

Environmental, Nutritional, Psychosocial, and Neuroendocrine Determinants as Potential Promoters of Precocious Puberty- An Integrated Review

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Abstract

Precocious puberty—defined as the onset of secondary sexual characteristics before age 8 in girls and 9 in boys—is rising globally and reflects a complex interplay of genetic, metabolic, environmental, psychosocial, and lifestyle factors. The hypothalamic–pituitary–gonadal (HPG) axis activation is governed centrally by the pulsatile release of gonadotropin-releasing hormone (GnRH), regulated by the KNDy (kisspeptin/neurokinin B/dynorphin) neuronal network. This review synthesizes current understanding of how metabolic cues (leptin, insulin), endocrine-disrupting chemicals (EDCs), psychosocial stress via limbic–hypothalamic–pituitary–adrenal (HPA) axis pathways, and circadian/sleep disruptions converge on the GnRH pulse generator to precipitate early puberty. The limbic system’s role in emotional regulation and stress responsiveness further modulates neuroendocrine signals influencing pubertal timing. Early puberty carries significant long-term physical and psychological risks. Addressing this multifactorial condition requires integrated clinical, lifestyle, environmental, and psychosocial interventions.

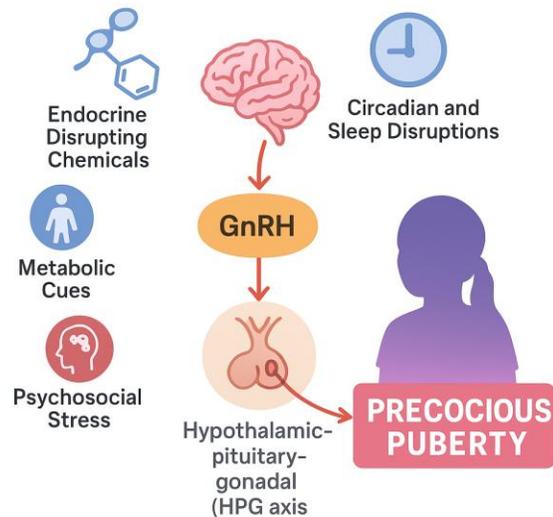


Figure 1: Schematic illustrating the GnRH pulse generator in the arcuate nucleus regulated by KNDy neurons (kisspeptin, neurokinin B, dynorphin).

Introduction

Puberty is a critical developmental window marked by activation of the hypothalamic–pituitary–gonadal (HPG) axis and emergence of secondary sexual characteristics. While normal pubertal timing ranges broadly, precocious puberty—appearance of pubertal signs before age 8 in girls and 9 in boys—is increasingly reported worldwide. This phenomenon reflects complex interactions among genetic susceptibility, metabolic status, environmental exposures, psychosocial stressors, and circadian rhythm alterations.

This review integrates recent advances in neuroendocrinology, toxicology, nutrition, psychology, and chronobiology to elucidate mechanisms triggering early pubertal onset. We highlight the central role of KNDy neurons as the GnRH pulse generator’s pacemaker and explore how metabolic inputs, EDCs, limbic–HPA stress pathways, and circadian disruption converge on this system.

Physiology of Pubertal Onset: The GnRH Pulse Generator and KNDy Neurons

Gonadotropin-releasing hormone (GnRH) is produced by neurons in the hypothalamus, a region of the brain. These neurons are located in the preoptic area and the arcuate nucleus of the hypothalamus. GnRH is released into the bloodstream and travels to the pituitary gland, where it stimulates the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the anterior pituitary gland [Lehmann, J., & Sönksen, P. H. \(2001\). GnRH neurons and the regulation of puberty. *Frontiers in neuroendocrinology*, 22\(3\), 291-314. <https://doi.org/10.1006/frne.2001.0207>](#). Pubertal onset depends on reactivation of pulsatile secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus. These two hormones then stimulate the gonads, either the ovaries in females or the testes in males, to

produce and secrete sex hormones, such as estrogen and testosterone. These hormones then drive the development of secondary sexual characteristics and the onset of puberty. (Rhees, R. W., & Seeley, R. J. (2017). *Central regulation of puberty. Handbook of clinical neurology*, 145, 389-409.

The KNDy neuronal network is composed of group of neurons co-expressing kisspeptin, neurokinin B (NKB), and dynorphin in the arcuate nucleus, which act as GnRH pulse generatory rhythmic pulsatility of GnRH. Kisspeptin acts as a potent stimulator of GnRH neurons, while dynorphin inhibits them, and NKB modulates the synchronization of the network. Merzenich, H., Schmitz, M., & Rahn, T. (2010). *The hypothalamic regulation of puberty. Progress in brain research*, 185, 79-93.

