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Obesogens in food as a health risk for obesity

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Aleksandra Janikowska[a], Dariusz Nowak[b] ✉

[a] *Aleksandra Janikowska,*[1] *https://orcid.org/0009-0008-2896-8706*
[b] *Dariusz Nowak,*[1] *https://orcid.org/0000-0002-5493-1949*
[1] *Department of Nutrition and Dietetics, Faculty of Health Sciences, Ludwik Rydygier Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Toruń, Poland*
✉ *Corresponding author: d.nowak@cm.umk.pl*

Abstract: Modern society is confronted on a daily basis with the problem of obesity, which is one of the greatest threats of the 21st century. Factors in the development of obesity are well known, although one of the lesser-known but equally important factors is exposure to chemicals called obesogens. These compounds are found in cosmetics, food and plastics, among others, and exposure can occur through the skin, the respiratory tract and the gastrointestinal tract. The results of many studies highlight the need for increased control and regulation of chemicals contained in consumer products. Understanding the role of obesogens as a factor in the development of obesity can help formulate better and more effective prevention and treatment strategies, which is crucial for public health.

Keywords: adipose tissue, chemicals, environmental factors, obesogens, obesity

1. Introduction

Today's society is facing the growing problem of obesity, which is recognised as one of the greatest health threats of the 21st century. It has been identified by the World Health Organization as a pandemic. Obesity is defined as a body mass index (BMI)>30 kg/m2, while normal BMI values by standards are 18.5–24.8 kg/m2. In 2021, the European Commission defined obesity as a chronic and recurrent disease that leads to a series of different non-communicable, diet-related diseases, including type 2 diabetes, cardiovascular disease and cancer. Additionally, obesity is associated with a 20-year reduction in life expectancy and other consequences such as unemployment and increased economic burden on society (Yilmaz et al., 2025). The mechanism of obesity development is complex and multifactorial. A high-calorie diet combined with a lack of or insufficient physical activity is presented as the basis for the onset of obesity (Burki, 2021; Piché et al., 2020). However, the development of the epidemic of this disease is due to a number of other lifestyle factors. These factors include: biological factors (endocrine disorders affecting the regulation of energy balance), behavioural factors (inappropriate, high-energy diet combined with insufficient physical activity), social factors (socioeconomic status, lifestyle, urbanisation) and environmental factors (stress, disturbed diurnal rhythm) (Blüher, 2019; Piché et al., 2020). The risk factors are well known by the public, but some of the lesser-known factors influencing weight gain and thus the development of obesity to date are chemicals called obesogens. The first mention of external compounds that may impair energy homeostasis regulation, promoting weight gain and the development of obesity, was made by Grün and Blumberg, who published two article on this topic in 2006 and 2009 (Grün & Blumberg, 2006, Grün & Blumberg, 2009a; Micić et al., 2021). This has led to an increase in interest in obesogens and has triggered a number of studies to understand their sources, mechanism of action and health effects.

The aim of this study is to present the role of obesogens in the development of obesity and to assess their impact on the population.

2. Methodology

This review was conducted according to the principles of narrative and systematic literature review. A comprehensive search of the Google Scholar, PubMed and Web of Science databases was performed from 2017 up to June 2025. The search strategy combined free-text words related to 'obesogens' and 'obesogens in food'. The bibliographic entries were selected on the basis of an individual assessment of the title and abstract of the literature searched. Reports not available full text, chapters in books, text in language other than English and case studies were excluded.

3. Results and discussion

3.1. Definition of obesogens

Obesogens are defined as chemical compounds, both dietary, endogenous and pharmaceutical and xenobiotic, which, together with a number of genetic and environmental factors such as excess energy intake relative to energy expenditure, cause a predisposition to weight gain and subsequent obesity and the subsequent development of metabolic diseases (Li et al., 2011). Obesogens can be natural (e.g., metals, viruses), anthropogenic prescription drugs (e.i. antibiotics, hormonal drugs), environmental (insecticides, plastics, household chemicals, particulate matter), or food components (fructose, trans-fats, preservatives, emulsifiers)(Heindel et al., 2024). These compounds impede the proper functioning of the endocrine system by affecting metabolism and thus disrupting adipose tissue homeostasis and causing weight gain (Entezari et al., 2022). Currently, more than 1,300 substances have been identified as possibly capable of disrupting endocrine regulation (Li, 2011; Nicolaou et al., 2024).

