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Integrating Chemical Stressors into Paradigms for Women's Physical and Mental Health

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CHAPTER 1: INTRODUCTION

Integrating Chemical Stressors in to Paradigms for Women's Physical and Mental Health

Feminists have introduced analysis of important sociocultural variables to the study of women's health and behavior. However, missing from this analysis is consideration of the influence of ubiquitous chemical exposure in our western lifestyle. An exception is community psychology which has considered the destructive influence of environmental chemical stressors and crises on whole communities. The most famous example of the community impact of chemical stressors is the case of Love Canal, where in l954 an elementary school and residential neighborhood were built on land that had been previously used by Hooker Chemical Corporation as a chemical dump site (Hess & Wandersman, l985). Not until l978 did the New York Department of Health begin to investigate the disproportionate number of birth defects, miscarriages, and illnesses reported by families exposed to over 200 dangerous chemical pollutants. At this point community psychologists, having become aware of the impact of chemical exposure on the residents of Love Canal, began to intervene in attempt to alleviate terror and to restore a sense of home for these 237 families.

Other branches of psychology such as developmental psychology, neuropsychology, and the developing field of neurobehavioral toxicology have studied the effects of toxicants on adults and children, for example, the effects of lead on children's neurological growth. However, even these developments have been slow. Hartman (l987) pointed out that as recently as l985, an international conference on the neurobehavioral effects of solvent exposure had only three U.S. neuropsychologists in attendance despite the fact that it was held in North Carolina. Animal research has to some extent contributed to the knowledge of physiological effects of chemicals on biological systems. And on the level of policy-making, the American Psychological Association has actively urged increased neurobehavioral testing of chemicals prior to approval (Travis, McLean, & Ribar, l989). However, despite encouragement for psychologists to become involved in issues of environmental health (Weiss, l983), attentiveness to toxicants has not impacted paradigms for branches of psychology such as health and clinical psychology that directly

assess and impact individuals. Bryce-Smith (l986) has addressed the tendency of the social sciences to ignore chemical influences:

> The subject of behavior is of course relevant to all human affairs, and is therefore of interdisciplinary importance. Yet within the political and social sciences, psychiatry and to a lesser extent psychology, behavioral and social phenomena are conventionally assumed to result mainly from social influences, or disorders of mind and thought. The causation of such phenomena is not normally seen as relevant to a 'hard' science such as chemistry despite the widespread medical use of neuroactive drugs to treat behavioral symptoms. Thus, while it would be almost comically absurd to suggest that neurosis is a symptom of, say, Valium deficiency, little thought seems to be given to the possibility that real nutrient deficiencies affecting brain function, or other influences from the chemical environment, could be playing a part. (p. 116)

Similarly, though branches of medicine such as occupational and environmental medicine consider toxicants as causes of physical illness, this understanding has yet to be integrated into mainstream medicine. In addition, the psychosomatic paradigm has created among many researchers a mindset primed to look at mind-body influences, but which ignores the opposite, or somatopsychic, influence. A truly inclusive understanding of human health must include knowledge of the deleterious effects of chemical exposures. Likewise, clinical and health psychology must incorporate chemical stressors into their understanding of stress and individuals.

Pervasive Chemicals

We live in an environmentally precarious time. The number of toxic chemicals that are present in our air, water, and food multiplies with growing industrialization. Land, water, work places, and homes are cluttered with artificial substances that the general public mistakenly

assumes are safe. The EPA reports that toxic chemicals implicated in cancer, birth defects, brain damage, and other serious health effects are now present in the bodies of almost every American as a result of air, water, and land contamination (Duehring & Wilson, l994). The National Institute of Environmental Health Sciences (NIEHS) estimates that approximately 85,000 chemicals are in commercial use in the world today (Lucier & Schecter, l998). In l984 the National Research Council (NRC) reported that no toxicity data were available for approximately 39,000 commercially used chemicals (Warner, l988, cited in Duehring & Wilson, l994). Of the 1,500 new chemicals that are registered with the EPA each year, the National Toxicology Program can provide toxicological evaluations on only 10 to 20 per year (Lucier & Schecter, l998).

These untested chemicals are commonly used in food products, cosmetics, and pesticides that consumers use on a regular basis, and over 160,000 industrial plants discharge them into public water supplies (Conservation Foundation, l987). A l987 National Academy of Sciences (NAS) study found that only 17% of the chemicals used as food additives, and none of those used in cosmetics, were tested for neurotoxic effects (Duehring & Wilson, l994). However, the National Institute for Occupational Safety and Health (NIOSH) discovered 884 neurotoxic chemical compounds that are used in cosmetics and perfumes (U.S. House of Representatives, cited in Duehring & Wilson, l994).

In l986, Congress mandated that the Toxics Release Inventory (TRI) collect data on the number of chemicals disbursed into the environment. In Illinois alone, the release of 24 million pounds of potentially cancer-causing chemicals was subsequently reported (Lawson, l993). The EPA estimates that over 69 million Americans breathe air that exceeds smog standards (Duehring & Wilson, l994) and this chemical air pollution is not found only in large industrial cities. Studies show that only a small percentage of the pesticides sprayed on crops arrive at their intended destinations (Moses et al., l993). Large water supplies are also contaminated by toxic chemicals that are carried in the air. For example, DDT, still legal in Mexico and Central America, has contaminated water sources as far away as the Great Lakes (Lawson, l993). Toxicants present risks even in isolation. For example, the EPA permits contamination of

drinking water with arsenic at 50 parts per billion resulting in a cancer risk rate of 1 person in every 50 from this variable alone (Raloff, l992, cited in Duehring & Wilson, l994). And persons are exposed to hundreds of chemicals on a daily basis with no data regarding the cumulative or combined effects of these exposures.

Although conditions of outdoor air, water, and land provide cause for concern, the modern American home contains dozens of dangerous chemicals. Persons may be at greater risk for breathing polluted air indoors than out, as the EPA has ranked indoor air pollution as greater than that encountered outdoors even at toxic waste dumps (Lawson, l993). There are more chemicals today in the average American home than in the average chemical laboratory one hundred years ago (Lawson, l993). Household items such as particleboard, glues, carpeting, insulation materials, paint, moth balls, cleaners, air fresheners and also stain-repellents and flame retardants used to treat fabrics and mattresses, contain chemicals that are potential health threats (Lawson, l993; Marinelli & Bierman-Lytle, l995). Chemical contamination of the environment is therefore not limited to air, water, and land pollution, but is integral to our industrial lifestyle. Regenstein (l982) summarized it this way: "The amount of toxic wastes produced in the U.S. each year amounts to over 600 pounds for every American and 90% of these deadly wastes are disposed of improperly" (p. 17). In Chapter 2 of this volume, Lynn Lawson provides a more detailed overview of the prevalence of chemical exposures.

