

Diabetes: Time to Look Beyond Gluttony and Laziness

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Introduction

According to European Action on Global Life Sciences (EAGLES) diabetes report 2008, 80% people with diabetes in the world hail from developing countries. This number is predicted to grow by 150% in the next 25 years. According to the International Diabetes Federation estimates, about 3.8 million people died of diabetes in 2007 and predict that without urgent action, this may increase by 50% in the next 10 years. This estimate is more than the deaths from HIV/AIDS and nearly four times more than the deaths from malaria.⁽¹⁾

Population-based surveys of 75 communities in 32 developing countries observed that the disease diabetes is rare in communities where traditional lifestyle has been preserved. By contrast, however, some Arab, Asian Indian, Chinese, and US Hispanic communities that have undergone westernization and urbanization are at a higher risk. In these populations, prevalence of diabetes ranges from 14% to 20%.⁽²⁾ India leads the world with the greatest number of diabetics (42 million) which is projected to expand up to 70 million⁽³⁾ by the year 2025 due to the compounding effect of increasing modernization coupled with the adoption of sedentary lifestyle in urban as well as in rural India.⁽⁴⁾ In addition, compared with the other ethnic groups around the world, Indians are identified to be more insulin resistant,⁽⁵⁾ secrete more insulin in response to glucose,⁽⁶⁾ demonstrate a higher glycemic response to all the

foods,⁽⁷⁾ and develop type 2 diabetes at younger age.⁽⁸⁾ Therefore, Indian physicians opine that the modern antidiabetic prescriptions developed based on European physiological conditions require different handling in the Indian framework.⁽⁹⁾

Nonetheless, despite the understanding that diabetes is a lifestyle related disease caused by laziness and gluttony and availability of array of drugs and devices,⁽¹⁰⁾ in the 21st century, the disease is taking the shape of a global epidemic. Mankind has known this disease for centuries, and it was treated as a disease of the wealthy.^(11,12) However, in this century, diabetes is not restricted to the rich people only; instead it has spread its tentacles in every strata of the society. In fact, in the course of modernization and wealth generation to improve the living standards through the process of industrialization, chemical, green and white revolutions, human activities have produced several non-natural pollutants also. These pollutants have affected the life of rich and the poor indiscriminately. For decades, toxicologists have been warning about the harmful effects of these pollutants. The time has come now to seriously look beyond the conventional understanding of diabetes pathogenesis in finding out other sets of factors that contribute to the epidemic of diabetes in modern era.

Persistent Organic Pollutants and Risk for the Development of Diabetes

A growing group of scientists the world over have now started believing that the main culprit for the development of diabetes in the mass may be the family of toxic chemicals known as persistent organic pollutants (POPs). It has been observed recently that there is a striking, monotonic and additive dose-response relationship between serum concentrations of POPs and prevalence of diabetes in the US population with background

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environmental exposure to POPs.⁽¹³⁾ This National Health and Examination Survey 1999–2002 observed a strong dose–response relation between serum concentrations of six POPs [2,2',4,4',5,5'-hexachlorobiphenyl (HCB); 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin (HpCDD); 1,2,3,4,6,7,8,9-octachlorodibenzo-*p*-dioxin (OCDD); oxychlorodane; *p,p'* – dichlorodiphenyltrichloroethane (DDE); and *trans*-nonachlor] and the prevalence of diabetes. The POPs belonging to organochlorine (OC), pesticides (*trans*-nonachlor and oxychlorodane) or polychlorinated biphenyls (PCBs) subclasses were strongly associated with diabetes.

Interestingly, in this study, it was noticed that obesity alone did not increase the prevalence of type 2 diabetes among subjects without detectable levels of POPs. It has been argued, therefore, that the increased risk for the development of diabetes in the obese people may be due the presence of high level of POPs.⁽¹⁴⁾ Presence of abundant adipose tissues and lipid-rich environment in a obese person may serve as the transport vehicle for lipophilic POPs and hence facilitate their accumulation in obese individuals. These explanations have been substantiated by a recent study which disclosed that even after adjustment for the known type 2 diabetes risk factors like obesity, environmental exposure to some POPs substantially increased the risk of future development of type 2 diabetes in elderly population.⁽¹⁵⁾ In this regard, it is interesting to note that the relationship between adiposity and type 2 diabetes has been weakly associated.⁽¹⁶⁾ Therefore, it has been proposed that similar to the known pancreatic β -cells toxicants like streptozotocine, alloxan and vacor,⁽¹⁵⁾ the long-term exposure of even low dose of POPs may disrupt the mitochondrial function of pancreatic β -cells resulting in compromised insulin secretion, as proper mitochondrial functioning is a key factor for pancreatic β -cells to secrete insulin.⁽¹⁷⁾

