



## Committee: World Health

# Question of: Combating the International Obesity and Diabetes Epidemic

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### Introduction:

Obesity is the presence of excessive body fat. Obesity has well-known deleterious effects on human health by increasing the risk of various chronic diseases including Type 2 diabetes, cardiovascular diseases, and some cancers. Failure to address the continued increase in obesity could result in an erosion of the health gains observed since the early 20th century.

Diabetes mellitus, which is simply termed diabetes throughout this report, is a disease characterised by high glucose in the blood. Insulin is a natural hormone secreted by pancreatic  $\beta$ -cells to decrease blood glucose levels. It acts by communicating with the skeletal muscle and adipocytes to take up glucose, and with the liver to block glucose production.

Type 1 diabetes occurs when insulin is no longer produced in sufficient quantities due to an autoimmune attack against pancreatic  $\beta$ -cells, and therefore glucose homeostasis is highly disrupted.

Type 2 diabetes occurs when cells fail to use insulin properly because of insulin resistance together with an inadequate response of the  $\beta$ -cells. In the early stage of Type 2 diabetes, the predominant abnormality is insulin resistance.

At first, high blood glucose is not observed because  $\beta$ -cells can compensate for insulin resistance by increasing insulin secretion or  $\beta$ -cell mass, but insufficient compensation ultimately leads to the onset of Type 2 diabetes.

The major complications of both types of diabetes include cardiovascular disease – not least the high risk of heart attack, blindness and renal failure. While this report touches briefly on Type 1 diabetes, its focus is on Type 2, which is subsequently referred to as just diabetes in Sections 2-5 of this report. Although much more research is still needed on the role environmental chemicals may play in Type 2 diabetes, data relating to Type 1 diabetes are even more scant.

## The Issue:

### Obesity:

The evidence that chemical exposures can affect weight gain in animals is compelling.

The term “environmental obesogens” refers to man-made chemicals that can disrupt normal controls over the formation of fat and energy balance. Chemicals implicated in causing weight gain have been identified both in experiments on animals and in cell-based studies, and include a variety of chemicals with diverse physical and chemical properties such as persistent organic pollutants (POPs – e.g., dioxins, polychlorinated biphenyls (PCBs) and certain organochlorine pesticides (OCPs), perfluorinated chemicals (PFCs) and brominated flame retardants (BFRs)), bisphenol A (BPA), organotins, diethylstilbestrol (DES), phthalates, organophosphate pesticides, lead, pre-natal nicotine exposure, diesel exhaust and some antipsychotic drugs.

It is therefore likely that there are other chemicals in the environment that increase the risk of obesity, which have yet to be recognised.

### Diabetes:

Type 2 diabetes is characterised by the body becoming more resistant to the action of the hormone insulin (which is secreted by the pancreas and works to balance the body’s glucose levels) and pancreatic-cell insufficiency. It is particularly alarming that the incidence of Type 2 diabetes is increasing in young people as well as in the older generations.

Type 1 diabetes is due to an immune attack on insulin-producing cells in the pancreas; it is characterised by low or absent endogenous insulin and has a peak age of onset during childhood.

While some researchers have tentatively suggested that both Type 1 and Type 2 may represent a spectrum of disease, the CHEM Trust report focuses on the role of environmental chemicals in Type 2 diabetes. This is because little information is available on the relationship between human contamination with chemicals and the risk of Type 1 diabetes.

Possible candidate environmental diabetogenic agents include POPs (such as dioxins, PCBs, some organochlorine pesticides and some brominated flame retardants), arsenic, BPA, some phthalates, organotins and organophosphate and carbamate pesticides.

It should be noted that diabetes itself has not been caused in animals exposed to these chemicals in laboratory studies, but metabolic disruption closely related to the pathogenesis of Type 2 diabetes has been reported for many chemicals.

### Developed markets:

While substantial laboratory evidence shows chemicals can affect weight gain in animals and therefore supports the hypothesis that EDCs (Endocrine disrupting properties of chemicals) promote

or otherwise influence obesity, the evidence in humans is still limited. When human studies are classified into in utero vs. adult exposures, the former studies were prospective and mainly focused on persistent chemicals while the latter studies were cross-sectional or prospective and dealt with persistent or non-persistent chemicals.

Findings from epidemiological studies on the effects of in utero exposure to environmental pollutants on body weight and size varied from negative to positive associations, depending on the chemical. Some studies have reported positive associations with inadvertent exposure to chemicals. For example, in utero exposure to organochlorine pesticides such as DDE or hexachlorobenzene has been associated with future obesity, but other studies did not replicate these findings. Also, positive associations tended to be different in subgroups, particularly by gender. Mixed results have also been reported for PCB exposure in relation to body mass index (BMI). Smoking in pregnancy has been associated with giving birth to offspring more likely to put on excess weight as they grow up. To the best of our knowledge, as yet there has been no study in humans on the effects of in utero exposure to non-persistent chemicals, such as BPA or phthalates. Given the ubiquitous exposure of pregnant women to these chemicals, such studies are now warranted.

As mentioned before, differences and inconsistencies in results by gender or other characteristics are to be expected when different risk factors are measured in the studies under comparison, and when different measured and unmeasured interactions influence the outcome of interest.