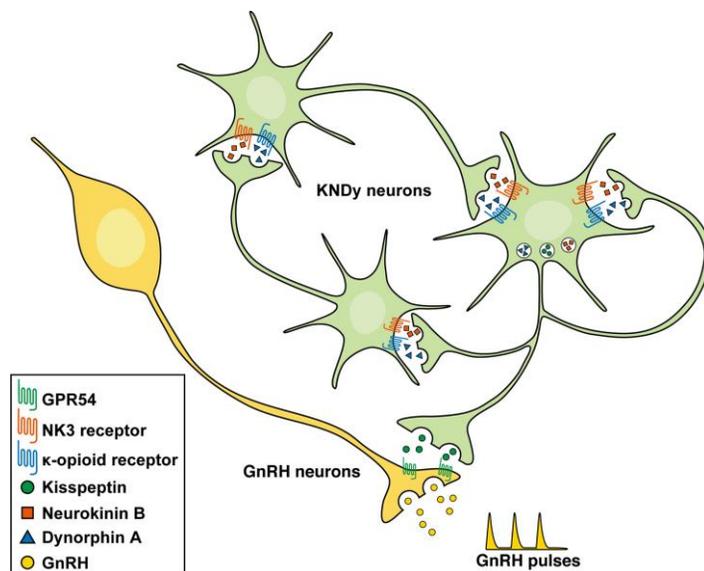


Figure: Mechanism of GnRH pulsation

The production and release of GnRH is regulated by several factors, including:

1. GnRH production can be influenced by various neurotransmitters, including dopamine, serotonin, and norepinephrine. These neurotransmitters act on GnRH neurons in the hypothalamus to regulate GnRH secretion.
2. The levels of estrogen and testosterone can also affect GnRH production and release. High levels of these hormones can inhibit GnRH release, while low levels can stimulate it.
3. Changes in nutritional status can also affect GnRH production and release. For example, low body weight has been shown to increase GnRH secretion, while obesity can reduce it.
4. Hormonal imbalances, such as those caused by hypothyroidism, can also alter GnRH production and release.

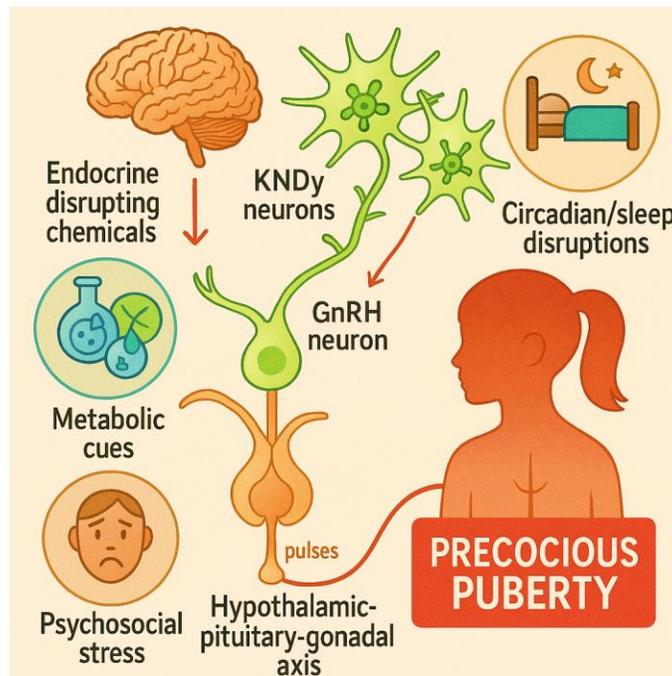
5. Environmental factors, such as stress, can affect GnRH production and release by influencing neurotransmitter levels and other hormonal pathways.

These are some of the main factors that can influence GnRH production and release, but the exact mechanisms are still not fully understood and further research is needed.

Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3816002/>

Disruption or premature activation of this system—due to genetic, epigenetic, or external stimuli—can precipitate early puberty by means of initiation of GnRH pulsation before time. The KNDy neurons integrate feedback from sex steroids and signals reflecting energy balance, stress, and circadian inputs.

Factors that promote Precocious puberty



A. Malnutrition

Nutritional status can play a significant role in the onset of precocious puberty. When children do not receive adequate nutrition, their bodies may respond by accelerating the process of puberty in order to ensure reproductive success later in life. Malnutrition can affect various physiological processes in children, including growth, development, and metabolism. In particular, it can disrupt endocrine function and alter the

production of hormones, such as leptin and ghrelin, which play a crucial role in regulating energy balance and metabolism. In some cases, malnutrition can also lead to changes in the regulation of hormones and the onset of puberty, including the secretion of growth hormone, insulin-like growth factor 1 (IGF-1), and gonadotropins, which stimulate the gonads. However, more research is needed to fully understand the specific mechanisms by which malnutrition can lead to premature puberty, and more studies are needed to establish this relationship conclusively. Additionally, malnutrition can impair immune function, leading to increased susceptibility to infections and inflammation, which can disrupt the normal physiological processes involved in puberty and development.

(World Health Organization. (2020). Nutrition and growth. https://www.who.int/nutrition/topics/nutrition_and_growth/en/

Konishi T, Kitazawa R. (2011). The effects of malnutrition on endocrine function. *Experimental Biology and Medicine*, 236(7), 791-798. <https://journals.sagepub.com/doi/abs/10.1258/ebm.2011.011041>

Several studies have suggested that malnutrition and malnutrition-related conditions, such as underweight or stunted growth, can affect the hypothalamic-pituitary-gonadal (HPG) axis and disrupt the normal timing of puberty. Additionally, malnutrition can cause oxidative stress and inflammation, which can disrupt the normal functioning of the endocrine system and affect pubertal development

Low body weight has been shown to increase gonadotropin-releasing hormone (GnRH) secretion. The exact mechanisms by which this occurs are still not fully understood, but several hypotheses have been proposed.