3.2. Mechanism of action

Substances categorised as obesogens interfere with various signaling pathways in the body that affect tissues crucial to whole-body metabolism at their endpoints, resulting in altered adipose tissue function (Chamorro-Garcia & Veiga-Lopez, 2021; Shahnazaryan et al., 2019). Multiple mechanisms have been shown through which obesogens can affect adipose tissue activity (Ćurić et al., 2021). Obesogens cause weight gain by altering lipid metabolism to promote adipogenesis and lipid accumulation (Gupta et al., 2020). Obesogens inappropriately stimulate adipogenesis and fat storage in vivo in two ways: directly or indirectly (Lee & Blumberg, 2019). Direct action may occur by increasing the number of adipocytes, increasing the size of adipocytes or altering hormonal pathways that are responsible for controlling fat development (Darbre, 2017). Indirect effects may include altering hormones that regulate appetite, satiety and food preferences and altering basal metabolism (Darbre, 2017). These disturbances cause changes in various organs such as the brain, pancreas, adipose tissue, gastrointestinal tract and muscle, altering energy balance to promote calorie storage and altering the composition of the microbiome (Lee & Blumberg, 2019).

3.3. Sources of obesogens

The number of newly discovered chemicals produced and marketed is increasing every year. The introduction of these substances into the environment occurs through industry, manufacturing practices, agriculture and human activities such as the use of medicines, personal care products and cleaning products (Barra et al., 2022). While some of these compounds occur naturally in nature, most are substances that have been released into the environment as a result of human activity without knowledge of their impact on the environment and human health. Obesogens include solvents – polychlorinated biphenyls (PCBs); pesticides – e.g., dichlorodiphenyltrichloroethane (DDT), chlorpyrifos, diazinon, permethrin, neonicotinoids; non-stick coatings – e.g., per- and polyfluorinated substances (PFAS); clothing and furniture protectants; food preservatives/additives/emulsifiers – e.g., parabens, monosodium glutamate, carboxymethylcellulose, 3-tert-butyl-4-hydroxyanisole (3-BHA)); personal care products – e.g., phthalates, parabens;

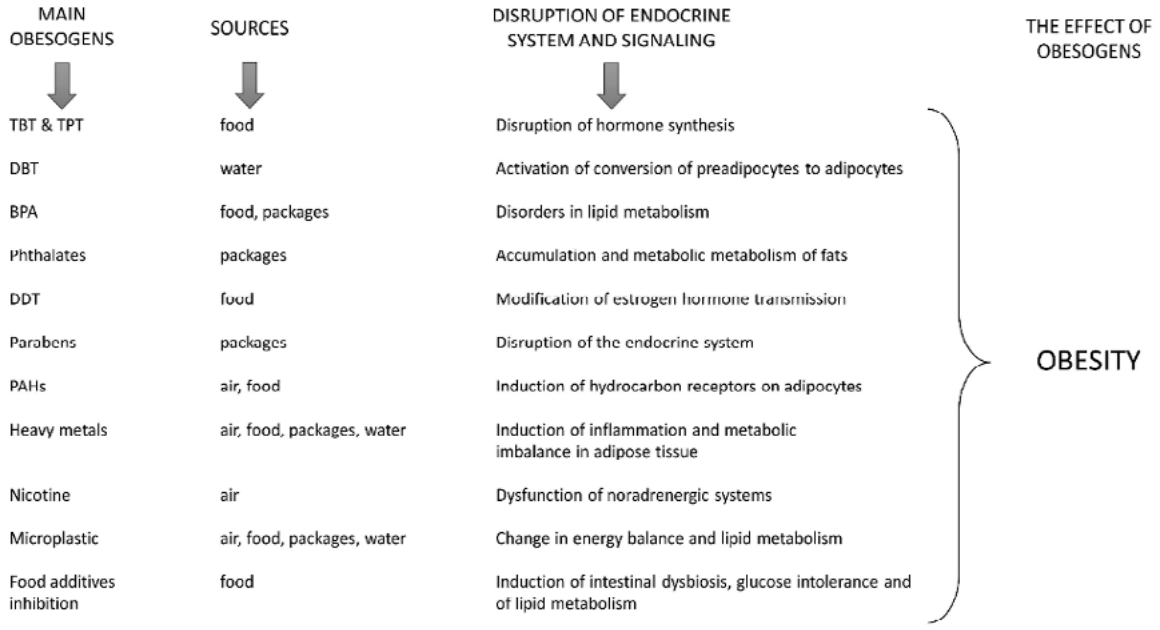

Figure 1. Sources of obesogens and their action. Complited by the author from data search on: Jaskulak et al. 2025; Kladnicka et al., 2022; Wang et al. 2024.

plastics – e.g., phthalates, bisphenols; resins and can linings – e.g., bisphenols; and air pollutants – e.g., polycyclic aromatic hydrocarbons (PAHs) (Heindel et al., 2024; Veiga-Lopez et al., 2018; Wang et al., 2024). Exposures can occur via air, water, food, skin contact or dust inhalation (Darbre, 2017; Heindel et al., 2024).

