

Heavy Metal-Induced Beta Cell Dysfunction: A Converging Pathway to Type 2 Diabetes and COVID-19 Complications

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Abstract

Emerging evidence indicates that chronic exposure to environmental toxicants, particularly cadmium and lead, may play a significant role in the development and exacerbation of Type 2 Diabetes (T2D). This review highlights that 100% of analysed fast food samples contained cadmium at levels ranging from 74% to over 1100% higher than the U.S. EPA's safety limit for drinking water, suggesting that fast food is a substantial and under recognized source of cadmium exposure. Such exposure is particularly concerning in communities experiencing a "double burden" of high fast-food density and limited access to healthier dietary options. Cadmium and lead have both been shown to impair pancreatic beta cell function through oxidative stress, inflammation, mitochondrial dysfunction, and interference with insulin secretion and signalling pathways. These mechanisms contribute directly to insulin resistance and beta cell apoptosis, increasing the risk of T2D onset and progression. Furthermore, these metals compete with essential elements such as zinc, further disrupting glucose metabolism.

The COVID-19 pandemic has revealed a dangerous synergy between diabetes and viral infection. SARS-CoV-2 targets ACE2 receptors on beta cells, leading to cellular damage, insulin deficiency, and acute hyperglycaemia. In individuals with pre-existing diabetes or those already exposed to cadmium or lead, this interaction may intensify beta cell dysfunction and increase the severity of COVID-19 outcomes. These findings strengthen the hypothesis that environmental exposure to cadmium and lead not only contributes to metabolic disease but also enhances vulnerability to infectious agents such as SARS-CoV-2. The evidence underscores the need for targeted public health strategies to reduce toxic metal exposure, especially in high-risk populations, and calls for further interdisciplinary research into the combined effects of environmental toxicants and viral infections on metabolic health.

Keywords: Cadmium exposure; Lead toxicity; Type 2 diabetes; Pancreatic beta cells; COVID-19

Introduction

According to the findings, every single fast-food sample that was analyzed-100% of them-was found to contain cadmium at concentrations significantly exceeding safe thresholds. Specifically, the cadmium levels detected ranged from 74% to an astonishing 1158% higher than the maximum limit permitted by the U.S. Environmental Protection Agency (EPA) for cadmium in drinking water [1]. This suggests that frequent consumption of fast food may be a substantial and under recognized source of cadmium exposure.

Moreover, communities that experience what is referred to as a "double burden"-where there is a high density of fast-food restaurants and a simultaneous lack of healthier or more diverse dining options-could be at a particularly heightened risk for developing chronic diseases, such as type 2 diabetes [2]. In these areas, limited food choices may force individuals to rely heavily on fast food, increasing their exposure to harmful substances like cadmium. Scientific research has established a link between cadmium exposure and a greater likelihood of developing type 2 diabetes. Numerous studies have observed a positive correlation between

elevated cadmium levels in the body and an increased risk of this metabolic disorder. Although researchers are still working to fully understand the biological mechanisms involved, current evidence suggests that cadmium may disrupt the body's ability to regulate glucose and impair insulin sensitivity [3,4].

In particular, cadmium has been shown to have a toxic effect on pancreatic beta cells, the specialized cells responsible for producing and secreting insulin. Damage to these cells can result in diminished insulin production, thereby contributing to the onset and progression of type 2 diabetes. This toxic metal may, therefore, play a significant role in the metabolic dysfunction associated with excessive fast-food consumption. Exposure to lead (Pb) is associated with an increased risk of diabetes, particularly Type 2 Diabetes (T2D). Lead can disrupt glucose uptake, contribute to insulin resistance, and potentially impact glycaemic control, thus increasing the risk of developing diabetes and complicating existing diabetes [5,6].

Discussion

Cadmium's effects on beta cells are as follows

Disrupts lipid metabolism: Cadmium exposure can lead to an elevation of pro-inflammatory lipids, impacting insulin secretion and potentially worsening diabetes.

Induces oxidative stress: Cadmium exposure can cause oxidative stress in beta cells, leading to damage and impaired function.

Induces inflammation: Cadmium can trigger inflammation in pancreatic beta cells, contributing to their dysfunction and potentially leading to cell death.

Mitochondrial dysfunction: Cadmium can disrupt mitochondrial function, which is crucial for insulin secretion, potentially leading to impaired glucose homeostasis.

Impairs insulin secretion: Studies have shown that cadmium exposure can lead to decreased insulin secretion from pancreatic beta cells.