One theory is that low body weight serves as a signal of environmental stress and nutrient deprivation. When the body senses that resources are limited, it may respond by increasing GnRH secretion in an attempt to maintain normal reproductive function. This is thought to be regulated by the hypothalamic-pituitary-gonadal (HPG) axis, which is a complex network of hormones and neurotransmitters that regulate reproductive function.

Another theory is that low body weight can alter the levels of neurotransmitters, such as dopamine, serotonin, and norepinephrine, which are involved in regulating GnRH secretion. For example, low levels of serotonin have been shown to increase GnRH secretion, while high levels can inhibit it.

Some other studies have suggested that low body weight can also affect the levels of sex steroids, such as estrogen and testosterone, which can also alter GnRH secretion.

Overall, while the exact mechanisms by which low body weight increases GnRH secretion are not fully understood, it is thought to involve a complex interplay of hormones, neurotransmitters, and nutritional status.

Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3816002/>

Stress is associated with nutrient deprivation and poor health. When the body experiences stress, it can respond, including the onset of puberty. The exact biochemical processes involved in this relationship are complex and not fully understood, but it has been established that:

- i. The regulation of puberty is controlled by a delicate balance of hormones, including gonadotropin-releasing hormone (GnRH), luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Low body weight can disrupt this balance and lead to the early release of GnRH, which can trigger the onset of puberty. Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6126934/>
- ii. Low body weight can also activate the adrenal glands, which produce hormones such as cortisol. Cortisol can affect the release of GnRH and other hormones that regulate puberty, leading to precocious onset of maturation. Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4555197/>
- iii. Insulin-like growth factor (IGF-1) is involved in the regulation of puberty and other aspects of growth and development. Children with low body weight often have lower levels of IGF-1, which can contribute to the early onset of puberty. Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6126934/>
- iv. Protein is a key component in the regulation of puberty, and children who do not receive enough protein in their diets may experience precocious puberty as a result. Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4994775/>
- v. Children who consume diets high in processed foods, sugar, and unhealthy fats may be at a higher risk for precocious puberty. This is because these diets can disrupt the delicate hormonal balance necessary for normal puberty onset. Reference: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6126934/>

B. Metabolic Influences: Leptin, Insulin, and Obesity

Childhood obesity is a leading factor associated with early puberty, particularly in girls. Excess adipose tissue increases peripheral aromatase activity, converting androgens into estrogens. Elevated leptin levels, signaling sufficient energy stores, sensitize hypothalamic neurons, lowering the activation threshold for GnRH secretion. [Vickers, M. H., Breier, B. H., Cutfield, W. S., Hofman, P. L., &](#)

Wallace, E. M. (2014). Insulin resistance in obesity, type 2 diabetes, and the metabolic syndrome. *Front Endocrinol (Lausanne)*, 5, 4.

Insulin resistance and increased insulin-like growth factor-1 (IGF-1) also promote ovarian steroidogenesis, advancing sexual maturation. Conversely, chronic malnutrition can also disturb hormonal homeostasis. Reduced IGF-1, altered ghrelin secretion, and overall energy imbalance may prompt adaptive mechanisms that influence HPG axis reactivation. Historical and population-based studies reveal that both undernutrition and overnutrition disrupt normal pubertal timing.

World Health Organization. (2020). Nutrition and growth. https://www.who.int/nutrition/topics/nutrition_and_growth/en/

Konishi T, Kitazawa R. (2011). The effects of malnutrition on endocrine function. *Experimental Biology and Medicine*, 236(7), 791-798. <https://journals.sagepub.com/doi/abs/10.1258/ebm.2011.011041>

a. Dietary Patterns and Chemical Intake Through Food

In recent decades, changes in diet composition and food processing have introduced new factors that affect hormonal balance, particularly involving estrogen metabolism and endocrine function. These dietary elements interact complexly with the gut microbiota and endocrine systems, collectively impacting hormone regulation.

i. Ultra-Processed Foods and Gut Microbiota Alteration

Ultra-processed foods are commonly enriched with preservatives, emulsifiers, and artificial additives designed to extend shelf life and improve texture. These compounds can disrupt the gut microbial ecosystem, known as the gut microbiota, which plays a critical role in metabolizing estrogens. The gut bacteria express enzymes such as β -glucuronidase that regulate the estrobolome—the collection of microbial genes capable of metabolizing estrogens.

Disruption of this microbial balance (dysbiosis) can lead to altered estrogen metabolism, influencing the levels of circulating estrogens and their activity in the body. Such shifts may contribute to hormonal imbalances that can affect reproductive health and potentially increase risks for estrogen-sensitive conditions. Lustig, R. H. (2009). The toxic truth about sugar. *Nature*, 458(7239), 757-764.