3.4. Important compounds among the obesogens

Organotins are persistent organic pollutants widely used as pesticides, disinfectants and in aquatic systems as marine antifouling agents (Grün & Blumberg, 2009b). These compounds are harmful to the endocrine system by interfering with hormone synthesis (Kladnicka et al., 2022). One of the most studied substances in this group is tributyltin (TBT) and its related compound triphenyltin (TPT). Exposure to tributyltin occurs through dietary sources, more specifically seafood and shellfish (Lee & Blumberg, 2019). Indirectly, humans are exposed to TBT through house dust (Kladnicka et al., 2022). The obesogenic mechanism of these compounds is through stimulation of adipogenesis and lipogenesis (Núñez-Sánchez et al., 2023).

Dibutyltin (DBT) is the main breakdown product of TBT. This substance is more prevalent in the environment due to its presence in high concentrations in polyvinyl chloride (PVC) plastics. It may pose a risk to humans due to the penetration of this compound into drinking water from PVC pipes. Based on studies, prenatal exposure to this compound led to increased white adipose tissue (WAT) in the offspring and led to insulin resistance (Egusquiza & Blumberg, 2020).

Bisphenol A (BPA) is a phenolic compound used in the production of polycarbonate plastics and epoxy resins, which are ubiquitous in consumer products such as water bottles, water pipe linings, food and beverage can coatings, car parts and baby bottles (Darbre, 2017). Based on studies, it can be concluded that its dietary intake is the main route of human exposure (Kladnicka et al., 2022). Bisphenol A is the most extensively studied and documented obesogenic compound in relation to the occurrence of epigenetic changes associated with the onset and development of obesity (Núñez-Sánchez et al., 2023). Monomers

of this compound can leach from the plastic food and beverage containers in which they are packaged and distributed and thus integrate into hormonal and metabolic pathways in the body (Alharbi et al., 2022). Bisphenol A is a well-known compound that disrupts the endocrine system. It accumulates in white adipose tissue (WAT) and thus can affect the metabolic function of fat cells. Bisphenol A causes weight gain by disrupting the body's lipid balance, stimulating adipogenesis and lipid accumulation, and modulating the activity of nuclear receptors that accumulate lipid flux, thereby causing obesity (Martinez-Esquivel et al., 2022).

Phthalates are phthalic acid esters, which are mainly used as softeners to increase the flexibility and durability of plastic materials. They can be found in many everyday products such as adhesives, paints, children's toys, electronics, medical equipment, personal care products, air fresheners, pharmaceuticals and various textiles (Darbre, 2017). These compounds pose a risk in children because toys that contain phthalates in their composition can enter the child's body when they place them in the mouth (Kladnicka et al., 2022). The mechanism of these compounds is through their effects on fat accumulation and metabolism. Phthalates affect the function of nuclear receptors and alter the regulation of energy balance, leading to abnormal glucose metabolism and an increased risk of the onset and development of obesity and other related metabolic disorders (Amato et al., 2021; Wang et al. 2024).

Dichlorodiphenyltrichloroethane (DDT) and its breakdown product *dichlorodiphenyltrichloroethylene* (DDE) are two compounds in a group of pollutants widely distributed in the environment (Darbre, 2017). The greatest exposure to DDT and its metabolites occurs through the consumption of meat, meat products and fish, and it is also detectable in leafy vegetables, which are richer in DDT compared to other types of vegetables.

DDT and DDE act as nuclear estrogen receptor agonists (DDT), androgen receptor antagonist (DDE) and can bind to certain GPCRs. The result may be a change in estrogen signaling (Mohajer et al., 2021).

Parabens are alkyl esters of p-hydroxybenzoic acid characterised by antimicrobial and antifungal properties. They are typically used in personal care cosmetics and pharmaceuticals, which are the main source of human exposure. These compounds are endocrine disruptors (Núñez-Sánchez et al., 2023). Thus, they stimulate the formation of adipose tissue in preadipocytes and mesenchymal stem cells cells (Amato et al., 2021).