May induce apoptosis: Cadmium exposure can trigger apoptosis (programmed cell death) in beta cells, further reducing their number and function.

Cadmium role in diabetes development

Type 2 diabetes: Cadmium's impact on beta cells, particularly their ability to secrete insulin, can contribute to the development of type 2 diabetes, where the body either doesn't produce enough insulin or can't effectively use it.

Exacerbates diabetes: In individuals with pre-existing diabetes, cadmium exposure may worsen the condition by further impairing beta cell function.

Competition with zinc: Cadmium can interfere with zinc, an essential element for beta cell function, potentially disrupting zinc-dependent processes.

Disrupting insulin signalling: Cadmium may affect insulin signalling pathways, impacting how cells respond to insulin and take up glucose.

Altering gene expression: Cadmium can alter the expression of genes involved in beta cell function and glucose metabolism [7-26]. COVID-19 infection primarily modulates immune and inflammatory responses, and may cause a cytokine storm, resulting in possible lethal outcomes in diabetics. An experimental report suggests that ACE expressed in the pancreas and the SARS-CoV-2 virus invariably destroy β -cells which contain ACE-2 receptors and results in acute diabetes. Moreover, COVID-19 also causes hyperglycaemia in an individual with diabetes which may be related to insulin resistance and destruction of β -cells during SARS-CoV-2 infection [27]. A hypothesis stated that COVID-19 is a Man-made pandemic: Lead and Cadmium mutate Influenza virus and Produce: SARS COV-2 [28].

Lead Pb: Lead (Pb) exposure can negatively impact pancreatic beta cells, potentially contributing to the development of diabetes. Lead can interfere with insulin release and glucose metabolism and may also trigger inflammation and oxidative stress within these cells [29].

Influenza: People with diabetes face a higher risk of severe influenza complications, including hospitalization and even death, even when their diabetes is well-managed. The flu can also make it more difficult to control blood sugar levels in individuals with diabetes [30].

Conclusion

Current evidence highlights a concerning link between environmental toxicants-particularly cadmium and lead-and the development and progression of type 2 diabetes. Cadmium disrupts pancreatic beta cell function through oxidative stress, inflammation, mitochondrial damage, and impaired insulin secretion. Similarly, lead exposure interferes with glucose metabolism and insulin regulation. These mechanisms not only increase the risk of developing diabetes but can also worsen outcomes in individuals with existing metabolic disorders.

Importantly, diabetes significantly heightens vulnerability to infectious diseases, including COVID-19. SARS-CoV-2 targets ACE2 receptors expressed in pancreatic beta cells, leading to beta cell destruction, insulin deficiency, and acute hyperglycaemia. This viral impact may exacerbate or even trigger diabetes in infected individuals. Furthermore, COVID-19-related cytokine storms and immune dysregulation are more severe in those with pre-existing diabetes, increasing the risk of complications and mortality. The overlapping effects of environmental toxins and viral infection suggest a compounded threat to beta cell health and metabolic regulation. These findings underscore the need for integrated public health strategies that address environmental exposures and enhance metabolic resilience, particularly during global health crises like the COVID-19 pandemic. The role of the anti-aging gene Sirtuin 1 is critical to the prevention of type 2 diabetes and vulnerability to COVID-19. Xenobiotics in food, air and water may

inactivate Sirtuin1 with relevance to the severity of COVID-19 and type 2 diabetes [31-33]. The findings presented strongly strengthen the hypothesis that chronic exposure to toxic metals such as cadmium and lead contributes to the development of type 2 diabetes and may intensify the impact of viral infections like COVID-19. Scientific evidence demonstrates that both cadmium and lead impair pancreatic beta cell function, reduce insulin secretion, and promote insulin resistance-key factors in the onset and progression of diabetes.

Moreover, the intersection of diabetes and COVID-19 reveals a dangerous synergy. SARS-CoV-2 infects beta cells through ACE2 receptors, leading to their destruction and worsening glycaemic control. In individuals already exposed to cadmium or lead, the combined effects may accelerate beta cell dysfunction and immune dysregulation, contributing to more severe COVID-19 outcomes. These findings support the hypothesis that environmental exposure to cadmium and lead plays a critical role in both metabolic disease and susceptibility to viral infection. They also highlight the urgent need for further research and public health measures to reduce toxic metal exposure and protect at-risk populations, particularly in communities disproportionately reliant on fast food or with limited access to healthy dietary options.

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