

Casual Friday Series

# FM Perspectives on Body Fat

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(Lifestyle + Genetics) x Time = Chronic Health Outcomes



# POP's, Destroyers of Physiology

Many POPs were widely used during the boom in industrial production after World War II, when thousands of synthetic chemicals were introduced into commercial use. Many of these chemicals proved beneficial in pest and disease control, crop production, and industry. These same chemicals, however, have had unforeseen effects on human health and the environment.

Many people are familiar with some of the most well-known POPs, such as PCBs, DDT, and dioxins. POPs include a range of substances that include:

1. Intentionally produced chemicals currently or once used in agriculture, disease control, manufacturing, or industrial processes. Examples include PCBs, which have been useful in a variety of industrial applications (e.g., in electrical transformers and large capacitors, as hydraulic and heat exchange fluids, and as additives to paints and lubricants) and DDT, which is still used to control mosquitoes that carry malaria in some parts of the world.



[Environ Health Perspect](#). 2013 Feb; 121(2): 162–169.

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Review

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## Toxicological Function of Adipose Tissue: Focus on Persistent Organic Pollutants

[Michele La Merrill](#),<sup>1</sup> [Claude Emond](#),<sup>2,3</sup> [Min Ji Kim](#),<sup>4,5,6,7</sup> [Jean-Philippe Antignac](#),<sup>8</sup> [Bruno Le Bizec](#),<sup>8</sup>  
[Karine Clément](#),<sup>9,10,11,12</sup> [Linda S. Birnbaum](#),<sup>13,14</sup> and [Robert Barouki](#)<sup>4,5,6</sup>

**Discussion:** As a storage compartment for lipophilic POPs, AT plays a critical role in the toxicokinetics of a variety of drugs and pollutants, in particular, POPs. By sequestering POPs, AT can protect other organs and tissues from POPs overload. However, this protective function could prove to be a threat in the long run. The accumulation of lipophilic POPs will increase total body burden. These accumulated POPs are slowly released into the bloodstream, and more so during weight loss. Thus, AT constitutes a continual source of internal exposure to POPs. In addition to its buffering function, AT is also a target of POPs and may mediate part of their metabolic effects. This is particularly relevant because many POPs induce obesogenic effects that may lead to quantitative and qualitative alterations of AT. Some POPs also induce a proinflammatory state in AT, which may lead to detrimental metabolic effects.

## Case 1: 57-year-old female

| TESTS  | RESULT       | FLAG        | UNITS  | REFERENCE INTERVAL | LAB |
|--|--------------|-------------|--------|--------------------|-----|
| AST (SGOT)                                       | 30           |             | IU/L   | 0-40               | 01  |
| <b>ALT (SGPT)</b>                                | <b>37</b>    | <b>High</b> | IU/L   | 0-32               | 01  |
| GGT  | 42           |             | IU/L   | 0-60               | 01  |
| Iron Bind.Cap. (TIBC)                            | 280          |             | ug/dL  | 250-450            |     |
| UIBC   | 204          |             | ug/dL  | 131-425            | 01  |
| Iron   | 76           |             | ug/dL  | 27-159             | 01  |
| Iron Saturation                                  | 27           |             | %      | 15-55              |     |
| <b>Ferritin, Serum</b>                           | <b>293</b>   | <b>High</b> | ng/mL  | 15-150             | 01  |
| Cholesterol, Total                               | 172          |             | mg/dL  | 100-199            | 01  |
| Triglycerides                                    | 43           |             | mg/dL  | 0-149              | 01  |
| HDL Cholesterol                                  | 66           |             | mg/dL  | >39                | 01  |
| VLDL Cholesterol Cal                             | 9            |             | mg/dL  | 5-40               |     |
| LDL Cholesterol Calc                             | 97           |             | mg/dL  | 0-99               |     |
| <hr/>  |              |             |        |                    |     |
| Homocyst(e)ine                                   | 11.4         |             | umol/L | 0.0-14.5           | 01  |
| <b>**Please note reference interval change**</b> |              |             |        |                    |     |
| <b>TSH</b>                                       | <b>0.008</b> | <b>Low</b>  | uIU/mL | 0.450-4.500        | 01  |
| Thyroxine (T4)                                   | 8.9          |             | ug/dL  | 4.5-12.0           | 01  |
| T3 Uptake  | 39           |             | %      | 24-39              | 01  |
| Free Thyroxine Index                             | 3.5          |             |        | 1.2-4.9            |     |
| Triiodothyronine (T3)                            | 155          |             | ng/dL  | 71-180             | 01  |
| <b>Triiodothyronine (T3), Free</b>               | <b>4.7</b>   | <b>High</b> | pg/mL  | 2.0-4.4            | 01  |
| Reverse T3, Serum <sup>A</sup>                   | 22.5         |             | ng/dL  | 9.2-24.1           | 02  |

