A Review of a Two-Phase Population Study of Multiple Chemical Sensitivities

Stanley M. Caress[1] and Anne C. Steinemann[2]

[1]State University of West Georgia, Carrollton, Georgia, USA; [2]Georgia Institute of Technology, Atlanta, Georgia, USA

In this review we summarize the findings of a two-phase study of the prevalence, symptomatology, and etiology of multiple chemical sensitivities (MCS). We also explore possible triggers, the potential linkage between MCS and other disorders, and the lifestyle alterations produced by MCS. The first phase of the study consisted of a random sampling of 1,582 individuals from the Atlanta, Georgia, metropolitan area to determine the reported prevalence of a hypersensitivity to common chemicals. In this phase, 12.6% of the sample reported a hypersensitivity. Further questioning of individuals with a hypersensitivity indicated that 13.5% (1.8% of the entire sample) reported losing their jobs because of their hypersensitivity. The second phase was a follow-up questioning of the respondents who initially reported hypersensitivity. In this phase, we found that individuals with hypersensitivity experience a variety of symptoms and triggers. A significant percentage (27.5%) reported that their hypersensitivity was initiated by an exposure to pesticides, whereas an equal percentage (27.5%) attributed it to solvents. Only 1.4% had a history of prior emotional problems, but 37.7% developed these problems after the physical symptoms emerged. This suggests that MCS has a physiologic and not a psychologic etiology. *Key words:* chemical injury, environmental illness, MCS, multiple chemical sensitivities, TILT, toxicant-induced loss of tolerance. *Environ Health Perspect* 111:1490–1497 (2003). doi:10.1289/ehp.5940 available via *http://dx.doi.org/* [Online 9 April 2003]

Numerous government agencies, medical organizations, and researchers have stressed the need for additional epidemiologic research on multiple chemical sensitivities (MCS) (Ashford and Miller 1998). In a federal government publication on MCS , Mitchell (1995) outlined the need for epidemiologic research "to characterize the cases sufficiently for further work" and "to establish the magnitude of the problem caused by the MCS phenomenon in the population." Additionally, a federal government report indicated that the uncertainties surrounding the etiology, dynamics, and symptomatology of MCS could only be solved by a dramatic increase in research efforts (Interagency Workgroup on Multiple Chemical Sensitivity 1998).

This study consists of a two-phase investigation of the prevalence, symptomatology, and etiology of MCS. The initial phase focuses on the prevalence of MCS in the metropolitan Atlanta, Georgia, area. The second phase is a more extensive follow-up questioning of the positive respondents from the first phase, which focuses on symptomatology, etiology, and other aspects of hypersensitivity.

MCS Prevalence

MCS is generally acknowledged to be a condition where individuals have an acute hypersensitivity to low levels of chemicals found in everyday substances such as household cleaning agents, pesticides, fresh paint, new carpeting, synthetic building materials, newsprint, perfume, and numerous other petrochemical-based products (Davidoff et al. 2000). MCS can produce a wide range of symptoms, and individuals with hypersensitivity can encounter great difficulty functioning in normal working and living environments (Lax and Henneberger 1995).

Estimates of the number of people who have MCS vary widely. A National Academy of Sciences (NAS) report initially speculated that up to 15% of the American public could be experiencing a heightened sensitivity to common chemical products (NAS 1981). Subsequent research that used a variety of methodologies, however, produced different prevalence rates.

Previous prevalence research. Existing research on the prevalence of MCS tends to fall into two broad categories based on the characteristics of the individuals used as subjects. The first category includes subjects who are either self-selected or have common characteristics that place them in specific subgroups. Subgroups have consisted of individuals who either sought treatment at a medical clinic or had previously indicated that they had a medical condition. Other subgroups have been based on demographic characteristics such as age. The second category is a sample composed of randomly selected individuals from the general public. Studies in this category use a probability-based subject selection process to ensure that every member of the population had an equal chance of being included in the study.

Early MCS prevalence studies fall into the first category because they used research subjects who either were self-selected or had special characteristics. More recent epidemiologic research on MCS, however, typically falls into the second category.

A number of investigations have been conducted since 1990 that attempted to assess the prevalence of MCS in the U.S. population. In an early study, based exclusively on anecdotal evidence from conversations with medical personnel, Mooser (1987) suggested that 2–10% of the population suffered disruptive effects because of a hypersensitivity to chemical substances. Additional studies, however, questioned the validity of anecdotal evidence and suggested that this prevalence rate was an underestimate. One of the first studies to project the prevalence of MCS from a subgroup used a sample composed of 705 medical clinic patients (Kipen et al. 1995). Two subsequent studies also used subjects from specialized subgroups: one sample group was composed of 809 young adult college students (Bell et al. 1993) and one group comprised 160 elderly persons (Bell et al. 1997), both in Arizona. In these studies (Bell et al. 1993, 1997), approximately 15% of the younger sample and more than 37% of the elderly group reported a hypersensitivity.

In a random telephone survey of 1,027 residents in rural North Carolina, Meggs et al. (1996) determined that 33% of respondents reported becoming sick after smelling chemical odors (e.g., perfume, pesticide, fresh paint, car exhaust, newsprint). Although this study used a random sample that could be representative of the general public, the wording of the key questions did not distinguish between a normal aversion to harsh chemical odors and a true hypersensitivity to common substances at low levels.

The California Department of Health Services (CDHS) conducted the most extensive epidemiologic research on MCS to date (Kreutzer et al. 1999). The federally funded CDHS asked experts familiar with MCS to suggest optimum wording for questions to be included in a state-conducted medical survey. The survey, administered in 1998, took samples from different regions of the state and included more than 4,000 respondents. Kreutzer et al. (1999) found that 15.9% of respondents reported unusual sensitivity to

Address correspondence to S.M. Caress, State University of West Georgia, 1601 Maple St., Pafford Building, Room 121, Carrollton, GA 30118 USA. Telephone: (770) 836-6504. Fax: (770) 836-4665. E-mail: scaress@westga.edu

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common chemicals, which tended to confirm the original NAS estimate (NAS 1981). The CDHS study (Kreutzer et al. 1999) also included data on sex, educational level, marital status, and racial/ethnic linkage with MCS. Earlier anecdotal evidence had suggested that white women of higher educational status disproportionately reported MCS (Cullen 1992). The CDHS study (Kreutzer et al. 1999), however, found a heterogeneous distribution of MCS that cuts across gender, race, and educational categories.

