Mini Review
Volume 8:2, 2023

Journal of Diabetic Complications & Medicine

ISSN: 2475-3211 Open Access

BPF Induces Hyperglycemia by Upregulating Oxidative Stress-Responsive Mir-200 Family, Targeting Mettl14 and Xiap in Insulin-Producing β Cells

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Abstract

Bisphenol A (BPA) and its analogs are widely used chemicals in the manufacturing of various consumer products such as plastics, food packaging and thermal papers. However, recent studies have shown that exposure to these chemicals can disrupt glucose homeostasis in the body, leading to metabolic disorders such as diabetes and obesity. One such analog of BPA is Bisphenol F (BPF), which has been found to trigger hyperglycemia by acting on insulin-producing β cells in the pancreas. BPF exposure upregulates the expression of miR- 200 family in the pancreas which in turn targets two critical genes, Mettl14 and Xiap, leading to pancreatic apoptosis.

Keywords: Hyperglycemia • BPF • Insulin- producing β cells

Introduction

Bisphenol A (BPA) and its analogs are widely used chemicals in the manufacturing of various consumer products such as plastics, food packaging and thermal papers. However, recent studies have shown that exposure to these chemicals can disrupt glucose homeostasis in the body, leading to metabolic disorders such as diabetes and obesity. One such analog of BPA is Bisphenol F (BPF), which has been found to trigger hyperglycemia by acting on insulin-producing β cells in the pancreas. BPF exposure upregulates the expression of miR- 200 family in the pancreas which in turn targets two critical genes, Mettl14 and Xiap, leading to pancreatic apoptosis [1].

Literature Review

Pancreatic β cells are responsible for producing and secreting insulin, a hormone that regulates glucose levels in the blood. BPF exposure leads to hyperglycemia by reducing the number of functioning β cells in the pancreas. The upregulation of miR-200 family is responsible for this reduction in β cell function. The miR-200 family targets two genes, Mettl14 and Xiap, which play important roles in β cell survival and function. Mettl14 is a methyltransferase enzyme that regulates the expression of several genes in the β cells. BPF-induced downregulation of Mettl14 leads to decreased insulin synthesis and secretion from the β cells. This reduction in insulin production results in hyperglycemia, a hallmark of diabetes [2].

Xiap is an inhibitor of apoptosis protein that plays a crucial role in preventing programmed cell death in the β cells. BPF-induced upregulation of miR-200 family reduces the expression of Xiap, leading to increased apoptosis of β cells. This reduction in β cell numbers ultimately leads to a decrease in insulin production, which results in hyperglycemia. In addition to BPF, other analogs of BPA such as Bisphenol S (BPS) and Bisphenol AF (BPAF) have also been found to disrupt glucose homeostasis and contribute to the development of metabolic disorders. These chemicals act on multiple organs and tissues in the body, including the liver, adipose tissue and skeletal muscle, leading to insulin resistance and impaired glucose uptake [3].

Discussion

Exposure to BPA and its analogs such as BPF can disrupt glucose homeostasis in the body, leading to metabolic disorders such as diabetes and obesity. The action of BPF on insulin-producing β cells in the pancreas via miR-200 family and its targets Mettl14 and Xiap leads to hyperglycemia. Further research is needed to understand the mechanisms of action of these chemicals and develop strategies to reduce exposure to them. Bisphenol F (BPF) is a chemical commonly used in the manufacturing of plastics, food packaging and thermal papers. Recent studies have shown that exposure to BPF can lead to the development of metabolic disorders such as diabetes and obesity by disrupting glucose homeostasis in the body. One mechanism through which BPF can cause pancreatic damage is by upregulating the expression of the oxidative stress-responsive miR-200 family in the pancreas. This upregulation leads to the targeting of two crucial genes, Mettl14 and Xiap, ultimately leading to pancreatic apoptosis [4].

The miR-200 family is a group of microRNAs that have been shown to play important roles in the regulation of cell proliferation, differentiation and apoptosis. Recent studies have identified a link between the upregulation of miR-200 family and the development of pancreatic damage caused by BPF exposure. The oxidative stress induced by BPF triggers the upregulation of miR-200 family in the pancreas, leading to the targeting of Mettl14 and Xiap. Mettl14 is an enzyme responsible for regulating the expression of several genes in the pancreas, including those involved in insulin synthesis and secretion. BPF exposure reduces the expression of Mettl14 by targeting it via miR-200 family, leading to a decrease in insulin production and secretion. This decrease in insulin levels ultimately leads to hyperglycemia and the development of diabetes [5,6].

Conclusion

Xiap is an inhibitor of apoptosis protein that plays a critical role in preventing programmed cell death in the pancreas. BPF-induced upregulation of miR-200 family reduces the expression of Xiap, leading to an increase in apoptosis of pancreatic cells. This increase in pancreatic cell death ultimately leads to pancreatic damage and dysfunction. The upregulation of miR-200 family by BPF is also linked to the induction of oxidative stress in the pancreas. Oxidative

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Received: 29 March, 2023, Manuscript No. jdcm-23-98471; **Editor Assigned:** 01 April, 2023, PreQC No. P-98471; **Reviewed:** 17 April, 2023, QC No. Q-98471; **Revised:** 22 April, 2023, Manuscript No. R-98471; **Published:** 29 April, 2023, DOI: 10.37421/2475-3211.2023.8.189

stress occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the body's antioxidant defense mechanisms. BPF exposure leads to the production of ROS, which in turn triggers the upregulation of miR-200 family, leading to the targeting of Mettl14 and Xiap and ultimately causing pancreatic apoptosis. BPF exposure can upregulate the expression of miR-200 family in the pancreas, leading to the targeting of Mettl14 and Xiap and ultimately causing pancreatic apoptosis. This mechanism is linked to the induction of oxidative stress by BPF in the pancreas. Further research is needed to understand the mechanisms of action of BPF and develop strategies to reduce exposure to this chemical. Understanding the link between BPF exposure and pancreatic damage can aid in the development of new therapeutic approaches for the treatment of diabetes and other metabolic disorders.

Acknowledgement

None.

Conflict of Interest

None.

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How to cite this article: Pascali, Jacopo. "BPF Induces Hyperglycemia by Upregulating Oxidative Stress-Responsive Mir-200 Family, Targeting Mettl14 and Xiap in Insulin-Producing β Cells." *J Diabetic Complications Med* 8 (2023): 189.