Differential Vulnerabilities

Women may be differentially affected by particular chemicals with biological differences contributing to the vulnerability. For example, women have less of the enzyme alcohol dehydrogenase (Freeza et al., l990) which metabolizes alcohol, carbohydrates, and chemicals (Rogers, l990). Women, but not men, who use gas cooking appliances report greater constriction of airways and reduced lung function compared with those who do not (Jarvis, Chinn, Luczynska, & Burney, l996). Additionally women have smaller body mass, a higher respiration rate, and a higher percentage of body fat that has been shown to store fat-soluble chemicals. Cycles of weight loss during dieting may release toxicants from storage in fat tissue

just as bone loss in old age may release metals previously stored in bone (Setlow, Lawson, & Woods, l998).

Ethnic minorities, as well, may have biological predispositions for greater vulnerability to chemical damage. A deficiency of the enzyme G-6-PD (glucose-6-phosphate dehydrogenase) predisposes 16% of African American men to greater risk from environmental oxidants such as ozone and nitrogen dioxide (Rios, Poje, & Detels, l993) much as sickle cell trait makes persons vulnerable to aromatic amino and nitro compounds, carbon monoxide, and cyanide.

Differential Exposure Patterns

Social factors that may contribute to women's greater toxicant exposure include use of more fragrances, cosmetics, and cleaning products containing solvents. Women are exposed to others' personal products while performing clerical work in crowded workplaces, as well as to fumes from photocopy machines, carbonless paper, and pesticides. See the statement of Pat Helinsky and Brenda Smith regarding their experiences with illness from carbonless paper in the appendix. Additionally, the use of artificial sweeteners is promoted to women for weight loss. Yet Aspartame is a known neurotoxin that breaks down into formaldehyde when sweetened drinks are stored, with longer storage and warmer temperature associated with faster breakdown (Cohen, l997). Women also use more nonsteroidal anti-inflammatory drugs than do men thus liver and kidney detoxification systems may be affected (Setlow et al., l998).

Poor and minority persons experience concentrated exposures to toxicants because their communities are afforded unequal protection from toxic emissions (Bullard, l994; Hansen & Lurie, l995). Austin and Schill (l994) have elucidated three patterns of industrialization that have led to disproportionate amounts of toxic exposures in communities of color. In one pattern, whites who originally lived in housing communities near sources of industrial emissions relocate to better and more distal housing when they can afford to, leaving cheaper and more contaminated housing to people of color. In other cases housing projects for minorities have been built either near, or, in some cases, on top of contaminated areas where land is cheap (Oliver, l994). The third pattern involves the targeting of minority communities for placement of polluting activities. Because pollution attracts more pollution (e.g., waste management and

landfills become necessary to dispose of industrial waste) a locale becomes more contaminated over time. Polluters look for communities unlikely to generate opposition; low income and low education of the populace is therefore an attractive combination. Wright, Bryant and Bullard's (l994) description of life for African American communities in Cancer Alley, the collection of towns along the Mississippi River between Baton Rouge and New Orleans, is an example of this pattern. Many of the minority communities here are poor communities that were settled by former slaves who received the promise but not the delivery of good jobs from industries settling too close to their domiciles for safety. In one locale 18 petrochemical plants exist in a 9.5 square mile area exposing inhabitants to high levels of carcinogens, mutagens, and embryotoxins. Cancer, birth defects, and vinyl chloride in children's blood are the results. See Herrera's discussion of the consequences of chemical contamination for Mexican American women in Chapter 5. Still other examples of targeting people of color for poisoning include attempts to confiscate resources discovered on Indian lands such as Peabody Coal's mining on the Black Mesa which contaminates Hopi water supplies (Hall, l994). Commonly, industries attempt to situate waste disposal sites on Indian land through manipulation of tribal councils which are in some cases not even officially recognized by the nation in question (Mander, l991). The political and economic vulnerability of Indian nations is thus exploited by corporations resulting in the sacrifice of Indian health. For example, there is now suggestion that diabetes, which has a high incidence among Native Americans, may be caused by dioxin (Montague, l997b).

It is important, however, to understand that chemical use has become so ubiquitous that all persons are exposed to unknown quantities of toxicants, regardless of income or SES. See the statement by Amy Svoboda, former EPA attorney, in the appendix of this volume for a discussion of the illnesses that resulted when EPA workers, both clerical and professional, were exposed to a broad mixture of chemicals including high levels of 4-phenylcyclohexane (4-PC) in new carpet backing during pervasive renovations at EPA headquarters.

Effects of Chemicals

Toxicants can and do affect physiological and mental health in ways about which health providers and psychologists must become aware. Most are familiar with the research on lead and other heavy metal exposure in children and adults (Travis et al., l989). Adverse effects from lead poisoning have been well documented for virtually every system in the body. Children are especially sensitive to exposure to even low levels of lead that were previously thought to be harmless (Chaiklin, 1979). Such exposure to lead has been widely implicated in central nervous system (CNS) dysfunction (David, Grad, McGann & Koltun, 1982; Marlowe et al., 1985). Damage to the CNS early in development results in impaired performance on IQ tests (Rosen, 1995; Banks, Ferretti & Shucard, 1997), learning disabilities (Marlowe, Cossairt, Welch & Errera, 1984), and mental retardation (David, Grad, McGann, & Koltun, 1982; Marlowe, Folio, Hall, & Errera, 1982). High blood lead levels have been linked to emotional disturbance (Marlowe, Errera, Ballowe & Jacobs, 1983) and behavioral problems including aggression, disturbed peer relations, distractibility, immaturity (Marlowe & Errera, 1982; Marlowe, 1986), hyperactivity (David, Hoffman, Sverd, Clark, & Voeller, l976), and attention-deficit hyperactivity disorder (Needleman, Riess, Tobin, Biesecker, & Greenhouse, 1996). Recent studies suggest that lead-contaminated house dust and yard soil are at least one source of children's lead exposure (Lanphear et al., 1996; Aschengrau, Beiser, Bellinger, Copenhafer, & Weitzman, 1997). Lead can also be passed to infants after birth during breast feeding, as lactational bone loss may trasfer lead from a woman's bones to her breast milk. Muschak (l998) has cited breast milk as "an especially toxic source of lead," (p. 629) as lead levels in breast milk mirror levels in the blood.

