, 1987). A mere contingency between stimulus and response is no longer necessary or sufficient to establish a learned response. Rather, both conscious and automatic cognitively mediated processes, such as expectations, beliefs and especially inferences about causal relationships, are the processes that define conditioning (Rescorla, 1988). In the case of EI/MCS, odor is a significant trigger to symptom response, but the locus of control underlying the association to odor is not yet clear (Selner & Staudenmayer, 1992b). One basis could be the irritant properties of the chemical, the unconditioned stimulus (UCS) paired with the perceptual properties of that

chemical, the conditioned stimuli (CS), as is the case in some well-documented cases of toxic exposure (Bolla-Wilson et al., 1988; Shuster-man, Balmes, & Cone, 1988). Another basis for the learning, and one which is consistent with the psychological profile of EI/MCS patients, however, could be a psychophysiological response, the unconditioned response (UR), to a conscious or unconscious memory of trauma (UCS) which has been paired with the stimulus properties of the chemical (CS).

The subjective 'realness' of somatoform disorders can result in an amplification of bodily sensations to the point of experienced symptoms, making it extremely difficult to separate effects induced by beliefs from true physiologic responses (Barsky et al., 1988; Pennebaker & Watson, 1991). Thus, diagnosis and treatment may be more than a matter of cataloging physical symptoms and may instead necessitate a thorough understanding of the patient's belief system as it relates to specific symptoms.

Mental representation in health and illness

The study of individuals' mental representations of illness and health increased substantially in recent years. Leventhal, Meyer, and Nerenz (1980) provide the inspiration for much of the recent work with their theoretical analysis and empirical data showing that people understand health threats and regulate their health behavior by means of 'illness schemata,' implicit theories of illnesses, causes and cures. An edited volume by Skelton and Croyle (1991), *Mental Representation in Health and Illness*, brings together much of the research on this topic to that time.

Although it seems apparent that people's conceptions of illness should have a major impact on their reactions to illness and in their illness-related behaviors, the systematic investigation and application of this idea is relatively recent. Much of the work on the topic has not yet reached the point where it has clear implications for medical practice, but the potential for application in both diagnosis and treatment seem profound. For example, predicting outcomes is a practical implication of the work in mental representations. Lacroix (1991) developed methods for assessing illness schemata in patients who suffer from low back pain and is able to predict the likelihood that patients would return to work from the accuracy of their illness schemata. Those with more accurate schemata were more likely to resume working in shorter periods of time. Additionally, in some cases, treatment may involve attempts to change people's conceptions as a prelude to applying medical interventions.

Finally, in many cases of psychosomatic disorders, treatment depends primarily on changing beliefs. Diagnosis, in these cases, is largely a matter of assessing beliefs together with establishing the lack of a medical basis for patients' complaints. The problem of assessing and adequately characterizing beliefs or schemata, however, is a major limiting factor in the study of mental representations of illness. Many studies rely on interviews and questionnaires to provide the information required to characterize individuals' schemata. These approaches are costly in the time and effort required and also suffer from limitations of self-report such as patients' ability and willingness to self-disclose accurately and without bias (Mendelson, 1987), as well as limits on memory processes which may result in partial, rather than full, reporting of symptoms. Such factors are particularly germane to EI/MCS patients (Staudenmayer & Selner, 1995). Because these patients frequently encounter disbelief when dealing with doctors, many become wary of dealing with the mainstream medical community. For these patients, a more indirect method than direct questioning may be an attractive alternative. The approach we propose has potential for decreasing the patients' reactivity to the process of obtaining information, as well as demands on recall.

In summary, many of the studies in this area are concerned with the impact of people's conceptions of illness on their behaviors regarding the illness. Although this work is clearly related to our work on the role of beliefs in psychosomatic disorders, our emphasis is more on the causal role of beliefs in such illness than on the role of beliefs or schemata in patients' reactions to medical conditions. We now turn to a discussion of empirically derived network representations and their potential applicability as a means for assessing the mental representation of beliefs.

An empirical approach to representing beliefs

The Pathfinder network scaling algorithm (Schvaneveldt, 1990; Schvaneveldt, Durso, & Dearholt, 1989) generates empirically derived network representations of the associative structure among a set of concepts. Pathfinder takes distance estimates (e.g. relatedness ratings) as input to use in generating networks where each concept is represented by a node, and the relations between concepts are represented by links between nodes. The algorithm examines distances between nodes and includes a link in a Pathfinder network if the link provides the shortest path between the two nodes it connects as compared to the set of alternatives. In other words, a link is included when there is no alternative path (through other concepts) which is shorter than the direct link. The Pathfinder algorithm uses two parameters, r and q, to determine the method for computing the length of alternative paths. The first parameter, the Minkowski r-metric, determines how distance between two nodes not directly linked is computed. When $r =$ infinity, the length of a path is equal to the magnitude of the maximum link on the path. This means that only ordinal assumptions are required of the data because when $r =$ infinity the same links will be included for any monotonically increasing transformation of the data. The second parameter, q, limits the number of links allowed in searching for shorter alternative paths. When $q = n - 1$, where n equals the number of concepts being compared, there is essentially no limit on the number of links allowed in paths because the longest possible path has $n - 1$ links.