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**FINAL REPORT**

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## Serum Polychlorinated Biphenyls Increase and Oxidative Stress Decreases with a Protein-Pacing Caloric Restriction Diet in Obese Men and Women

Feng He,<sup>1,2,†</sup> Li Zuo,<sup>3,†</sup> Emery Ward,<sup>1</sup> and Paul J. Arciero<sup>1,\*</sup>

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### 6.1. Weight Loss, Phase 1 (WL; P-CR, Weeks 0–12)

#### PCBs and Oxidative Stress Markers

PCBs were reported as organic pollutants which have toxic effects on the human body such as endocrine disruption and neurotoxicity [18]. As such, we performed an extensive blood panel (CBC, TSH, bilirubin, ALT, alkaline phosphatase) on all study participants and all values were within normal ranges. There was no difference at the baseline level (week-12) as well as the post intervention (week-64) for any of the hematology parameters between P-CR and HH participants (Table S1). Consistent with previous studies [17,24,25,30], we found levels of total PCBs in serum increased after 12 weeks of WL intervention in both sexes. Therefore, there are two aspects of WL in humans; the benefits include the decrease of adipose tissue mass whereas the possible harms are due to the increase of serum concentrations of lipophilic chemicals like PCBs.

However, there may be time differences for these two opposite aspects to reveal their biological effects in humans. In particular, the improved redox status of marked reduction of TBARS and an increase in TAC after Phase 1 WL period despite the increase of PCBs may not be surprising. For this perspective, possible benefits due to the decrease of adipose tissue mass may be more immediate, but possible harms due to increased lipophilic chemicals may need more time. WL via diet intervention is a novel approach to study the effect of PCBs on human health. Hitherto, few other studies investigated the relationship between oxidative stress and PCBs following WL in humans. In the present study, 12 weeks of a P-CR intervention suppressed lipid peroxidation (i.e., TBARS) and boosted total antioxidant capacity equally in obese men and women.



## Case 2: 66-year-old female

### Ordered Items

CMP14+LP+TP+TSH+5AC+CBC/D/P...; Insulin; Venipuncture; Cardiovascular Report

| TESTS                                 | RESULT  | FLAG        | UNITS | REFERENCE INTERVAL | LAB |
|---------------------------------------|---|-------------|-------|--------------------|-----|
| <b>CMP14+LP+TP+TSH+5AC+CBC/D/P...</b> |   |             |       |                    |     |
| Chemistries                           |   |             |       |                    | 01  |
| <b>Glucose</b>                        | <b>322</b>                                      | <b>High</b> | mg/dL | 65-99              | 01  |
| <b>Hemoglobin A1c</b>                 | <b>11.8</b>                                     | <b>High</b> | %     | 4.8-5.6            | 01  |
| Please Note:                          |   |             |       |                    | 01  |
|                                       | Prediabetes: 5.7 - 6.4                          |             |       |                    |     |
|                                       | Diabetes: >6.4                                  |             |       |                    |     |
|                                       | Glycemic control for adults with diabetes: <7.0 |             |       |                    |     |

