EXPOSURE TO CHEMICAL-RELATED ENVIRONMENT POLLUTION AND DIABETES MELLITUS

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Abstract
Human health and environmental health are inextricably entwined, and the ways in which we grow, process, package, transport, market, and consume food are critical factors for both human and environmental health. The use of chemicals in modern agriculture has significantly increased productivity. But it has also significantly increased the concentration of pesticides in food and in our environment, with associated negative effects on human health. Annually there are dozens of million cases of pesticide poisonings worldwide. Moreover, it is now better that pesticides have significant chronic health effects, including cancer, neurological effects, diabetes, respiratory diseases, fetal diseases, and genetic disorders. Diabetes is the single most important metabolic disease which can affect nearly every organ system in the body. In India it is estimated that presently 19.4 million individuals are affected by this deadly disease, which is likely to go up to 57.2 million by the year 2025. However, few researches have been conducted that show association of diabetes with environmental factors like pollution, exposure to chemicals (mercury, arsenic), psychological condition (depression, stress) and socio-economic conditions (occupation, earnings) etc.

KEYWORDS: Chemicals, Environment Pollution, Diabetes mellitus, Human health.

Diabetes mellitus
Diabetes mellitus (DM), long considered a disease of minor significance to world health, is now taking its place as one of the major threat to human health in the 21st Century. It is most common non communicable disease of the world and fourth to fifth leading cause of death in developed countries. The past two decades have seen an explosive increase in the number of people diagnosed with diabetes worldwide (Zimmet, 2001 and Amos et al, 1997). The term diabetes mellitus describes a metabolic disorder with heterogeneous aetiologies which is characterized by chronic hyper glycaemia and disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The long-term relatively specific effects of diabetes include development of retinopathy, nephropathy and neuropathy (WHO, 2011). The prevalence of diabetes is increasing rapidly worldwide and the World Health Organization (2011) has predicted that by 2030 the number of adults with diabetes would have almost doubled worldwide, from 177 million in 2000 to 370 million. India
leads the world with largest number of diabetics earning the dubious distinction of being termed the "diabetes capital of the world". The current prevalence of type 2 diabetes is 2.4% in rural India and 8.2% in urban population of India. Prevalence of impaired glucose tolerance is much higher (Ramchandran, 2004; and Mohan, 2009). According to the Diabetes Atlas (2006), published by the International Diabetes Federation, the number of people with diabetes in India currently is around 40.9 million and is expected to rise to 69.9 million by 2025, unless urgent preventive steps are taken. The so called "Asian Indian Phenotype" refers to certain unique clinical and biochemical abnormalities in Indians which include increased insulin resistance, greater abdominal adiposity i.e., higher waist circumference despite lower body mass index and lower adiponectin levels. This phenotype makes Asian Indians more prone to diabetes and premature coronary artery disease. A part of this is due to genetic factors. However, the primary driver of the epidemic of diabetes is the rapid epidemiological transition associated with changes in dietary patterns and decreased physical activity as evident from the higher prevalence of diabetes in the urban population. Even though the prevalence of micro vascular complications of diabetes like retinopathy and nephropathy are comparatively lower in Indians, the prevalence of premature coronary artery disease is much higher in Indians compared to other ethnic groups. Type 1 diabetes mellitus is an auto immune disorder genetically mediated, while type 2 is more of a life style induced disorder although the role of genetic susceptibility, infections are also equally strong. Diabetes is diagnosed if the (venous) fasting plasma glucose (FPG) value is >126 mg/dl, or if the plasma glucose value 2 hours after a 75g oral load of glucose is > 200 mg/dl (WHO, 2006).

Environmental Health
The scientific community’s definition of “environmental health” also has changed in recent years. Two decades ago, the study of environmental health focused almost exclusively on chemical toxicants and their relationship to cancer, Diabetes, Cardiovascular disease, asthma and other illnesses. Now the definition of environmental health is much broader, and researchers are studying the effects on human health of the physical and social environment, which includes issues related to urban and rural development, appropriate uses of land, agriculture and pesticide use, public transportation systems, and industrial development (Olden, 1998).