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of compounds that are formed during incomplete combustion of coal, oil, gas, wood, tobacco and intensive heat treatment of meat. The main sources of exposure are the inhalation of tobacco and wood smoke and the consumption of food contaminated with PAHs. One PAH is benzopyrene, which is formed by incomplete combustion and is present in grilled or smoked foods. This compound is correlated with the development of obesity through the induction of hydrocarbon receptors in adipocytes (Martinez-Esquivel et al., 2022).

Heavy metals, some of which are involved in metabolic dysfunction including the metabolic imbalance between BAT and WAT (Mohajer et al., 2021). The main source of exposure to heavy metals is water (Martinez-Esquivel et al., 2022). Arsenic is commonly found in water, soil and sediments (Mohajer et al., 2021). Sources include water and food that has been prepared using contaminated water. Arsenic promotes lipid storage and induces inflammatory processes, and induces insulin resistance and thus the development of type II diabetes (Martinez-Esquivel et al., 2022). In the case of cadmium, the population is exposed through water, air, tobacco smoke, plastics and food. This metal shows obesogenic properties through metabolic changes in adipose tissue. Cadmium reduces the bacterial diversity of the intestinal microbiota, so exposure to this metal early in life is particularly harmful, as the bacterial flora is not adequately developed during this period (Mohajer et al., 2021). Lead pollution comes from mining, industry and the burning of fossil fuels. Lead exposure is associated with obesity because it disrupts endocrine pathways, increasing oxidative stress and inducing inflammation (Martinez-Esquivel et al., 2022).

Nicotine is a substance contained in the tobacco plant. Epidemiological studies indicate that maternal smoking during pregnancy is a major factor in the development of obesity in the offspring. Prenatal nicotine overload leads to dysfunction of the noradrenergic systems, which results in increased appetite and causes fat accumulation (Darbre, 2017).

Microplastics (MPs) are found in a wide range of products and pose a risk through inhalation of contaminated air and dust, ingested bottled water and contaminated food. At least 45 different plastics are now commonly used, including polypropylene (PP), polyethylene (PE), polyethylene terephthalate (PET) and polyvinyl chloride (PVC). Humans are estimated to ingest tens of thousands to millions of microplastic particles annually, or on the order of several milligrams daily (Kannan & Vimalkumar, 2021). The main sources of microplastic exposure are believed to come from contaminated food and water. Dietary sources of microplastic include seafood (clams, shellfish, fish, seaweed) and sea salt. Diet is identified as the third main source of microplastic, next to drinking water and inhaling polluted air. Microplastics are also commonly used in cosmetic products such as scrubs and exfoliating gels, shower gels, shampoos and creams. The microplastic content in such products can vary. Microplastic has been shown to affect adipocyte differentiation after accumulation in the liver and kidneys, resulting in changes in energy balance and lipid metabolism (ibidem).

Food additives (preservatives, antioxidants, stabilisers, colourings and sweeteners) are among the risk factors. Preservatives are ingredients that are designed to prevent potential food damage, such as oxidation, rancidity. Research results indicate that sodium benzoate, when used as a preservative, can affect glucose homeostasis. This compound, together with sodium sulphate, inhibits the release of leptin from fat cells. This results in inadequate leptin levels, which reduces metabolic rate while increasing appetite and weight gain, resulting in the development of diabetes (Ravichandran et al., 2022). Sweeteners also known as non-nutritive sweeteners (NNS) are a common dietary supplement. They can act as obesogens. Commonly used in foods and beverages, acesulfame K, sucralose, saccharin can alter the composition of the intestinal barrier and induce glucose intolerance through intestinal dysbiosis (Mohajer et al., 2021).