There may be no threshold for the effects of lead on the fetus. Among normal births, lead levels in the placenta correlate negatively and highly significantly ($p = .00002$) with head circumference, and placental cadmium is strongly negatively correlated with both head circumference ($p = .00015$) and birth weight (Bryce-Smith, l986). The author concludes: "It is chilling to realize that this prenatal brain damage has been, and is, occurring among a high proportion of the children born in industrialized countries" (p. 144).

Lead can interfere with processes as basic as neuronal firing by serving as a calcium antagonist, and can inhibit dendritic growth and obstruct the synthesis of myelin required to insulate the nerve axon. Additionally, lead interferes with the release of the neurotransmitters acetylcholine, dopamine, noradrenalin, and GABA (Bryce-Smith, l986). In adults, exposure to lead has been associated with poor cognitive functioning (Muldoon et al., 1996). Schottenfeld & Cullen (1984) found occupational lead intoxication to be linked to organic affective illness which results in fatigue, irritability, insomnia, nervousness, headache, weakness and depression. Regarding the potential for misdiagnosis, Hartman (l987) has said: "Because the emotional concomitants of lead intoxication can resemble major affective disorder, misdiagnosis potentially can occur and delay appropriate administration of chelation therapy" (p. 51). Additionally, emotional depression may occur as a secondary reaction to the cognitive losses.

Other heavy metals also impair cognitive abilities and affect the activity level of the nervous system. Copper can induce demyelination, and both lead and molybdenum can encourage copper deficiency, which (like an excess) can contribute to demylenation (Bryce-Smith, l986). Likewise, the effect of mercury toxicity on health is well known. Mercury poisoning is associated with respiratory, gastrointestinal, and skin problems, as well as anxiety and personality disorders (Perez-Comas, 1991). Recent research examining mercury exposure from dental amalgam fillings has shown that levels of mercury in the brain, blood, and urine as a result of tooth fillings is consistent with low absorption of mercury (Mackert & Berglund, 1997; Kingman, Albertini & Brown, 1998). Furthermore, Vimy, Hooper, King and Lorscheider (1997) found that mercury levels in breast milk of women were significantly correlated with the number of amalgam fillings. Concentration of total mercury in infant brains was also found to be highly correlated with levels of mercury in the mothers' hair (Cernichiari et al., 1995). Thus, maternal exposure to mercury poses a great risk to the fetus.

Despite (perhaps insufficient) gains in understanding the effects of metals, there are many other toxicants that affect health. Examples are solvents, pesticides, formaldehyde and others. Many poorly understood but well established conditions are now thought to be at least in part caused by toxicants.

Formaldehyde

Formaldehyde is widely used in construction materials such as insulation and particleboard, and in clothing, carpeting, and a myriad of other products (Wantke, Demmer, Tappler, Gotz, & Jarisch, 1996). It is present in all homes, and in particularly high levels in mobile homes, likely to be inhabited by low income persons and the elderly. Formaldehyde is a water soluble irritant that easly enters the bloodstream via inhalation, ingestion, or skin absorption, and exposure can contribute to bronchial asthma, menstrual irregularities, and hypersensitivity (Indoor Air Pollution in Massachusetts, l989). The EPA considers formaldehyde a group B1 probable human carcinogen. Studies have shown a positive relationship between formaldehyde levels and severity of symptoms such as sinus infection, headache, fatigue, depression, insomnia, and chest and abdominal pain (Godish, 1990). Chronic, long-term exposure to formaldehyde has been associated with increased levels of autoantibodies,
potentially leading to increased sensitivity to other toxins and a Lupus-like disorder (Thrasher, Broughton, & Madison, 1990). Although an ear, nose, and throat irritant, meta-analytic studies have found conflicting results regarding the relationship between prolonged formaldehyde exposure (e.g., occupational exposure) and the incidence of nasal and nasopharyngeal cancers (Collins, Acquavella, & Esmen, 1997; Partanen, 1993). Children are more sensitive to formaldehyde than are adults and are likely to experience more negative effects from formaldehyde inhalation (Krzyzanowski, Quackenboss, & Lebowitz, 1990; Wantke et al., 1996). Children attending schools in which formaldehyde was present in the particleboard wall paneling showed respiratory dysfunction and other physical ailments such as headaches, rhinitis (inflammation of the nasal mucous membrane), and epistaxis (nosebleeds) (Wantke et al., 1996). Krzyzanowski, Quackenboss, and Lebowitz (1990) found that children whose homes had higher levels of formaldehyde reported increased complaints of asthma, chronic bronchitis, wheezing, and chronic coughing.

There is much overlap between toxicants and illnesses, and direct correspondence between an illness and a particular chemical is often difficult to demonstrate due to multiple exposures, lack of follow-up studies for exposed populations, and persons' lack of knowledge

regarding what have been their exposures. Pesticides are a good example of the blurred health effects from various factors. Moreover, they deserve special mention because of the magnitude of the threat posed by their repeated and ubiquitous use.

Pesticides and Health

In l991, U.S. agricultural growers used 817 million pounds of active pesticide ingredients (National Research Council, l993), and active ingredients generally comprise only 1-5% of preparations. The remaining ingredients, referred to as "inerts" may legally include Chicago sludge (human waste from treatment plants), other pesticides whose effects may be potentiative with the active ingredients, and even radioactive and hazardous wastes. Inerts, seen as trade secrets, are not revealed on packages, and it is only because of a lawsuit against the EPA by the Northwest Coalition for Alternatives to Pesticides (NCAP) that information is now available regarding ingredients that are legally allowed to be included in pesticide mixtures. Food itself is the greatest source of pesticide exposure for the general public, due not only to use of chemicals in growing, storing, and transporting, but to the fact that food packaging can be penetrated by household and retailer insecticide use (Nriagu & Simmons, l990). Pesticides come to us in food and water, are used in restaurants, homes, hospitals, and workplaces, are present in air from agricultural drift, and are sprayed directly on passengers on international air flights. Persons who live in agricultural regions are likely to have pesticide residues in their house dust (Simcox, Fenske, Woiz, Lee, & Kalman, l995). Although they are implicated in disorders including cancer, and immune and reproductive disorders, use has not diminished. In fact, the U.S. exports approximately 450 million pounds of pesticides per year and imports 150 million (Pimentel & Lehman, l993). It is extremely difficult to explore the health effects of individual pesticides for several reasons. Pesticides fall into several categories, each with different mechanisms of action. Effects are blurred by multiple exposures, chemical combinations, the addition of inerts, and the general lack of knowledge of the public regarding the timing and content of their exposures. Many of the markers of pesticide exposure damage, such as cholinesterase inhibition, serum enzyme elevations, heme biosynthesis alterations, and chromosome alteration are not specific to one particular chemical (Brewster, Hulka, & Lavy,

l992). It is therefore possible to demonstrate exposure, and even harm, to individuals from pesticide exposure, but difficult to definitively tie the response to a particular pesticide.