Human health and environmental health
Human health and environmental health are inextricably entwined, and the way in which the food is grown, processed, packaged, transported, marketed, and consumed is critical for both human and environmental health (American Public Health Association, 2007). In exploring the impact of the built environment on public health, research indicates that the burden of illness is greater among minorities and low income communities (Bashir, 2001; Goran & Treuth 2001 and Kawachi 2001). Lower–socioeconomic status communities usually have limited access to quality housing and live in neighborhoods that do not facilitate outdoor activities or provide many healthy food options (King et al.,2000) and this low SES is a strong predictor of diabetes (Brancati et al.,1996). Inequities in construction and maintenance of low income housing, especially for older persons, persons with disabilities, and immigrants, have resulted in insufficient housing, poor quality housing, overcrowding, and higher levels of population density and health problems. Consequently, these communities may experience greater rates of respiratory disease, developmental disorders, obesity, chronic illnesses like diabetes, CVD, hypertension. Understanding linkages between socioeconomic inequity and health is essential to reducing exposures to environmental hazards as well as disparities in health (Taylor & Harrell 2001 and Fullilove, 2000). In the literature on socioeconomic status in relation to health, the skill-level attributed to different occupations has been linked to the exposure to deleterious working conditions, with the greatest exposures found among the least skilled (i.e. comparing professional, managerial, skilled, semi-skilled and unskilled occupational grades). Mechanisms of these relationships include exposure to physical, chemical and microbiological toxins, as well as lack of worker autonomy leading to psychosocial stress. Education, income, immigration, ethnicity and gender influence the determination of which populations obtain low-skilled occupations and are exposed to environmental risks. Viewed macro-economically, technological development and economic growth are main sources of occupational structure and health. The international recession portends potential damage to occupational and environmental health through losses in employment and income, and loss of financial capacity to protect worker health based on use of new technology (WHO, 2010).
Environment & Diabetes mellitus

The use of chemicals in modern agriculture has significantly increased productivity. But it has also significantly increased the concentration of pesticides in food and in our environment, with associated negative effects on human health. Annually there are dozens of millions of pesticide poisonings worldwide (Richter, 2002). Moreover, it is now better understood that pesticides have significant chronic health effects, including cancer, neurological effects, diabetes, respiratory diseases, fetal diseases, and genetic disorders (Anderson, 2014). Chemical factors like Mercury and Arsenic are well-known toxic agents that induce oxidative stress and produce various types of cell & tissue damage. They increase oxidative stress thus inducing both insulin-dependent and non-insulin dependent diabetes. It also reduces the anti-oxidant capacity, depletes glutathione and thus induces insulin resistance (Longnecker & Daniels, 2001). All forms of diabetes have very serious effects on health. In addition to the consequences of abnormal metabolism of glucose there are a number of long-term complications associated with the disease. These include cardiovascular, peripheral vascular, ocular, neurologic and renal abnormalities, which are responsible for morbidity, disability and premature death in young adults. Furthermore, the disease is associated with reproductive complications causing problems for both mothers and their children. Although improved glycemic control may decrease the risk of developing these complications, diabetes remains a very significant cause of social, psychological and financial burdens in populations worldwide (Cather, 2015). Health effects of pesticides first focused on the risks of acute intoxication among people with direct exposure. The availability of longitudinal data shifted the main concern to the risks of chronic intoxication and environmental contamination. More recently the concern moved to diseases such as cancer for which the risk is not associated with instant effects of pesticides but with chronic exposure. Nowadays, the concern about latent effects is not only on people with direct exposure but also on subjects with indirect exposure such as consumers or residents of rural communities. Different risks associated with pesticides are often classified based on whether they have short-term effects (such as diarrhea, abdominal pain, headaches, nausea, vomiting, etc.) or long-term effects (such as skin diseases, cancer, depression, neurological deficits, diabetes, genetic disorders, or even death) (Andersson, 2014 and Dadvand, 2013).

