

Gripshover, T. C., Wahlang, B., Head, K. Z., Young, J. L., Luo, J., Mustafa, M. T., Kirpich, I. A., & Cave, M. C. (2023). The environmental pollutant, polychlorinated biphenyl 126, alters liver function in a rodent model of alcohol-associated liver disease. *Alcohol Clinical and Experimental Research*, 47(1), 60-75. <https://doi.org/10.1111/acer.14976>

Definitions

- **Polychlorinated biphenyl (PCB) 126:** A harmful chemical pollutant that doesn't break down easily in the environment.
- **Alcohol-associated liver disease (ALD):** Liver damage caused by drinking too much alcohol.
- **Hepatic steatosis:** Build-up of fat in the liver.
- **Hepatomegaly:** Enlarged liver.
- **Glycogen:** A form of stored glucose in the liver and muscles.
- **Aryl hydrocarbon receptor (Ahr):** A protein that binds pollutants and can change how genes are expressed.

Key Findings

- PCB 126 worsens liver damage in mice that also consume alcohol.
- PCB 126 increases fat and decreases sugar storage in the liver.
- PCB 126 and alcohol together lead to more severe liver damage than either alone.
- Changes in liver function due to PCB 126 and alcohol include increased fat storage, reduced fat breakdown, and lower blood sugar levels.

Introduction

This study investigates how the pollutant PCB 126 affects liver disease caused by alcohol consumption. Alcohol-associated liver disease (ALD) is a major health issue, and PCB 126 is a toxic chemical that may worsen this condition. Understanding these interactions is crucial for public health.

Main Content

Background

Alcohol-associated liver disease (ALD) is a type of liver damage that happens because of excessive alcohol intake. It can start as fat build-up in the liver (steatosis) and progress to more serious conditions like inflammation and scarring. PCB 126 is a harmful pollutant that doesn't break down easily and can accumulate in the environment. It has been linked to various health problems, including liver damage.

Methods

- **Animal Model:** Male C57BL/6J mice were used.
- **Chemical Exposure:** Mice were given 0.2 mg/kg PCB 126 or corn oil (control) by oral gavage.
- **Diet:** Four days after chemical exposure, mice were fed an ethanol-containing diet using a chronic-binge model.
- **Group Division:** Mice were divided into four groups:
 - Pair-fed + vehicle
 - Pair-fed + PCB 126
 - Ethanol-fed + vehicle
 - Ethanol-fed + PCB 126
- **Measurement of Liver Function:**
 - Liver weight was recorded.
 - Hepatic triglycerides and glycogen levels were measured.
 - Gene expression analysis was performed using qRT-PCR.
 - Blood chemistry was analyzed for liver enzymes, lipids, glucose, and insulin levels.

Results

- **Liver Weight:**
 - Ethanol feeding increased liver weight.
 - PCB 126 exposure further increased liver weight in ethanol-fed mice.
- **Hepatic Triglycerides:**
 - Elevated in ethanol-fed groups.
 - Highest in the ethanol + PCB 126 group.
- **Glycogen Levels:**
 - Decreased in both PCB 126 and ethanol-fed groups.
 - Lowest in the ethanol + PCB 126 group.
- **Gene Expression Changes:**
 - Increased expression of Cd36 (fatty acid translocase) due to PCB 126 and ethanol.
 - Decreased expression of Alb (albumin), Fabp1 (fatty acid-binding protein), Fasn (fatty acid synthase), Scd-1 (stearoyl-CoA desaturase 1), and Srebf1 (sterol regulatory element-binding transcription factor 1) in ethanol + PCB 126 group.
- **Blood Chemistry:**
 - Increased plasma triglycerides in ethanol-fed groups.

- Decreased glucose levels in the ethanol + PCB 126 group.
- Decreased insulin levels in the ethanol + PCB 126 group.
- **Immune Response and Inflammation:**
 - Increased granulocyte infiltration in ethanol-fed groups.
 - Elevated markers of endoplasmic reticulum stress and apoptosis in liver tissue.

Conclusion

The study concluded that PCB 126 worsens the effects of alcohol on the liver, leading to increased fat accumulation and disrupted sugar metabolism. This combination of pollutants and lifestyle factors can significantly harm liver health. The findings highlight the need for awareness and regulation of environmental pollutants to protect against compounded health risks.

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