3.5. Health risks

Obesogens are chemicals that promote obesity by increasing the number of fat cells and the amount of fat stored in existing fat cells (Janesick & Blumberg, 2012). Obesogens affect the function of the hypothalamus, the part of the brain responsible for controlling eating behaviour. Bisphenol A induces presynaptic and postsynaptic changes in signaling pathways, thus causing compulsive eating behaviour against a background of increased dietary intake, contributing to obesity (Shahnazaryan et al., 2019). A drastic increase in the development of many metabolic diseases has been attributed to chemicals called EDCs. It is noteworthy in this case that many of them are lipophilic, which favours their accumulation in adipose tissue over years. Such bioaccumulation promotes increased fat storage, resulting in increased retention of lipophilic obesogens, which in turn leads to increased adipose tissue and a renewed influx of contaminants (Darbre, 2017). It has been hypothesised that obesogens may adversely affect male reproductive health via "modulation" of the endocrine system (Rato & Sousa, 2021; Sousa et al., 2022). These chemical compounds alter male reproductive function, mainly via adverse effects on the central nervous system. There is a known link between obesogen exposure and reduced testosterone levels, as these compounds alter the steroidogenic function of luteal cells (LCs) and all pathways involved in sex steroid formation (Rato & Sousa, 2021). Much evidence has been found that endocrine disruptors affect the neuroendocrine system, specifically the hypothalamic-pituitary-thyroid (HPT) axis, also referred to as the reproductive axis. Different chemicals present in the environment can target the HPT axis, at different locations and with different intensities, disrupting its regulation (Rato & Sousa, 2021). It has long been debated whether obesogens could be an environmental factor in the etiology of PCOS (polycystic ovary syndrome). Chronic and

prolonged exposure to an obesogen such as bisphenol A (BPA) leads to its bioaccumulation and activation of signaling pathways that can alter metabolic and reproductive functions in women. BPA can directly cause androgen production, in ovarian cells, resulting in hyperandrogenism, it also interacts with receptors in adipose tissue, beta-pancreas cells causing hyperinsulinemia. All these changes have a negative effect on the ovarian follicles, causing the symptoms of PCOS (Urbanetz et al., 2023). Bisphenol A is responsible for the development of metabolic disorders such as obesity, type II diabetes, and fatty liver disease (Lewis et al., 2024; Oliviero et al., 2022). For this reason, it has been banned in many countries, and chemicals such as bisphenol S, bisphenol F (BPF), bisphenol AF and tetramethyl BPF have been used as its substitutes Oliviero et al., 2022). Many compounds that may be obesogens are currently being analysed. These include preservatives, antimicrobials, antibiotics and bisphenols. Epidemiological studies provide indisputable evidence linking exposure to new pollutants with the risk of obesity in humans (Wu et al., 2025). Many of these compounds can disrupt endocrine and metabolic functions and contribute to the development of obesity and other metabolic disorders, such as type 2 diabetes, coronary heart disease and hypertension (Heindel, 2019; Oliviero et al., 2022). Numerous epidemiological studies have shown a positive correlation between exposure to bisphenol A (BPA), phthalates, pesticides, alkylphenols and the incidence of cardiovascular disease, diabetes and weight gain (Oliviero et al., 2022; Shahnazaryan et al., 2019). There is also much discussion about microplastic (MP) consumption and the potential risks associated with it. Biomonitoring studies of human stool, fetus, and placenta provide direct evidence of MP exposure in infants and children. MPs <20 μm were reported to cross biological membranes (Kannan & Vimalkumar, 2021). Studies involving laboratory animals have revealed the occurrence of various forms inflammation, immunological response, endocrine disruption, alteration of lipid and energy metabolism, and other disorders after exposure to microplastics. Microplastics may contain a number of compounds, such as phthalates, bisphenols, and organotins, leading to oxidative stress, cytotoxicity, immunotoxicity, thyroid hormone disruption, and altered adipogenesis and energy production. It is suspected that ubiquitous microplastics are a potential factor in obesity (Kannan & Vimalkumar, 2021).

A recent study reports that children are also a particularly vulnerable group when it comes to obesogens. Obesogens disrupt the endocrine system in many ways. They affect appetite, promote inflammation, disrupt the functioning of the gut microbiome, and contribute directly or indirectly to the development of obesity (Kapama et al., 2025). Therefore, in addition to excessive calorie intake and physical inactivity, obesogens should be added to the existing factors predisposing to obesity (Nicolaou et al., 2024). Another recent review of 75 studies showed that early exposure to certain obesogens increases the risk of obesity later in life (Jaskulak et al., 2025). Epidemiological studies provide evidence of a link between prenatal or early exposure and an increased risk of obesity in offspring. Certain compounds such as phthalates, parabens and bisphenols disrupt hormone balance (EDCs), predisposing children and women to obesity or metabolic disorders. Phthalates are key compounds that pose a threat to children (Jaskulak et al., 2025).

