# Supplementary Material

Supplementary Material is provided online.

**Supplementary material 1: systematic review description**

**New Prisma diagram in separate file**

In accordance with the EFSA guidance, this review question was described in terms of four key items: population (P), exposure (E), comparator (C) and outcome (O) (PECO). A combination of search terms for exposure and outcome with the Boolean operator “AND” was used. To identify available data to answer this review question, a literature search was performed in Scopus and Pubmed with the following search terms: triphenyl phosphate, diphenyl phosphate, hydroxyl-triphenyl phosphate and hydroxyl-diphenyl phosphate in the title, abstract and keywords, and limited to studies published until 5 July 2021 in PubMed and Scopus. A complementary search was run in PubMed and Scopus on 3 September 2021 to include additional metabolites of TPP; namely, meta-hydroxy-phenyl diphenyl phosphate, para-hydroxy-phenyl diphenyl phosphate, mono-hydroxyl triphenyl phosphate, di-hydroxyl triphenyl phosphate and monophenyl phosphate. For the outcomes, the following search terms were used for obesity: “obesogen\*”, “obesit\*”, “obesity”, “adiposity” and “adipose tissue”. For the outcomes receptor, the following search terms were used: “receptor\*”, “receptor protein-tyrosine kinases”, “receptor activity-modifying proteins”, “receptor-like protein tyrosine phosphatases”, “receptors, cell surface”, "receptor activity-modifying proteins", "nuclear receptor interacting protein 1", "nuclear receptor coactivators" and"insulin receptor substrate proteins". For omics, the following search terms were used: "genom\*", "genomics", "glucom\*", "glycom\*", "glycomics", "metabolom\*", "metabolomics", "omic\*", "proteom\*", "proteomics", "transcriptom\*" and "transcriptome". Some of the research string have been created by identifying the most appropriate MeSH terms, based on the National Center of Biotechnology Information MeSH Classification (https://www.ncbi.nlm.nih.gov/mesh/). For population (P), screening the abstracts and the full-text of the studies allowed us to identify the relevant experimental animal or human studies. Considering the limited number of terms searched for the outcomes obesity, the following additional terms like “fat” or “fat pad” for the *in vivo* literature and “fat”, “overweight”, “BMI” or “body mass index” for human studies might have retrieved more relevant literature.

### Selection of publications using inclusion and exclusion criteria

Relevant studies were selected in a two-tiered approach using the inclusion and exclusion criteria described in Table below**Error! Reference source not found.** In Tier 1, the title and abstracts were screened. Studies judged as relevant in Tier 1 were carried forward to Tier 2 where the full-text of the studies was examined. The CADIMA tool (https://www.cadima.info) was used for screening in Tier 1 and 2. As CADIMA takes only exclusion criteria with 3 possible answers: *Yes*, *Unclear* or *No*, when the answer to the exclusion criteria was *Unclear* or *No*, the article was excluded. Two reviewers were involved. Possible disagreements were resolved after discussion involving a third reviewer. Then, the studies were exported in an EndNote database. 37 publications were considered for further evaluation and appraisal.

**Exclusion and inclusion criteria table:**

|  |
| --- |
| **Exclusion and inclusion criteria.** |
| ***Tier 1 Inclusion and exclusion criteria (based on title, abstracts and keywords:*** |
| *Iterative exclusion criteria:* |
| * Is the article purely editorial (letter to editor, response to an article)? * Is the article specific to an effect of TPP or its metabolite? * Is the article related to human exposure assessment or human risk assessment only? * Is the article related to metabolic disruption effect? * Is the article related to obesity? * Is the article related to a case report? |
| *Iterative inclusion criteria:* |
| * Is the article related to *in vivo*/*ex vivo* rodents, or *in vivo* zebrafish, or *in vitro* study, or human epidemiological study, or *in silico* (e.g. molecular modelling or QSAR), or Adverse Outcome Pathway (AOP) development, or omics (proteomics, metabolomics, … )? * Is the article a review on obesity/adipogenesis? |
| ***Tier 2 Inclusion criteria (based on full texts):*** |
| *Iterative inclusion criteria:* |
| * *Is the article written in English?* * *Are the specific effects of TPP or a metabolite identifiable?* * *Does the article deal with obesity?* |
| *Exclusion criteria:* |
| * *Has the article been retracted after its initial publication?* |

***Filtering step:***

For receptor and omics literature search, a filtering step was applied in EndNote on any field with PDF and notes of the articles, thanks to the integrated filtering tool. For omics, a sequential filtering was applied with a first filter with “NMR”, “MS”and “Mass Spectrometry” and then with “metabolom”, “lipidom”, “lipid”, “steroidom”, “steroïdom”, “chemometric” or “metabolic finger print”. For receptor, the following filters were used “PPAR gamma”, “PPAR-gamma”, “PPARγ”, “Peroxisome proliferator-activated receptor-γ”, “PPAR-γ”, “Peroxisome proliferator-activated receptor gamma”, “pparg” or “peroxisome proliferator-activated receptor-gamma”.

**Supplementary material 2: literature references selected based on title and abstract reading (n=37):**

(Alam et al. 2010; Alam et al. 2012; Boyle, Buckley, and Quirós-Alcalá 2019; Cano-Sancho, Smith, and La Merrill 2017; Q. Chen et al. 2021; Cui et al. 2020; Fang et al. 2015; Green et al. 2017; Gu et al. 2019; Hao et al. 2019; Hoffman et al. 2017; Hogberg et al. 2021; Hu et al. 2017; Kassotis, Hoffman, and Stapleton 2017; Kim et al. 2020; Kim et al. 2021; Kojima et al. 2013; Lockhart et al. 1975; D. Luo, Liu, et al. 2020; K. Luo, Zhang, et al. 2020; Mitchell et al. 2018; Negi et al. 2021; Nicole 2014; Pillai et al. 2014; Reddam et al. 2019; Rock et al. 2020; Scanlan et al. 2015; Selmi-Ruby et al. 2020; Soubry et al. 2017; Tung, Ahmed, et al. 2017; Tung, Peshdary, et al. 2017; Van den Eede, Cuykx, et al. 2015; D. Wang et al. 2019; D. Wang, Zhu, et al. 2018; X. Wang et al. 2020; Y. Wang et al. 2021; Y. Zhang et al. 2020)

**Supplementary material 3: literature references selected based on full-text reading (n=17)**

(Boyle, Buckley, and Quirós-Alcalá 2019; Cano-Sancho, Smith, and La Merrill 2017; Cui et al. 2020; Fang et al. 2015; Green et al. 2017; Hao et al. 2019; Hoffman et al. 2017; Hu et al. 2017; Kassotis, Hoffman, and Stapleton 2017; Kim et al. 2020; Kojima et al. 2013; D. Luo, Liu, et al. 2020; Pillai et al. 2014; Tung, Ahmed, et al. 2017; Tung, Peshdary, et al. 2017; D. Wang et al. 2019; D. Wang, Zhu, et al. 2018)