In summary, the Pathfinder network scaling method starts from estimates of distances between all nodes (or concepts). In the resulting network, links are included if and only if the distance associated with the link is the shortest distance between the nodes compared to the set of alternatives. The r-parameter determines how distance in multiple link alternative paths is computed, and the q-parameter determines the maximum number of links in the alternative paths.

Readers familiar with work on cognition will recognize that Pathfinder networks are related to semantic networks. However, the primary method for constructing semantic networks is theoretical in nature (Collins & Loftus, 1975; Meyer & Schvaneveldt, 1976; Quillian, 1969). Pathfinder generates networks empirically from distance estimates, obtained by having subjects judge the strength of the relationship between two concepts. The use of relatedness ratings, in combination with techniques for determining the most salient relationships among the concepts may provide a means for better reflecting a patient's system of beliefs than would the more traditional method of eliciting answers to specific questions about symptomology. For example, relatedness judgments may represent a more indirect, but possibly objective, approach to discovering a patient's system of beliefs because it does not suffer from the vagaries of recall or specific context effects. Additionally, use of Pathfinder networks reduces the relatedness data to only the most salient relations among concepts.

Pathfinder is used for representing knowledge in a number of domains; this includes basic research in memory (Cooke, 1992; Cooke, Durso, & Schvaneveldt, 1986), learning (Gomez & Schvaneveldt, 1994), problem-solving (Durso, Rea, & Dayton, 1994), representation of the cognitive structures underlying expertise (Cooke & Schvaneveldt, 1988), and assessment of knowledge growth for academic subject matter (Goldsmith & Johnson, 1990; Housner, Gomez, & Griffey, 1993a, 1993b). In the research reported here, Pathfinder is used to generate belief representation for various populations, including EI/MCS patients, allergy patients with and without asthma, doctors (allergists) and introductory psychology controls to determine whether the patterns of belief in EI/MCS patients differ from those manifested by other subject groups.

The first step in producing Pathfinder networks requires generating a list of concepts for representing the domain of inquiry. In the present study 24 key concepts were identified by a psychologist on our research team who is experienced in treating EI/MCS patients. The concept list contained 14 symptoms frequently identified in interviews with EI/MCS patients together with 10 causal factors (shown in Table 2).

Next relatedness data are obtained by having subjects rate every pair of concepts on a numerical scale. Once produced, the networks can be compared to various reference networks. Reference networks are generated on the same set of concepts but are intended to reflect specific patterns of belief. These networks can be generated empirically (based on averaged data from a group of subjects) or theoretically. An example of a theoretically generated network is the EI/MCS prototype shown in Figure 1. The theoretical basis for this network comes from extensive observation and research on EI/MCS patients. Such research reflects a prototypical tendency for EI/MCS patients to associate the majority of symptoms and causal factors with reactivity to low-level *multiple chemical* (MULTICHEM) exposure.

Our purpose here is to examine the feasibility of using relatedness ratings and Pathfinder networks to characterize the system of beliefs held by EI/MCS patients, physicians who treat these patients, asymptomatic controls and patients with more specific patterns of symptoms (allergy and asthma patients). If EI/MCS patients differ markedly from other research participants in their patterns of belief about their illness then these differences should be apparent in comparison to other populations who make relatedness ratings on the same set of concepts.

Table 2: Concepts used for ratings

Symptoms
ATTENTION difficulty, thinking or concentrating
poor MEMORY
PAIN in back, joints or extremities
muscle WEAKNESS
HEADACHES
PALPITATIONS, irregular heartbeat, pain in chest
BREATHING difficulty, shortness of breath
NAUSEA, stomach or abdominal pain
DIARRHEA or constipation
SLEEP problems
ANXIETY, nervous or panic
DEPRESSION, helpless or hopeless
extreme FATIGUE
SEXUAL difficulties