Heindel et al. (2024) present other models leading to obesity in addition to obesogens that disrupt hormonal signalling. The energy balance model, the carbohydrate-insulin model, and the oxidation-reduction model also lead to obesity. The first is based on increased energy supply, the second on increased supply of rapidly digestible carbohydrates causing increased insulin response promoting lipogenesis, and the last on oxidative stress resulting from an excess of reactive oxygen species (ROS).

Recent research warns consuming food additives may result in serious health risks, not only for adults but also for children. Studies of food additives in various experimental animals, cell cultures, and the human population demonstrate numerous risk factors, including obesity, dyslipidemia, weight gain, hyperglycaemia, insulin resistance, glucose intolerance, energy imbalance, and hormonal intervention (Ravichandran et al., 2022). Reducing the marketing of energy-dense snacks to children and increasing

the promotion of healthier foods, such as fruits and vegetables, may be an effective and necessary instrument to improve the dietary intake of children and reduce the risk of their experiencing some chronic diseases later in life (Folkvord et al., 2021).

Conclusion

From the analysis of studies available in the scientific literature, it can be concluded that obesogens have a significant impact on fat metabolism and the body's endocrine system, which contributes to the development of obesity. It has been noted that these substances are present in many everyday products, including food, cosmetics and plastics. The results of many studies highlight the need for greater control and regulation of chemicals used in consumer products. Further research is also recommended to identify new obesogens and to study their mechanisms of action. It is also important to develop a strategy to reduce exposure to these substances, mainly in vulnerable groups such as children and pregnant women. Other factors leading to obesity should not be overlooked either. Preventive measures are also important. The availability of healthy food and a proper diet combined with physical activity affect the gut microbiota and play a key role in obesity prevention.

References

Alharbi, H. F., Algonaiman, R., Alduwayghiri, R., Aljutaily, T., Algheshairy, R. M., Almutairi, A. S., Alharbi, R. M., Alfurayh, L. A., Alshahwan, A. A., Alsadun, A. F., & Barakat, H. (2022). Exposure to bisphenol A substitutes, bisphenol S and bisphenol F, and its association with developing obesity and diabetes mellitus: A narrative review. *International Journal of Environmental Research and Public Health, 19*(23), 15918. https://doi.org/10.3390/ijerph192315918

Amato, A. A., Wheeler, H. B., & Blumberg, B. (2021). Obesity and endocrine-disrupting chemicals. *Endocrine Connections, 10*(2), 87 – 105. https://doi.org/10.1530/EC-20-0578

Barra, G. N., Kwon, H. Y., Morrison, M. K., Steinberg, R. G., Wade, G. M., Khan, M. M., Schertzer, D. J., & Holloway, C. A. (2022). Increased gut serotonin production in response to bisphenol A structural analogs may contribute to their obesogenic effects. *American Journal of Physiology – Endocrinology and Metabolism, 323*(1), 80-91. https://doi.org/10.1152/ajpendo.00049.2022

Blüher, M. (2019). Obesity: Global epidemiology and pathogenesis. *Nature Reviews Endocrinology, 15*(5), 288-298. https://doi.org/10.1038/s41574-019-0176-8

Burki, T. (2021). European Commission classifies obesity as a chronic disease. *The Lancet Diabetes & Endocrinology, 9*(7), 418. https://doi.org/10.1016/S2213-8587(21)00145-0

Chamorro-García, R., & Veiga-Lopez, A. (2021). The new kids on the block: Emerging obesogens. *Advances in Pharmacology, 92*, 457 – 484. https://doi.org/10.1016/bs.apha.2021.05.003

Ćurić, M., Majanović, S. K., Detel, D., Ružić, A., & Štimac, D. (2021). Obesogens – new global health problem? *Periodicum Biologorum, 123*(3 – 4), 49-53. https://doi.org/10.18054/pb.v123i3-4.5032

Darbre, D. F. (2017). Endocrine disruptors and obesity. In *Etiology of Obesity, 6*(1), 18 – 27. https://doi.org/10.1007/s13679-017-0240-4

Egusquiza, R. J., & Blumberg, B. (2020). Environmental obesogens and their impact on susceptibility to obesity: New mechanisms and chemicals. *Endocrinology, 161*(3), Article bqaa024. https://doi.org/10.1210/endocr/bqaa024

Entezari, B., Bozdağ, D., & Orhan, H. G. (2022). Obesogens: Definition, mechanisms of action, potential industrial chemicals and pharmaceuticals. *Istanbul Journal of Pharmacy, 52*(2), 215-225. https://doi.org/10.26650/IstanbulJPharm.2022.102702