**Supplementary material 4: identification of additional potentially relevant publications for TPP evaluation or for the establishment of the biological plausible link(n=59)**:

(Arukwe, Carteny, and Eggen 2016; Belcher et al. 2014; Brooke et al. 2009; Butt et al. 2014; Butt et al. 2016; Carignan et al. 2013; Cequier et al. 2015; G. Chen et al. 2015; Cooper et al. 2011; Du et al. 2016; Fang, Webster, and Stapleton 2015; Farhat et al. 2013; Fu et al. 2005; Grapov et al. 2012; Grün and Blumberg 2006; Grün et al. 2006; He et al. 2018; Heindel et al. 2017; Heindel et al. 2015; Honkakoski et al. 2004; Hu et al. 2017; Janesick and Blumberg 2011; Jin, Chen, and Fu 2016; Kim et al. 2015; Krivoshiev et al. 2016; Krumm et al. 2018; La Merrill and Birnbaum 2011; Lefterova et al. 2014; Liu, Ji, and Choi 2012; Lu et al. 2017; Ma et al. 2018; McGee et al. 2013; Meeker and Stapleton 2010; Morris et al. 2014; Niemelä et al. 2008; Papalou et al. 2019; Patisaul et al. 2013; Preston et al. 2017; Romano et al. 2017; Rosen et al. 2002; Rosen and MacDougald 2006; Saillenfait et al. 2018; Schang, Robaire, and Hales 2016; Su et al. 2014; Suzuki et al. 2013; Takahashi, Kamei, and Ezaki 2005; Tontonoz et al. 1994; Tontonoz and Spiegelman 2008; Van den Eede, Heffernan, et al. 2015; van der Veen and de Boer 2012; Vrang et al. 2010; L. Wang, Huang, et al. 2018; Q. Wang et al. 2015; Yan et al. 2017; Q. Zhang et al. 2016; Q. Zhang et al. 2014; Q. Zhang et al. 2017; Zoeller et al. 2012; Wang Y. 2016).

**Supplementary material 5: publications selected among the additional potentially relevant publications for TPP evaluation or for the establishment of the biological plausible link (n=21):**

(Belcher et al. 2014; G. Chen et al. 2015; Fang, Webster, and Stapleton 2015; Grün and Blumberg 2006; Grün et al. 2006; Heindel et al. 2017; Heindel et al. 2015; Honkakoski et al. 2004; Janesick and Blumberg 2011; Kim et al. 2015; Lefterova et al. 2014; Ma et al. 2018; Papalou et al. 2019; Rosen et al. 2002; Su et al. 2014; Suzuki et al. 2013; Takahashi, Kamei, and Ezaki 2005; Tontonoz et al. 1994; Tontonoz and Spiegelman 2008; Vrang et al. 2010; Wang Y. 2016)

**Supplementary material 6: TPP corpus data set**

|  |  |  |
| --- | --- | --- |
| **Literature reference** | **Study types** | **Final allocation** |
| (Belcher et al. 2014) | *In vitro* | Question 2 |
| (Boyle, Buckley, and Quirós-Alcalá 2019) | Epidemio | Question 1 |
| (Cano-Sancho, Smith, and La Merrill 2017) | *In vitro* | Question 2 |
| (G. Chen et al. 2015) | *In vivo* | Question 1 |
| (Cui et al. 2020) | *In vivo* | Question 2 |
| (Fang et al. 2015) | *In vitro* | Question 2 |
| (Fang, Webster, and Stapleton 2015) | *In vitro* | Question 2 |
| (Green et al. 2017) | *In vivo* | Question 1 and question 2 |
| (Hao et al. 2019) | *In vitro* & *in silico* | Question 2 & in silico |
| (Hoffman et al. 2017) | Epidemio | Question 1 |
| Honkakoski et al., 2004 | *In vitro* | Not kept in question 2 since PPARγ was not investigated |
| (Hu et al. 2017) | *In vitro* & *in silico* | Question 2 & in silico |
| (Kassotis, Hoffman, and Stapleton 2017) | *In vitro* | Question 2 |
| (Kim et al. 2020) | *Iin vivo* & omics | Question 1and question 2 &omics |
| (Kojima et al. 2013) | *In vitro* | Question 2 |
| (D. Luo, Liu, et al. 2020) | Epidemio | Question 1 |
| (Pillai et al. 2014) | *In vitro* & *in silico* | Question 2 & in silico |
| (Romano et al. 2017) | Epidemio | Question 1 |
| (Su et al. 2014) | *In vitro* | Question 2 |
| (Suzuki et al. 2013) | *In vitro* | Question 2 |
| (Tung, Ahmed, et al. 2017) | *In vitro* | Question 2 |
| (Tung, Peshdary, et al. 2017) | *In vitro* | Question 2 |
| (Y. Wang et al. 2016) | *In vitro* | Question 2 |
| (D. Wang, Zhu, et al. 2018) | *In vivo* | Question 1  and question 2 |
| (D. Wang et al. 2019) | Omics and in vivo | Question 1  and Question 2 &omics |

**Supplementary material 7: Grid of evaluation used during the preliminary analysis of in silico, in vitro, in vivo, omics and epidemiological studies**

See: Archives\Tableaux Figures\Grid of evaluation\_Goliath.xlsx

**Supplementary material 8: Arguments (either in favour, against or reflecting uncertainties) used by Group 1 to answer question 1**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 1** | Moderate evidence from animal data (3 *in vivo* studies) that TPP promotes weight increase and adipogenicity. | For | *In vivo* animals | 7 |
| Epidemiological data weak or non-relevant. | Against | Epidemio | 6 |
| Two relevant epidemiological studies but reliability issues. | Uncertainty | Epidemio | 5 |
| Omics evidence is weak (Reported effects not discussing obesity markers/weight gain/adipogenesis etc.; too few replicates). | Against | Omics | 4 |
| Effects observed *in vivo* after fœtal exposure different from adult exposure | For | *In vivo* animals | 3 |
| Not enough epidemiological studies. | Against | Epidemio | 2 |
| The 2 relevant epidemiological studies come to different conclusions. | Against | Epidemio | 2 |
| Two *in vivo* studies with weak relevance: Chen et al., 2015; Green et al., 2017. | Against | *In vivo* animals | 2 |
| Omics data not appropriate to decipher on adverse effect. | Uncertainty | Omics | 2 |
| Careful study design to elucidate adverse transgenerational epigenetic effects. | Uncertainty | Not classified | 2 |