Environmental risk factors are thought to act as either ‘initiators’ or ‘accelerators’ of beta cell autoimmunity, or ‘precipitators’ of overt symptoms in individuals who already have evidence of beta cell destruction. They also may function by mechanisms that are directly harmful to the pancreas, or by indirect methods that produce an abnormal immune response to proteins normally present in cells. The Type 1 Diabetes Mellitus (T1DM) environmental risk factors that have received most attention are viruses and infant nutrition. The role of hygiene in the etiology of T1DM is also currently being explored. It has been hypothesized that delayed exposure to microorganisms due to improvements in standard of living hinders the development of the immune system, such that it is more likely to respond inappropriately when introduced to such agents at older (compared to younger) ages (Marshall et al., 2004; McKinney et al., 1997). In recent years, most populations experience a continuous supply of calorie-dense processed foods, as well as a decrease in physical activity. This likely explains the rise in Type 2 Diabetes Mellitus (T2DM) prevalence worldwide. The major environmental risk factors for T2DM are obesity (> 120% ideal body weight or a body mass index > 30 k/m2 ) and a sedentary lifestyle (Shaw and Chisholm, 2003). Thus, the tremendous increase in the rates of T2DM in recent years has been attributed, primarily, to the dramatic rise in obesity worldwide (Zimmet et al., 2001). It has been estimated that approximately 80% of all new T2DM cases are due to obesity (Lean, 2000). This is true for adults and children. In addition to general obesity, the distribution of body fat, estimated by the ratio of waist-to-hip circumference (WHR), also has an impact on T2DM risk. WHR is a reflection of abdominal (central) obesity, which is more strongly associated with T2DM than the standard measures of obesity, such as those based on body mass index. The other major T2DM risk factor is physical inactivity. In addition to controlling weight, exercise improves glucose and lipid metabolism, which decreases T2DM risk. Physical activity, such as daily walking or cycling for more than 30 minutes, has been shown to significantly reduce the risk of T2DM (Hu et al., 2003). Junk food simply means an empty calorie food. An empty calorie food is a high calorie or calorie rich food which lacks in micronutrients such as vitamins, minerals, or amino acids, and fiber but has high energy (calories). These foods don’t contain the nutrients that the body needs to stay healthy. Dense sugar content can cause type 2 diabetes mellitus (Nisar et al., 2009). Unfortunately, meals consisting of junk food don’t fill up for
Air pollution has been linked to a number of health problems, including heart, circulatory and lung diseases. It has also been shown to be particularly harmful to vulnerable groups of people, such as those with diabetes. The risk associated with exposure to traffic-related Particulate Matter (PM) was assessed with diabetes. The risk associated with exposure to traffic-related Particulate Matter (PM) was assessed

Recent studies also suggest that exposure to chronic traffic and air pollution influences the development and progression of atherosclerosis (Bauer et al., 2010 & Kanzli et al., 2010), possibly via systemic oxidative stress and low-grade inflammation in endothelial cells and macrophages (Brook et al., 2010). Air pollution exposure increases the risk of cardiopulmonary morbidity, acute events and mortality (Gan et al., 2011 and Ruckerl et al., 2011). Developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of developing diabetes (Kohei 2010 and Dorman 2003).

Aging, obesity, insufficient energy consumption, alcohol drinking, smoking, etc. are independent risk factors of pathogenesis. Obesity (particularly visceral fat obesity) due to a lack of exercise is accompanied by a decrease in muscle mass, induces insulin resistance, and is closely associated with the rapid increase in the number of middle- and high-aged patients. The changes in dietary energy sources, particularly the increase in fat intake, the decrease in starch intake, the increase in the consumption of simple sugars, and the decrease in dietary fiber intake, contribute to obesity and cause deterioration of glucose tolerance. A variety of factors, the most important of which may be that different gene-environment interactions operate different populations may be responsible in increasing the risk of developing diabetes (Kohei 2010 and Dorman 2003). Air pollution exposure increases the risk of cardiopulmonary morbidity, acute events and mortality (Gan et al., 2011 and Ruckerl et al., 2011). Recent studies also suggest that exposure to chronic traffic and air pollution influences the development and progression of atherosclerosis (Bauer et al., 2010 & Kanzli et al., 2010), possibly via systemic oxidative stress and low-grade inflammation in endothelial cells and macrophages (Brook et al., 2010). Air pollution has been linked to a number of health problems, including heart, circulatory and lung diseases. It has also been shown to be particularly harmful to vulnerable groups of people, such as those with diabetes. The risk associated with exposure to traffic-related Particulate Matter (PM) was assessed

Environment must be conducive to a way of life that provides adequate time for sleep, rest, relaxation, social interactions, employment, safe and efficient means of everyday transportation to work. Anxiety was correlated significantly and negatively with Energy and Positive Wellbeing (Sridhar et al., 2010). Psychological condition (depression) and socio-economic conditions (occupation, earnings), alcohol consumption and current drinkers were strongly associated with stress symptoms. Studies have shown that tobacco and drinking are used as means of coping with life’s stresses (Bressert, 2012). The mechanism behind these effects is related to the so-called "stress hormones." These hormones, that include adrenaline and cortisol, have as one of their primary effects, the mobilization of stored energy including glucose and fatty acids. Direct effects of stress on the nerves controlling the pancreas can also inhibit insulin release. Energy mobilization is part of the "fight or flight" response and is useful to prepare individuals to deal with stressors. In individuals who do not have diabetes, these energy sources can be quickly utilized. However, in people with diabetes, the lack of insulin or the presence of significant insulin insensitivity causes the newly released glucose to build up in the blood stream. Stress has long been considered an important factor in Type 2 diabetes. However, it is only recently that research has demonstrated that stress may play a role in the onset of Type 2 diabetes in individuals predisposed to diabetes and in blood glucose control in people with established diabetes. Simple stress management techniques can have a significant impact on long-term blood glucose control and can constitute a useful tool in the management of this common condition (Surwit, 2002). Stress has been reported to increase blood glucose and decrease insulin activity (Surwit et al., 1992). Golmohammadi et al., in 2006, carried out a study to look into association of stress with diabetes mellitus type 2.