Causes
reactive to low level
 MULTIPLE CHEMICAL exposure
DISCOMFORT, warm, cold, dry, humid or stuffy
physical OVEREXERTION
EMOTIONAL upset or stress response
NEUROLOGIC or brain abnormality
INFECTION, flu
HEART/LUNG disease
GASTROINTESTINAL disease
MUSCLE injury
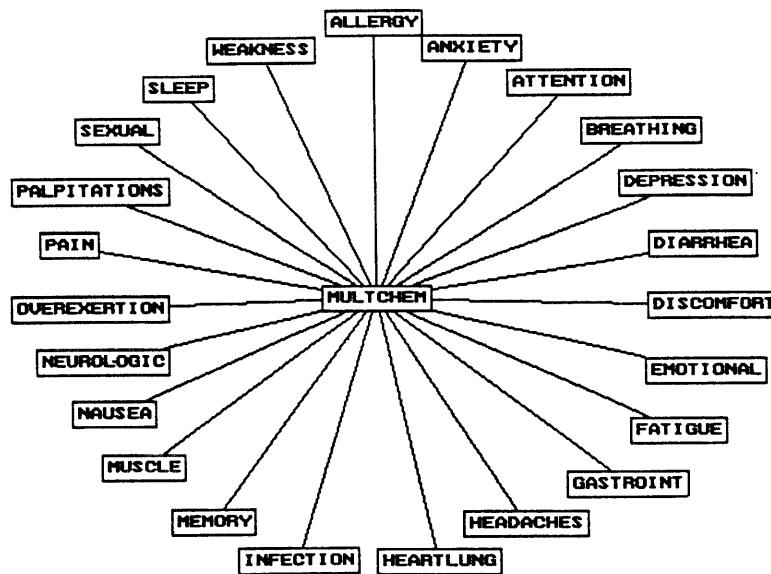ALLERGY to pollens, mold, dust, animals

Fig. 1. EI/MCS prototype (reference) network.

In addition, the networks generated for EI/MCS patients should share more similarity with the prototype EI/MCS network as compared to the other groups.

Method

Participants There were five groups of participants consisting of 87 persons in all: 23 allergy patients without asthma; 20 allergy patients with asthma; 11 EI/MCS patients; 11 allergists; and 22 asymptomatic controls. The allergy, asthma and EI/MCS patients were recruited as outpatients at the Allergy Respiratory Institute of Colorado. Participants in all three patient groups were identified based on a physician's diagnosis. The EI/MCS patients all had records of attributing multiple symptoms to reactivity to multiple chemicals with no traceable physiological basis for their symptoms. All EI/MCS patients who were actively being considered for evaluation or treatment by physicians at the Allergy Respiratory Institute of Colorado during the autumn of 1992 were contacted by phone, followed by a cover letter and ratings questionnaire in the mail. A total of 21 ratings questionnaires were mailed to 15 females and 6 males. The EI/MCS patients who responded were 9 females and 2 males. The women ranged in age from 36 to 60 ($M = 47.67$). The men were between 47 and 55. The allergy participants were all receiving immunotherapy for allergies at the Allergy Respiratory Institute. These patients, who routinely received allergy shots and were recruited over a 2-week period, completed the ratings in the allergy-shot room while waiting for the resolution of allergy injections. The allergy group contained 14 women ranging from 27 to 57 years of age ($M = 37.64$), and 9 men ranging in age from 28 to 64 years ($M = 39.78$). The asthma group, in addition to allergies, also had medically diagnosed, reversible obstructive airways disease (asthma). These patients were recruited and completed the ratings under the same circumstances as the allergy patients. The asthma group contained 15 women ranging in age from 32 to 61 ($M = 45.73$) and 5 men ranging in age from 31 through 50 ($M = 39.80$). The doctors were recruited from a group of internationally renowned experts in allergy and immunology

who attended the 1992 annual Aspen Allergy Conference. The doctors ranged in age from 38 to 72 years; 10 were male ($M = 48$) and the remaining doctor was female (age 46). The control participants were 22 undergraduate college students enrolled in an introductory psychology course at New Mexico State University who were screened for symptoms related to chemical sensitivities. The students participated in this experiment in partial fulfillment of course credit.

Materials and procedure The relatedness ratings were collected by on-line computer or by a paper-pencil-ratings questionnaire administered in the form of a booklet. The questionnaire consisted of an informed consent form, a list of the concepts to be rated (shown in Table 2), and all pairwise combinations of the concepts. Participants were asked to examine the list of concepts before rating them (the concepts were not explicitly labeled as symptoms and causes as they are in the table). Participants then went on to rate every possible pairwise combination of the terms appearing in Table 2. The ratings were collected on a 9-point relatedness scale, where '1' corresponded to 'unrelated' and '9' corresponded to 'highly related'. The pairs were presented in a random order.

Results and Discussion

Group average networks First, average networks for each group were computed from averaging the z-score transformations of each individual participant's data. In all analyses, r was set at infinity, and q was set to $n - 1$ (or 23). These parameter values only require ordinal assumptions of the data, and the networks with the fewest links are produced. The average EI/MCS patient network is shown in Figure 2. Average networks for the allergy and asthma patients, doctors and controls are shown in Figures 3 through 6, respectively. Examination of the networks reveals marked differences between groups.