***Supplementary material 9:*** ***Arguments (either in favour, against or reflecting uncertainties) used by Group 2 to answer question 1***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 1** | Low evidence based on epidemiological studies. | Against | Epidemio | 8 |
| Epidemiological studies not directly comparable: difference in studied population, not the same studied endpoint (adiposity in Luo et al., 2020 and low body weight (LBW) in Boyle et al., 2019). | Uncertainty | Epidemio | 4 |
| Moderate to strong evidence from animal studies that TPP increased body weight (BW) or body fat mass (BFM). | For | *In vivo* animals | 4 |
| *In vivo* studies not particularly of high-quality/reliability/relevance. Kim study: ip, high doses, solvent. | Uncertainty | *In vivo* animals | 4 |
| Endpoints assessed well-related to obesity although not the same within all the dataset. | For | *In vivo* animals | 2 |
| Contradictory results in Wang D et al. (Environ Pollut 2019, increased BW (exacerbated with high-fat diet (HFD)), while Kim S. et al. (Arch Toxicol 2020) showed no increase in BFM with a standard diet and even reduced BFM in condition of HFD. | Against | *In vivo* animals | 2 |
| There is some inconsistency in the link between birth weight and obesity in adult (Zhao et al. 2012). | Uncertainty | Epidemio | 2 |

***Supplementary material 10: Arguments (either in favour, against or reflecting uncertainties) used by Group 1 to answer question 2***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 2** | TPP binds to PPARγ1. | For | Binding / activation PPARγ | 5 |
| Consistently demonstrated TPP effects in different cell lines in varied level of confidence. | For | *In vitro* / cell line | 4 |
| No *in vivo* study linking TPP to PPARγ activation and obesogenic effects. | Against | *In vivo* / hormone /adverse effect | 4 |
| Not sure that PPARγ activation is the only mechanism. | Uncertainty | Binding to / activation of PPARγ | 4 |
| *In silico* models confirm interaction of TPP with PPARγ (docking). | For | *In silico*/molecular | 3 |
| Not all studies have proper control or description of methods. | Uncertainty | Control / transparency | 3 |
| Activation of PPARγ only at very high concentration and not sure it will be sufficient to activate the whole pathway. | Uncertainty | Binding to/ activation of PPARγ | 3 |
| Level of activation weaker than by known agonists. | Against | Binding to/ activation of PPARγ | 2 |
| One study on omics (Tung et al., 2017b) reliable with good overlap (83%) of genes activated by TPP and troglitazone. | For | Omics | 2 |
| Lack of reproducibility or reliability in omics studies. | Against | Omics | 2 |

***Supplementary material 11: Arguments (either in favour, against or reflecting uncertainties) used by Group 1 to answer question 2***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 2** | Overall, the studies are in agreement regarding the effects. | For | Overall | 5 |
| In the 3 *in vitro* studies that studied adipogenesis, the mechanisms were not examined in detail. | Uncertainty | *In vitro*/cell line | 4 |
| TPP interacts with PPARγ (molecular docking) in Hu W et al., 2017 in Environ Sci Technol I and Hao Z et al., 2019 in Chem Res Toxicol. | For | *In silico* /molecular | 3 |
| Large number of convincing data on a clear interaction between TPP and PPARγ in several species e.g. 'Fang papers and Wang 2016'. | For | *In vitro*/cell line | 3 |
| No relevant omic studies: no data about the expression of lipogenic genes/proteins associated with an activation of the PPARγ signalling pathway. | Uncertainty | Omics | 3 |
| Poor *in vivo* data. | Against | *In vivo* | 3 |
| TPP binds PPARγ in all studies. | For | Binding to /activation of PPARγ | 2 |
| Moderate to strong evidence that TPP activates PPARγ. | For | *In vitro*/cell line | 2 |
| Data do not allow to conclude regarding the mechanism and a potential action of TPP through PPARγ activation in the adipose tissue. | Uncertainty | Omics | 2 |
| High concentrations used *in vivo* studies. | Against | *In vivo* | 2 |
| Two relevant and well performed studies which agree in direction of effects (Wang 2018 - weak, Wang et al., 2019 moderate). | For | *In vivo* | 2 |
| Overall agreement, but not relevant to the involvement of PPARγ pathways. | Uncertainty | *In vivo* | 2 |

***Supplementary material 12: Arguments (either in favour, against or reflecting uncertainties) used by Group 1 to answer question 3***

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 3** | Lack of investigations to demonstrate if it is the only MoA for obesity induction in human. | Uncertainty | Link PPARγ activation and adverse effect | 6 |
| PPARγ is inducing obesity in human, well known rosiglitazone, troglitazone. | For | Link PPARγ activation and adverse effect | 5 |
| Lack of clinical trials to demonstrate the link in population level. | Uncertainty | Type of studies relevance | 4 |
| PPARγ involve in mechanism of obesity development but not the only way to induce obesity. | For | Link PPARγ activation and adverse effect | 4 |
| Clear links of PPARγ activation pathway and obesogenic effects, show cased with multiple levels of controls (positive, negative, knock-out). | For | Link PPARγ activation and adverse effect | 2 |
| Clear links of PPARγ and modification of glucose insulin pathway. | For | Link PPARγ and endocrine MoA | 2 |
| Other MoA could be involved. | Uncertainty | Link PPARγ activation and adverse effect | 2 |
| PPARγ activation is crucial for adipogenicity. | For | Link PPARγ activation and adverse effect | 2 |
| No large agreement in the scientific community concerning link between PPARγ and obesity. | Against | Link PPARγ activation and adverse effect | 2 |

**Supplementary material 13: Arguments (either in favour, against or reflecting uncertainties) used by Group 2 to answer question 3**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 3** | Evidence that PPARγ agonists may elicit energy metabolism (metabolomic effects/ body weights and key obesity markers in animals. | For | *In vivo*/ animal evidence (other than TPP) | 6 |
| Activation of PPARγ *per se* is insufficient to classify a compound as an obesogen. | Against | Specificity of mechanism /MoA | 6 |
| How relevant the data is to humans and how well the results in mice, *in silico* and *in vitro* can really be extended to meaningful weight gain in humans. | Uncertainty | Overall (other than TPP) | 6 |
| Moderate biological plausibility. | For | Specificity of mechanism /MoA | 5 |
| It is likely that other factors (e.g., hormonal regulation of satiety and appetite) play a role in the manifestation of the (apical) adverse outcome of obesity. | Uncertainty | Specificity of mechanism /MoA | 5 |
| Indirect or direct effects question still open. | Uncertainty | Specificity of mechanism /MoA | 3 |
| Similar metabolic shifts in obesity cohorts and in metabolomics approaches. | For | Human/epi evidence (other than TPP) | 2 |