First Phase

In the first phase of this study—a population-based prevalence study conducted in 1999–2000 (Caress and Steinemann. In press), we investigated the prevalence of a hypersensitivity to common chemical products and the extent of the medical diagnosis of MCS in the Atlanta, Georgia, metropolitan area. This stage of the study also included a preliminary exploration of the severity and potential causes (initiations) of hypersensitivity. Additionally, we examined lifestyle changes, age of onset, and potential linkages between MCS and the demographic variables of sex, age, and educational level.

Methods

The construction of the research design for this phase required the development of a measurement instrument, selection of a target population, and determination of sampling techniques and sample size. In addition, concerns of reliability and operational and external validity had to be addressed.

The Interagency Work Group on Multiple Chemical Sensitivities (1998) concluded that "questionnaires are one of the most useful tools in epidemiologic investigations." The questionnaire used in our study was constructed to investigate both the medical diagnosis and symptomatology of MCS, as well as other facets of chemical hypersensitivity. To ensure external validity of the questionnaire (the assumption that the results of this study may be applicable to other populations), we replicated the exact wording of the key questions used in the CDHS questionnaire (Kreutzer et al. 1999). This replication also facilitates an analytical comparison between our study and Kreutzer et al.

The process of determining the sample size to ensure operational validity is contingent on the degree of random error associated with the measurement. Consequently, a sufficiently large sample was required. The number of cases necessary to ensure validity is based on the desired confidence levels and confidence intervals; therefore, a confidence level of 95% is acceptable for this type of research. The degree of accuracy of the findings (confidence interval) of 3% is normally desirable for epidemiologic inquiries (O'Sullivan and Rassel 1995).

We used both confidence level and degree of accuracy in a standard probability formula to determine the size of the sample. The standard formula is as follows:

$$n^2 = \text{proportion}^2 \times (1 - \text{proportion}) \times z$$

where the n is the sample size and z is the z-score (standard score corresponding to the appropriate confidence level) (O'Sullivan and Rassel 1995). This formula indicated that for a study to achieve a confidence level of 95% with a 3% degree of accuracy, it must have at least 1,067 cases. Therefore, a phone list of 2,000 telephone numbers was generated to ensure a sufficiently large sample. A total of 1,582 respondents ultimately completed the questionnaire; thus, the sample size of this study exceeded the size necessary to obtain the desired confidence level and degree of accuracy. For the data-gathering procedure in this study, we used random sampling methods to protect from any systematic bias in the data. Phone numbers randomly selected (lottery method) from lists generated by the local phone company were used to construct the sample. The target population of the Atlanta, Georgia, metropolitan area was covered by using telephone numbers from the 770 and 404 area codes.

We increased the reliability of the questionnaire by ensuring that the data demonstrated stability. The data, therefore, were gathered in three separate cohorts. We administered the questionnaire to 496 individuals in the summer of 1999. We surveyed a second cohort of 322 individuals in fall 1999 and a third cohort of 764 individuals in the winter and spring of 2000. The winter and spring cohort was larger because the data-gathering covered a longer period. The findings of each separate cohort were examined to identify any significant deviation. Because the results of all three cohorts displayed only minor variation (essentially equivalent given the 3% confidence interval), we judged the data to be stable and combined data from the three cohorts. Thus, all results in the study are an aggregation of the three cohorts.

Prior to gathering data, we conducted a pretest to evaluate the face validity of the questionnaire (respondents' belief that the questions asked them accurately reflected what was being studied). A test group of 253 individuals was used to evaluate the original questionnaire, which was lengthier than the final version. An unexpectedly large number of respondents terminated the interview before its completion because they found the questionnaire too time-consuming or tiring. The face validity evaluation and a subsequent item analysis provided the impetus for shortening the questionnaire. Less-pertinent questions were removed, and the final version contained 12 health-related questions and three additional demographic questions.

The final version of the questionnaire initially inquired if the respondent has ever been diagnosed with MCS or environmental illness. A subsequent key question, which used the same wording as the CDHS, was

> compared to other people, do you consider yourself unusually sensitive to everyday chemicals like those in household cleaning products, perfume, detergents, insect spray and things like that?

Respondents who replied positively to the key question were asked several additional questions that investigated symptom severity, origin (initiation), age of onset, and behavior modifications. Demographic questions on age, sex, and educational level were asked of all respondents.

Findings

The aggregated data showed that 12.6% (n = 199) of the respondents reported an unusual sensitivity to common chemical substances (Table 1). An additional 1.4% (n = 22) were not certain if they had hypersensitivity. Of the respondents, 3.1% (n = 49) reported that they had been medically diagnosed as having either environmental illness or MCS.

Of the individuals who reported that they were unusually sensitive to common chemicals, or suspected it, 42.7% (n = 93) could identify an original cause (initiation) of their hypersensitivity. Of these, the cause of hypersensitivity was reported to be chemical exposure by 12.4% (n = 27); an exposure to pesticides by 5% (n = 11); other types of exposure by 11.5% (n = 25); and other causes by 13.8% (n = 30).

Of the respondents who reported sensitivities, 45.1% (n = 106) received medical treatment (Table 1). A majority of the respondents (61.5%, n = 142) reported taking some

Table 1. First-phase data: prevalence of sensitivity and behavior modifications.[a]

Question	Yes % (n)	No % (n)	Not sure % (n)	Refuse % (n)
Sensitive to chemicals	12.6 (199)	85.7 (1,351)	1.4 (22)	0.3 (4)
MCS diagnosed	3.1 (49)	95.3 (1,504)	1.6 (25)	0.1 (1)
Received treatment[b]	45.1 (106)	47.2 (111)	5.5 (13)	2.1 (5)
Take precautions at home[b]	61.5 (142)	30.3 (70)	5.2 (12)	2.6 (6)
Difficulty shopping[b]	29.9 (64)	65 (139)	5.1 (11)	0 (0)
Lost job[b]	13.5 (29)	84.7 (182)	0.5 (1)	1.4 (3)

[a]Unequal totals result from rounding and/or missing data. [b]Asked only of subjects who reported a hypersensitivity to chemicals.

precautions at home because of their hypersensitivity. Somewhat less than one-third (29.9%, $n = 64$) indicated that their hypersensitivity made it difficult to shop in stores in a normal manner. Moreover, 13.5% ($n = 29$) of the respondents lost their jobs because their hypersensitivity prevented them from functioning adequately in their workplace. The number of respondents who lost employment because of their hypersensitivity represents approximately 1.8% of the entire sample. We also asked positive respondents at what age their symptoms first appeared; responses were as follows: before 20 years of age, 32.4% ($n = 70$); 21–36 years of age, 35.2% ($n = 76$); 26–50 years of age, 14.8% ($n = 32$); and after 50 years of age, 9.7% ($n = 21$) (Table 2).