If EI/MCS patients relate their symptoms to low-level *multiple chemical* exposure, then a representation of their belief system should reflect this. As expected, the average EI/MCS patient network reflects a radial structure with a
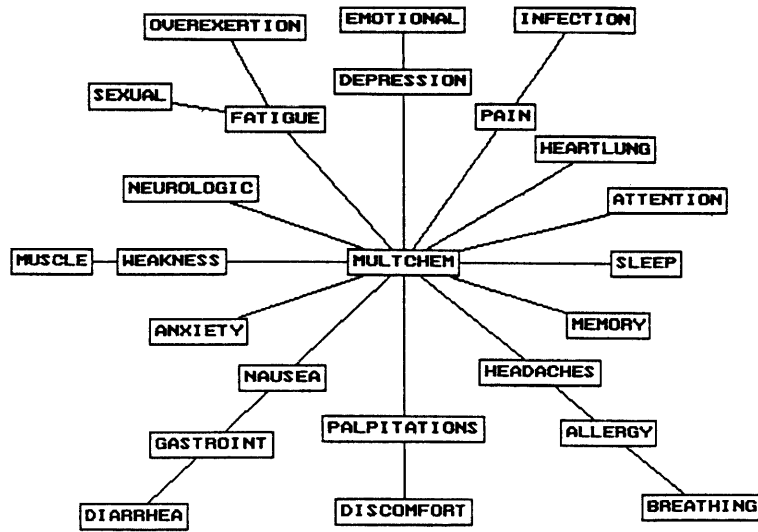
Fig. 2. Average EI/MCS patient network.

number of concepts centering on the concept reactivity to low-level *multiple chemical* exposure. Brodsky (1983) was the first to define MCS patients on the criterion that their lives revolved around their illness. The network shown in Figure 2 does indeed have the concept of reactivity to low-level *multiple chemical* exposure at the center of the belief system.

In contrast to the average EI/MCS network, the average doctor network (Figure 3) is organ-

ized around several key categories reflecting the relationships between emotional and physical disorders. In particular, the doctors' network demonstrates a highly organized and systematic framework where reactivity to low-level *multiple chemical* exposure plays a peripheral role, with only one direct connection to the rest of the network, through *emotional* upset or stress response.

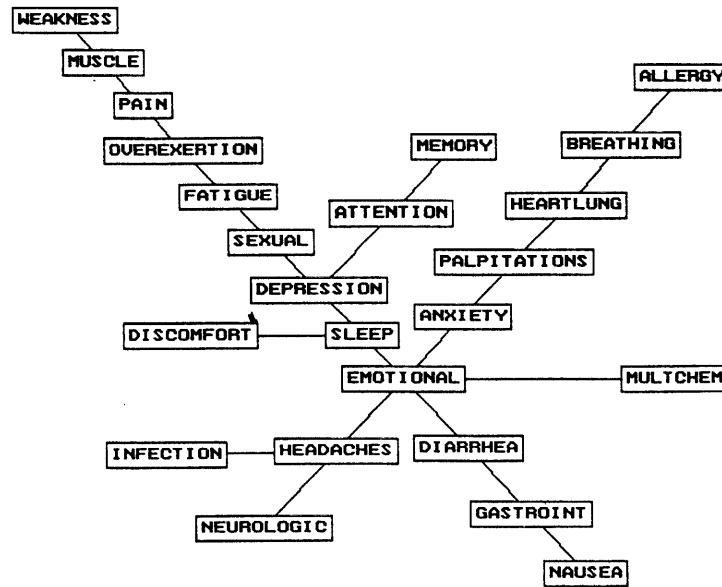The allergy and asthma patients' networks (Figures 4 and 5 respectively) also differ mark-

Fig. 3. Average doctor network.