The interpretation of cross-sectional studies showing associations between serum concentrations of persistent chemicals such as POPs and adiposity is problematic because adiposity itself delays the metabolism of these chemicals and prolongs their half-lives. However, there are data which strongly support cross-sectional findings of a relationship between POPs and obesity. For example, one prospective study of 90 subjects who were diabetes-free during 18 years of follow-up observed that some POPs (including p,p'-DDE and PCBs) predicted the future risk of obesity. It is important to note that the dose-response curves between serum concentrations of some POPs and BMI were exactly inverted U-shaped: as serum concentrations of POPs at the baseline increased, BMI increased until a critical low dose; above this dose, BMI did not increase, and it even started to decrease as serum concentration of POPs increased. This shape of the association confirms what had been expected from experimental studies on EDCs in animals. Another prospective study among the elderly reported positive associations between levels of the less chlorinated PCBs, p,p'-DDE or dioxins and abdominal obesity, while the highly chlorinated PCBs inversely predicted future risk of abdominal obesity.

Concerning non-persistent but ubiquitous compounds, some metabolites of phthalates were positively associated with adiposity, even though the associations were also different depending on gender and age. However, the concentrations of phthalates in serum or urine primarily reflect recent exposure, making the interpretation of cross-sectional findings more difficult.

Even though population-based studies in humans are essential to confirm the relevance of environmental obesogens, testing hypotheses on the relationships between chemical exposures and obesity in humans is particularly difficult because of the major roles that both diet and physical activity play in obesity.

## Key Events

Event/Date	Explanation
1975-	Worldwide prevalence of obesity tripled until today
2000-	Number of overweight children in Africa has increased by 50% until today
2004	WHO publishes the "WHO Global Strategy on Diet, Physical Activity and Health", which underlines the actions needed to support healthy diets and regular physical activity
2011	<ul style="list-style-type: none"> <li>• The United Nations General Assembly on the Prevention and Control of Noncommunicable Diseases recognizes the critical importance of reducing unhealthy diet and physical inactivity</li> <li>• "Global Action Plan for the Prevention and Control of Noncommunicable Diseases 2013-2020" with the aim to achieve greater awareness of the problem is developed (see Previous attempts to solve the Issue for details)</li> </ul>
2012	Cardiovascular diseases (heart diseases & stroke) were the leading death cause
2016	<ul style="list-style-type: none"> <li>• more than 1.9 (39%) billion adults (18+) were overweight, 650 million (13%) were obese</li> <li>• 41 million children (under 5) were overweight/obese</li> <li>• 340 million children (5-19) were overweight/obese</li> <li>• half of the children under 5 who were overweight/obese lived in Asia</li> <li>• WHO Assembly welcomed the report of the Commission of Ending Childhood Obesity and its 6 recommendations</li> </ul>
2017	WHO welcomed the implementation plan to guide countries to act and implement recommendations of the Commission of Ending Childhood Obesity



## Previous Attempts to Solve the Issue

### WHO Global Strategies on Diet, Physical Activity and Health (2004)

In 2004, the WHO published this paper that describes the actions needed to support healthy diets and regular physical activity. This paper calls upon all members to take action on local, regional and global level to improve not only diets, but especially also physical activity patterns throughout the whole population.

### The Political Declaration of the High-Level Meeting of the United Nations General Assembly on the Prevention and Control of Noncommunicable Diseases (September 2011)

This declaration recognizes the critical importance of reducing unhealthy diet and physical inactivity, and is committed to advancing the implementation of the “*WHO Global Strategy on Diet, Physical Activity and Health*”. This includes, where possible, the introduction of policies and actions aimed at promoting both healthy diets and increasing physical activity.

### Global Action Plan for the Prevention and Control of Noncommunicable Diseases 2013-2020

This action plan’s aim is to achieve the commitment of the UN Political Declaration of Noncommunicable (chronic) diseases (NCDs). The plan will further contribute to progress on 9 global targets to fight NCDs by 2025, as well as a 25% relative reduction of premature mortality from NCDs by 2025 a halt in the raise of the global obesity rate.

### Commission of Ending Childhood Obesity (2016)

The WHO welcomed this report and its 6 concrete recommendations to address critical periods in the life course to tackle childhood obesity. The report featured an implementation plan to guide countries in acting against obesity and to implement the recommendations of the Commission. This plan was welcomed in 2017.

## Bibliography

“Diabetes Programme.” *World Health Organization*, World Health Organization, 13 Nov. 2018, [www.who.int/diabetes/en/](http://www.who.int/diabetes/en/).

James, W. Philip T. “Response to the Draft WHO Report on Diet, Nutrition and the Prevention of Chronic Disease.” International Obesity Taskforce.

“Obesity and Overweight.” *World Health Organization*, World Health Organization, [www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight](http://www.who.int/en/news-room/fact-sheets/detail/obesity-and-overweight).

“Our Policy Priorities.” *World Obesity Federation | Home*, [www.worldobesity.org/what-we-do/our-policy-priorities](http://www.worldobesity.org/what-we-do/our-policy-priorities).

“3rd International Conference on Obesity, Nutrition and Endocrinology.” *International Conference on Obesity & Diabetes - 2018 | Obesity Events | Obesity Meetings on May 14-16, 2018 at Melbourne, Australia*, [www.obesityconferences.org/](http://www.obesityconferences.org/).