Pesticides are categorized into classes according to mechanism of action. A number of pesticides (chlorinated hydrocarbons) are nervous system stimulants, and exposures may engender anxiety, nervousness, overexcitability, or even seizures. Nervousness and noise sensitivity are known indicators of organophosphate poisoning because organophosphates interfere with acetylcholinesterase (AcE) and cause a build up of acetylcholine at the nerve junction. AcE testing may reveal evidence of exposure, particularly if baseline measures are available. However, Reidy, Bowler, Rauch, and Pedroza (l992) have cited studies that show that even if appropriate medical help is forthcoming, initial AcE tests may be in the "normal" range even in the presence of symptoms, with AcE inhibition increasing in the ensuing weeks.

Sherman (l995) summarized the health effects of the organophosphates (OPO4) which stimulate both muscarinic and nicotinic receptors. Stimulation at cholinergic sites results in "wheezing, nausea, vomiting, abdominal cramps, frequent and/or involuntary defecation and urination, and visual disturbance" (p. 34). Nicotinic stimulation may cause "weakness, fatigue, muscle cramps, and involuntary muscle twitching and fasciculation" (p. 34). Delayed neurological damage may include sensory disturbances, motor weakness, ataxia, and paralysis. Central nervous system symptoms in patients may include "headache, anxiety, confusion, slurred speech, tremors, incoordination, generalized weakness, restlessness, sleep disturbance, nightmares and excessive dreaming, emotional instability, neurosis, apathy and seizures" (p. 34). In spite of the severity of these symptoms, Sherman points out that accurate diagnosis is made difficult because the patient may have any combination of the symptoms. For example, in 41 patients exposed to the organophosphate chlorpyriphos (Dursban), Sherman found that despite having had serious symptoms that included congestion, nausea, vomiting and/or diarrhea, CNS effects that included memory loss, confusion and others, and despite seeking care, "only rarely was a diagnosis of OPO4 poisoning made, and even more rarely were the patients treated with atropine. Costly and ineffective treatments were prescribed, while exposure continued in most cases, as signs and symptoms persisted" (p. 35). In Chapter 10

Elizabeth Sigmund discusses organophosphate use in the United Kingdom and related health effects, with particular attention to sheep farmers, obliged by law to dip their animals in organophosphate solutions.

The carbamate pesticides consist of methyl and dimethyl carbamates which have acetylcholinesterase-inhibiting activity, and the dithiocarbamates, which do not. The methyl (sold as Sevin or Carbaryl) and dimethyl carbamates are used in insecticides, miticides, rodenticides, and in drugs for glaucoma and for myasthenia gravis. The AcE inhibition is referred to as reversible, with effects similar to organophosphates, but shorter-acting. Toxic doses engender "salivation, lacrimation, urination, defecation, miosis, muscle spasms, general muscular weakness, prostration, and convulsions of the tonic and or clonic type" (Miller, l982, p. 780). Insecticidal carbamates exhibit additional effects including interference with thyroid activity, liver metabolism, or phospholipid synthesis in the brain, and changes in blood levels of serotonin (Miller, l982). Dithiocarbamates can also inhibit alcohol-metabolizing enzymes and prevent the synthesis of norepinephrine (NE) from dopamine (DA). In animals dietary exposure engenders ataxia with peripheral nerve demyelination and degeneration, and hind limb paralysis. Carbon disulfide, a metabolite of the dithiocarbamates lowers NE levels in the brain, heart, and adrenal glands. The prevention of synthesis of NE from DA may be a bio substrate for the memory consolidation interference seen in persons with pesticide-induced neuotoxicity (Miller, l982).

There are many other classes into which the thousands of individual pesticides may fall. This discussion cannot be exhaustive, but is meant to illustrate the variety and severity of possible health effects consequent to exposure. Most difficult to treat, and perhaps most dangerous for those exposed, are combinations of pesticides. Reidy et al. (l992) found performance decrements in motor speed and coordination, visuospatial memory, and increased anxiety, depression and physical complaints in 21 Hispanic migrant farm workers with documented exposures to a pesticide combination compared with matched controls. The exposed group exhibited more anxiety and depression with no improvement one year later. An eight-

year study in Spain found that organophosphates and paraquat may lower lithium levels in

animals thereby having possible ramifications for mood level (Perron, cited in "Pesticides may effect", l996). Mood symptoms, as well as anxiety may appear psychiatric.

Pesticides, most of which contain solvents, are implicated in additional neurological changes including Parkinson's Disease, which is associated with rural living and pesticide exposure. Semchuk, Love, and Lee (l992) found found that occupational herbicide use was associated with a three-fold increase in Parkinson's Disease with a "concomitant increase in PD risk with each 10-year increment in the cumulative exposure to agricultural work" (p. 1330). Paraquat induces Parkinsonian tremors in amphibians (Briggs, l992). Either acute or low level chronic exposure to methyl bromide has been reported to cause irreversible neurological changes including psychoses (Moses et al., l993).

Physicians and psychologists often fail to consider or recognize pesticides as a cause of illness/dysfunction, thus pesticide poisoning is underreported and hence invisible. When persons are exposed to large amounts of pesticides, as in spills or inappropriate working conditions for migrant workers, there is no tracking system to monitor their health in the ensuing months. Indeed, migrant workers have severe exposures, and often no medical care is available (Perfecto & Velasquez, 1992). In Chapter 11 Margaret Reeves and Lucy Rosas discuss pesticides, health, and the lives of Hispanic women farm workers.

Conditions of Ill Health with Toxicant Contributors

Despite issues of overlapping exposures and the lack of tracking systems, some understanding of chemical contributions to ill-health has been gained through epidemiological work, clinical investigations of those with recent exposures, animal research, and other means. The chemical problem has been studied through focus on classes of chemicals and their various effects, and by looking at associations between chemical exposures and the development of particular conditions in either animals or humans.

Reproductive Problems

Diethylstilbestrol (DES) was given to millions of pregnant women between the years of l948 and l971 in order to prevent spontaneous abortions. This estrogen-like drug caused severe congenital defects such as vaginal deformities in girls and abnormally small penises and

undescended testicles in boys. Thalidomide was also readily given to pregnant women as a sedative and resulted in extreme fetal deformities such as the absence of limbs. These examples underscore that the medical community has not adequately protected women from harmful chemicals in the past. It is therefore not valid to assume that chemicals released into the environment, found in our household products and food, or prescribed by our doctors are safe. A connection between environmental contaminants and infertility and birth defects has been supported by a number of studies. We now know that the fetus is more vulnerable than is the mother to the effects of toxicants (Lappe, l991). Serious birth defects including mental retardation have been traced to Dursban, a pesticide commonly used to kill household fleas, ants, and roaches (Sherman, l995). Animal research also connects toxins to reproductive problems. For example, an inverse relationship was found between the amount of dioxin female rhesus monkeys ingested and their ability to conceive and deliver a healthy infant monkey (Rier, Martin, Bowman, Dmowski, & Becker, l993).