| TESTS                     | RESULT   | FLAG        | UNITS | REFERENCE INTERVAL | LAB |
|---------------------------|--|-------------|-------|--------------------|-----|
| AST (SGOT)                | 17   |             | IU/L  | 0-40               | 01  |
| ALT (SGPT)                | 18   |             | IU/L  | 0-32               | 01  |
| GGT                       | 28   |             | IU/L  | 0-60               | 01  |
| Iron Bind.Cap. (TIBC)     | 290  |             | ug/dL | 250-450            |     |
| UIBC                      | 225  |             | ug/dL | 118-369            | 01  |
| Iron                      | 65   |             | ug/dL | 27-139             | 01  |
| Iron Saturation           | 22   |             | %     | 15-55              |     |
| <b>Ferritin, Serum</b>    | <b>188</b>   | <b>High</b> | ng/mL | 15-150             | 01  |
| <b>Cholesterol, Total</b> | <b>260</b>   | <b>High</b> | mg/dL | 100-199            | 01  |
| <b>Triglycerides</b>      | <b>506</b>   | <b>High</b> | mg/dL | 0-149              | 01  |
| HDL Cholesterol           | 47   |             | mg/dL | >39                | 01  |
| VLDL Cholesterol Cal      |  |             | mg/dL | 5-40               |     |
|                           | The calculation for the VLDL cholesterol is not valid when triglyceride level is >400 mg/dL. |             |       |                    |     |
| LDL Cholesterol Calc      |  |             | mg/dL | 0-99               |     |
|                           | Triglyceride result indicated is too high for an accurate LDL cholesterol estimation.        |             |       |                    |     |

|                |      |  |        |          |    |
|----------------|------|--|--------|----------|----|
| <b>Insulin</b> | 22.6 |  | uIU/mL | 2.6-24.9 | 01 |
|----------------|------|--|--------|----------|----|





Case 2: 66-year-old female

| TESTS   | RESULT       | FLAG        | UNITS  | REFERENCE INTERVAL | LAB |
|---|--------------|-------------|--------|--------------------|-----|
| Free Thyroxine Index  | 2.1          |             |        | 1.2-4.9            |     |
| Triiodothyronine (T3)   | 139          |             | ng/dL  | 71-180             | 01  |
| Triiodothyronine (T3), Free   | 3.4          |             | pg/mL  | 2.0-4.4            | 01  |
| Reverse T3, Serum <sup>A</sup>  | 18.1         |             | ng/dL  | 9.2-24.1           | 02  |
| T4, Free (Direct)   | 1.23         |             | ng/dL  | 0.82-1.77          | 01  |
| <b>Thyroid Peroxidase (TPO) Ab</b>                                    | <b>57</b>    | <b>High</b> | IU/mL  | 0-34               | 01  |
| <b>Thyroglobulin Antibody</b>   | <b>1.0</b>   | <b>High</b> | IU/mL  | 0.0-0.9            | 01  |
| <b>Thyroglobulin Antibody measured by Beckman Coulter Methodology</b> |              |             |        |                    |     |
| <b>C-Reactive Protein, Cardiac</b>                                    | <b>11.97</b> | <b>High</b> | mg/L   | 0.00-3.00          | 01  |
| <b>Relative Risk for Future Cardiovascular Event</b>                  |              |             |        |                    |     |
| <b>Low</b>  |              |             |        | <b>&lt;1.00</b>    |     |
| <b>Average</b>  |              |             |        | <b>1.00 - 3.00</b> |     |
| <b>High</b>   |              |             |        | <b>&gt;3.00</b>    |     |
| Homocyst(e)ine  | 6.5          |             | umol/L | 0.0-17.2           | 01  |
| <b>**Please note reference interval change**</b>                      |              |             |        |                    |     |
| TSH   | 4.280        |             | uIU/mL | 0.450-4.500        | 01  |
| Thyroxine (T4)  | 8.1          |             | ug/dL  | 4.5-12.0           | 01  |
| T3 Uptake   | 26           |             | %      | 24-39              | 01  |



## Serum Polychlorinated Biphenyls Increase and Oxidative Stress Decreases with a Protein-Pacing Caloric Restriction Diet in Obese Men and Women

Feng He,<sup>1,2,†</sup> Li Zuo,<sup>3,†</sup> Emery Ward,<sup>1</sup> and Paul J. Arciero<sup>1,\*</sup>