In addition, results from the National Health and Nutrition Examination Survey 1999–2002 revealed that the serum concentrations of OC pesticides were strongly and positively associated with the phenomenon of insulin resistance, a frequent pathogenic precursor of type 2 diabetes among non-diabetic subjects.⁽¹⁸⁾ It is suggested, therefore, that the background environmental exposure to some POPs, especially OCs and chlordane pesticides, may be critically involved in the pathogenesis of diabetes through pathways involving insulin resistance. Another study from National Health and Environment Effect Research Laboratory, USA, observed increased mortality from Ischemic Heart Disease (IHD) and type 2 diabetes mellitus in four wheat producing states of USA and established the association between increased environmental exposure to chlorophenoxy herbicides used in great wheat producing states and

the mortality from type 2 diabetes in the general population of the county.⁽¹⁹⁾ This study supported earlier observations on the effects from exposure to dioxin and dioxin-like compounds, and increased mortality from IHD and diabetes in the general rural population of agricultural counties in states like Minnesota, Montana, North and South Dakota. Furthermore, association between increased prevalence of self-reported diabetes in people working in pesticide plants and farming occupations and exposure to high level of β -hexachlorocyclohexane (β -HCH), HCB, 2,2'-bis(4-chlorophenyl)-1,1,1-trichloroethane (*p,p'*-DDT) and oxychlorodane was also observed.⁽²⁰⁾

A recent study with 20 years of follow-up to the exposure of POPs contaminant with non-diabetic individuals observed that even low dose exposure to POPs [like *trans*-nonachlor, oxychlorodane, mirex, highly chlorinated biphenols and polybrominated biphenyl (PBB)-153] was associated with increased risk of diabetes development.⁽²¹⁾ Impaired insulin secretion is an important aspect of impaired glucose metabolism and diabetes development. Exposure to high level of contaminants like PCBs in non-diabetic people has been found to be associated with low level of insulin but higher blood glucose values.⁽²²⁾

Despite the fact that global phase-out of 12 most hazardous POPs such as like PCB, dioxins, and furan began in 2004 under the Stockholm Convention,⁽²³⁾ a combination of factors has led to their continued production, trade, use, and the release into the environment in India.⁽²⁴⁾ OC and organophosphate (OP) pesticides are likely to pollute lipid containing food sources like milk owing to their lipophilicity. Recently, the presence of the high concentrations of benzene hexachloride (BHC), aldrin, dieldrin, endrin and heptachlor has been reported from Malsinghwa village, Chandigarh (India).⁽²⁵⁾ In a study on 147 samples of bovine milk collected from over 14 districts of Haryana (India), presence of HCH, DDT, endosulfan, and aldrin was detected in 100%, 97%, 43%, and 12% samples, respectively. Concentrations of β -HCH and 2,2'-bis(4-chlorophenyl)-1,1-dichloroethylene (*p,p'*-DDE) were more as compared to other isomers and metabolites of HCH and DDT.⁽²⁶⁾ BHC (75%), methyl parathion (37.5%), dieldrin and DDE (12.5% each) were also found in bovine milk samples collected from small-scale rural and urban dairies of Allahabad region in Uttar Pradesh (India).⁽²⁷⁾ Rural dairy samples carried higher loads of methyl parathion, which may be attributed to the large-scale agricultural practices in the area.⁽²⁷⁾ Table 1 presents some of the important POPs that have displayed statistically significant association for the development of type 2 diabetes.

Table 1: Major environmental persistent organic pollutants (POPs) that have displayed significant association with the development of diabetes

PCBs	Dioxins	OC pesticides	Others
PCB-74, PCB-126, PCB-153, dioxin-like PCBs, highly chlorinated PCBs	PCDDs, PCDFs, total dioxins, HpCDD, OCDD	<i>p,p'</i> -DDE, <i>p,p'</i> -DDT, HCB, β -HCH, oxychlorane, <i>trans</i> -nonachlor, heptachlor, aldrin, cyanazide	NO ₂ , PM, BPA, PBB-153, PBDE-153, dichlovos, trichlorfon, cadmium, arsenic

PCBs, polychlorinated biphenyls; PCDDs, polychlorinated dibenzo-*p*-dioxins; PCDFs, polychlorinated dibenzofurans; HpCDD, 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin; OCDD, 1,2,3,4,6,7,8,9-octachlorodibenzo-*p*-dioxin; OC, organochlorine; *p,p'*-DDE, 2,2'-bis(4-chlorophenyl)-1,1-dichloroethylene; *p,p'*-DDT, 2,2'-bis(4-chlorophenyl)-1,1,1-trichloroethane; HCB, hexachlorobenzene; β -HCH, β -hexachlorocyclohexane; NO₂, nitrogen dioxide; PM, particulate matter; BPA, bisphenol A; PBB, polybrominated biphenyl; PBDE, polybrominated diphenyl ether