It has been well supported that there are environmental chemicals and pharmaceuticals that can mimic female sex hormones and cause a number of medical illnesses (Weiss, l994). Estrogen mimickers may disrupt delicate hormonal balances through binding with and either blocking or facilitating estrogenic effects (Colborn, Dumanoski, & Myers, l996). Dioxin in particular has been linked directly to endometriosis, a condition characterized by extreme pain and often infertility that affects more than 5 million reproductive-aged women in the U.S. (Gibbons, l993). Endometriosis is poorly understood, misdiagnosed, and often attributed to psychological factors by physicians even though both animal and human studies have correlated level of exposure to dioxin with the development of this condition (Ballweg, l992). Mary Lou Ballweg provides a detailed discussion of environment and endometriosis in Chapter 4.

Chemicals with estrogen-mimicking properties include DDE (Weiss, l994) which is found in the insecticide dicofol sprayed on food crops in the United States, nonylphenols that are components in spermicides and hair coloring products, and endosulfan which is a pesticide used on U.S. vegetables. PCBs, although now illegal to produce in this country, were released as waste products into the air and water by industrial plants and presently persist in the

environment. PCBs can accumulate in human fat tissue and are secreted in breast milk, which allows the passing of these chemicals on to the next generation. Dioxins and other harmful chemicals are ubiquitous in the U.S. The simplest household and personal products are contaminated with these chemicals for aesthetic or other purposes. This is evident in the case of TCDD which is present in all chlorine-bleached paper products including sanitary napkins, tampons, toilet paper, diapers, and coffee filters. Although a body of research linking reproductive disorders with environmental pollutants is amassing, chemical use has not decreased, and some scientists have argued that continued use may result in pervasive severe reproductive abnormalities (Colborn et al., l996).

Chemical Exposure and Cancer

Chemicals have been implicated in virtually every form of cancer (Montague, 1997a). It has been estimated that 90-95% of all cancers are caused by carcinogen exposure (Proctor, 1995, as cited in Montague, 1997a). While the influence of some chemicals is uncontrollable (e.g., the influence of radioactive chemicals from the solar system), industries synthesize and knowingly release other cancer-causing chemicals into the immediate environment. Even very small amounts of these chemicals retained in the body may pose danger. For example, dioxin has been shown to be lethal to lab animals in extremely small doses, and dioxin herbicides have been linked with Hodgkin's Disease, non-Hodgkin's lymphoma, thyroid cancer, and sarcoma (Colborn et al., l996). At particular risk for cancer from pesticides may be children. Davis, Brownson, Garcia, Bentz, and Turner (l993) found an association between childhood brain cancer and family pesticide use, including termite treatment, diazinon in the garden, lawn herbicides, Kwell shampoo, indoor pesticide treatment, and even flea collars. Likewise, Gold, Gordis, Tonascia, and Szklo (l979) found that family insecticide use was one of five variables associated with the development of childhood brain tumors.

Fat soluble chemicals such as Dioxin, DDT, and PCBs can accumulate in the body and may be passed along to other species in the food chain (Colborn et al., 1996; Montague, 1997b). Further, many chemicals are interactive both with other chemicals and with endogenous

hormones. Organochlorines and other toxins interact with the body's natural estrogen system in ways that may lead to the development of cancer. These chemicals, so-dubbed "xenoestrogens," are manufactured chemicals that may mimic or block estrogen (Colborn et al., 1996). Dioxins interact with sex-linked hormones in the development of particular cancers. For example, animal research has shown that only female rats develop cancer following particular carcinogen exposures, and, if a rat's ovaries are removed, some cancers then do not develop.

Of particular interest is the interaction between chemicals and hormones in understanding the cause of breast cancer. It has been shown that chlorinated chemicals or "organochlorines" (found in everyday compounds such as pesticides and plastics) interfere with hormone balances in both animals and humans. Women who work with chemicals (e.g., vinyl chloride) develop breast cancer at disproportionate rates when compared to women with lesser contact. In addition, women with breast cancer have higher levels of pesticides in their breast tissue than do women with benign breast disease (Falk et al.,1992, cited in Moses et al. , l993; Thornton, 1993). Inhaling low doses of vinyl chloride fumes has been shown to induce breast cancer in female rats (Steingraber, 1997). Further, a cohort study of female NYC residents showed that exposure to DDE was directly related to incidence of breast cancer (Wolff, Toniolo, Lee, Rivera, & Dubin, 1993). Given that breast cancer is associated with high estrogen levels, the strong preliminary evidence that hormone-disrupting chemicals raise estrogen levels, and the evidence that breast cancer does indeed develop in greater incidence in women with particular chemical exposures, some scientists have advocated for invoking the precautionary principle and thus limiting our exposures to hormone-disrupting chemicals (Thornton, l993; Colborn et al., l996). Joe Thornton further discusses the research on chemical exposure and breast cancer in Chapter 3.

Given the preponderance of evidence that chemical exposure causes breast cancer, it is surprising that little media attention has been directed toward its prevention. Ample research points to the fact that a primary aspect of prevention would be for women to avoid exposure to various chemicals. Instead, most breast cancer education involves the early detection of cancer rather than prevention. One possible reason for the lack of discussion of chemicals is that large

chemical companies are the primary sponsors of Breast Cancer Awareness Month (Paulsen, l993). As sponsors, pharmaceutical companies maintain editorial control over the education campaigns. The misdirected educational effort of Breast Cancer Awareness Month underscores the trend in which chemical companies threaten public welfare for the sake of monetary gains.

Despite the strong suggestive nature of the evidence for toxicants as causal in cancer, and despite the variation in cancer from one nation to another and the change in cancer statistics for groups who migrate (Landrigan, l983), almost all of the work on all cancers in Health Psychology has explored psychological variables in a model hypothesizing their unidirectional influence on physical health.