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edly from the EI/MCS patients' network. For example, even though *allergy* to pollens, mold, dust and animals should be a central concept for both these groups, the pattern of relationships among concepts is clearly more specific than it was for EI/MCS patients (who exhibited a radial pattern of connections to the central concept in their illness). Also noteworthy, allergy, asthma and EI/MCS groups share the same connections among the concepts *breathing* difficulty, *allergy, headaches*, and reactivity to low-level *multiple chemical* exposure. However, in contrast to the EI/MCS patients but consistent with the doctors' network, reactivity to low-level *multiple chemical* exposure for both the allergy and the asthma group is only peripherally connected in the network.
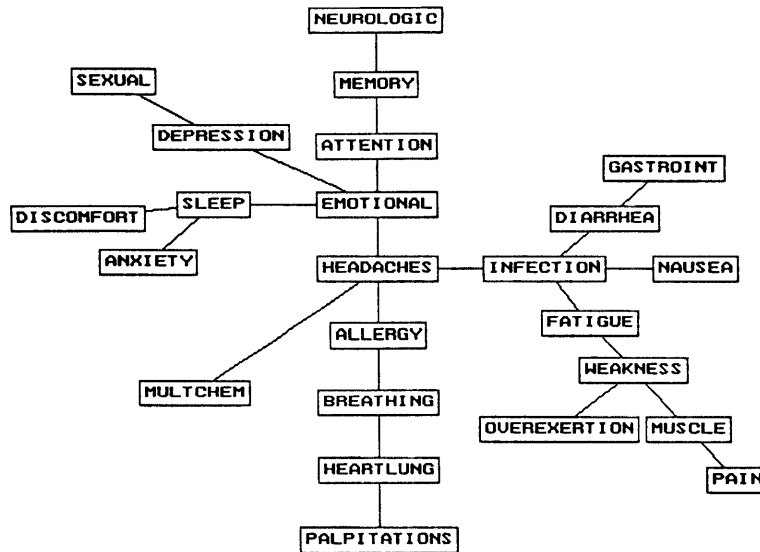
The average control participants' network
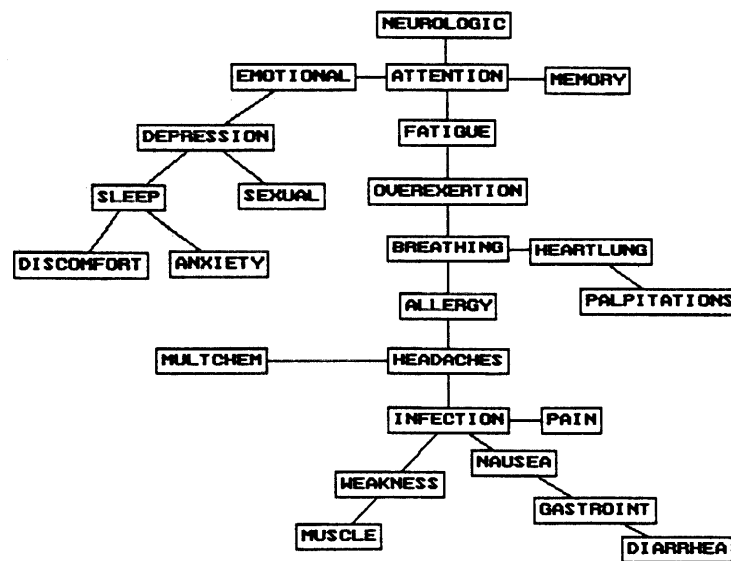

Fig. 4. Average allergy patient network.

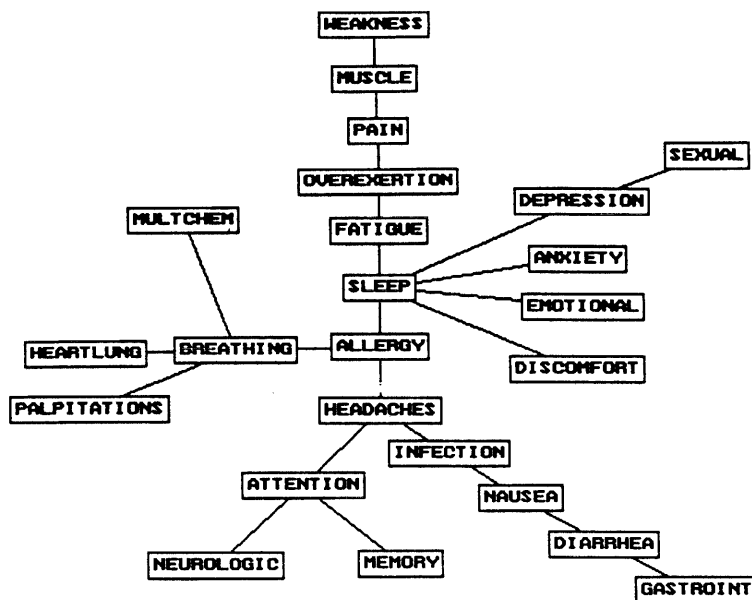Fig. 5. Average asthma patient network.

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Fig. 6. Average control subject network.

(Figure 6) also results in a very different organization as compared to the EI/MCS patients' network. Interestingly, the control network reflects everyday assumptions about the relationships between symptoms and causes. For example, the concepts muscle *weakness, muscle injury, pain*, physical *overexertion* and *fatigue* are all thematically linked, as are the concepts *headaches, attention* difficulty, poor *memory* and *neurologic* or brain abnormality. The cluster of concepts containing *headaches, nausea*, and *gastrointestinal* disease appear to be related to beliefs about colds and flu. There are also several concepts extending from the node *sleep* problems, suggesting that this is a salient concept for undergraduate university students. Finally, reactivity to low-level *multiple chemical* exposure is only peripherally connected to the network, and in this case, the link is through *breathing* difficulty which may well represent irritant effects from pollution and indoor air (e.g. cigarette smoke).