The educational level distribution of the entire sample was evenly spread; 10.1% ($n = 52$) had less than a high school education, 24.7% ($n = 374$) were high school graduates, 25.7% ($n = 389$) had some college, 31.5% ($n = 477$) were college graduates, and 7.9% ($n = 120$) had postgraduate education (Table 3). The sex distribution of the sample was 59.8% female and 38.8% male (Table 3).

A cross-tabulation with sensitivity and education level indicates that positive respondents were also fairly evenly distributed across all education levels, with a minor bias toward higher educational levels (Table 4). People with a high school education or less made up 36% ($n = 69$) of the total number of positive respondents, with college graduates and individuals with postgraduate education making up 33% ($n = 64$), and people with some college comprising 31% ($n = 60$) (Table 4). The educational level distribution of positive respondents, therefore, is comparable to the education level of the entire sample. The cross tabulations also indicate a sex distribution slanted somewhat toward females; respondents who reported an unusual sensitivity to chemicals were 71.7% female and 28.3% male (Table 4).

Discussion: First-Phase Findings

Although a 12.6% positive response rate (Kreutzer et al. 1999) is below the level found in the CDHS study, when we consider a sampling error of 3%, the rates are essentially statistically equivalent. These findings are consistent with the NAS estimate (NAS 1981) that up to 15% of Americans have a hypersensitivity to low levels of common chemicals. The potential of a sex linkage was suggested in the earlier clinic-based studies, with the speculation that MCS is primarily a female condition. The CDHS study (Kreutzer et al. 1999) found a higher incidence of females in its total sample reporting a hypersensitivity (16% of females sampled vs. 6.9% of the males). Females also comprise 71.7% of the respondents who reported unusual hypersensitivity in our study. This would initially suggest that females are disproportionately more likely to have the symptomatology of MCS than males. This proportion, however, is less dramatic when the female bias in the sample is considered. The total sample in our study was 59.8% female, which is somewhat higher but statistically congruent with the female population of northwestern Georgia, which is 51.3% female. The CDHS study also had a larger number of female respondents (59%) than in the general population of California, but this is also well within acceptable parameters. The actual percentage of males in our study who experienced MCS, when adjusted for the sample bias, was approximately one-third, which suggests that although females report a higher incidence of hypersensitivity, it affects both sexes.

Early clinic-based studies suggested that a hypersensitivity is more common in individuals with a higher level of education (Cullen et al. 1992). Critics of this conclusion argue that highly educated individuals are only more likely to seek treatment and be diagnosed with MCS, whereas less-educated people are more likely to remain undiagnosed. Our study tends to support the latter observation. The data are similar to the CDHS results (Kreutzer et al. 1999), which indicate that a hypersensitivity to chemicals is widely distributed across education levels (Table 4), which, as noted in the CDHS study, suggests that a universal etiology is probable.

Second Phase

The second phase, conducted in the spring of 2000, consisted of an extensive follow-up examination of respondents who reported a hypersensitivity to chemicals in the initial phase. (Caress et al. 2002). It explored symptomatology, etiology, potential triggering agents, and linkages to other disorders. The second phase also examined the potential linkage between the onset of reactions and specific chemical substances, as well as lifestyle modifications made by hypersensitive individuals.

Theories of Etiology and Dynamics

Current research suggests that MCS exhibits a two-step process of initiation (causation) and triggering (subsequent reactions) (Ashford and Miller 1998). Hypersensitivity emerges after initiation, which can result from a massive exposure to a specific toxic agent (Rea et al. 1978) or a chronic exposure to one or more toxic substances, even at low levels (Miller et al. 1997). After initiation, triggering occurs, which involves reactions to a wider range of substances.

Theories about the nature of initiation have been based on one or more of the following systems: neurologic, immunologic, endocrine, and psychologic (Interagency Workgroup on Multiple Chemical Sensitivity 1998). Fiedler et al. (1992) suggested that neurologic disorders are connected to MCS, and other studies have indicated that MCS is associated with immunologic dysfunctions. Some researchers contend, however, that MCS does not follow the same pattern as immunologic disorders (Ziem 1992), which has led other researchers to examine the connection between MCS and immune dysfunction linked to the neuroendocrine system (Meggs 1992). It also has been suggested that inflammation of the respiratory tract (Meggs 1995) and disorders such as porphyria are potential causative factors (Ellefson and Ford 1996). Other researchers have examined the

Table 2. First-phase data: onset age and etiology.[a]

Question	% (*n*)
Age of onset of hypersensitivity (years)[b]	
< 20	32.4 (70)
21–35	35.2 (76)
36–50	14.8 (32)
> 50	7.9 (17)
Refuse/don't know	9.7 (21)
Original cause of hypersensitivity[b]	
Chemical exposure	12.4 (27)
Pesticide exposure	5.0 (11)
Other exposure	11.5 (25)
Other cause	13.8 (30)
Don't know	57.3 (125)

[a]Unequal totals result from rounding and/or missing data. [b]Only asked subjects who had or suspected hypersensitivity.

Table 3. First-phase data: respondent demographics (entire sample).[a]

Question	% (*n*)
What is your age?	
< 20 years	5.8 (89)
21–35 years	24.3 (373)
36–50 years	33.2 (510)
> 50 years	34.5 (530)
Refuse/don't know	2.3 (35)
What is your sex?	
Male	38.8 (600)
Female	59.8 (926)
No answer	1.4 (22)
What is your educational level?	
Did not complete high school	10.1 (152)
High school graduate	24.7 (374)
Some college	25.7 (389)
College graduate	31.5 (477)
Professional/graduate school	7.9 (120)

[a]Unequal totals result from rounding and/or missing data.