Corvalán, C., Uauy, R., & McMichael, A. J. (2013). How susceptibility to the obesity epidemic varies globally. *Obes Rev*, 14 Suppl 2, 22-33.

Rodríguez-Bernal, C. L., Palou, M., Sánchez-Villegas, A., García, A., & Martínez-González, M. A. (2017). Processed food consumption and risk of obesity, type 2 diabetes and cardiovascular disease. *Crit Rev Food Sci Nutr*, 57(7), 1445-1461.

ii. Pesticide Residues with Estrogenic and Anti-Androgenic Activities

Fruits and vegetables, even when healthy in general, may carry residues of pesticides known as endocrine-disrupting chemicals (EDCs). Many pesticides act as xenoestrogens—chemicals that mimic or interfere with natural estrogens—and some exhibit anti-androgenic effects, inhibiting male hormone activity. These compounds can bind to hormone receptors, disrupting normal hormone signaling pathways, thereby altering reproductive hormone balance, fertility, and possibly contributing to developmental and metabolic disorders.

iii. Excessive Consumption of Phytoestrogens

Phytoestrogens are naturally occurring plant compounds found in foods like soy, flaxseed, and legumes. These compounds, including isoflavones and lignans, structurally resemble human estrogens and can bind to estrogen receptors, though with much weaker affinity.

When consumed in very high amounts, phytoestrogens may exert mild estrogenic effects, which could influence hormone-sensitive tissues. While moderate intake is generally considered beneficial, excessive consumption might affect menstrual cycles, fertility, or the risk of hormone-related cancers in susceptible individuals.

iv. High Sugar Diets and Insulin-Mediated Hormonal Effects

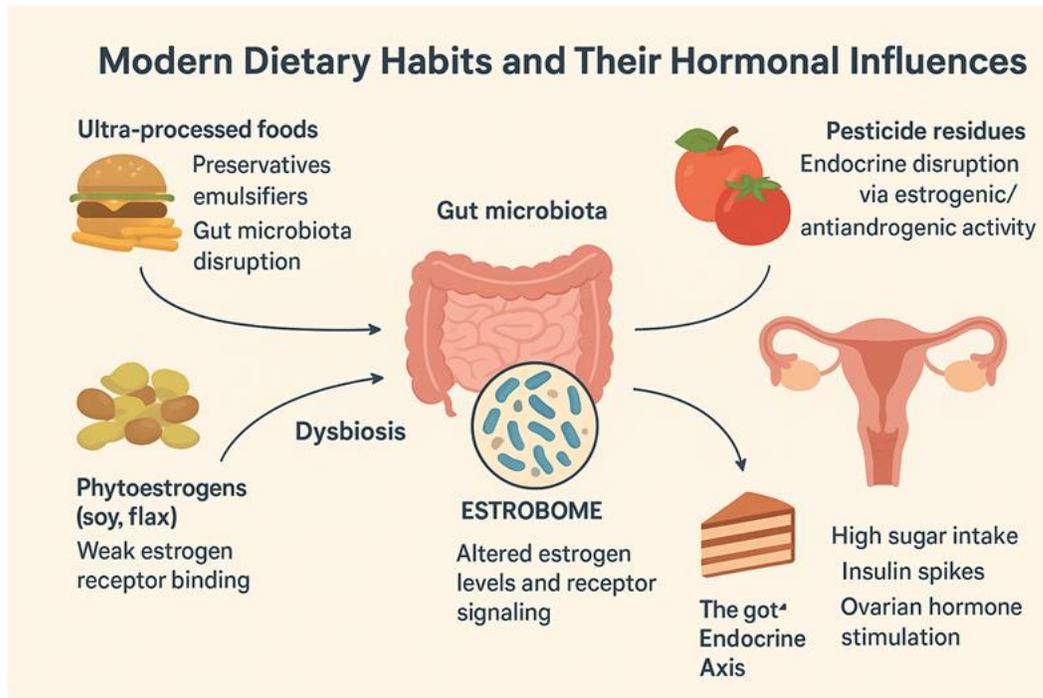
Diets high in refined sugars and simple carbohydrates cause rapid spikes in blood glucose, leading to increased insulin secretion. Insulin, beyond its metabolic role, acts as a hormone that can influence the hypothalamic-pituitary-ovarian axis by stimulating ovarian theca cells to produce androgens, which are then converted to estrogens by granulosa cells.

Chronic hyperinsulinemia may thus elevate ovarian hormone production, contributing to conditions such as polycystic ovary syndrome (PCOS) characterized by hormonal imbalance, irregular menstruation, and infertility.

v. The Gut–Endocrine Axis: A Crucial Mediator

Emerging research highlights the gut–endocrine axis, where gut microbiota interacts with the endocrine system to regulate hormone levels and actions. The estrobolome subset of gut bacteria is particularly involved in estrogen metabolism by deconjugating estrogens excreted into the gut, allowing their reabsorption and recycling (enterohepatic circulation).

Dysbiosis, or an imbalance in gut microbiota, can impair estrobolome function, resulting in disrupted estrogen recycling. This leads to altered systemic estrogen levels, impacting reproductive health, metabolic regulation, and susceptibility to hormone-related diseases.