Chemicals and the Immune System

Health psychologists have been instrumental in demonstrating the interplay between psychological variables and immunity. However, a more thorough investigation would incorporate toxins as direct influences on immune competence. Prolonged exposure to various chemicals may affect the immune system in a variety of ways, from immune suppression to the development of autoimmunity. Both pre-and postnatal chemical exposures have been shown to weaken the immune system of both animals and humans (Colborn et al., 1996; Thornton, 1993). Small amounts of PCBs have been shown to cause immune system suppression in female seals, and other synthetic estrogens (e.g., DES) have been linked with transgenerational cancers and immune system suppression. The immune system suppression caused by dioxins and other hormone disrupters may predispose persons to a wide range of effects including greater susceptibility and thus more deaths attributed to other diseases.

Other chemicals, including formaldehyde, solvents, hydrocarbons, and organochlorines have been shown to suppress immune system functioning in humans (Thornton, 1993; Vojdani, Ghoneum, & Brautbar, 1992, as cited in Duehring, 1993). Though animal studies of carbaryl (a methyl carbamate pesticide) in the l960s and 1970s showed multiple indices of immune suppression, carbaryl (Sevin) is now one of the three most commonly used pesticides in the U.S. Used on crops, animals, plants, and indoors, its effects are neurological, immunological, reproductive, carcinogenic, and mutagenic (Cox, no date). Persons taking cimitedine (Tagamet)

for ulcer are at increased risk for harm upon exposure, as Tagamet inhibits carbaryl breakdown. Additionally, persons with G-6-PD deficiency (including 14% of African-American males) are at risk for red blood cell degeneration from carbaryl exposure, a life-threatening effect. Further, chronic exposure to particular chemicals (e.g., chlordane/heptachlor) has been associated with auto-immune disorders (Broughton, Thrasher, & Madison, 1990.)

Foreign materials in the body may result in a variety of physiological responses, ranging from encapsulation to auto-immune suppression. Encapsulation, most often associated with breast implants (Davis, 1995), is the process by which the body responds to foreign matter by forming a wall of fibrous tissue. This tissue then becomes rock-hard, necessitating removal during which the tissue is actually chiseled from the chest wall. Greater complications arise when breast implants rupture (Davis, 1995; Levenson, Greenberger, & Murphy, 1996; Spiera & Spiera, 1997). Indeed ruptured silicone breast implants have been linked with eosinophilia, hyperimmunoglobulinemia A, fatigue, scleroderma, rheumatoid arthritis, and lupus (Levenson, Greenberger, & Murphy, 1996; Spiera & Spiera, 1997). "Gel bleed" may also occur when the silicone gradually leaks into the individual's body, and in some studies, has been associated with respiratory problems, arthritis, connective tissue damage, and irreparable auto-immune system damage (Walsh, Solomon, & Espinoza, 1989; Weiss, 1991; Goldblum, Relley, & O'Donnell, 1992, as cited in Davis, 1995). See Susan Franks' review of the literature on health effects of silicone breast implants in chapter 7 of this volume.

<u>De-Legitimized Illnesses</u>

Other conditions, not so readily labeled or accepted as diabetes or cancer, also are contributed to or caused by chemicals. Because of the lack of understanding and acceptance for these conditions, persons who suffer from them are often regarded as having psychological problems. Sick Building Syndrome (SBS) was initially attributed to mass hysteria (Soine, l995) and researchers attempted to search for a personality constellation that would render a person susceptible to SBS. Yet no psychological constellation has emerged. Instead, SBS is associated with offgassing from indoor contaminants (including volatile organic hydrocarbons) involved in remodeling materials such as carpet, paint, formaldehyde, and pesticides (Rogers, l989).

Scandinavian studies have found that the following are all associated with the onset of symptoms in sick buildings: gender, pre-existing asthma or rhinitis, a history of atopy, job category, photocopying, VDT use, and handling carbonless paper (cited in Ashford et al., 1995). See Pat Helinski and Brenda Smith's account of the effect of carbonless paper on office worker health in this volume (appendix). Multiple chemical sensitivity (MCS), moreso than any other condition, perhaps suggests a direct relationship between chemicals and illness. Persons acquire an intolerance for a particular chemical or class of chemicals that then tends to spread to more and more incitants (Ashford & Miller, 1991; Randolph & Moss, 1982). A large portion of persons with MCS attribute their illness to pesticide exposure. Others cite remodeling, low-level ongoing exposures, or lack of ventilation in the workplace as causal in development of their sensitivities (Gibson, under review). An international study of multiple chemical sensitivity found that organic solvents, pesticides, amalgam/mercury, formaldehyde, renovated buildings, paints/lacquers, pentachlorophenol/wood preservative, and stress/psychological factors were all associated with illness onset in at least three countries (Ashford et al., 1995). Persons who acquire MCS experience severe difficulties including alarming symptoms, inability to work, lack of access to medical care, dwindling finances, and ill-treatment by others uneducated about the condition (Gibson, 1993; Gibson, Cheavens, & Warren, 1996). See Pam Gibson's discussion of the life impacts of having MCS in Chapter 6.

Neurological Sequelae of Chemical Exposure

The nervous system is particularly sensitive to toxics. According to Bryce-Smith (1986) "There are good grounds for regarding neurotoxins as potentially the most dangerous of all chemical pollutants since they can affect our most critical organ, the brain, and thence even the way we think" (p. 123). Lead and other heavy metals and pesticides have neurological effects and were discussed earlier. Here attention will be given to organic solvents, prevalent in vehicle fuels, industry, paints, carpet, cleaners, wood preservatives, cosmetics, fabrics, dry cleaning, and other contexts.

Given that solvents are de-fatters or de-greasers, it is not surprising that they are able to impair neurological functioning. The report from the 1990 Conference on Organic Solvents and

the Nervous System concluded that solvent-exposed workers were at a higher risk for neuropsychological symptoms, lower performance on neurobehavioral tests, and were more likely to receive disability for a neuropsychiatric disorder (Baker, l994). A number of studies have documented neurological sequelae of solvent-exposure including difficulties with anxiety and depression, as well as impairments in memory, concentration, abstraction, and reaction time (Moses et al., l993). Irritability, fatigue, loss of ability to smell, postural difficulties, and reduction of cerebral blood flow may also occur (Baker, l994). Solvent exposure can slow central nervous system processing and thus cause intellectual deterioration (Bang, l984). On EEG with event-related potentials, Morrow, Steinhauer, and Hodgson (l992) found a delay in the P300 wave indicating a delay in cognitive processing in persons with occupational solvent exposure compared with controls, with those with longer exposures being more affected. Both auditory (Varney, Kubu, & Morrow, l998) and visual (Morrow, l994) attentional processes seem to be disrupted by solvent exposure. Morrow, Ryan, Hodgson, and Robin (l990) described a pattern of CNS dysfunction following solvent exposure that included decreased activity on a PET scan, EEG asymmetries, impairment in learning, memory, attention, and psychomotor speed, and elevations on every clinical scale on the MMPI. Hooisma, Hanninen, Emmen, and Kulig (l994) documented more problems among painters than controls in the neurologically-related symptoms categories of mood disturbance, absentmindedness, sensorimotor disturbances (upper and lower), sleep problems, fatigue, and somatic complaints. Effects of solvents in combination may be potentiative or multiplicative, and thus harmful to health even if below Threshold Limit Values (TLVs). Iregren (l982) found that workers exposed to the single solvent toluene showed statistically significant detriments only in reaction time compared with controls, while workers exposed to solvent mixtures showed showed detriments in the additional areas of perceptual speed, manual dexterity, and memory. Neurobehavioral testing often reveals effects at exposures between the levels that induce irritant and those that cause narcotic effects. Most laboratory and on-site testing of solvents has been done with participants in a resting state even though solvent uptake is known to increase during exercise (Dick, l988). But, a number of chemicals show performance detriments at levels at or below TLVs even with participants in the