Table 4. Cross-tabulations of first-phase data: education and sensitivity to chemicals and sex and sensitivity to chemicals.[a]

Are you sensitive to chemicals?	Yes % (*n*)	No % (*n*)
Education		
Did not complete high school	14 (27)	9 (120)
High school graduate	22 (42)	25 (325)
Some college	31 (60)	25 (321)
College graduate	24 (47)	33 (425)
Graduate/professional school	9 (17)	8 (100)
Sex		
Male	28.3 (53)	42.2 (539)
Female	71.7 (142)	57.8 (763)

[a]Respondents who answered "not sure" or "refuse" were not included.

role of the limbic system (Bell et al. 1995) and metabolic mechanisms in MCS (Byers et al. 1988). Examinations of a relationship between MCS and other conditions such as systemic lupus, chronic fatigue syndrome, and fibromyalgia have also been conducted (Ashford and Miller 1998; Interagency Workgroup on Multiple Chemical Sensitivity 1998). Additional studies focus on the role of psychologic factors. These psychologically based studies speculate that hypersensitivity to low levels of chemicals may be a somatization disorder (Black et al. 1990) or a conditioned response (Siegel and Kreutzer 1997). Psychogenic theories, however, have been criticized for methodologic weaknesses, such as biased patient selection and the lack of presymptom data (Davidoff and Fogarty 1994).

Miller and Mitzel (1995) conducted an experiment that investigated the genesis and other aspects of chemical sensitivity. They divided questionnaires from 112 individuals who had previously reported a chemical sensitivity into two subgroups based on the origin of the condition. One subgroup consisted of questionnaires from individuals who developed their sensitivity after a major exposure to organophosphate pesticides, whereas the other subgroup was composed of questionnaires from people who traced their sensitivity to an exposure to building materials. The authors postulated that the degree of neurotoxicity would be greater from pesticide exposure than from an exposure to the class of chemicals used in building materials. A comparison of the two subgroups indicated that regardless of exposure origin, individuals in both subgroups experienced similar symptoms. There was, however, a considerable difference between the subgroups in the severity of the symptoms, with the pesticide-origin subgroup experiencing more severe symptoms than the building material–origin subgroup. This differentiation of symptom severity between subgroups led the authors to conclude that chemical sensitivity has specific physical dynamics inconsistent with somatoform disorders. The results of Mitzel and Miller (1995), therefore, suggest that chemical sensitivity has a physiologic genesis and is not psychogenic.

Table 5. Second-phase data: nature of symptoms.[a]

Question	% (*n*)
What is the severity of your symptoms?	
Severe	23.2 (16)
Somewhat severe	29.0 (20)
Mild	42.0 (29)
No problem	5.8 (4)
What is the length of time after exposure that symptoms appear?	
Immediately	42.0 (29)
Within an hour	24.6 (17)
Many hours or more	5.8 (4)
Different lengths of time	26.1 (18)
Don't know	1.4 (1)
What is the duration of your symptoms?	
Several hours or less	47.8 (33)
Several days	40.6 (28)
Week or more	11.6 (8)
Are your reactions always the same?	
Always	68.1 (47)
Usually	18.8 (13)
Sometimes	8.7 (6)
Seldom or never	2.9 (2)
Don't know	1.4 (1)

[a]Unequal totals result from rounding and/or missing data.

Table 6. Second-phase data: symptoms.[a]

Symptom	Yes % (*n*)	No % (*n*)	Don't know % (*n*)
Headache	88.4 (61)	11.6 (8)	0 (0)
Burning eyes	76.8 (53)	23.2 (16)	0 (0)
Concentration	31.9 (22)	65.2 (45)	2.9 (2)
Nausea/stomach	55.1 (38)	43.5 (30)	1.4 (1)
Muscle pain	30.4 (21)	65.2 (45)	4.3 (3)
Dizziness	46.4 (32)	52.2 (36)	1.4 (1)
Fever	17.4 (12)	82.6 (57)	0 (0)
Unconsciousness	7.2 (5)	92.8 (64)	0 (0)
Asthma	59.4 (41)	40.6 (28)	0 (0)
Other	50.7 (35)	49.3 (34)	0 (0)

[a]Unequal totals result from rounding and/or missing data.

Methods

The research design of the second phase of our study required the construction and administration of an expanded measurement instrument and the implementation of statistical measurements to evaluate its reliability and validity. In addition, we compared our findings with the results of Miller and Mitzell (1995) to evaluate the external validity of the data.

The second phase questionnaire had 71 questions and was administered only to individuals who had previously reported a hypersensitivity to common chemicals in the first phase. The sample used in this second phase was a derivative of the random survey, thus ensuring that it was representative of the target population. The initial phase located 199 individuals who reported a hypersensitivity to chemicals. These individuals became the pool of potential subjects for the follow-up study in the second phase. Subjects were called a few months after the completion of the initial survey and asked if they would answer a longer and more detailed questionnaire. Between the two phases, a number of potential subjects had moved, had become too ill to participate, or otherwise declined. The loss of these potential subjects ultimately reduced the size of the second phase sample to 69—approximately one-third of the 199 persons reporting hypersensitivity.

Despite its random genesis, the sample is too small to ensure randomness. In addition, because the sample is small, it could not be divided into seasonal cohorts. We evaluated the reliability of the findings of the second phase, however, by using statistical measures of internal consistency. Measurements of Cronbach's coefficient of α and other measures of interitem correlation were used on several clusters of related questions to determine consistency levels of the responses. A subsequent item analysis was conducted to further evaluate the findings and promote the integrity of the study. For the Cronbach and other interitem analysis, questions about reaction triggers as well types of symptoms were clustered. We also used an additional cluster consisting of questions about behavior modifications.

To ensure face validity, we pretested the expanded questionnaire. The pretest uncovered no significant problems and the measurement instrument was judged acceptable. External validity was promoted by constructing the

Table 7. Second-phase data: reaction triggers and severity.[a]

Question	No % (*n*)	Severe % (*n*)	Medium % (*n*)	Mild % (*n*)	Total yes % (*n*)
What produces your symptoms and how severe are they?					
Perfume	18.8 (13)	21.7 (15)	31.9 (22)	27.5 (19)	81.2 (56)
Cleaners	11.6 (8)	27.5 (19)	39.1 (27)	21.7 (15)	88.4 (61)
Fresh ink	69.6 (48)	10.1 (7)	8.7 (6)	7.2 (5)	26.1 (18)[a]
Appliances	87.0 (60)	2.9 (2)	4.3 (3)	2.9 (2)	10.1 (7)[a]
Pesticides	14.5 (10)	34.8 (24)	27.5 (19)	18.8 (13)	81.2 (56)[a]
Chlorine/water	55.1 (38)	7.2 (5)	15.9 (11)	15.9 (11)	39.1 (27)[a]
Tobacco smoke	17.4 (12)	33.3 (23)	27.5 (19)	21.7 (15)	82.6 (57)
New carpet	37.7 (26)	20.3 (14)	15.9 (11)	17.4 (12)	53.6 (37)[a]
Furniture	53.6 (37)	14.5 (10)	13.0 (9)	11.6 (8)	39.1 (27)[a]
Salon/barber	33.3 (23)	21.7 (15)	15.9 (11)	23.2 (16)	60.9 (42)[a]
Public parks	21.7 (15)	20.3 (14)	15.9 (11)	15.9 (11)	52.2 (36)[a]
Car exhaust	20.3 (14)	26.1 (18)	20.3 (14)	26.1 (18)	72.5 (50)[a]
What actions of others produce your symptoms and how severe are they?					
Laundry	53.6 (37)	5.8 (4)	4.3 (3)	8.7 (6)	18.8 (13)[a]
Lawn pesticides	46.4 (32)	4.3 (3)	13 (9)	14.5 (10)	31.9 (22)[a]
Running car	60.9 (42)	4.3 (3)	2.9 (2)	7.2 (5)	14.5 (10)[a]
Others' smoke	47.8 (33)	5.8 (4)	5.8 (4)	21.7 (15)	33.3 (23)[a]
Barbecue grill	44.9 (31)	14.5 (10)	10.1 (7)	14.5 (10)	39.1 (27)[a]