### 7. Conclusions

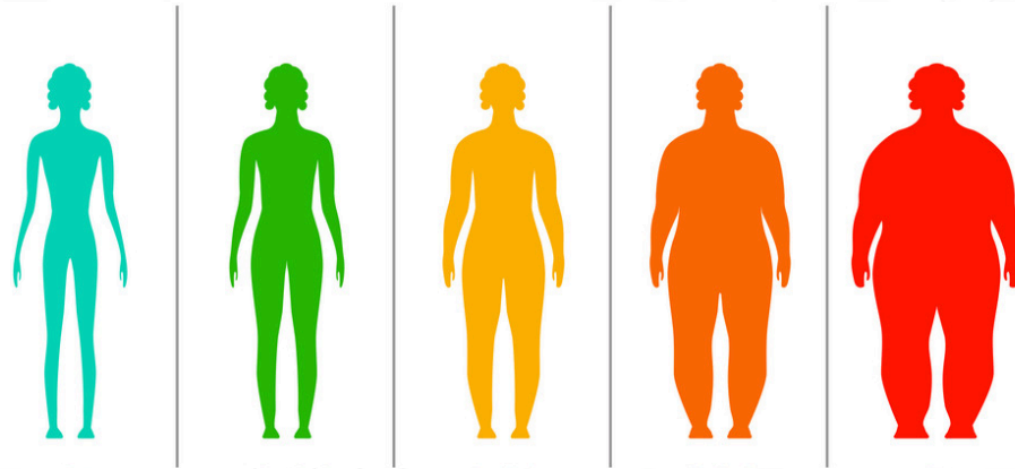
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In summary, a 12-week P-CR diet effectively induced WL, favorably altered redox status, and increased circulating levels of serum PCBs to a similar extent in both obese women and men. Moreover, we provide novel findings showing that a 52-week mP-CR intervention prevents weight relapse in the absence of adverse effects (i.e., increased oxidative stress, abnormal blood panel) induced by elevated PCBs compared to a traditional HH diet. The current study demonstrates that a P-CR nutritional intervention, when coupled with close observation of compliance and dietary counseling, should be regarded as an effective dietary plan to mobilize stored PCBs and improve redox status. Future studies should investigate the mechanistic pathway (e.g., oxidative stress mediated pathway) of WL-induced PCB elevations and subsequent elimination from the body. Effective strategies such as the combination of proper nutrition and exercise should be designed in order to maintain the improved body composition and redox status along with the mobilization of stored PCBs during WL.