Folkvord, F., Naderer, B., Coates, A., & Boyland, E. (2021). Promoting fruit and vegetable consumption for childhood obesity prevention. *Nutrients, 14*(1), 157. https://doi.org/10.3390/nu14010157

Grün, F., & Blumberg, B. (2009a). Endocrine disrupters as obesogens. *Molecular and Cellular Endocrinology, 304*(1 – 2), 19 – 29. https://doi.org/10.1016/j.mce.2009.02.018

Grün, F., & Blumberg, B. (2009b). Minireview: The case for obesogens. *Molecular Endocrinology, 23*(8), 1127-1134. https://doi.org/10.1210/me.2008-0485

Grün, F., & Blumberg, N. (2006). Environmental obesogens: Organotins and endocrine disruption via nuclear receptor signaling. *Endocrinology, 147*(6), 50-55. https://doi.org/10.1210/en.2005-1129

Gupta, R., Kumar, P., Fahmi, N., Garg, B., Dutta, S., Sachar, S., Mathaur, A. S., & Vimaleswaran, K. S. (2020). Endocrine disruption and obesity: A current review on environmental obesogens. *Current Research in Green and Sustainable Chemistry, 3*, 100-102. https://doi.org/10.1016/j.crgsc.2020.06.002

Heindel, J. J. (2019). History of the obesogen field: Looking back to look forward. *Frontiers in Endocrinology, 10*, 14. https://doi.org/10.3389/fendo.2019.00014

Heindel, J. J., Lustig, R. H., Howard, S., & Corkey, B. E. (2024). Obesogens: A unifying theory for the global rise in obesity. *International Journal of Obesity (London), 48*(4), 449-460. https://doi.org/10.1038/s41366-024-01460-3

Janesick, A., & Blumberg, B. (2012). Obesogens, stem cells and the developmental programming of obesity. *International Journal of Andrology, 35*(3), 437-448. https://doi.org/10.1111/j.1365-2605.2012.01247.x

Jaskulak, M., Zimowska, M., Rolbiecka, M., & Zorena, K. (2025). Understanding the role of endocrine disrupting chemicals as environmental obesogens in the obesity epidemic: A comprehensive overview of epidemiological studies between 2014 and 2024. *Ecotoxicology and Environmental Safety, 299*, 118401. https://doi.org/10.1016/j.ecoenv.2025.118401

Kannan, K., & Vimalkumar, K. (2021). A review of human exposure to microplastics and insights into microplastics as obesogens. *Frontiers in Endocrinology, 12*, Article 724989. https://doi.org/10.3389/fendo.2021.724989

Kapama, A., Stefanaki, C., Mastorakos, G., & Papagianni, M. (2025). The role of endocrine disruptors in childhood obesity: Unraveling the obesogens. *Hormone Research in Paediatrics, 26*, 1-14. https://doi.org/10.1159/000545043

Kladnicka, I., Bludovska, M., Plavinova, I., Muller, L., & Mullerova, D. (2022). Obesogens in foods. *Biomolecules, 12*(5), 680. https://doi.org/10.3390/biom12050680

Lee, M. K., & Blumberg, B. (2019). Transgenerational effects of obesogens. *Basic & Clinical Pharmacology & Toxicology, 125*(S3), 44-57. https://doi.org/10.1111/bcpt.13214

Lewis, N., Abdulkadir, A., Kandel, S., Rosby, R., & Hossain, E. (2024). Per- and polyfluoroalkyl substances (PFAS) as emerging obesogens: Mechanisms, epidemiological evidence, and regulatory challenges. *Physiologia, 4*(4), 517-567. https://doi.org/10.3390/physiologia4040035

Li, X., Ycaza, J., & Blumberg, B. (2011). The environmental obesogen tributyltin chloride acts via peroxisome proliferator-activated receptor gamma to induce adipogenesis in murine 3T3-L1 preadipocytes. *Journal of Steroid Biochemistry and Molecular Biology, 127*(1-2), 9-15. https://doi.org/10.1016/j.jsbmb.2011.03.012

Martinez-Esquivel, A., Trujillo-Silva, J. D., & Cilia-Lopez, G. V. (2022). Impact of environmental pollution on the obesogenic environment. *Nutrition Reviews, 80*(7), 1787-1799. https://doi.org/10.1093/nutrit/nuac003