[a]Unequal totals result from "Don't know" or refused answers and/or missing data.

questionnaire to conform with recommendations made by the federal Interagency Workgroup on Multiple Chemical Sensitivity (1998), thus allowing comparisons with subsequent studies.

Findings

Severity and reaction duration of symptoms. The first question asked about the severity of a subject's sensitivity to common chemicals, with the four response categories being "severe," "somewhat severe," "mild," and "little problem." There was close to an even split between the two more intense responses ("severe" and "somewhat severe"), with 52.2% (n = 36), and the less intense responses ("mild" and "little problem"), with 47.8% (n = 34). The distribution of answers was 23.2% (n = 16) "severe," 29% (n = 20) "somewhat severe," 42% (n = 29) "mild," and only 5.8% (n = 4) "no problem" (Table 5).

In subsequent questions, respondents were asked about the time reactions took to manifest. The largest group of respondents (42%, n = 29) reported that their reactions began almost immediately after an exposure: "within an hour" was the answer given by 24.6% (n = 17) of the subjects, and only 5.8% (n = 4) said it took many hours to react. "Different times depending on the type of exposure" was the answer given by 26.1% (n = 18) of the sample, and 1.4% (n = 1) did not know. When asked questions regarding duration of reactions and if reactions to substances that made them sick were always the same, 47.8% (n = 33) of the respondents said reactions lasted several hours, 40.6% (n = 28) reported several days, and 11.6% (n = 8) said several weeks. Additionally, 68.1% (n = 47) replied that reactions were "always the same," 18.8% (n = 13) said that they "usually respond the same way," and 8.7% (n = 6) indicated that they "sometimes react the same way." Only 2.9% (n = 2) said that "they seldom or never react in the same way," and 1.4% (n = 1) did not know.

Types of symptoms. Responses to questions about the type of symptoms experienced after an exposure to an offending substance (Table 6) were as follows: headaches (88.4%, n = 61), burning eyes (76.8%, n = 53), stomach distress/nausea (55.1%, n = 38), dizziness (46.4%, n = 32), loss of mental concentration (31.9%, n = 22), and muscle pain (30.4%, n = 21). Fever was a less common symptom (17.4%, n = 12), and loss of consciousness affected 7.2% (n = 5) of the people in the survey; 59.4% (n = 41) of the respondents experienced asthma-like symptoms such as breathing difficulty after an exposure to an irritating substance, and 50.7% (n = 35) of the respondents indicated that they suffered from a variety of other symptoms.

Triggering mechanisms and etiology. We asked several questions designed to identify the triggers that set off reactions and also to determine their magnitude (Table 7). After reading a list of substances and products to the respondents, interviewers asked which substances made them sick and how serious their negative reactions were. The products that made the largest percentage of respondents sick were cleaning agents (88.4%, n = 61), pesticides (81.2%, n = 56), and perfume (81.2%, n = 56). Car exhaust (72.5%, n = 50), barber shops/beauty salons (60.9%, n = 42), new carpets (53.6%, n = 37), new furniture (39.1%, n = 27), chlorine in household water (39.1%, n = 27), and fresh ink (26.1%, n = 18) were also common triggers.

Several additional questions were asked to determine if the product usage or behavior of other people could act as a triggering mechanism for reactions. The most frequently cited behaviors of others that triggered reactions were smoke from a neighbor's fireplace, wood stove, or barbecue grill (39.1%, n = 27); secondhand tobacco smoke (33.3%, n = 23); a neighbor's use of pesticide or weed killers (31.9%, n = 22); or use of laundry products (18.8%, n = 13).

In this phase we also investigated the potential origin (initiation) of hypersensitivity (Table 8). The percentage of respondents who reported that they were "sure" of the original cause of their hypersensitivity made up 14.5% (n = 10) of the sample, and an additional 26.1% (n = 18) replied that they were "pretty sure" what caused (initiated) their symptoms. The subjects who could identify or suspect a probable cause of their hypersensitivity were asked additional questions to help uncover the etiology of MCS. The respondents reported that their original hypersensitivity was produced by exposure to pesticides, 27.5% (n = 19); harsh cleaners or solvents, 27.5% (n = 19); new construction (building materials), 17.4% (n = 12); and gasoline or other petroleum products, 15.9% (n = 11).

A cross-tabulation of cause (initiation) of symptoms with severity of symptoms indicates that respondents who could identify the cause of their symptoms were more likely to report that they were severe (50%, n = 5) than those who did not know the cause (16%, n = 6) (Table 9).

Linkage to other medical conditions. Several studies speculated that MCS is either a product of or connected to other disorders. The questionnaire, therefore, inquired about

Table 8. Second-phase data: etiology.

Question	Yes % (n)	No % (n)	Maybe % (n)	Refused or missing % (n)
Do you know what originally caused your symptoms?	14.5 (10)	55.1 (38)	26.1 (18)	4.3 (3)
Do you know or suspect the following as the original cause?[a]				
Pesticides	27.5 (19)	34.8 (24)	33.3 (23)	4.3 (3)
Solvents	27.5 (19)	30.4 (21)	37.7 (26)	4.3 (3)
Building materials	17.4 (12)	43.5 (30)	34.8 (24)	4.3 (3)
Petroleum products	15.9 (11)	43.5 (30)	36.2 (25)	4.3 (3)

[a]Multiple answers possible.

Table 9. Cross-tabulations of second-phase data: cause (initiation) of sensitivity with symptom severity.