In summary, it is interesting to note how the average networks for the allergy and asthma patients, doctors and control subjects all shared the feature that reactivity to low-level *multiple chemical* exposure was only peripherally connected to the network. This is in contrast to the average EI/MCS patient network where a large number of general malaise symptoms are all attributed to reactivity to low-level *multiple chemical* exposure.

Individual networks The group differences represented in the average networks are supported by statistical analyses of the data from individuals. Table 3 shows group averages of four different indices computed from individuals' data: the number of links connected directly to the *multiple chemical* node; the average number of links intervening between the *multiple chemical* node and all other nodes (which reflects the centrality of the *multiple chemical* node); network similarity (NETSIM); and the pairs index. These four indices are not independent of one another, but they represent different views on the organization of belief structures. The first two measures were used to investigate the hypothesis that EI/MCS patients attribute multiple symptoms to low levels of *multiple chemical* exposure. According to this hypothesis, EI/MCS networks should have more direct connections to the *multiple chemical* node (Table 3), as well as fewer intervening links between the *multiple chemical* node and every other node (Table 3). Both predictions were supported in the data. First, individuals in the allergy, asthma, doctor and control groups

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Table 3: Averages of four indices on individuals in each participant population

		Index:			
		Number of links to MCS node	Average number of intervening links to MCS node	NETSIM with MCS Prototype	Critical pairs index
Group	N	M (SD)	M (SD)	M (SD)	M (SD)
EI/MCS	11	10.45 (4.59)	1.66 (0.41)	0.12 (.08)	.32 (.25)
Allergy	23	2.70 (3.04)	2.60 (0.95)	−0.02 (.03)	−.26 (.16)
Asthma	20	3.05 (2.19)	2.64 (0.70)	−0.01 (.04)	−.12 (.19)
Doctors	11	2.64 (4.25)	2.42 (0.92)	−0.02 (.07)	−.34 (.15)
Controls	22	3.09 (2.58)	2.72 (0.90)	−0.03 (.02)	−.54 (.13)

showed no differences in the number of concepts directly linked with *multiple chemical*, $F(3, 72) = 0.12$, $MSE = 0.99$, $p = .950$, but these individuals showed significantly fewer links to the *multiple chemical* node as compared to the EI/MCS patients, $t(85) = -7.51$, $p \le .001$. Similarly, allergy and asthma patients, doctors and controls showed no differences in the average number of intervening links between *multiple chemical* and the other 23 concept nodes, $F(3, 72) = 0.31$, $MSE = 0.23$, $p = .818$, but individuals in these groups showed significantly more intervening links to *multiple chemical* than EI/MCS patients $t(85) = 3.64$, $p \le .001$.

Next, individual Pathfinder networks were compared to the prototype EI/MCS network (see Figure 1) using a network similarity index (NETSIM). NETSIM is based on the expected similarity between networks and is computed in the following manner. First, the observed similarity is computed by dividing the number of links shared by both networks (those in the intersection of the links) by the number of links in either network (the number of links in the union). Next, because the probability that two networks will share x links can be computed from the hypergeometric probability distribution, this information can be used to compute the expected number of links in common between two networks. The expected similarity is subtracted from the observed similarity between two networks to get the NETSIM index which is relative to the chance level of similarity (NETSIM = 0). Thus, networks more similar than chance will yield positive NETSIM values,

and networks less similar than chance will yield negative NETSIM values. Comparing individuals to the EI/MCS prototype (shown in Figure 1), we should find higher NETSIM values for EI/MCS patients than for the other subject groups.

The mean NETSIM values are shown in Table 3 (third column). Planned t-tests compared the NETSIM values for each group to the chance level of similarity (or zero). Because the analyses consisted of five comparisons, the significance criterion was set at $p = .01$ using the Bonferroni method of controlling for family-wise error rates ($EF/k = .05/5 = .01$). The only group with greater than chance levels of similarity to the EI/MCS prototype was the EI/MCS patient group, $t(10) = 5.23$, $p \le .001$. The NETSIM values for allergy patients and control subjects were close to chance, but, due to the consistency between individuals, these small values were significantly below chance, $t(22) = -3.81$ and $t(21) = -5.36$, both $p \le .001$. The NETSIM values for the asthma patients and the doctors did not differ from chance, $t(19) = -0.84$, $p = .409$ and $t(10) = -0.74$, $p = .479$. An ANOVA comparing the NETSIM values for the allergy, asthma, doctor, and control subjects showed no differences between groups, $F(3, 72) = 0.90$, $MSE = .001$, $p = .445$, but individuals in these groups were significantly lower on network similarity to the EI/MCS prototype than EI/MCS patients, $t(85) = -9.56$, $p \le .001$.