**Supplementary material 14: Arguments (either in favour, against or reflecting uncertainties) used by Group 1 to answer question 4**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 4** | The PPAR signalling pathway is involved in adipogenesis and obesity, (specificity). | For | Specificity | 5 |
| Epidemiological evidence is limited. | Uncertainty | Consistency | 5 |
| Lacking relevant data that could be used for human. | Uncertainty | Consistency | 3 |
| Essentiality depending on the dose and period of exposure; not clearly evaluated in the studies. | Uncertainty | Essentiality | 3 |
| There are several mechanisms implicated, not only PPARγ activation. | For | Empirical support and quality | 2 |
| The relationship between TPP and lipid metabolism modulation is quite clear. | For | Empirical support and quality | 2 |
| Several studies are not relevant for generalization to the human population: the doses are often large compared to human exposure and cell/animal models. | Against | Relevance | 2 |
| Human relevance remains largely unclear based on the *in vitro*/*in vivo* data. | Against | Relevance | 2 |
| Not enough human *in vitro* models. Omics data are not informative; rat strain not a good one; not always on human models for *in vitro* studies; controversies between studies. | Uncertainty | Empirical support and quality | 2 |
| The fact that TPP effect on lipid metabolism is linked to PPARγ activation is not proved. | Against | Empirical support and quality | 2 |
| Less evidence for question 1. | Against | Consistency | 2 |
| TPP induces increase in body weight in dose dependent fashion, in mice exposed in utero or during lactation Wang 2019. | For | Dose response + temporal | 2 |

**Supplementary material 15: Arguments (either in favour, against or reflecting uncertainties) used by Group 2 to answer question 4**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Questions** | **Text** | **Category** | **Type of studies** | **Number of “like”** |
| **Question 4** | Lack of clear evidence from epidemiological studies (mostly due to lack of data). | Uncertainty | Empirical support and quality | 5 |
| Consistency: moderate: There is high consistency between rodent and human *in vitro* systems. | For | Consistency | 5 |
| Specificity weak /moderate: TPP may exert its effects via different mechanisms linked, for instance, with metabolic adaptation or increased food intake and/or reduced energy expenditure via alteration of hypothalamic peptidergic circuits. | Uncertainty | Specificity (ED/PPARγ) | 5 |
| Possible but not necessarily via direct PPARγ activation. | Uncertainty | Specificity (ED/PPARγ) | 4 |
| Essentiality strong: blocking PPARγ and inhibition of TPP binding prevent TPP effects (*in silico* question 2 activation PPARγ). | For | Essentiality | 3 |
| Specificity: strong evidence that PPARγ activation is related to obesity and TPP may act via PPARγ to induce adipogenesis. | For | Specificity (ED/PPARγ) | 3 |
| Animal data is moderate, role of PPARγ is strong from *in vitro* studies. | For | Empirical support and quality | 2 |
| How relevant the data is to humans and how well the results in mice, *in silico* and *in vitro* can really be extended to meaningful weight gain in humans. | Uncertainty | Overall/ general/ not classified | 2 |
| Moderate: question 2 and question 3 clearly indicate that TPP has the potentiality to cause obesity via PPARγ activation in humans. | For | Empirical support and quality | 2 |
| Analogy weak: clearly not sufficient, only ROZI *in vitro* / *in vivo*; no evidence of obesity via PPARγ for organic phosphate. | Against | Analogy | 2 |

References

Alam, T. M., M. K. Alam, M. Neerathilingam, D. E. Volk, S. Sarkar, G. A. S. Ansari, and B. A. Luxon. 2010. "1H NMR metabonomic study of rat response to tri-phenyl phosphate and tri-butyl phosphate exposure." *Metabolomics* 6 (3): 386-394. <https://doi.org/10.1007/s11306-010-0205-z>.

Alam, T. M., M. Neerathilingam, M. K. Alam, D. E. Volk, G. A. S. Ansari, S. Sarkar, and B. A. Luxon. 2012. "1H nuclear magnetic resonance (NMR) metabolomic study of chronic organophosphate exposure in rats." *Metabolites* 2 (3): 479-495. <https://doi.org/10.3390/metabo2030479>.

Arukwe, A., C. C. Carteny, and T. Eggen. 2016. "Lipid peroxidation and oxidative stress responses in juvenile salmon exposed to waterborne levels of the organophosphate compounds tris(2-butoxyethyl)- and tris(2-chloroethyl) phosphates." *J Toxicol Environ Health A* 79 (13-15): 515-25. <https://doi.org/10.1080/15287394.2016.1171978>.

Belcher, S. M., C. J. Cookman, H. B. Patisaul, and H. M. Stapleton. 2014. "In vitro assessment of human nuclear hormone receptor activity and cytotoxicity of the flame retardant mixture FM 550 and its triarylphosphate and brominated components." *Toxicol Lett* 228 (2): 93-102. <https://doi.org/10.1016/j.toxlet.2014.04.017>.

Brooke, D., M. Crookes, P. Quarterman, and J. Burns. 2009. "Environmental risk evaluation report: Triphenyl phosphate (CAS No. 115-86-6)." *Environment Agency, Bristol, UK*: 140.

Butt, C. M., J. Congleton, K. Hoffman, M. Fang, and H. M. Stapleton. 2014. "Metabolites of organophosphate flame retardants and 2-ethylhexyl tetrabromobenzoate in urine from paired mothers and toddlers." *Environ Sci Technol* 48 (17): 10432-8. <https://doi.org/10.1021/es5025299>.

Butt, C. M., K. Hoffman, A. Chen, A. Lorenzo, J. Congleton, and H. M. Stapleton. 2016. "Regional comparison of organophosphate flame retardant (PFR) urinary metabolites and tetrabromobenzoic acid (TBBA) in mother-toddler pairs from California and New Jersey." *Environ Int* 94: 627-634. <https://doi.org/10.1016/j.envint.2016.06.029>.

Carignan, C. C., M. D. McClean, E. M. Cooper, D. J. Watkins, A. J. Fraser, W. Heiger-Bernays, H. M. Stapleton, and T. F. Webster. 2013. "Predictors of tris(1,3-dichloro-2-propyl) phosphate metabolite in the urine of office workers." *Environ Int* 55: 56-61. <https://doi.org/10.1016/j.envint.2013.02.004>.

Cequier, E., A. K. Sakhi, R. M. Marcé, G. Becher, and C. Thomsen. 2015. "Human exposure pathways to organophosphate triesters - a biomonitoring study of mother-child pairs." *Environ Int* 75: 159-65. <https://doi.org/10.1016/j.envint.2014.11.009>.

Chen, G., Y. Jin, Y. Wu, L. Liu, and Z. Fu. 2015. "Exposure of male mice to two kinds of organophosphate flame retardants (OPFRs) induced oxidative stress and endocrine disruption." *Environ Toxicol Pharmacol* 40 (1): 310-8. <https://doi.org/10.1016/j.etap.2015.06.021>.

Chen, Q., X. Lian, J. An, N. Geng, H. Zhang, J. K. Challis, Y. Luo, Y. Liu, G. Su, Y. Xie, Y. Li, Z. Liu, Y. Shen, J. P. Giesy, and Y. Gong. 2021. "Life Cycle Exposure to Environmentally Relevant Concentrations of Diphenyl Phosphate (DPhP) Inhibits Growth and Energy Metabolism of Zebrafish in a Sex-Specific Manner." *Environ Sci Technol*. <https://doi.org/10.1021/acs.est.1c03948>.