Environmental Exposures

A. Endocrine Disrupting Chemicals (EDCs)

Children have different levels of hormones compared to adults. For example, children have higher levels of growth hormone, which promotes growth and development, and lower levels of sex hormones, such as testosterone and estrogen, compared to adults. Their endocrine systems respond differently to stimuli compared to adults. For example, children's insulin sensitivity and glucose tolerance is lower compared to adults, which may contribute to differences in metabolism and energy balance.

Children's endocrine systems are undergoing rapid development and change as they grow and mature. For example, the onset of puberty is associated with changes in hormone levels and the activation of the hypothalamic-pituitary-gonadal (HPG) axis, which regulates the production and secretion of hormones related to sexual maturation. Children are more susceptible to the effects of environmental factors, such as endocrine-disrupting chemicals (EDCs), due to their still-developing endocrine systems and higher exposure levels. EDCs have been shown to alter the timing of puberty and disrupt hormone balance in children.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5513855/https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4890687/>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4126201/>

Endocrine-disrupting chemicals (EDCs) are synthetic or naturally occurring substances that interfere with the normal functioning of the endocrine system—the body’s complex network responsible for hormone production, regulation, and signaling. Hormones regulate a wide range of physiological processes, including growth, metabolism, reproduction, and immune responses. When EDCs enter the body, they can disrupt hormone balance by mimicking natural hormones, blocking hormone receptors, altering hormone synthesis, or modifying hormone metabolism and clearance. This disruption can lead to adverse health effects such as reproductive disorders, developmental abnormalities, metabolic syndromes, immune dysfunction, and even increased risk of certain cancers. :

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5513855/>

EDCs are pervasive in the environment due to widespread industrial and agricultural use, as well as their presence in everyday consumer products. Some of the most common and well-studied EDCs include:

a. **Bisphenol A (BPA):**

BPA is a chemical primarily used in manufacturing polycarbonate plastics and epoxy resins. It is commonly found in food and beverage containers, such as water bottles and the linings of canned foods and beverages. BPA can leach into food and drinks, especially when containers are heated or damaged. Structurally similar to estrogen, BPA can bind to estrogen receptors, disrupting normal hormonal signaling. Studies have linked BPA exposure to reproductive abnormalities, altered brain development, and increased risk of metabolic disorders.

Hauser R, Meeker JD, Calafat AM, et al. Phthalates and couple fecundity: a prospective study of 1,101 couples from a fertility clinic. *Environmental Health Perspectives*. 2010;118(3):289-294.

Trasande L, Attina TM, Blustein J. Association Between Urinary Bisphenol A Concentration and Obesity Prevalence in Children and Adolescents. *JAMA*. 2012;308(11):1113-1121.

Johnson-Down L, O'Loughlin J, Garant MP, et al. Sugar-Sweetened Beverage Consumption and Cardiovascular Risk in First Nations Children and Youth: A Systematic Review. *Nutrients*. 2018;10(11):1649.

b. **Phthalates:**

Phthalates are a group of chemicals used as plasticizers to increase the flexibility and durability of plastics. They are commonly found in personal care products like shampoos, lotions, and perfumes, as well as in children’s toys, medical tubing, and packaging materials. Phthalates can interfere with androgen hormones, critical for male reproductive development. Exposure has been associated with reduced fertility, developmental defects in male reproductive organs, and potential impacts on thyroid function. Hauser R, Meeker JD, Calafat AM, et al. Phthalates and couple fecundity: a prospective study of 1,101 couples from a fertility clinic. *Environmental Health Perspectives*. 2010;118(3):289-294.

Trasande L, Attina TM, Blustein J. Association Between Urinary Bisphenol A Concentration and Obesity Prevalence in Children and Adolescents. *JAMA*. 2012;308(11):1113-1121.

Johnson-Down L, O'Loughlin J, Garant MP, et al. Sugar-Sweetened Beverage Consumption and Cardiovascular Risk in First Nations Children and Youth: A Systematic Review. *Nutrients*. 2018;10(11):1649.

c. Parabens:

Parabens are preservatives widely used in cosmetics, toiletries, and some food products to prevent microbial growth. Common parabens include methylparaben and propylparaben. They possess weak estrogenic activity and can mimic natural estrogen by binding to estrogen receptors. Chronic exposure to parabens has raised concerns about their potential role in breast cancer development and endocrine-related reproductive issues. Hauser R, Meeker JD, Calafat AM, et al. Phthalates and couple fecundity: a prospective study of 1,101 couples from a fertility clinic. *Environmental Health Perspectives*. 2010;118(3):289-294.

Trasande L, Attina TM, Blustein J. Association Between Urinary Bisphenol A Concentration and Obesity Prevalence in Children and Adolescents. *JAMA*. 2012;308(11):1113-1121.