	Degree of symptoms[a]				
Cause	Severe % (n)	Somewhat severe % (n)	Mild % (n)	No problem % (n)	Total n
Known					
Yes	50 (5)	20 (2)	20 (2)	10 (1)	10
No	16 (6)	32 (12)	45 (17)	8 (3)	38
Maybe	22 (4)	28 (5)	50 (9)	0	18
Refused/missing	–	–	–	–	2
Pesticide					
Yes	37 (7)	32 (6)	21 (4)	11 (2)	19
No	25 (6)	21 (5)	46 (11)	8 (2)	24
Don't know	13 (3)	30 (7)	57 (13)	0	23
Refused/missing	–	–	–	–	2
New construction					
Yes	33 (4)	33 (4)	25 (3)	8 (1)	12
No	20 (6)	30 (9)	47 (14)	3 (1)	30
Don't know	25 (6)	25 (6)	50 (12)	0	24
Refused/missing	–	–	–	–	3
Petroleum products					
Yes	46 (5)	36 (4)	18 (2)	0	11
No	17 (5)	30 (9)	47 (14)	7 (2)	30
Don't know	24 (6)	24 (6)	52 (13)	0	25
Refused/missing	–	–	–	–	3

[a]Unequal totals result from rounding and/or missing data.

other medical problems (Table 10). Subjects were first asked if they had any medical conditions in addition to their sensitivity to chemicals, and those who answered "yes" were asked if they suffered from any of the following conditions: gastrointestinal problems, fibromyalgia, chronic fatigue, or any other condition. All subjects were then asked about their allergies to natural substances such as pollen, animal hair, dust/dust mites, molds, and other natural allergens.

A majority of the respondents (53.6%, n = 37) replied that they had another medical condition that could be related to their hypersensitivity, and an additional 7.2% (n = 5) were not sure. Gastrointestinal problems were experienced by 26.1% (n = 18) of the subjects, and 21.7% (n = 15) said they have fibromyalgia. In addition, 18.8% (n = 13) indicated that they suffer from chronic fatigue or other immunologic troubles, and 27.5% (n = 19) answered that they have another related medical condition. The cumulative response of the subjects to all of the related conditions was > 54.4% because several gave more than one positive answer.

A larger percentage (73.9%, n = 51) indicated that they had allergies to natural substances. Pollen was an irritant for 65.2% (n = 45) of the subjects, 52.2% (n = 36) reported an allergy to animal hair or dander, 55.1% (n = 38) had an allergy to dust or dust mites, and 49.3% (n = 34) reacted to molds. Moreover, 44.9% (n = 31) said they were allergic to other natural allergens. Again, the cumulative numbers exceeded the total number of subjects with a positive reply to allergies because of multiple responses.

Linkage to mental illness. We asked questions related to mental illness because of the contention by some researchers that MCS is psychogenic (Gots 1995). The questions, however, were constructed to investigate if mental problems preceded or followed the development of symptoms (Table 11).

Only 1.4% (n = 1) of the respondents reported experiencing depression, anxiety, or other emotional problems before the onset of their symptoms. An additional 5.8% (n = 4) replied that they did not know if they had these emotional symptoms or not before they developed their hypersensitivity. Only 4.3% (n = 3) of the respondents had ever taken any medication for emotional problems before the onset of their chemical hypersensitivity symptoms. In contrast, 37.7% (n = 26) of the respondents said that they experienced depression, anxiety, or other emotional problems after they developed their hypersensitivity, and 27.5% (n = 19) had taken some medication for these emotional problems after the emergence of their condition.

Lifestyle modifications. The necessity of avoiding offending substances can force a hypersensitive person to make numerous lifestyle changes; thus, several questions were asked to determine the extent of these alterations. Subjects were asked if they had to change their residence or alter their home, and if so, in what manner (Table 12). Of the respondents, 13% (n = 9) moved from their homes because of their hypersensitivity. A much larger percentage made major adjustments to their living environment; 34.8% (n = 24) reported that they removed carpeting or furniture from their home, and 47.8% (n = 33) stated that they had installed air and/or water filtration systems. About three-fourths of the respondents (76.8%, n = 53) said they had changed their cleaning and personal hygiene supplies, and 15.9% (n = 11) said they had switched from gas appliances to electric appliances. An additional 33.3% (n = 23) reported making other changes to their residences.

Demographics. The final questions asked about the race/ethnicity, family income, marital status, age, and sex of the respondents (Table 13). Whites made up 66.7% (n = 46) of individuals with a hypersensitivity, blacks comprised 27.5% (n = 19), and Hispanics were 2.9% (n = 2) of this group. An additional 2.9% (n = 2) replied "other" or refused to identify their race or ethnicity.

The annual household incomes of people with a hypersensitivity were evenly spread over the various levels, with 11.6% (n = 8) reporting an income of > $100,000; 23.2% ($n$ = 16) with an income level of $50,000–$100,000; 26.1% (n = 18) with $20,000–50,000; and 27.5% (n = 19) reporting an income < $20,000.

Regarding marital status, 52.2% (n = 36) of the individuals in the sample were married or living as a couple, 13% (n = 9) were divorced or separated, 14.5% (n = 10) were widowed, and 18.8% (n = 13) had never been married. An additional 1.4% (n = 1) refused to specify their marital status. The age range of the sample was reasonably well distributed with a minor bias toward upper age groups, with 33.3% (n = 23) of the subjects ≥ 60 years of age, 39.1% (n = 27) 40–59 years of age, 23.2% (n = 16) 20–39 years of age, and only 4.3% (n = 3) < 20 years of age. The sample was 79.7% (n = 55) female and 18.8% (n = 13) male, with some data missing.

Table 10. Second-phase data: related medical problems.

Question	Yes % (n)	No % (n)	Don't know % (n)	Refused/ missing % (n)
Do you have any other related medical problems?	53.6 (37)	37.7 (26)	7.2 (5)	1.4 (1)
Do you have any of the following medical problems?[a]				
Gastrointestinal/stomach	26.1 (18)	63.8 (44)	2.9 (2)	7.2 (5)
Fibromyalgia	21.7 (15)	69.6 (48)	1.4 (1)	7.2 (5)
Chronic fatigue	18.8 (13)	72.5 (50)	1.4 (1)	7.2 (5)
Other	27.5 (19)	60.9 (42)	4.3 (3)	7.2 (5)
Do you have any allergies to natural substances; if so, to what?[a]				
Total	73.9 (51)	23.2 (16)	2.9 (2)	0 (0)
Pollen	65.2 (45)	24.6 (17)	4.3 (3)	5.7 (4)
Animal hair/dander	52.2 (36)	37.7 (26)	2.9 (2)	7.1 (5)
Dust/dust mites	55.1 (38)	33.3 (23)	5.8 (4)	5.7 (4)
Molds	49.3 (34)	40.6 (28)	4.3 (3)	5.7 (4)
Other	44.9 (31)	43.5 (30)	5.8 (4)	5.7 (4)

[a]Multiple answers possible.