Cooper, E. M., A. Covaci, A. L. van Nuijs, T. F. Webster, and H. M. Stapleton. 2011. "Analysis of the flame retardant metabolites bis(1,3-dichloro-2-propyl) phosphate (BDCPP) and diphenyl phosphate (DPP) in urine using liquid chromatography-tandem mass spectrometry." *Anal Bioanal Chem* 401 (7): 2123-32. <https://doi.org/10.1007/s00216-011-5294-7>.

Du, Z., Y. Zhang, G. Wang, J. Peng, Z. Wang, and S. Gao. 2016. "TPhP exposure disturbs carbohydrate metabolism, lipid metabolism, and the DNA damage repair system in zebrafish liver." *Sci Rep* 6: 21827. <https://doi.org/10.1038/srep21827>.

Fang, M., T. F. Webster, P. L. Ferguson, and H. M. Stapleton. 2015. "Characterizing the peroxisome proliferator-activated receptor (PPARγ) ligand binding potential of several major flame retardants, their metabolites, and chemical mixtures in house dust." *Environmental Health Perspectives* 123 (2): 166-172. <https://doi.org/10.1289/ehp.1408522>.

Fang, M., T. F. Webster, and H. M. Stapleton. 2015. "Activation of Human Peroxisome Proliferator-Activated Nuclear Receptors (PPARγ1) by Semi-Volatile Compounds (SVOCs) and Chemical Mixtures in Indoor Dust." *Environ Sci Technol* 49 (16): 10057-64. <https://doi.org/10.1021/acs.est.5b01523>.

Farhat, A., D. Crump, S. Chiu, K. L. Williams, R. J. Letcher, L. T. Gauthier, and S. W. Kennedy. 2013. "In Ovo effects of two organophosphate flame retardants--TCPP and TDCPP--on pipping success, development, mRNA expression, and thyroid hormone levels in chicken embryos." *Toxicol Sci* 134 (1): 92-102. <https://doi.org/10.1093/toxsci/kft100>.

Fu, M., T. Sun, A. L. Bookout, M. Downes, R. T. Yu, R. M. Evans, and D. J. Mangelsdorf. 2005. "A Nuclear Receptor Atlas: 3T3-L1 adipogenesis." *Mol Endocrinol* 19 (10): 2437-50. <https://doi.org/10.1210/me.2004-0539>.

Grapov, D., S. H. Adams, T. L. Pedersen, W. T. Garvey, and J. W. Newman. 2012. "Type 2 diabetes associated changes in the plasma non-esterified fatty acids, oxylipins and endocannabinoids." *PLoS One* 7 (11): e48852. <https://doi.org/10.1371/journal.pone.0048852>.

Green, A. J., J. L. Graham, E. A. Gonzalez, M. R. La Frano, S. E. Petropoulou, J. S. Park, J. W. Newman, K. L. Stanhope, P. J. Havel, and M. A. La Merrill. 2017. "Perinatal triphenyl phosphate exposure accelerates type 2 diabetes onset and increases adipose accumulation in UCD-type 2 diabetes mellitus rats." *Reprod Toxicol* 68: 119-129. <https://doi.org/10.1016/j.reprotox.2016.07.009>.

Grün, F., and B. Blumberg. 2006. "Environmental obesogens: organotins and endocrine disruption via nuclear receptor signaling." *Endocrinology* 147 (6 Suppl): S50-5. <https://doi.org/10.1210/en.2005-1129>.

Gu, J., F. Su, P. Hong, Q. Zhang, and M. Zhao. 2019. "(1)H NMR-based metabolomic analysis of nine organophosphate flame retardants metabolic disturbance in Hep G2 cell line." *Sci Total Environ* 665: 162-170. <https://doi.org/10.1016/j.scitotenv.2019.02.055>.

Hao, Z., Z. Zhang, D. Lu, B. Ding, L. Shu, Q. Zhang, and C. Wang. 2019. "Organophosphorus Flame Retardants Impair Intracellular Lipid Metabolic Function in Human Hepatocellular Cells." *Chemical Research in Toxicology* 32 (6): 1250-1258. <https://doi.org/10.1021/acs.chemrestox.9b00058>.

He, C., K. English, C. Baduel, P. Thai, P. Jagals, R. S. Ware, Y. Li, X. Wang, P. D. Sly, and J. F. Mueller. 2018. "Concentrations of organophosphate flame retardants and plasticizers in urine from young children in Queensland, Australia and associations with environmental and behavioural factors." *Environ Res* 164: 262-270. <https://doi.org/10.1016/j.envres.2018.02.040>.

Heindel, J. J., F. S. Vom Saal, B. Blumberg, P. Bovolin, G. Calamandrei, G. Ceresini, B. A. Cohn, E. Fabbri, L. Gioiosa, C. Kassotis, J. Legler, M. La Merrill, L. Rizzir, R. Machtinger, A. Mantovani, M. A. Mendez, L. Montanini, L. Molteni, S. C. Nagel, S. Parmigiani, G. Panzica, S. Paterlini, V. Pomatto, J. Ruzzin, G. Sartor, T. T. Schug, M. E. Street, A. Suvorov, R. Volpi, R. T. Zoeller, and P. Palanza. 2015. "Parma consensus statement on metabolic disruptors." *Environ Health* 14: 54. <https://doi.org/10.1186/s12940-015-0042-7>.

Hoffman, K., A. Lorenzo, C. M. Butt, L. Adair, A. H. Herring, H. M. Stapleton, and J. L. Daniels. 2017. "Predictors of urinary flame retardant concentration among pregnant women." *Environment International* 98: 96-101. <https://doi.org/10.1016/j.envint.2016.10.007>.

Hogberg, H. T., R. de Cássia da Silveira E Sá, A. Kleensang, M. Bouhifd, O. Cemiloglu Ulker, L. Smirnova, M. Behl, A. Maertens, L. Zhao, and T. Hartung. 2021. "Organophosphorus flame retardants are developmental neurotoxicants in a rat primary brainsphere in vitro model." *Archives of Toxicology* 95 (1): 207-228. <https://doi.org/10.1007/s00204-020-02903-2>.

Honkakoski, P., J. J. Palvimo, L. Penttilä, J. Vepsäläinen, and S. Auriola. 2004. "Effects of triaryl phosphates on mouse and human nuclear receptors." *Biochem Pharmacol* 67 (1): 97-106. <https://doi.org/10.1016/j.bcp.2003.08.037>.

Hu, W., F. Gao, H. Zhang, Y. Hiromori, S. Arakawa, H. Nagase, T. Nakanishi, and J. Hu. 2017. "Activation of Peroxisome Proliferator-Activated Receptor Gamma and Disruption of Progesterone Synthesis of 2-Ethylhexyl Diphenyl Phosphate in Human Placental Choriocarcinoma Cells: Comparison with Triphenyl Phosphate." *Environ Sci Technol* 51 (7): 4061-4068. <https://doi.org/10.1021/acs.est.7b00872>.

