

Bassler, J., Ducatman, A., Elliott, M., Wen, S., Wahlang, B., Barnett, J., & Cave, M. C. (2019). Environmental perfluoroalkyl acid exposures are associated with liver disease characterized by apoptosis and altered serum adipocytokines. *Environmental Pollution*, 247, 1055-1063.

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Definitions

- **Perfluoroalkyl acids (PFAAs):** Man-made chemicals used in products like non-stick cookware and water-repellent fabrics.
- **Apoptosis:** A process of programmed cell death.
- **Serum adipocytokines:** Proteins released by fat cells into the blood that can signal other parts of the body.
- **Non-alcoholic fatty liver disease (NAFLD):** A liver condition not caused by alcohol, where fat builds up in the liver.

Key Findings

- Exposure to PFAAs is linked to liver disease.
- PFAAs cause liver cell death (apoptosis) and alter fat cell signaling proteins in the blood.
- PFAAs reduce some inflammatory responses in the body.

Introduction

This study explores how exposure to certain man-made chemicals, called perfluoroalkyl acids (PFAAs), affects liver health. PFAAs are found in many everyday products and can accumulate in the environment and human body. The research focuses on how these chemicals might contribute to liver diseases, particularly non-alcoholic fatty liver disease (NAFLD).

Main Content

Background

PFAAs are synthetic chemicals widely used in consumer products for their resistance to heat, water, and grease. These chemicals are persistent in the environment and have been found to accumulate in human bodies. Concerns have arisen about their potential health impacts, particularly on liver function, as studies have linked PFAA exposure to increased liver enzymes and liver disease.

Methods

- **Participants:** 200 adults from the C8 Health Study, aged 40-70, with varied body mass index (BMI) and a mix of African American and Caucasian individuals.
- **Exclusions:** Participants with hepatitis, excessive alcohol consumption (>3 drinks/day).

- **Sample Storage:** Serum samples stored at -80°C since 2006.
- **Biomarkers Measured:**
 - **Exposure biomarkers:** Levels of PFOA, PFOS, PFHxS, PFNA in serum.
 - **Disease biomarkers:** CK18 M30 and CK18 M65 (liver cell death), TNF α , IL-6, IL-8, IFN γ (inflammatory proteins), adiponectin, insulin, leptin (fat-related proteins), C3a, and PAI-1.
- **Analysis:**
 - **Statistical tools:** SAS 9.4 and R software.
 - **Adjustments for confounders:** Age, sex, BMI, alcohol consumption, and kidney function (eGFR).
 - **Data transformation:** Natural log transformation for normality.
 - **Cut-off determination:** Classification and regression tree (CART) analysis for biomarker levels.

Results

- **Liver Cell Death:**
 - Higher PFAA levels were associated with increased CK18 M30 and CK18 M65, indicating more liver cell death.
- **Inflammatory Response:**
 - TNF α levels were significantly lower with higher PFAA exposure.
 - IL-8 levels were lower with increased PFOS and PFNA exposure.
 - IFN γ levels were higher with PFOA and PFNA exposure.
- **Adipocytokines:**
 - Adiponectin levels were higher with PFHxS, PFOS, and PFNA exposure.
 - Leptin levels were higher with PFHxS and PFNA exposure.
- **Sex Differences:**
 - Different responses in inflammatory and adipocytokine biomarkers between males and females, particularly with IL-8, IFN γ , and C3a.

Conclusion

The study shows that PFAA exposure is linked to liver damage and changes in inflammatory and fat-related proteins in the blood. These findings underscore the potential health risks of PFAAs and the need for further research to understand their impact on liver diseases like NAFLD. Additionally, the differences observed between males and females suggest that sex may play a role in how these chemicals affect the body.

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