Table 11. Second-phase data: emotional problems and/or use of medication (e.g., for depression or anxiety).[a]

Question	Yes % (n)	No % (n)	Don't know % (n)	Refused/missing % (n)
Trouble before symptoms appeared	1.4 (1)	92.8 (64)	5.8 (4)	0.0 (0)
Trouble after symptoms appeared	37.7 (26)	62.3 (43)	0.0 (0)	0.0 (0)
Medication used before symptoms appeared	4.3 (3)	94.2 (65)	1.4 (1)	0.0 (0)
Medication used after symptoms appeared	27.5 (19)	72.5 (50)	0.0 (0)	0.0 (0)

[a]Unequal totals result from rounding and/or missing data.

Table 12. Second-phase data: changes in the home since symptoms appeared.[a]

Changes since symptoms appeared	Yes % (n)	No % (n)	Refused/missing % (n)
Carpet/furniture	34.8 (24)	65.2 (45)	0 (0)
Water/air filtration	47.8 (33)	50.7 (35)	1.4 (1)
Cleaning/hygiene supplies	76.8 (53)	23.2 (16)	0 (0)
Gas appliances	15.9 (11)	84.1 (58)	0 (0)
Other changes	33.3 (23)	65.2 (45)	1.4 (1)
House/apt. (moved)	13.0 (9)	85.5 (59)	1.4 (1)

[a]Unequal totals result from rounding and/or missing data.

Discussion: Second-Phase Findings

The Cronbach's α coefficients for the three clusters of variables indicate differing degrees of internal consistency between similar questions. The highest α coefficient (0.7028) was for the cluster of questions on triggers, with the cluster dealing with behavior modifications having a coefficient of 0.6882. The α for questions about symptoms was the lowest at 0.5054. (Supplemental information is available at http://ehpnet1.niehs.nih.gov/members/2003/5940/supplemental.pdf). All of these indicate at least a moderate amount of internal consistency. Our analysis suggests that individuals with a hypersensitivity displayed greater consistency in what triggered a reaction than in the type of symptoms they experienced after an exposure. A subsequent item analysis revealed that the trigger cluster coefficient was significantly influenced by the inclusion of electrical appliances; when this potential trigger was removed from the analysis, the internal consistency of the trigger cluster was greater. The item analysis also suggested that these findings have more relevance to MCS as a condition than to the appropriateness of the measurement instrument. Because the questions used in this phase were derived from numerous anecdotal studies of the symptoms, triggers, and behavior of MCS sufferers, the α coefficients indicated that individuals with MCS exhibit a variety of symptomologies and behavior adjustments. The analysis also suggested that not all individuals with a hypersensitivity react in the same manner when exposed to a triggering substance and that these individuals can take different actions to accommodate their hypersensitivity.

Table 13. Second-phase sample: demographics.

Question	% (*n*)
Race/ethnicity	
Black	27.5 (19)
Hispanic	2.9 (2)
Caucasian	66.7 (46)
Other	2.9 (2)
Household income	
< $20,000	27.5 (19)
$20,000–50,000	26.1 (18)
$50,000–100,000	23.2 (16)
> $100,000	11.6 (8)
Refused	11.6 (8)
Marital status	
Married	52.2 (36)
Divorced/separated	13 (9)
Widowed	14.5 (10)
Never married	18.8 (13)
Refused	1.4 (1)
Age (years)	
< 20	4.3 (3)
20–39	23.2 (16)
40–59	39.1 (27)
≥ 60	33.3 (23)
Sex	
Male	18.8 (13)
Female	79.7 (55)
No answer	1.4 (1)
Level of education	
Did not complete high school	11.6 (8)
High school graduate	26.1 (18)
Some college	29 (20)
College graduate or higher	31.9 (22)
No answer	1.4 (1)

We generated cross-tabulations of data in the second phase of this study to evaluate the external validity of the data in relationship with Miller and Mitzel's findings (Miller and Mitzel 1995). Although data on etiology, symptomatology, and severity were cross-tabulated with symptom severity, it is important to note that the method of data-gathering in this study differs considerably from Miller and Mitzel's methods. Their entire sample consisted of respondents who could definitively identify the origin (initiation) of their sensitivity, whereas 55.9% (n = 38) of the 69 subjects in our study could not identify what originally initiated their sensitivity. Only 14.7% (n = 10) of the subjects in our study reported knowing with certainty what caused their sensitivity, with an additional 26.5% (n = 18) having some idea. Consequently, the number of applicable cases in this study is limited and impedes any analytical comparison with Miller and Mitzel's findings.

The most significant cross-tabulation is between the severity of symptoms and subjects who either knew or did not know the origin of their sensitivity. Individuals who could identify the origin of their sensitivity were far more likely to report severe symptoms than people who did not know the original cause. Of the 10 respondents who could identify the origin, 50% (n = 5) described their symptoms as severe. Only 16% (n = 6) of the 38 respondents who did not know the cause say severe, with 22% (n = 4) of the 18 respondents who suspected a cause reporting severe symptoms. These results provide some substantiation for Miller and Mitzel's data (Miller and Mitzel 1995). The number of cases on the other cross-tabulations of severity with pesticide etiology, petroleum products etiology, and building material etiology is too limited, however, for a meaningful analysis. The findings of this phase indicate that for a substantial number of the respondents, their hypersensitivity is disruptive and life-altering, and a majority experience symptoms described as being either severe or somewhat severe.

Very few of the respondents (1.4%) had a history of mental or emotional problems prior to the onset of their hypersensitivity, even though over one-third (37.7%) experienced some emotional troubles after their hypersensitivity manifested. These results are relevant to the question of etiology and tend to support Miller and Mitzel's conclusion (Miller and Mitzel 1995) that MCS is inconsistent with somatoform disorders. The difference between the presymptom and postsymptom findings weakens the notion that MCS is psychogenic, or that a chemical hypersensitivity is a product of emotional disturbance. These findings indicate, in contrast, that the physical problems emerge first and emotional problems develop only afterward. It is plausible that hypersensitivity could be so disruptive that it produces substantial mental stress as the individuals attempt to cope with the limitations it produces. Another explanation may be that exposures to toxic agents can affect brain functions related to mood and emotions (Bell et al. 1997). This is an area that needs considerably more research (Ashford 1999).