Figure 7 shows the NETSIM values for each subject in the study. If the lowest NETSIM value for EI/MCS patients is taken as the criterion for classifying participants as EI/MCS
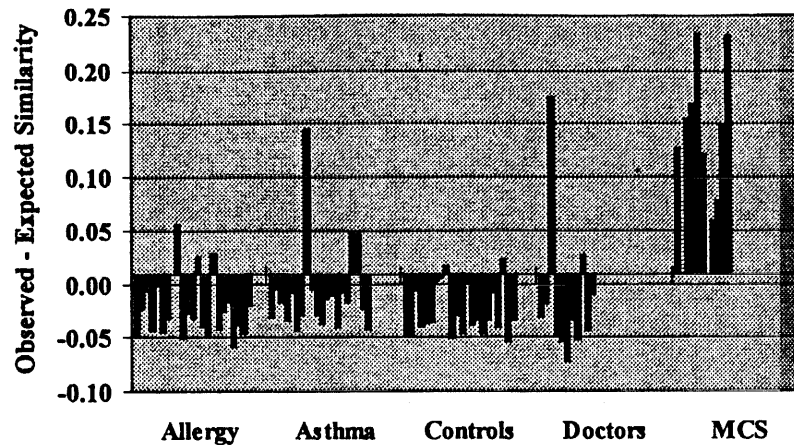
Fig. 7. Distribution of observed minus expected similarity (NETSIM values) to the EI/MCS prototype.

patients, 3 allergy patients, 3 asthma patients, 2 controls and 2 doctors are incorrectly classified. Thus, the data from individuals strongly reflect the differences found in the average data.

Critical concept pairs Finally, we attempted to identify a small number of critical concept pairs from the differences in the relatedness ratings given by control subjects and EI/MCS patients. A diagnostic tool requiring only a small number of judgments is clearly preferable to one requiring all 276 concept pairs.

Using z-score transformations of the ratings, an analysis of all 276 concept pairs revealed that EI/MCS and control participants gave markedly different ratings on many concept pairs. We attempted to find a small number of pairs that reliably distinguished EI/MCS patients and controls. Forty-seven pairs produced the largest differences between the groups (all $ps \leq .006$).

For 27 of these pairs, control subjects gave higher ratings than EI/MCS patients. EI/MCS patients gave higher ratings for the remaining 20 pairs. The *multiple chemical* concept was in 11 of these 20 pairs. A pairs index was created using these 47 pairs. This index is the average rating of the 20 pairs on which EI/MCS patients gave higher ratings minus the average of the 27 pairs on which control subjects gave higher ratings. By its construction, the score for EI/MCS patients should be higher than for controls.

The reason for isolating these particular con-

cept pairs from the set of all pairs in generating a diagnostic score should be clear. We are attempting to target concept relations which are seen in fundamentally different ways between subject populations. By developing the index from the EI/MCS and control groups, we can assess its validity by applying it to the other groups.

The critical pairs index was computed for each individual. The mean critical pairs indices are shown in Table 3. Planned comparisons showed that the critical pairs indices were significantly greater than zero for EI/MCS patients, $t(10) = 4.26$, $p \leq .002$, whereas critical pairs indices for the remaining groups were all significantly less than zero: $t(22) = -7.72$, $p \leq .001$ for allergy patients; $t(19) = -2.86$, $p \leq .01$ for asthma patients; $t(21) = -19.19$, $p \leq .001$ for controls; and $t(10) = -7.30$, $p \leq .001$ for doctors.

Figure 8 shows values for individual subjects for the critical pairs index. It is not surprising that the controls differed from the EI/MCS patients given the method used to generate the pairs index. However, the results for the other subject groups validate the index because these subjects were not used in the construction of the index. Only 2 asthma patients were incorrectly classified as EI/MCS patients. Although further confirmation of these results is required, we were encouraged to find such clear differences between individual EI/MCS patients and individuals in all of the other groups.
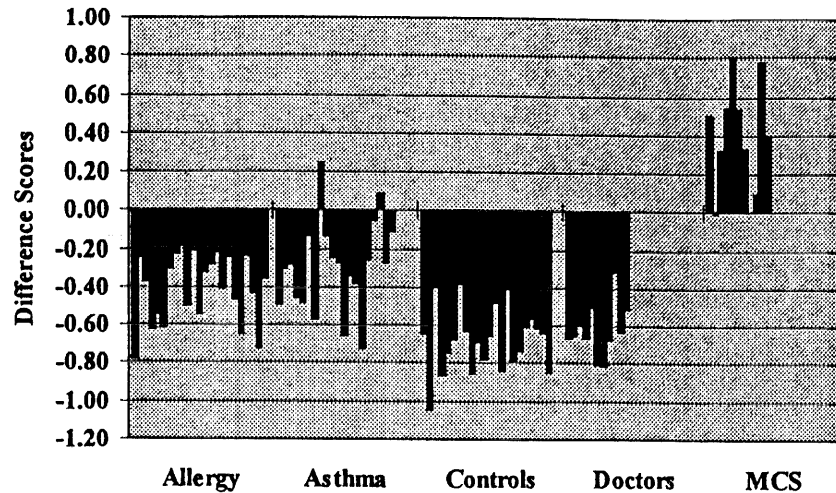
Fig. 8. Distribution of pairs index-based on 47 critical concept pairs.