Table 2: Arsenic contaminated cities and towns from the states of India⁽³¹⁻³⁷⁾

States	Cities/Towns
Andhra Pradesh	Medak, Pathancheru
Arunachal Pradesh	Dibang valley was affected the maximum, including six other districts
Assam	Karimganj, Jorhat, Dhamji, Golaghat, and Lakhimpur were affected the maximum amongst the 21 affected districts
Bihar	Bhagalpur, Munger, Buxer, Khagaria, Samastipur, Vaishali, Begusarai, Lakhisarai, Bhojpur, Patan, Saran (Chapra), Katihar, Purvi and Paschimi Champaran, Sitamarhi, Madhubani, Supaul, Araria, Kishanganj, Purnia
Chhattisgarh	Rajanandgaon, Durg, Ambagarhchawki, Raipur
Delhi	New Delhi
Jharkhand	Sahibganj
Karnataka	Raichur, Sindhanur, Lingsugur, Deodurg, Manvi, Maski
Madhya Pradesh	Kaner, Betul
Manipur	Thuobal
Nagaland	Mon, Mokochung
Punjab	Patiala, Chandigarh
Tripura	Dhalai, West and North Tripura districts
Uttar Pradesh	Lakhimpur-Kheri, Ballia, Kanpur Nagar, Bahraich, Balarampur, Gorakhpur, Chandauli, Ghazipur, Varanasi, Unnao, Faizabad, S. R. Nagar, Lucknow, Pilibhit, Shravasti, Siddharth Nagar, Maharajanj, Kushinagar
West Bengal	Maldah, Murshidabad, North and South 24 Parganas, Nadia, Howrah, Hoogly, Bardhaman, Kolkata, Koochbihar, North and South Dinajpur

Arsenic Exposure and Risk for the Development of Diabetes

The association between exposure to inorganic arsenic and incidence of diabetes is a relatively new toxicological observation. Up to now, there are 20 epidemiologic studies linking diabetes mellitus with acute as well as chronic exposure to arsenic from environmental and occupational sources. Cross-sectional and prospective follow-up studies from Taiwan indicated the association between arsenic exposure from drinking water and prevalence of diabetes in a dose-dependent manner. Further reports from Sweden and Bangladesh supported these studies. However, except the studies from Taiwan and Bangladesh, other studies could not show consistency in a systematic analysis.⁽²⁸⁾ Recently, in a cross-sectional study from National Health and Nutrition Examination Survey (USA) 2003–2004, it was observed that after adjustment for biomarkers of seafood intake that constitute organic arsenicals, total urine arsenic level was positively associated with increased prevalence of type 2 diabetes.⁽²⁹⁾ This finding supports the hypothesis that accumulative effect of even low level exposure to inorganic arsenic in drinking water may be responsible for the prevalence of diabetes epidemic.^(29,30) In India, arsenic contamination of ground water is a common phenomenon along the Bengal-Delta plain, covering the

states of West Bengal, Jharkhand, Bihar, Uttar Pradesh, Assam and other northern states.⁽³¹⁾ Recently, from southern part of India, ground water of several villages in Raichur district of Karnataka state has been found to be affected heavily with arsenic contamination.⁽³²⁾ Table 2 presents arsenic contaminated districts and towns from different states of India. Arsenic consumption has been very common in the region of Chandigarh (northern India) where prolonged consumption occurs as a result of intake of arsenic contaminated water, vegetables, adulterated opium, Ayurvedic and indigenous medicines and homemade brews.⁽³³⁾ Increased and indiscriminate uses of fertilizers, insecticides and industrial activities also cause increased release of arsenic in Indian soil.⁽³¹⁾

Concentrations of and exposure to POPs and inorganic arsenic in many societies around the world may be different. Therefore, the risk of diabetes development may vary and could be graver than that contemplated. Diabetes is a lifelong debilitating disease. It was mentioned in the beginning of the article that the projected number of diabetics in India by 2025 would be around 70 million.⁽³⁾ If the exposure to arsenic contamination plays an important and causal role in diabetes development, nearly 50 million people living in 3200 villages alone in 9 of the total 18 districts of West Bengal are affected with arsenic contaminated

drinking water.⁽³¹⁾ Rice and vegetable fields irrigated by such ground water further receive large amount of arsenic in this area.⁽³⁴⁾ Recently, 30,000 people were found chronically exposed to arsenic contaminated water from a small town called Maski alone in Karnataka state.⁽³²⁾