resting state: 1,1,1-trichloroethane, tetrachloroethylene, acetone, and styrene. When activity is added other chemicals show neurological effects at levels lower than in the traditional stationary paradigm: i.e., toluene, xylene. The implications for exposure in the workplace that include movement and/or physical exertion are that TLVs may not protect workers engaged in physical activity. See Rosalie Ackerman's discussion of diagnostic issues for psychologists seeing solvent-exposed clients in Chapter 9.

Although toxic encephalopathy (dementia induced by toxicants) generally is thought to occur following several years of exposure, and to plateau or improve with avoidance, some follow-up studies show that those exposed to large amounts of solvents retain their neurological difficulties several months later (Morrow, Ryan, Hodgson, & Robin, l991) or even continue to deteriorate (Welsh, Kirshner, Heath, Gilliland, & Broyles, l991). Even those who improve symptomatically often continue to exhibit detriments on objective tests such as Memory Span, Digit Symbol, Block Design, Trail-Making A and B (Baker, l994), or even to further deteriorate (Juntunen, Antti-Poika, Tola, & Partanen, l982).

Functional difficulties often are evident before structural. Thus, in a two-year prospective study, White, Proctor, Echeverria, Schweikert, and Feldman (l995) found higher acute solvent exposure associated with poorer performance on visual short-term memory and manual motor dexterity tasks even when clinical disease was not evident. Lindstrom (l973) found that both workers with solvent poisoning and those with exposure but not poisoning showed inferior performance compared with controls on tests of visual perception and sensorimotor speed. However, even though brain damage from solvents and other chemicals (i.e., toxic encephalopathy) typically is not evident on CT or MRI scans, SPECT and PET scans frequently show abnormalities in the temporal and frontal lobes, and basal ganglia (Callender, Morrow, Subramanian, Duhon, & Ristovv, l993).

The pattern of psychological disturbance on the MMPI in solvent-exposed workers resembles that of the World War II prisoners of war with elevations in anxiety, depression, somatic concerns, thought disturbance, and social isolation. Longer exposure was associated with higher scores on thought disturbance, social alienation, and anxiety (Morrow, Ryan,

Goldstein, & Hodgson, l989; Morrow, Ryan, Hodgson, & Robin, l990). The anxiety is congruent with Dager, Holland, Cowley, and Dunner's (l987) finding that panic disorder can be elicited by exposure to organic solvents in the workplace. Sleep disturbances such as difficulty falling asleep (DFA), poor quality of sleep, daytime sleepiness, and the use of hypnotics are elevated in workers exposed to organic solvents (Lindelof, Almkvist, & Gothe, l992). Yet sleep disturbances have long been seen as symptoms of anxiety (e.g., DFA) or depression (early morning awakening, EMA) (Detre & Jereki, l971). Hence, the tendency to attribute the problems to psychological causes is great.

All of these findings are in agreement with Lundberg's (l996) summary of the literature on air pollution and psychiatric problems which found air pollution levels to be positively correlated with psychiatric emergencies, calls received by police, aggressive behavior, and feelings of hopelessness. In fact, solvent exposures may precipitate subtle effects upon well-being not generally attributed to exposure. Seeber, Blaszkewicz, Golka, and Kiesswetter (l997) found a dose-effect relationship between solvent exposure and ratings of annoyance and complaints, but not tiredness or tension, in both exposure chambers and a factory setting. The implications for general well-being are considerable, as exposure levels accounted for 34% of the variance.

Backlash Against Awareness of Toxicants

Even though the environmental state of affairs is beginning to be recognized, there is a backlash among some fields of medicine and psychology in that attempts are made to label/define any problems associated with chemical exposures as psychosomatic. This backlash is both fueled by and congruent with industrial interests. For example the attempt to construct a psychological profile for persons with Sick Building Syndrome was unfruitful (Soine, l995), but nonetheless served to delegitimize persons made ill in the workplace and to delay helpful interventions. Gulf War Veterans are told that their problems are caused by stress even when those problems include caustic sperm and birth defects in their children (Hudson, Miller, & Briggs, l995; Radetsky, l997). See ___ in Chapter ___ for consideration of women and Gulf War Illness. The discrediting of physiological/neurological damage as purely psychological casts blame on individuals rather than on the true cause of their problems thus taking a toll on the

welfare of the persons in question. Persons are subject to the cumulative stress of not having their needs addressed as well as being perceived as psychologically unbalanced. Hess and Wandersman (l985) have discussed the exacerbation of collective stress brought on by the government's denial of the contamination at Love Canal. Beyond the terror involved in threats to physical health, these individuals had to deal with the fact that the government was denying the reality of the chemical exposure. Community psychologists recognized the impact of the fear of exposure, the lack of validation by the government, and the resulting loss of control experienced by the residents of Love Canal.

When wrong-doing is admitted, consequences and sequalae vary by race. In white communities greater penalties are imposed for hazardous waste sites, clean-ups begin sooner, and clean-up more often involves elimination rather than simply "containment" or walling off the area than in communities of color (Bullard, l994). When buyouts occur for relocating inhabitants of contaminated communities, polluters first buy out white communities, leaving African American communities to suffer, forcing them to resort to litigation for compensation (Wright et al., l994).

Likewise, persons injured by materials that are generally understood to be safe (despite a lack of testing) face formidable obstacles when pursuing justice and/or compensation for their injuries. Brenda Thomas and Pat Helinski (this volume) discuss the obstacles they faced following chemical injury in the workplace.