General discussion

This research used knowledge-representation techniques to assess beliefs in patients professing to have EI/MCS. These patients experience an unusually wide range of symptoms, all linked to chemical sensitivity, but in many cases it is impossible to find a medical diagnosis for the symptoms. This is in contrast to typical medical patients who exhibit specific symptoms with a true physiological basis. Psychologically, EI/MCS patients differ from normal patients in personality, attitudes, affect and, most relevant to this research, strength of belief about toxicogenic attribution. We hypothesized that if psychosomatic illness is mediated by belief, then methods for representing beliefs should show systematic differences between psychosomatic patients and other populations. Participants in this study made relatively simple judgments about the relatedness of concepts relevant to the EI/MCS phenomenon. Associative networks were generated from the relatedness judgments and were used to evaluate the pattern of beliefs held by EI/MCS patients. Inspection of the group average networks suggests that EI/MCS patients *do* view relations among concepts differently from allergy and asthma patients, doctors and asymptomatic controls. EI/MCS patients attribute a large number of their problems, relative to the other subject groups in this study, to reactivity to low levels of *multiple chemical* exposure.

Quantitative analyses also showed differences between the groups of participants. The networks from individual EI/MCS patients showed a pattern of many links to the *multiple chemical* node, as well as fewer intervening links to the *multiple chemical* node. Two measures of individual belief structure were discussed: one based on network comparisons to a theoretical prototype (NETSIM values) and one empirically derived from the differences in concept ratings for EI/MCS patients and undergraduate college students (critical pairs index). Both measures successfully differentiated individuals from different research participants based on patterns of belief. An advantage of the theoretical approach is that the EI/MCS prototype was generated using an independent source (based on the research and observations concerning the EI/MCS phenomenon in the medical community). Comparisons to the prototype were used independently to verify the a priori classifications of the subject populations in this study. A disadvantage of the theoretical approach is that rating 276 concept pairs is a rather time-consuming task. The empirical approach, on the other hand, is potentially much less time-consuming because the information necessary for computing the critical pairs index is based on only 47 comparisons. The disadvantage of the

critical pairs index is that it is dependent on the EI/MCS patients in this particular sample. However, it should be noted that this index did successfully classify most of the asthma and allergy patients, as well as the doctors (subject data which was clearly independent from the data used to formulate the index).

The fundamental discrimination that a physician makes in cases of multisystem complaints attributed to chemical exposure is between a disorder of belief and a real disease process. In most cases of this type, the physician begins with a thorough history, physical exam and appropriate laboratory tests (Selner, 1989). Psychological assessment is usually recommended only after the medical examinations and assessments are completed and often only if there is a failure to confirm a medical diagnosis (Selner & Staudenmayer, 1985). It would seem that some indication of the patient's beliefs at the beginning of the evaluation process instead of at the end could assist the physician in formulating an assessment strategy and a working diagnosis. Such a procedure could also help in estimating the likelihood of treatment viability and prognosis. The advantage of the methods used here over standard interviews is that an approach which does not rely on a direct line of questioning may prove useful for circumventing problems associated with recall as well as defense mechanisms which may cause patients to answer questions in ways that prevent an accurate diagnosis.

The research reported here is only a first step toward demonstrating differences in the belief systems of EI/MCS patients and other participants. These results will clearly need to be cross-validated using additional EI/MCS patients and other groups of participants. However, the approach outlined above could form a basis for developing techniques that can identify patients with psychosomatic illness. Future research will focus on the development of quantitative scales for distinguishing EI/MCS patients from other research participants based on patterns of belief. Such scales could be used to classify individuals who might be at risk of EI/MCS so that appropriate treatment can be targeted. Additionally, if there proves to be a relationship between the radial structure of an EI/MCS patient's network and the degree to which the patient is committed to the belief system, then this may provide an indication of treatment viability. Clinical experience with EI/MCS patients shows that degree of commitment to the belief system is a predictor of treatment viability. Finally, individual networks and the associated indices could also be used as an objective means for tracing changes in belief or insights experienced by EI/MCS patients as they progress through clinical treatment.

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