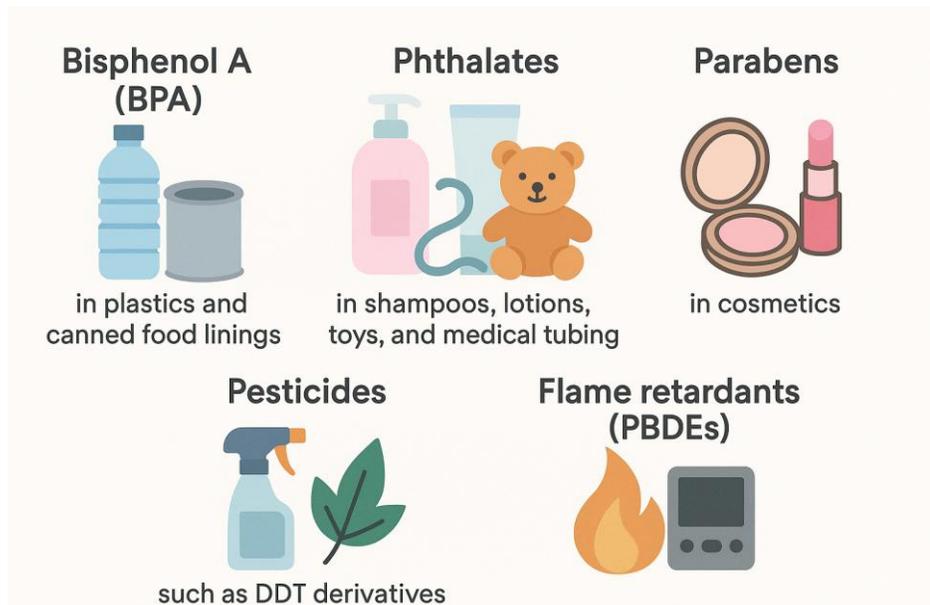
Johnson-Down L, O'Loughlin J, Garant MP, et al. Sugar-Sweetened Beverage Consumption and Cardiovascular Risk in First Nations Children and Youth: A Systematic Review. *Nutrients*. 2018;10(11):1649.

d. Pesticides (such as DDT derivatives):

Certain pesticides, including dichlorodiphenyltrichloroethane (DDT) and its breakdown products, are potent endocrine disruptors. Despite bans or restrictions in many countries, these persistent organic pollutants remain in the environment due to their long half-lives. DDT and related compounds can mimic estrogen or interfere with androgen signaling, leading to reproductive abnormalities, developmental delays, and wildlife population declines. Pesticide exposure has also been linked to neurodevelopmental disorders in children.

e. Flame Retardants (Polybrominated Diphenyl Ethers, PBDEs):

PBDEs are used in furniture, electronics, textiles, and building materials to reduce flammability. These compounds bioaccumulate in the environment and in human tissues. PBDEs can disrupt thyroid hormone homeostasis, which is critical for brain development and metabolism. Exposure to PBDEs has been associated with cognitive impairments, behavioral changes, and thyroid dysfunction.



These chemicals accumulate over time. Even low-dose, chronic exposure during early childhood—or even in utero—can alter epigenetic markers that regulate puberty-related genes.

B. Indoor Air Quality and Urban Exposure

In modern lifestyles, children tend to spend a significant portion of their time indoors—in homes, schools, and recreational centers—where they are exposed to a variety of indoor pollutants. Unlike outdoor air pollution, which has been extensively studied, indoor pollutants often reach higher concentrations due to confined spaces and limited ventilation. These pollutants include volatile organic compounds (VOCs), microplastic particles, secondhand smoke, and airborne phthalates. Emerging evidence indicates that these compounds can interfere with critical hormone pathways, particularly adrenal steroidogenesis and estrogen metabolism, which are pivotal in regulating puberty and overall endocrine function.

- a. **Volatile Organic Compounds (VOCs):**
 VOCs are a large group of carbon-based chemicals emitted as gases from products like furniture, paints, varnishes, adhesives, and cleaning agents. Common VOCs include formaldehyde, benzene, and toluene. When inhaled, VOCs can enter the bloodstream and influence hormone-producing glands such as the adrenal glands and ovaries. Specifically, VOC exposure can disrupt adrenal steroidogenesis—the process by which adrenal glands synthesize steroid hormones like cortisol and androgens—which play a role in the timing of puberty and stress responses. Moreover, VOCs can affect enzymes involved in estrogen metabolism, leading to altered levels of circulating estrogens.
- b. **Microplastic Particles:**
 Microplastics are tiny plastic fragments (less than 5 millimeters) that result from the breakdown of larger plastic items or are directly introduced through consumer products. Indoors, microplastics can accumulate in dust and air. Due to their small size, these particles can be inhaled or ingested, carrying with them chemical additives such as plasticizers and flame retardants. These additives often have endocrine-disrupting properties, capable of interfering with hormone receptors or

altering hormone synthesis. Exposure to microplastics and their associated chemicals has been linked to disturbances in reproductive hormone balance.

c. **Secondhand**

Smoke:

Tobacco smoke contains thousands of chemicals, many of which are toxic and hormonally active. Children exposed to secondhand smoke indoors absorb substances that can interfere with adrenal and gonadal hormone production. Components of smoke can inhibit or stimulate enzymes that regulate steroid hormone synthesis, thereby disrupting normal pubertal development. Studies have shown associations between secondhand smoke exposure and altered timing of puberty, as well as increased risk of respiratory and metabolic disorders. :

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5513855/>

d. **Airborne**

Phthalates:

Phthalates are plasticizers commonly found not only in products but also as airborne contaminants indoors, especially in dust and aerosols. Inhalation of airborne phthalates leads to systemic absorption, where they interfere with hormone signaling. Phthalates are known to disrupt estrogen and androgen pathways, and in children, this interference can skew the delicate hormonal balance that governs puberty onset. Research has linked phthalate exposure to premature thelarche (early breast development) and other signs of early puberty.