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separately. There was a 36% increased risk of developing type 2 diabetes for each additional 1 μg/m3 exposure to either PM 10 or PM 2.5 pollution from traffic. This suggests a higher toxicity of traffic-specific air pollution compared to that of total PM. The exact mechanisms underlying how air pollution influences the body's metabolic systems and insulin production, which regulates blood sugar levels, is not known. However, it is thought that PM causes inflammation in the body. This in turn has been linked to cells becoming resistant to insulin, which can lead to the development of type 2 diabetes. Older, obese participants were more likely to develop type 2 diabetes than other participants in this study. Being overweight and older has been linked to inflammation in the body in other studies, suggesting exposure to PM is an additional burden for such people (Weinmayr at et.,2015). These underlying biological mechanisms are also involved in the pathogenesis of type 2 diabetes mellitus, particularly in the progression of insulin resistance (Rajagopalan et al., 2012). It has also been shown in animal models that exposure to particulate matter 2.5 μm or less in diameter (PM2.5) for 24 weeks exaggerates insulin resistance, visceral inflammation and adiposity in diet induced obese mice (Sun et al.,2009). Adverse effects have been found to be associated with indoor air pollution exposure, for example between Environmental Tobacco Smoke (ETS) exposure and type 2 diabetes mellitus incidence and susceptibility among adults and adolescents. It thus seems biologically plausible that air pollution may be a risk factor for insulin resistance and type 2 diabetes mellitus. (Zhang et al.,2011 and Weitzman et al., 2005).

As far as the role of traffic-related air pollution exposure in the pathogenesis of insulin resistance is concerned, research suggests an association between traffic related air pollution and type 2 diabetes mellitus in adults, which has been investigated in several studies (Raaschou et al., 2013; Anderson et al., 2012; Dijkema et al.,2011 and Puett et al., 2011). Andersen et al.,(2014) reported borderline significant associations between confirmed diabetes cases and NO2 exposure in participants of the Danish Diet, Cancer and Health Cohort Study.

The observations that home proximity to a major road is associated with adverse effects in women (Puett et al., 2011 and Krämer et al., 2010), and that there are indications of a relationship between type 2 diabetes mellitus and traffic within a 250 m buffer, imply that traffic-related air pollution could be responsible for associations of air pollution with type 2 diabetes mellitus in adults (Dijkema et al., 2011). Insulin resistance levels tended to increase with increasing traffic-related air pollution exposure, and this observation remained robust after adjustment for several confounding factors, including BMI and passive smoking. Data suggest that air pollution exposure may increase the risk of insulin resistance at the age of 10 years. (Thiering et al.,2013). Studies on air pollution and insulin resistance in humans are scarce, but associations between air pollution and metabolic outcomes have been studied using several animal models. For example, PM2.5 increased HOMA-IR in rats fed a high-fat diet for 6 weeks but not in rats fed a normal diet (Yan et al., 2011). Furthermore, ambient air pollution exaggerated adipose inflammation and insulin resistance in diet induced obese mice (Sun et al., 2009). There is no doubt that Type 2 diabetes mellitus (Non Insulin Dependent Diabetes Mellitus) is associated with unhealthy lifestyle practices like physical inactivity, obesity, consumption of high carbohydrate, high fat and low fiber diet etc. Emphasis on the management of these factors, along with the medication is justified as these factors play a major role in controlling the disease. On the other hand, several other factors are underestimated and overlooked, which in fact do carry very significant role in causation, manifestation and progression of diabetes. These factors include stress, occupation, environmental pollution, chemical exposure, tobacco & alcohol consumption and low socioeconomic status. Thus, these factors also need equal emphasis if we are to control and effectively manage diabetes.

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