Janesick, A., and B. Blumberg. 2011. "Minireview: PPARγ as the target of obesogens." *J Steroid Biochem Mol Biol* 127 (1-2): 4-8. <https://doi.org/10.1016/j.jsbmb.2011.01.005>.

Jin, Y., G. Chen, and Z. Fu. 2016. "Effects of TBEP on the induction of oxidative stress and endocrine disruption in Tm3 Leydig cells." *Environ Toxicol* 31 (10): 1276-86. <https://doi.org/10.1002/tox.22137>.

Kassotis, C. D., K. Hoffman, and H. M. Stapleton. 2017. "Characterization of Adipogenic Activity of House Dust Extracts and Semi-Volatile Indoor Contaminants in 3T3-L1 Cells." *Environmental Science and Technology* 51 (15): 8735-8745. <https://doi.org/10.1021/acs.est.7b01788>.

Kim, S., J. Jung, I. Lee, D. Jung, H. Youn, and K. Choi. 2015. "Thyroid disruption by triphenyl phosphate, an organophosphate flame retardant, in zebrafish (Danio rerio) embryos/larvae, and in GH3 and FRTL-5 cell lines." *Aquat Toxicol* 160: 188-96. <https://doi.org/10.1016/j.aquatox.2015.01.016>.

Kim, S., N. Rabhi, B. C. Blum, R. Hekman, K. Wynne, A. Emili, S. Farmer, and J. J. Schlezinger. 2020. "Triphenyl phosphate is a selective PPARγ modulator that does not induce brite adipogenesis in vitro and in vivo." *Archives of Toxicology* 94 (9): 3087-3103. <https://doi.org/10.1007/s00204-020-02815-1>.

Kojima, H., S. Takeuchi, T. Itoh, M. Iida, S. Kobayashi, and T. Yoshida. 2013. "In vitro endocrine disruption potential of organophosphate flame retardants via human nuclear receptors." *Toxicology* 314 (1): 76-83. <https://doi.org/10.1016/j.tox.2013.09.004>.

Krivoshiev, B. V., F. Dardenne, A. Covaci, R. Blust, and S. J. Husson. 2016. "Assessing in-vitro estrogenic effects of currently-used flame retardants." *Toxicol In Vitro* 33: 153-62. <https://doi.org/10.1016/j.tiv.2016.03.006>.

Krumm, E. A., V. J. Patel, T. S. Tillery, A. Yasrebi, J. Shen, G. L. Guo, S. M. Marco, B. T. Buckley, and T. A. Roepke. 2018. "Organophosphate Flame-Retardants Alter Adult Mouse Homeostasis and Gene Expression in a Sex-Dependent Manner Potentially Through Interactions With ERα." *Toxicol Sci* 162 (1): 212-224. <https://doi.org/10.1093/toxsci/kfx238>.

La Merrill, M., and L. S. Birnbaum. 2011. "Childhood obesity and environmental chemicals." *Mt Sinai J Med* 78 (1): 22-48. <https://doi.org/10.1002/msj.20229>.

Lefterova, M. I., A. K. Haakonsson, M. A. Lazar, and S. Mandrup. 2014. "PPARγ and the global map of adipogenesis and beyond." *Trends Endocrinol Metab* 25 (6): 293-302. <https://doi.org/10.1016/j.tem.2014.04.001>.

Legler, J., D. Zalko, F. Jourdan, M. Jacobs, B. Fromenty, P. Balaguer, W. Bourguet, V. Munic Kos, A. Nadal, C. Beausoleil, S. Cristobal, S. Remy, S. Ermler, L. Margiotta-Casaluci, J. L. Griffin, B. Blumberg, C. Chesné, S. Hoffmann, P. L. Andersson, and J. H. Kamstra. 2020. "The GOLIATH Project: Towards an Internationally Harmonised Approach for Testing Metabolism Disrupting Compounds." *Int J Mol Sci* 21 (10). <https://doi.org/10.3390/ijms21103480>.

Liu, X., K. Ji, and K. Choi. 2012. "Endocrine disruption potentials of organophosphate flame retardants and related mechanisms in H295R and MVLN cell lines and in zebrafish." *Aquat Toxicol* 114-115: 173-81. <https://doi.org/10.1016/j.aquatox.2012.02.019>.

Lockhart, W. L., R. Wagemann, J. W. Clayton, B. Graham, and D. Murray. 1975. "Chronic toxicity of a synthetic tri aryl phosphate oil to fish." *ENVIRONM.PHYSIOL.BIOCHEM.* 5 (6): 361-369.

Lu, S. Y., Y. X. Li, T. Zhang, D. Cai, J. J. Ruan, M. Z. Huang, L. Wang, J. Q. Zhang, and R. L. Qiu. 2017. "Effect of E-waste Recycling on Urinary Metabolites of Organophosphate Flame Retardants and Plasticizers and Their Association with Oxidative Stress." *Environ Sci Technol* 51 (4): 2427-2437. <https://doi.org/10.1021/acs.est.6b05462>.

Ma, X., D. Wang, W. Zhao, and L. Xu. 2018. "Deciphering the Roles of PPARγ in Adipocytes via Dynamic Change of Transcription Complex." *Front Endocrinol (Lausanne)* 9: 473. <https://doi.org/10.3389/fendo.2018.00473>.

McGee, S. P., A. Konstantinov, H. M. Stapleton, and D. C. Volz. 2013. "Aryl phosphate esters within a major PentaBDE replacement product induce cardiotoxicity in developing zebrafish embryos: potential role of the aryl hydrocarbon receptor." *Toxicol Sci* 133 (1): 144-56. <https://doi.org/10.1093/toxsci/kft020>.

Meeker, J. D., and H. M. Stapleton. 2010. "House dust concentrations of organophosphate flame retardants in relation to hormone levels and semen quality parameters." *Environ Health Perspect* 118 (3): 318-23. <https://doi.org/10.1289/ehp.0901332>.

Mitchell, C. A., S. Dasgupta, S. Zhang, H. M. Stapleton, and D. C. Volz. 2018. "Disruption of Nuclear Receptor Signaling Alters Triphenyl Phosphate-Induced Cardiotoxicity in Zebrafish Embryos." *Toxicol Sci* 163 (1): 307-318. <https://doi.org/10.1093/toxsci/kfy037>.

Morris, P. J., D. Medina-Cleghorn, A. Heslin, S. M. King, J. Orr, M. M. Mulvihill, R. M. Krauss, and D. K. Nomura. 2014. "Organophosphorus flame retardants inhibit specific liver carboxylesterases and cause serum hypertriglyceridemia." *ACS Chem Biol* 9 (5): 1097-103. <https://doi.org/10.1021/cb500014r>.