The combined exposure to these indoor pollutants creates a biochemical environment in children that can accelerate the onset of puberty. Disruption of **adrenal steroidogenesis** affects the production of hormones like dehydroepiandrosterone (DHEA) and its sulfate (DHEAS), which are involved in adrenarche—the early phase of puberty. Meanwhile, alterations in **estrogen metabolism** influence the timing of puberty is not solely dictated by genetics and physical health but is also significantly influenced by psychosocial and emotional environments. Stress and early life adversity play a pivotal role in modulating neuroendocrine systems that control pubertal onset.

Psychosocial Stress and the Limbic–HPA Axis

Chronic stress leads to sustained elevations in cortisol, the primary stress hormone produced by the adrenal glands. Cortisol is regulated by the hypothalamic-pituitary-adrenal (HPA) axis, which closely interacts with the hypothalamic-pituitary-gonadal (HPG) axis responsible for reproductive development. When cortisol levels remain elevated over time due to adverse psychosocial conditions, the balance between these two axes is disrupted, accelerating pubertal maturation.

Children exposed to stressful environments such as: Family conflict, Parental absence, Emotional neglect, Physical or emotional trauma and/or Socioeconomic instability, often demonstrate earlier pubertal milestones, such as premature breast development, accelerated growth spurts, and earlier menarche. From an evolutionary biology perspective, early maturation in response to environmental stressors may represent an adaptive mechanism to maximize reproductive success in unpredictable or harsh environments. Essentially, early puberty might be the body's way of increasing reproductive potential when the future appears uncertain. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3816002/> McEwen, B.S. (1998). Protective and damaging effects of stress mediators. *New England Journal of Medicine*, 338(3), 171-179.

Epel, E.S., McEwen, B., Seeman, T., Matthews, K., Castellazzo, G., Ickovics, J.R., and Brownell, K.D. (2000). Stress and body shape: stress-induced cortisol secretion is consistently greater among women with central fat. *Psychosomatic Medicine*, 62(5), 611-619. The exact mechanisms by which stress affects GnRH secretion are not fully understood, but several theories have been proposed.

- i. One theory is that stress affects the release of neurotransmitters, such as dopamine, serotonin, and norepinephrine, which are involved in regulating GnRH secretion. For example, stress can increase the release of norepinephrine, which can stimulate GnRH secretion.
- ii. Another theory is that stress can alter the levels of sex steroids, such as estrogen and testosterone, which can also influence GnRH secretion. For example, stress has been shown to increase the levels of testosterone, which can stimulate GnRH secretion.

It's important to note that the effects of stress on GnRH secretion can be complex and depend on the type, duration, and severity of stress, as well as individual differences. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3816002>



Parenting, Attachment, and Family Dynamics

The quality of parenting and family relationships significantly impacts children’s stress responses and hormonal regulation. Warm, secure, and emotionally supportive environments foster stable oxytocin and cortisol patterns that help maintain a balanced neuroendocrine state, delaying premature puberty.

Conversely, harsh, inconsistent, or neglectful parenting styles can dysregulate stress hormone pathways. Altered oxytocin levels—known for their role in social bonding—and heightened cortisol secretion indirectly affect the secretion of gonadotropin-releasing hormone (GnRH), the key hormone triggering puberty. This hormonal imbalance can lower the threshold for pubertal initiation, leading to earlier onset.

Sleep and Circadian Rhythm Disruption

Biological rhythms governed by the circadian clock play a crucial role in coordinating hormonal secretions necessary for normal growth and development, including puberty. Melatonin, a hormone secreted by the pineal gland during darkness, suppresses GnRH secretion in childhood, helping delay pubertal onset. [S. S. Patel, A. J. Zhu, L. V. Kovalchuk, I. Kovalchuk, Light at night, insomnia and breast cancer, J Pineal Res 48 \(2010\), pp. 271–282.](#)

1. S. N. Davis, H. Mirick, R. M. Stevens, J. F. Thomas, Association between light at night, melatonin secretion, sleep deprivation, and the internal distribution of cancers in press (2001).
2. J. E. Herbert, P. G. Kaiserman-Abramof, A. M. Martins, M. I. Tamanaha, M. D. Mill Júnior, A. J. Klahr, C. Krieger, M. P. Zugaib, H. M. Hidalgo, G. B. Colli, M. J. Oliveira, Circadian desynchrony induces decreased pituitary sensitivity to GnRH in a female rat model, *Sci Transl Med* 5 (2013), p. 193ra91.