# Body fat accumulation is an adaptation response.



(Lifestyle + Genetics) x Time = Chronic Health Outcomes



(Lifestyle + Genetics) x Time = Chronic Health Outcomes



- Energy vs Expenditure
- Intake vs Clearance
- Longevity Vs Comfort



# Fat loss is an *adaptation* response.



(Lifestyle + Genetics) x Time = Chronic Health Outcomes











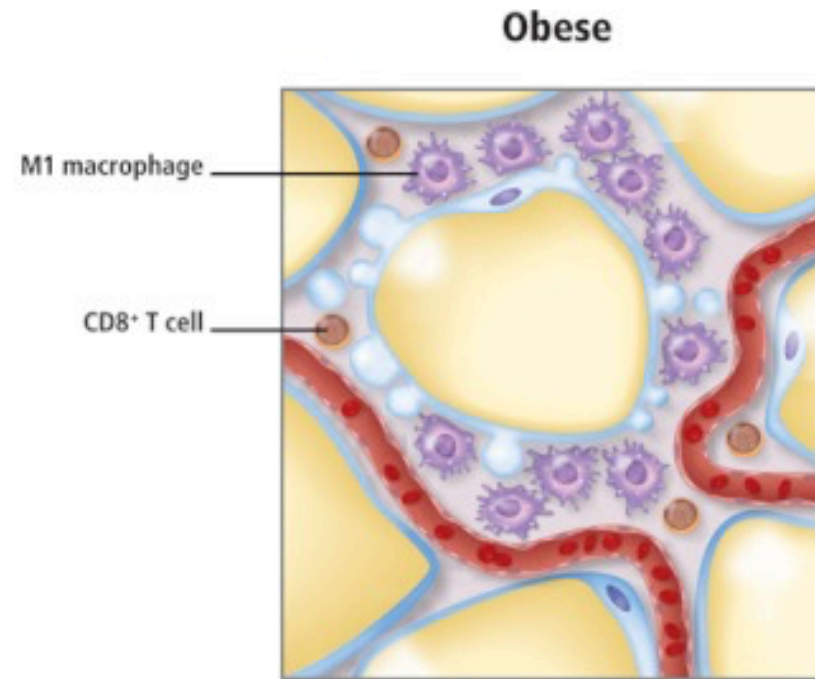
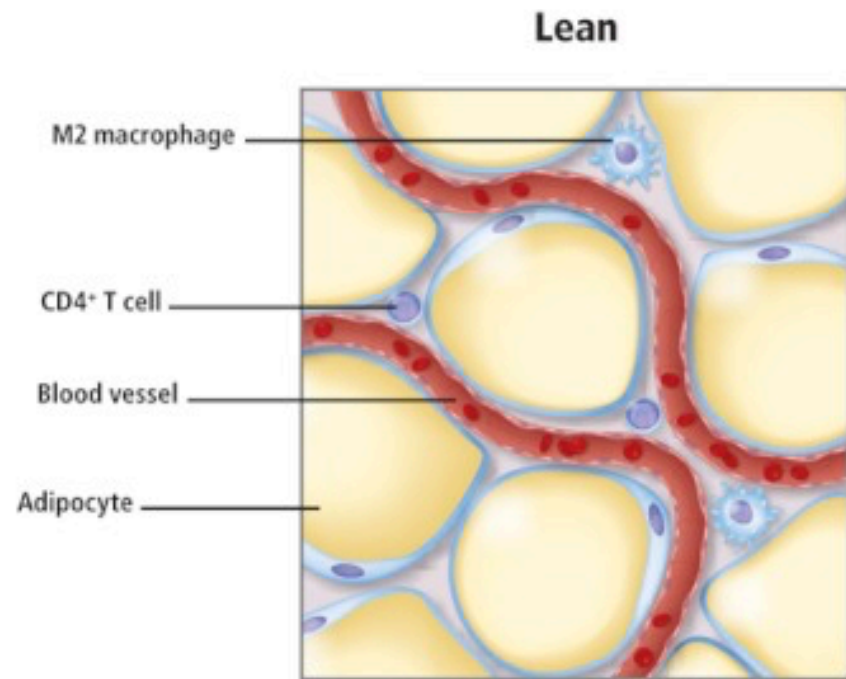
# POPs vs. Fat: Persistent Organic Pollutant Toxicity Targets and Is Modulated by Adipose Tissue