Negi, C. K., L. Bajard, J. Kohoutek, and L. Blaha. 2021. "An adverse outcome pathway based in vitro characterization of novel flame retardants-induced hepatic steatosis." *Environ Pollut* 289: 117855. <https://doi.org/10.1016/j.envpol.2021.117855>.

Nicole, W. 2014. "More fat, Less bone? Flame retardant may deliver a one-two punch." *Environmental Health Perspectives* 122 (11): A312. <https://doi.org/10.1289/ehp.122-A312>.

Niemelä, S., S. Miettinen, J. R. Sarkanen, and N. Ashammakhi. 2008. "Adipose tissue and adipocyte differentiation: molecular and cellular aspects and tissue engineering applications." *Topics in Tissue Engineering* 4 (1): 26.

Papalou, O., E. A. Kandaraki, G. Papadakis, and E. Diamanti-Kandarakis. 2019. "Endocrine Disrupting Chemicals: An Occult Mediator of Metabolic Disease." *Front Endocrinol (Lausanne)* 10: 112. <https://doi.org/10.3389/fendo.2019.00112>.

Patisaul, H. B., S. C. Roberts, N. Mabrey, K. A. McCaffrey, R. B. Gear, J. Braun, S. M. Belcher, and H. M. Stapleton. 2013. "Accumulation and endocrine disrupting effects of the flame retardant mixture Firemaster® 550 in rats: an exploratory assessment." *J Biochem Mol Toxicol* 27 (2): 124-36. <https://doi.org/10.1002/jbt.21439>.

Pillai, H. K., M. Fang, D. Beglov, D. Kozakov, S. Vajda, H. M. Stapleton, T. F. Webster, and J. J. Schlezinger. 2014. "Ligand binding and activation of PPARγ by firemaster® 550: Effects on adipogenesis and osteogenesis in vitro." *Environmental Health Perspectives* 122 (11): 1225-1232. <https://doi.org/10.1289/ehp.1408111>.

Preston, E. V., M. D. McClean, B. Claus Henn, H. M. Stapleton, L. E. Braverman, E. N. Pearce, C. M. Makey, and T. F. Webster. 2017. "Associations between urinary diphenyl phosphate and thyroid function." *Environ Int* 101: 158-164. <https://doi.org/10.1016/j.envint.2017.01.020>.

Reddam, A., C. A. Mitchell, S. Dasgupta, J. S. Kirkwood, A. Vollaro, M. Hur, and D. C. Volz. 2019. "mRNA-Sequencing Identifies Liver as a Potential Target Organ for Triphenyl Phosphate in Embryonic Zebrafish." *Toxicol Sci* 172 (1): 51-62. <https://doi.org/10.1093/toxsci/kfz169>.

Rock, K. D., G. St Armour, B. Horman, A. Phillips, M. Ruis, A. K. Stewart, D. Jima, D. C. Muddiman, H. M. Stapleton, and H. B. Patisaul. 2020. "Effects of Prenatal Exposure to a Mixture of Organophosphate Flame Retardants on Placental Gene Expression and Serotonergic Innervation in the Fetal Rat Brain." *Toxicol Sci* 176 (1): 203-223. <https://doi.org/10.1093/toxsci/kfaa046>.

Romano, M. E., N. L. Hawley, M. Eliot, A. M. Calafat, N. K. Jayatilaka, K. Kelsey, S. McGarvey, M. G. Phipps, D. A. Savitz, E. F. Werner, and J. M. Braun. 2017. "Variability and predictors of urinary concentrations of organophosphate flame retardant metabolites among pregnant women in Rhode Island." *Environ Health* 16 (1): 40. <https://doi.org/10.1186/s12940-017-0247-z>.

Rosen, E. D., C. H. Hsu, X. Wang, S. Sakai, M. W. Freeman, F. J. Gonzalez, and B. M. Spiegelman. 2002. "C/EBPalpha induces adipogenesis through PPARgamma: a unified pathway." *Genes Dev* 16 (1): 22-6. <https://doi.org/10.1101/gad.948702>.

Rosen, E. D., and O. A. MacDougald. 2006. "Adipocyte differentiation from the inside out." *Nat Rev Mol Cell Biol* 7 (12): 885-96. <https://doi.org/10.1038/nrm2066>.

Saillenfait, A. M., S. Ndaw, A. Robert, and J. P. Sabaté. 2018. "Recent biomonitoring reports on phosphate ester flame retardants: a short review." *Arch Toxicol* 92 (9): 2749-2778. <https://doi.org/10.1007/s00204-018-2275-z>.

Scanlan, L. D., A. V. Loguinov, Q. Teng, P. Antczak, K. P. Dailey, D. T. Nowinski, J. Kornbluh, X. X. Lin, E. Lachenauer, A. Arai, N. K. Douglas, F. Falciani, H. M. Stapleton, and C. D. Vulpe. 2015. "Gene transcription, metabolite and lipid profiling in eco-indicator daphnia magna indicate diverse mechanisms of toxicity by legacy and emerging flame-retardants." *Environ Sci Technol* 49 (12): 7400-10. <https://doi.org/10.1021/acs.est.5b00977>.

Schang, G., B. Robaire, and B. F. Hales. 2016. "Organophosphate Flame Retardants Act as Endocrine-Disrupting Chemicals in MA-10 Mouse Tumor Leydig Cells." *Toxicol Sci* 150 (2): 499-509. <https://doi.org/10.1093/toxsci/kfw012>.

Selmi-Ruby, S., J. Marín-Sáez, A. Fildier, A. Buleté, M. Abdallah, J. Garcia, J. Deverchère, L. Spinner, B. Giroud, S. Ibanez, T. Granjon, C. Bardel, A. Puisieux, B. Fervers, E. Vulliet, L. Payen, and A. M. Vigneron. 2020. "In Vivo Characterization of the Toxicological Properties of DPhP, One of the Main Degradation Products of Aryl Phosphate Esters." *Environ Health Perspect* 128 (12): 127006. <https://doi.org/10.1289/ehp6826>.

Soubry, A., C. Hoyo, C. M. Butt, S. Fieuws, T. M. Price, S. K. Murphy, and H. M. Stapleton. 2017. "Human exposure to flame-retardants is associated with aberrant DNA methylation at imprinted genes in sperm." *Environmental Epigenetics* 3 (1). <https://doi.org/10.1093/eep/dvx003>.

Su, G., D. Crump, R. J. Letcher, and S. W. Kennedy. 2014. "Rapid in vitro metabolism of the flame retardant triphenyl phosphate and effects on cytotoxicity and mRNA expression in chicken embryonic hepatocytes." *Environ Sci Technol* 48 (22): 13511-9. <https://doi.org/10.1021/es5039547>.

Suzuki, G., N. M. Tue, G. Malarvannan, A. Sudaryanto, S. Takahashi, S. Tanabe, S. Sakai, A. Brouwer, N. Uramaru, S. Kitamura, and H. Takigami. 2013. "Similarities in the endocrine-disrupting potencies of indoor dust and flame retardants by using human osteosarcoma (U2OS) cell-based reporter gene assays." *Environ Sci Technol* 47 (6): 2898-908. <https://doi.org/10.1021/es304691a>.