The demographic characteristics of individuals with hypersensitivity tend to reflect those of the general population in the area. The distribution of whites, blacks, and Hispanics in the sample approximates their proportions of the population in the Atlanta metropolitan area. Hypersensitivity also is widely distributed among education and income levels, even though it is more common in females. These findings tend to confirm the CDHS investigation (Kreutzer et al. 1999), which also found that hypersensitivity cuts across race/ethnicity, education, and income groupings. This study, therefore, contributes to the increasing evidence that MCS is widespread and serious and deserving of substantially more research.

References

Ashford NA. 1999. Low-level chemical sensitivity: implications for research and social policy. Toxicol Ind Health 15:421–427.

Ashford NA, Miller CS. 1998. Chemical Exposures: Low Levels and High Stakes. New York:John Wiley and Sons.

Bell IR, Hardin E, Baldwin G, Schwartz G. 1995. Increased limbic symptomatology and sensitizability of young adults with chemical and noise sensitivities. Environ Res 70:84–97.

Bell IR, Schwartz GE, Peterson JM, Amend D. 1993. Self-reported illness from chemical odors in young adults without clinical syndromes or occupational exposures. Arch Environ Health 48:6–13.

Bell IR, Walsh ME, Gersmeyer A, Schwartz GE, Kano P. 1997. Cognitive dysfunctions and disabilities in geriatric veterans with self-reported intolerance to environmental chemicals. J Chronic Fatigue Syndr 2:5–42.

Black D, Rathe A, Goldstein R. 1990. Environmental illness: a controlled study of 26 subjects with 20th century disease. JAMA 264:3166–3170.

Byers V, Levin AS, Ozonoff DM, Baldwin RW. 1988. Association between clinical symptoms and lymphocyte abnormalities in a population with chronic domestic exposure on industrial solvents-contained domestic water supply and a high incidence of leukemia. Cancer Immunol 27:77–81.

Caress SM, Steinemann AC. In press. The prevalence of multiple chemical sensitivities in a population based study. Am J Public Health.

Caress SM, Steinemann AC, Waddick C. 2002. Symptomatology and etiology of multiple chemical sensitivities in the southeastern United States. Arch Environ Health 57(5):429–436.

Cullen MR, Pace PE, Redlich CA. 1992. The experience of the Yale occupational and environmental medicine clinics with multiple chemical sensitivities, 1986–1991. Toxicol Ind Health 8:15–19.

Davidoff AL, Fogarty L. 1994. Psychogenic origins of multiple chemical sensitivities syndrome: a critical review of the research literature. Arch Environ Health 49:316–325.

Davidoff AL, Fogarty L, Keyl PM. 2000. Psychiatric inferences from data on psychological/psychiatric symptoms in multiple chemical sensitivities syndrome. Arch Environ Med 55:163–175.

Ellefson R, Ford R. 1996. The porphyrias: characteristics and laboratory tests. Regul Toxicol Pharmacol 24:S119–S125.

Fiedler N, Maccia C, Kipen H. 1992. Evaluation of chemically sensitive patients. J Occup Environ Med 34:529–538.

Gots RE. 1995. Multiple chemical sensitivity: distinguishing between psychogenic and toxicodynamic. Regul Toxicol Pharmacol 24:8–15.

Interagency Workgroup on Multiple Chemical Sensitivity. 1998. A Report on Multiple Chemical Sensitivity (MCS). Predecisional Draft 26-31. Washington, DC:Interagency Workgroup on Multiple Chemical Sensitivity. Available: http://web.health.gov/environment/mcs/toc.htm [accessed 31 June 2003].

Kipen HM, Hallman W, Kelly-McNeil K, Fiedler N. 1995. Measuring chemical sensitivity prevalence: a questionnaire for population studies. Am J Public Health 85:575–577.

Kreutzer R, Neutra RR, Lashuay N. 1999. Prevalence of people reporting sensitivities to chemicals in a population-based survey. Am J Epidemiol 150:1–12.

Lax MB, Henneberger PK. 1995. Patients with multiple chemical sensitivities in an occupational health clinic: presentation and follow-up. Arch Environ Health 51:425–431.

Levin AS, Byer VS. 1987. Environmental illness: a disorder of immune regulation. Occup Med State Art Rev 2:669–681.

Meggs WJ. 1992. MCS and the immune system. Toxicol Ind Health 8:203–214.

———. 1995. Multiple chemical sensitivity—chemical sensitivity as a symptom of airway inflammation. Clin Toxicol 33:107–110.

Meggs WJ, Dunn KA, Bloch RM, Goodman PE, Davidoff AL. 1996. Prevalence and nature of allergy and chemical sensitivity in a general population. Arch Environ Health 51:275–282.

Miller C, Ashford N, Doty R, Lamielle M, Otto D, Rahill A, et al. 1997. Empirical approaches for the investigation of toxicant-induced loss of tolerance. Environ Health Perspect 105:515–519.

Miller CS, Mitzel HC. 1995. Chemical sensitivity attributed to pesticide exposures versus remodeling. Arch Environ Health 50:119–129.

Mitchell F, ed. 1995. Multiple Chemical Sensitivity: A Scientific Overview. Atlanta, GA:Agency for Toxic Substances and Disease Registry.

Mooser SB. 1987. The epidemiology of multiple chemical sensitivities (MCS). Occup Med 2:663-681.

NRS (National Research Council), Assembly of Life Sciences. 1981. Indoor Pollutants. New York:National Academy Press. Available: http://www.nap.edu/books/POD277/html/R1.html [accessed 26 June 2003].

O'Sullivan E, Rassel GR. 1995. Research Methods for Public Administrators. White Plains, NY:Longman Publishers.

Rea WJ, Bell IR, Suits CW, Smiley RE. 1978. Food and chemical sensitivity after environmental chemical exposure: case histories. Ann Allergy 41:101–110.

Siegel S, Kreutzer R. 1997. Pavlovian conditioning and multiple chemical sensitivity. Environ Health Perspect 105:521–526.

Ziem G. 1992. Multiple chemical sensitivity: treatment and follow-up with avoidance and control of chemical exposures. Toxicol Ind Health 8:73–86.

Author/s:
Caress, SM; Steinemann, AC

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