Implications for Health Providers

Given the prevalence and seriousness of chemical exposures, and their potential health effects, it is appropriate for medical practitioners, and clinical and health psychologists to incorporate chemical stressors into the understanding of physical and mental health. Of primary importance is the need to educate health care providers regarding the deleterious effects of chemical exposure. Similarly, there is a great need for physicians to be trained to look for chemical exposure as a potential causal factor in a variety of syndromes. Currently, few medical schools require a course in toxicology. Furthermore, doctors must be made aware that a variety of physical syndromes may appear psychological in the early stages of the illness (Klonoff &

Landrine, 1997). Physicians must learn to screen for chemical exposure, particularly among minority and low SES populations and those occupationally exposed. Landrigan (l985) has said:

> The identification, diagnosis, and prevention of neurologic and mental disorders of toxic environmental origin will require that neurologists and psychiatrists consider the possibility of neurotoxic etiology in the evaluation of every patient who presents with neurologic or mental illness of unknown origin. (p. 59–60)

Beyond learning to identify when chemical exposure has occurred, health care providers must be trained to care for individuals who are facing the consequences of chemical exposure. For example, few physicians measure cholinesterase levels when a patient has had an organophosphate pesticide exposure. Health care providers must move beyond the "standard of care" when client need requires. Teresa Henry (l995; this volume, Chapter 12) has published primary care guidelines for considering and responding to pesticide exposure that include a summary of the acute and chronic effects of the major classes of pesticides, the essentials of taking a health history that includes questions about pesticide exposures, and suggestions for physical examination, diagnosis, and treatment. Standard health histories rarely include these elements, yet for persons who work with live near, or are exposed unintentionally to pesticide applications, they may be crucial. Brewster, Hulka, and Lavy (l992) provide an in-depth review of the research on biomarkers of pesticide exposure that may help providers develop an excellent knowledge base regarding the intricacies of deciding what to test for and when. O'Malley (l997) and Henry (l995) both discuss classes of pesticides, symptoms of exposures, and suggestions for interventions. Health care providers should know the rudiments of pesticide toxicology as the authors present it, and, minimally, know when to refer patients to an appropriate specialist. Of equal import is to refrain from invoking psychosomatic hypotheses for toxicant-induced physical illnesses. Endometriosis is only one of a large number of conditions that are often erroneously attributed to psychological factors to the detriment of women seeking health care. Klonoff and Landrine (l997) have reviewed a number of physical conditions, including multiple sclerosis, temporal lobe epilepsy and others, overrepresented in women that

may frequently present with psychological features thus confusing health care providers not well informed regarding physical illness.

Persons who are pesticide-exposed (and this includes everyone to a greater or lesser degree) are at risk for CNS damage and serious changes in quality of life. Yet they have no information and have not consented to, and in most cases do not benefit from the use of these chemicals. Agricultural crops, apartments, student dorms, workplaces, and most all public buildings and places of business (including physicians' offices) are sprayed regularly with chlorpyriphos (Dursban) for fleas, roaches, or ants. This occurs in spite of the recent EPA seven-point judgment against Dow Elanco for failing to reveal data related to health complaints against Dursban. Dow Elanco is required by the EPA to withdraw chlorpyriphos from the indoor broadcast flea control market, the indoor total release fogger market, the paint additive market, and from the direct application pet care market (shampoos, dips, sprays) among other controls (Goldman, l997).

With ubiquitous chemical exposures and such questionable consumer protection, health providers must be all the more vigilant for health effects engendered by toxicants. Likewise, Dick (l988) has encouraged the use of neurobehavioral testing to detect performance deficits indicative of mild effects that may be precursors of more severe, narcotic, or chronic effects of chemical exposure. As mentioned earlier, structural indicators (i.e., MRI, CAT) do not appear until considerable brain damage has occurred. Landrigan (l985) points out that

> The number of people potentially occupationally exposed to the known neurotoxins in an industrialized society is enormous. If only a small fraction of those exposed suffer neurologic or mental illness as a result of such exposure, then the problem of neurologic and mental illness caused by chemical neurotoxins will prove to be neither slight nor isolated. (p. 59)

Additionally, there is clearly a need for sensitivity when caring for individuals with delegitimized illnesses. Although these individuals may appear healthy, their lives have been altered drastically. In addition to the decline in physical health, individuals who have become ill due to chemical exposures must readjust their lives to varying degrees, ranging from a loss of a

job to the need to vacate their own homes. In such an environmentally dangerous time, these individuals understandably feel unsafe. Mental health providers may be particularly valuable to individuals facing the consequences of life upheaval due to chemical exposures. Indeed, these individuals must deal with declining health as well as a lack of understanding among the general population. Practitioners may have to make adjustments in facilities in order for these patients to have access to medical offices. Most conventional offices are not accessible to persons who are sensitive to perfumes, pesticides, and other chemicals. Some persons may have to be seen at alternative locations or even in their homes. Persons exposed to toxicants in the workplace, such as migrant workers may have no health care coverage and thus will not seek health care unless community outreach efforts are made to provide for affordable services.

In addition, there is room in psychology and in medicine to take on a form of public action. On both the individual and community level, health providers have the power and skills necessary to educate the public. For example, physicians provide pamphlets and other materials regarding well established conditions. However, conditions such as “house painters syndrome” need to be addressed in the same manner. Painters and others suffering from poisoning on the job need to know the cause of their symptoms and be urged to obtain help before impairment becomes irreversible.

Academics must integrate toxins into relevant coursework for future providers by first educating themselves, and then providing appropriate readings and case studies for their students. Courses in toxicology need to be required for all health providers in order to expand the current paradigm for causes of illness. To fail to address these issues, (and this has already happened), is to allow for injured persons to fall through the cracks of our system, losing their health and any chance of recovery in the process. Perhaps the most poignant chapter in this volume is Rhonda Zwillinger’s account in the appendix of the experiences of the homeless chemically injured. Zwillinger, author of The Dispossessed Project, describes her own injury from chemical exposure, her interviews of others who live nomadically in poverty, and the impact on the mental and physical health of the interviewees and their children. The presence of technological refugees such as Zwillinger’s informants, Herrera’s Mexican women injured by

chemicals, Reeves and Rosas' Hispanic farm workers, Gibson's research participants who have acquired MCS, and Hansen's Native American women with chemical injuries is evidence of technology's underside and our failure to address its shortcomings. When taken together, these populations form a substantive portion of our population that suffer injuries so that others may profit—a group with which a relevant and effective health care system will sooner or later have to contend. This collection is an effort to bring together writers from separate but related disciplines to the aim of providing a multidisciplinary discussion of the impact of chemical exposures on women's health and educating ourselves about technological issues that have too long been ignored.

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