Takahashi, M., Y. Kamei, and O. Ezaki. 2005. "Mest/Peg1 imprinted gene enlarges adipocytes and is a marker of adipocyte size." *Am J Physiol Endocrinol Metab* 288 (1): E117-24. <https://doi.org/10.1152/ajpendo.00244.2004>.

Tarazona, J. V., D. Court-Marques, M. Tiramani, H. Reich, R. Pfeil, F. Istace, and F. Crivellente. 2017. "Glyphosate toxicity and carcinogenicity: a review of the scientific basis of the European Union assessment and its differences with IARC." *Arch Toxicol* 91 (8): 2723-2743. <https://doi.org/10.1007/s00204-017-1962-5>.

Tontonoz, P., E. Hu, R. A. Graves, A. I. Budavari, and B. M. Spiegelman. 1994. "mPPAR gamma 2: tissue-specific regulator of an adipocyte enhancer." *Genes Dev* 8 (10): 1224-34. <https://doi.org/10.1101/gad.8.10.1224>.

Tontonoz, P., and B. M. Spiegelman. 2008. "Fat and beyond: the diverse biology of PPARgamma." *Annu Rev Biochem* 77: 289-312. <https://doi.org/10.1146/annurev.biochem.77.061307.091829>.

Tung, E. W. Y., S. Ahmed, V. Peshdary, and E. Atlas. 2017. "Firemaster® 550 and its components isopropylated triphenyl phosphate and triphenyl phosphate enhance adipogenesis and transcriptional activity of peroxisome proliferator activated receptor (Pparγ) on the adipocyte protein 2 (aP2) promoter." *PLoS One* 12 (4): e0175855. <https://doi.org/10.1371/journal.pone.0175855>.

Tung, E. W. Y., V. Peshdary, R. Gagné, A. Rowan-Carroll, C. L. Yauk, A. Boudreau, and E. Atlas. 2017. "Adipogenic Effects and Gene Expression Profiling of Firemaster® 550 Components in Human Primary Preadipocytes." *Environ Health Perspect* 125 (9): 097013. <https://doi.org/10.1289/ehp1318>.

Tversky, A., and D. Kahneman. 1974. "Judgment under Uncertainty: Heuristics and Biases." *Science* 185 (4157): 1124-31. <https://doi.org/10.1126/science.185.4157.1124>.

Van den Eede, N., M. Cuykx, R. M. Rodrigues, K. Laukens, H. Neels, A. Covaci, and T. Vanhaecke. 2015. "Metabolomics analysis of the toxicity pathways of triphenyl phosphate in HepaRG cells and comparison to oxidative stress mechanisms caused by acetaminophen." *Toxicol In Vitro* 29 (8): 2045-54. <https://doi.org/10.1016/j.tiv.2015.08.012>.

Van den Eede, N., A. L. Heffernan, L. L. Aylward, P. Hobson, H. Neels, J. F. Mueller, and A. Covaci. 2015. "Age as a determinant of phosphate flame retardant exposure of the Australian population and identification of novel urinary PFR metabolites." *Environ Int* 74: 1-8. <https://doi.org/10.1016/j.envint.2014.09.005>.

van der Veen, I., and J. de Boer. 2012. "Phosphorus flame retardants: properties, production, environmental occurrence, toxicity and analysis." *Chemosphere* 88 (10): 1119-53. <https://doi.org/10.1016/j.chemosphere.2012.03.067>.

Vrang, N., D. Meyre, P. Froguel, J. Jelsing, M. Tang-Christensen, V. Vatin, J. D. Mikkelsen, K. Thirstrup, L. K. Larsen, K. B. Cullberg, J. Fahrenkrug, P. Jacobson, L. Sjöström, L. M. Carlsson, Y. Liu, X. Liu, H. W. Deng, and P. J. Larsen. 2010. "The imprinted gene neuronatin is regulated by metabolic status and associated with obesity." *Obesity (Silver Spring)* 18 (7): 1289-96. <https://doi.org/10.1038/oby.2009.361>.

Wang, D., S. Yan, J. Yan, M. Teng, Z. Meng, R. Li, Z. Zhou, and W. Zhu. 2019. "Effects of triphenyl phosphate exposure during fetal development on obesity and metabolic dysfunctions in adult mice: Impaired lipid metabolism and intestinal dysbiosis." *Environmental Pollution* 246: 630-638. <https://doi.org/10.1016/j.envpol.2018.12.053>.

Wang, D., W. Zhu, L. Chen, J. Yan, M. Teng, and Z. Zhou. 2018. "Neonatal triphenyl phosphate and its metabolite diphenyl phosphate exposure induce sex- and dose-dependent metabolic disruptions in adult mice." *Environ Pollut* 237: 10-17. <https://doi.org/10.1016/j.envpol.2018.01.047>.

Wang, L., X. Huang, A. K. C. Laserna, and S. F. Y. Li. 2018. "Untargeted metabolomics reveals transformation pathways and metabolic response of the earthworm Perionyx excavatus after exposure to triphenyl phosphate." *Sci Rep* 8 (1): 16440. <https://doi.org/10.1038/s41598-018-34814-9>.

Wang, Q., J. C. Lam, J. Han, X. Wang, Y. Guo, P. K. Lam, and B. Zhou. 2015. "Developmental exposure to the organophosphorus flame retardant tris(1,3-dichloro-2-propyl) phosphate: estrogenic activity, endocrine disruption and reproductive effects on zebrafish." *Aquat Toxicol* 160: 163-71. <https://doi.org/10.1016/j.aquatox.2015.01.014>.

Wang, X., F. Li, J. Liu, C. Ji, and H. Wu. 2020. "Transcriptomic, proteomic and metabolomic profiling unravel the mechanisms of hepatotoxicity pathway induced by triphenyl phosphate (TPP)." *Ecotoxicol Environ Saf* 205: 111126. <https://doi.org/10.1016/j.ecoenv.2020.111126>.

Wang, Y., J. Hong, M. Shi, L. Guo, L. Liu, H. Tang, and X. Liu. 2021. "Triphenyl phosphate disturbs the lipidome and induces endoplasmic reticulum stress and apoptosis in JEG-3 cells." *Chemosphere* 275: 129978. <https://doi.org/10.1016/j.chemosphere.2021.129978>.

Wang, Y., Y. Kwon, L. An, C.N. Holmes, M. Haeba, and G.A. LeBlanc. 2016. "Differential interactions of the flame retardant triphenyl phosphate within the PPAR signaling network." *MOJ Toxicology* 2 (3).

Wang Y., Kwon Y., An L.,Holmes C.N., Haeba M., LeBlanc G.A. 2016. "Differential interactions of the flame retardant triphenyl phosphate within the PPAR signaling network." *MOJ Toxicology* 2 (3).