Contamination of Mother's Milk with Persistent Organic Pollutants

A newborn baby starts growing by feeding on the breast milk of its mother with lots of love and affection. It is agonizing to state that pesticides have not spared even mother's milk. High level of endosulfan-1, dieldrin, oxychlordane, β -HCH, *p,p'*-DDE, *p,p'*-DDT, and *cis*-heptachlore epoxide were detected in the breast milk of Danish and Finnish women.⁽³⁸⁾ High lipid solubility of these chemicals makes fat-rich milk an easy target for their transport. Physiologically, breast feeding in children is believed to help prevent development of diabetes in the future generation as well as in the feeding mothers by virtue of several hormonal, biochemical and physiological mechanisms.⁽³⁹⁾ A study of mothers' milk from Indian metropolitan city Chennai (Madras), Perugudi (situated at suburb of Chennai), Chidambaram (an agricultural town 250 km south of Chennai) and Parangipettai (a fishing village 15 km north of Chidambaram, Tamil Nadu, India) revealed the presence of measurable concentrations of HCH, DDTs, PCBs, chlordane-related compounds (CHL), and HCBs.⁽⁴⁰⁾ Authors report that concentrations of OC pesticides, HCHs and DDTs have increased in the breast milk of mothers of Chennai in the last decades.⁽⁴⁰⁾ A recent study that analyzed the contamination status of OCs in human milk across three major Indian metropolitan cities, New Delhi, Mumbai and Kolkata, found that the presence of DDT was predominant followed by HCHs and PCBs, though CHLs and HCB were noticed in lower concentrations.⁽⁴¹⁾ Based on the estimated daily intake, this study observed that some infants were exposed to OCs to a greater extent, particularly HCHs, than the guideline standards.

It is important to understand that metabolism of POPs in mammalian system is intractable and the half-life of compounds ranges from 7 to 10 years in humans.⁽⁴²⁾ Therefore, accumulative effect of even low dose exposure in the general population over time and their presence in the body may have long-term detrimental effects. Dioxins and 2,3,7,8-tetrachlorobenzo-*p*-dioxin (TCDD) are congeners of POPs and have been reported to modify glucose metabolism, increase insulin resistance,^(43,44) and cause hypoinsulinemia through the alteration of pancreatic membrane tyrosine phosphorylation,⁽⁴⁵⁾ suggesting their involvement in the pathogenesis of both type-1 and type-2 diabetes mellitus. Heavy presence of these POPs in bovine as well as human milk and

its consumption in India may find an association with altered glucose metabolism^(6,7) and increased incidence of insulin resistance⁽⁵⁾ in Indian adults as well as in the children population and relates to the increased incidence of diabetes. Milk is an important essential diet in Indian homes, be it urban or rural. These findings may explain the query as to why the incidence of disease is soaring not only in the meat-addicted West, but also in countries such as India where millions follow vegetarianism.⁽²³⁾

Persistent Organic Pollutants and Development of Metabolic Syndrome

The prevalence of clusters of risk factors, such as elevated blood pressure, triglycerides, impaired fasting blood glucose levels, insulin resistance, and waist circumference, responsible for the development of metabolic syndrome has been observed in non-diabetic adults with the background exposure to a mixture of POPs. OC pesticides have been observed to display strong and consistent association with the development of metabolic syndrome, especially elevated triglycerides and fasting blood glucose levels. Dioxin-like PCBs were found to be significantly associated with waist circumference, triglycerides and impaired fasting blood glucose levels. Polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans have been observed to be significantly associated with increased blood pressure among non-diabetic adults.⁽⁴⁶⁾ Insulin resistance is an important phenomenon of metabolic syndrome. It has increased at an alarming rate in the modern global society. A recent experimental study in Sprague-Dawley rats provides evidence that long-term consumption of lipophilic POPs leads to the development of insulin resistance and associated metabolic disorders.⁽⁴⁷⁾

The Journey Ahead

Despite the fact that globalization and industrialization has contributed significantly in improving the living standards of Indians, majority of the population still depends on agricultural practices. Increasing the production of food grains and other commodities is mandatory for India to feed the world's second largest population. It is apparent, therefore, that this goal could not be easily achieved without the help of chemical fertilizers, insecticides, pesticides and digging the ground for irrigation bore wells. Altogether, these activities play a role in exaggerating POPs and arsenic contamination. There is lack of diabetes prevalence and incidence data from POPs and arsenic affected regions of India. In view of the statement of Shaukat Sadikot that four out of five people with diabetes in rural and tribal India do not know that they have it,⁽¹⁾ it is quite possible that the real situation may be graver than predicted. In light of the above developments, it becomes imperative,

therefore, to look beyond the conventional belief that diabetes is merely a consequence of gluttony and laziness in this era. There is an urgent need to accelerate research in this area linking toxicological impacts of various pollutants, to have realistic data at a national level and to educate people regarding the cumulative detrimental and long-term harmful effects of even low exposure of these pollutants later in their life.

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