Julia R. Barrett

Published: 1 February 2013 | <https://doi.org/10.1289/ehp.121-a61> | Cited by: 4

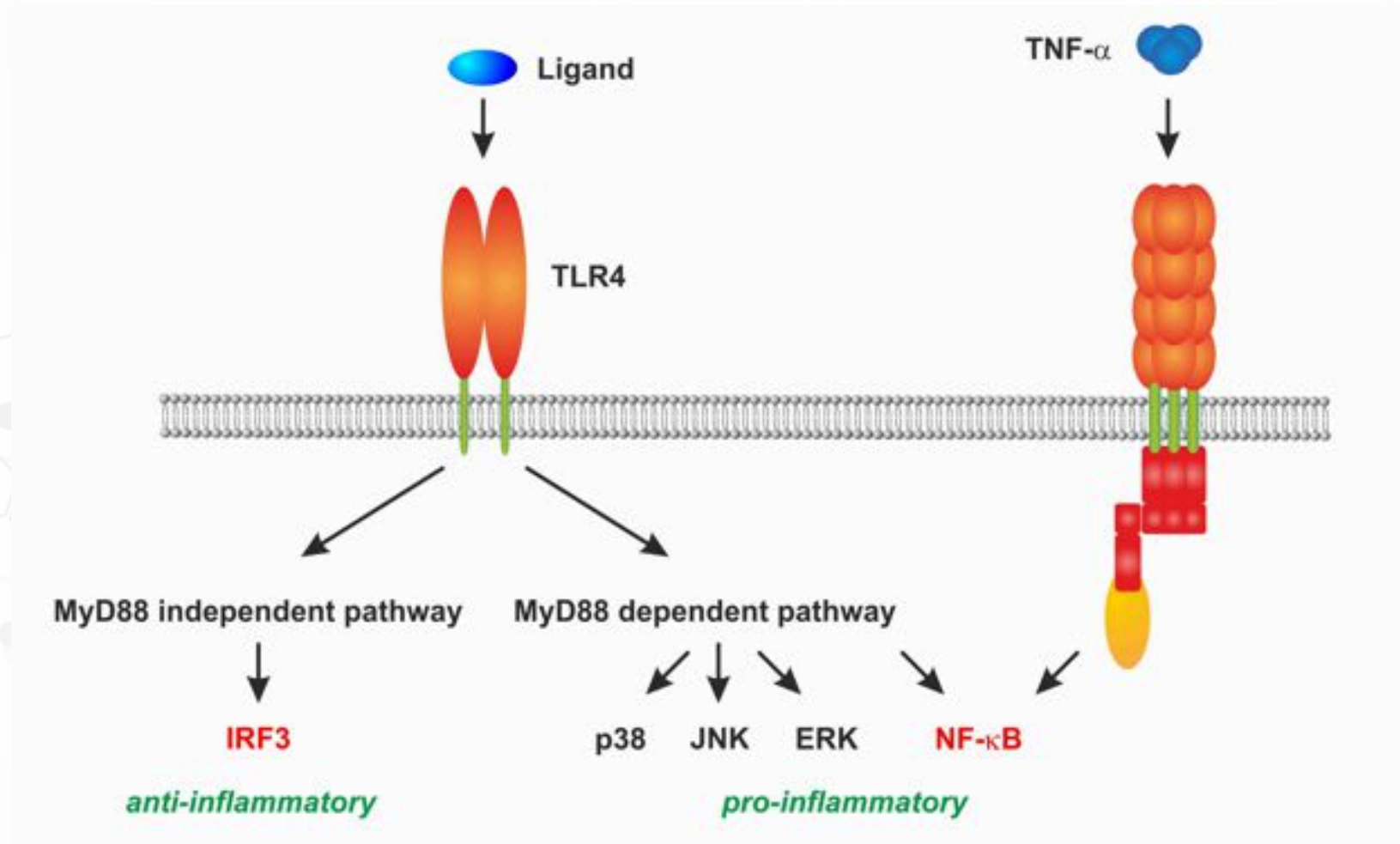
Adipose tissue itself may experience toxic effects, especially if exposure occurs within critical windows of susceptibility, such as during prenatal, early postnatal, or pubertal development. Developmental exposure could redirect gene expression, with effects that may not become apparent until later in life. This mechanism, among other possibilities, could explain how several POPs may act as obesogens, compounds that increase the risk of obesity, itself a risk factor for diabetes, liver and cardiovascular diseases, and cancer. Furthermore, recent studies show that POPs provoke an inflammatory state in adipose tissue, a condition associated with the metabolic side effects of obesity. POPs also appear to have a role in lipotoxicity, the accumulation of lipids in nonadipose tissues, leading to metabolic dysfunction characteristic of cardiovascular disease and heart disease.





<https://ehp.niehs.nih.gov/doi/10.1289/ehp.121-a61>





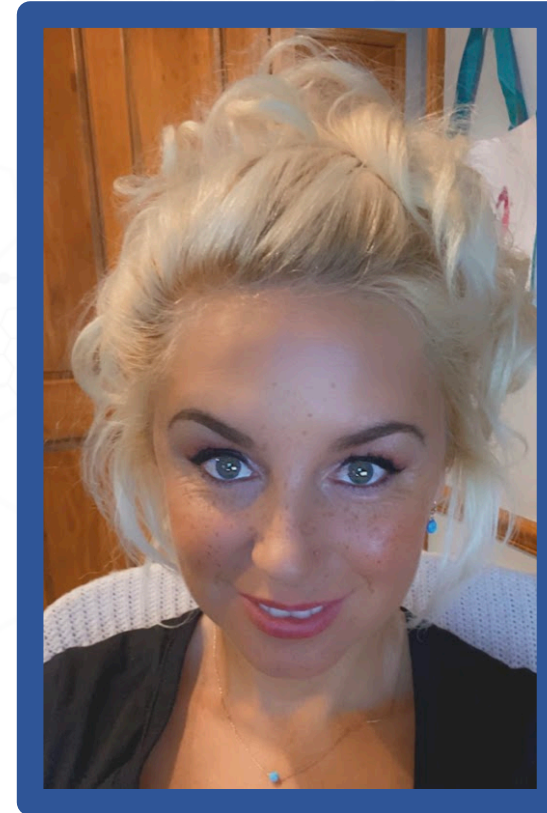
# 21 – Day Metabolic Clearing Program



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[zeb@biogenetix.com](mailto:zeb@biogenetix.com)



[kim@biogenetix.com](mailto:kim@biogenetix.com)