Micić, D., Polovina, S., Micić, D., & Macut, D. (2021). Endocrine disrupting chemicals and obesity: The evolving story of obesogens. *Acta Endocrinologica (Bucharest), 17*(4), 503-508. https://doi.org/10.4183/aeb.2021.503

Mohajer, N., Du, C. Y., Checkcinco, C., & Blumberg, B. (2021). Obesogens: How they are identified and molecular mechanisms underlying their action. *Frontiers in Endocrinology, 12*, 780888. https://doi.org/10.3389/fendo.2021.780888

Nicolaou, M., Toumba, M., Kythreotis, A., Daher, H., & Skordis, N. (2024). Obesogens in adolescence: Challenging aspects and prevention strategies. *Children, 11*(5), 602. https://doi.org/10.3390/children11050602

Núñez-Sánchez, A. M., Jiménez-Méndez, A., Suárez-Cortés, M., Martínez-Sánchez, A. M., Sánchez-Solís, M., Blanco-Carnero, E. J., Ruiz-Alcaraz, J. A., & Ramos-Molina, B. (2023). Inherited epigenetic hallmarks of childhood obesity derived from prenatal exposure to obesogens. *International Journal of Environmental Research and Public Health, 20*(6), 4711. https://doi.org/10.3390/ijerph20064711

Oliviero, F., Marmugi, A., Viguié, C., Gayrard, V., Picard-Hagen, N., & Mselli-Lakhal, L. (2022). Are BPA substitutes as obesogenic as BPA? *International Journal of Molecular Sciences, 23*(8), 4238. https://doi.org/10.3390/ijms23084238

Piché, M. E., Tchernof, A., & Després, J. P. (2020). Obesity phenotypes, diabetes, and cardiovascular diseases. *Circulation Research, 126*(11), 1477-1500. https://doi.org/10.1161/CIRCRESAHA.120.316101

Rato, L., & Sousa, A. C. A. (2021). The impact of endocrine-disrupting chemicals in male fertility: Focus on the action of obesogens. *Journal of Xenobiotics, 11*(4), 163-196. https://doi.org/10.3390/jox11040012

Ravichandran, G., Lakshmanan, D. K., Arunachalam, A., & Thilagar, S. (2022). Food obesogens as emerging metabolic disruptors: A toxicological insight. *Journal of Steroid Biochemistry and Molecular Biology, 217*, 106042. https://doi.org/10.1016/j.jsbmb.2021.106042

Shahnazaryan, U., Wójcik, M., Bednarczuk, T., & Kuryłowicz, A. (2019). Role of obesogens in the pathogenesis of obesity. *Medicina, 55*(9), 515. https://doi.org/10.3390/medicina55090515

Sousa, A. C. A., Alves, M. G., Oliveira, P. F., Silva, B. M., & Rato, L. (2022). Male infertility in the XXI century: Are obesogens to blame? *International Journal of Molecular Sciences, 23*(6), 3046. https://doi.org/10.3390/ijms23063046

Urbanetz, L. A. M. L., Soares Junior, J. M., Maciel, G. A. R., Simões, R. S., Baracat, M. C. P., & Baracat, E. C. (2023). Does bisphenol A (BPA) participate in the pathogenesis of polycystic ovary syndrome (PCOS)? *Clinics, 78*, 100310. https://doi.org/10.1016/j.clinsp.2023.100310

Veiga-Lopez, A., Pu, Y., Gingrich, J., & Padmanabhan, V. (2018). Obesogenic endocrine-disrupting chemicals: Identifying knowledge gaps. *Trends in Endocrinology & Metabolism, 29*, 607 – 625. https://doi.org/10.1016/j.tem.2018.06.003

Wang, X., Sun, Z., Liu, Q. S., Zhou, Q., & Jiang, G. (2024). Environmental obesogens and their perturbations in lipid metabolism. *Environment & Health, 2*(5), 253-268. https://doi.org/10.1021/envhealth.3c00202

Wu, S., Tong, C., & Liu, J. (2025). Obesogenic effects of six classes of emerging contaminants. *Journal of Environmental Sciences, 151*, 252-272. https://doi.org/10.1016/j.jes.2024.03.032

Yilmaz, B., Erdogan, C. S., Sandald, S., Kelestimur, F., & Carpenter, D. O. (2025). Obesogens and energy homeostasis: Definition, mechanisms of action, exposure, and adverse effects on human health. *Neuroendocrinology, 115*, 72-99. https://doi.org/10.1159/000542901