Blue light is a type of high-energy visible (HEV) light that is found in the blue part of the visible light spectrum. It is a natural component of sunlight, but can also be produced by electronic devices such as smartphones, laptops, and televisions.

Studies have shown that exposure to blue light can affect the circadian rhythm, or the body's internal "biological clock," which regulates the sleep-wake cycle and the secretion of hormones such as melatonin and cortisol. Disruptions to the circadian rhythm, including exposure to artificial light at night, have been linked to a range of health effects, including changes in metabolism, mood, and endocrine function.

There is some evidence to suggest that exposure to blue light, particularly at night, may contribute to early onset of puberty by disrupting the circadian rhythm and altering hormone levels. For example, exposure to blue light at night has been shown to suppress the secretion of melatonin, a hormone that regulates the sleep-wake cycle and has inhibitory effects on the hypothalamic-pituitary-gonadal (HPG) axis, which regulates the production and secretion of hormones related to sexual maturation.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5908672/>

- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4890687/>
- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7379495/>

Exposure to TV has been associated with early onset of puberty in some studies, although the evidence is not strong. It is believed that exposure to sexual content on TV and other media can lead to early onset of puberty in some children. This is because viewing such content can increase levels of hormones like testosterone, which can promote the onset of puberty.

Additionally, TV viewing has been linked to sedentary behavior, which can lead to weight gain and obesity, both of which are associated with early onset of puberty. Furthermore, TV viewing can also disrupt sleep patterns, which can affect the timing of puberty by altering the secretion of hormones like melatonin, which regulates the sleep-wake cycle.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2667214/>

- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5879448/>

The consequences of reduced melatonin include:

- **Earlier sleep disruption** due to difficulty falling asleep or fragmented sleep
- **Nocturnal hormonal imbalance** as the natural nighttime surge of melatonin diminishes
- **Increased adrenal activation** from stress-related pathways during night hours

Together, these changes lower the physiological threshold for initiating puberty, pushing pubertal development earlier than expected.

Excessive evening screen use further exacerbates circadian disruption by impacting:

- **REM sleep cycles**, essential for brain development and hormonal regulation
- **Melatonin amplitude**, reducing its overall nighttime availability
- **Cortisol regulation**, potentially causing elevated nighttime cortisol levels
- **Growth hormone secretion**, which predominantly occurs during deep sleep phases and is crucial for normal growth and development

Children suffering from chronic sleep deprivation or irregular sleep patterns exhibit a higher prevalence of early puberty. Sleep loss effectively stresses the neuroendocrine system, accelerating pubertal processes and influencing physical and emotional health. Melatonin, secreted at night, inhibits GnRH secretion during childhood. Chronic sleep deprivation destabilizes neuroendocrine rhythms, contributing to earlier activation of the HPG axis. Circadian disruption from late-night screen use and irregular sleep patterns is an emerging modifiable risk factor for precocious puberty.

Genetic and Congenital Determinants

Heritable Factors

The timing of puberty is strongly influenced by genetic factors, with studies showing a high degree of heritability. Specific genes have been identified that play crucial roles in regulating the initiation and progression of puberty. For example, *KISS1* and its receptor *KISS1R* encode kisspeptin and its receptor, which are essential for stimulating gonadotropin-releasing hormone (GnRH) neurons in the hypothalamus. This stimulation triggers the cascade of hormonal changes leading to puberty. Variations or mutations in these genes can either advance or delay pubertal onset. Similarly, the *TAC3* gene and its receptor *TACR3* regulate neurokinin B, a neuropeptide that modulates GnRH secretion. Mutations in *GNRHR*, which encodes the GnRH receptor itself, can impair or alter the hormonal signaling necessary for puberty. Lastly, *LIN28B* is implicated in timing biological developmental events, including puberty, and polymorphisms in this gene have been linked to variations in pubertal age. Families with histories of early puberty often display these genetic variants across multiple generations, indicating a strong hereditary influence.

Congenital and Pathological Causes

Certain congenital disorders and pathological conditions can precipitate early puberty by disrupting normal hormonal pathways:

- i. Congenital Adrenal Hyperplasia (CAH): This inherited enzyme deficiency leads to excess production of adrenal androgens from birth. Elevated androgens can cause premature development of secondary sexual characteristics, such as early pubic hair and accelerated growth.
- ii. Hypothalamic Hamartomas: These benign, tumor-like malformations in the hypothalamus can secrete GnRH independently of the body's normal regulatory mechanisms, triggering precocious puberty.
- iii. Central Nervous System (CNS) Lesions or Tumors: Tumors affecting the hypothalamus or pituitary gland can disrupt normal endocrine control, causing early activation of the HPG axis.
- iv. Chronic Endocrine Disorders: Conditions such as hypothyroidism or other hormonal imbalances can interfere with pubertal timing.
- v. Thyroid Dysfunction: Both hypothyroidism and hyperthyroidism affect metabolism and hormonal homeostasis and have been associated with altered pubertal onset.

Advanced neuroimaging, especially magnetic resonance imaging (MRI), has enabled detection of subtle hypothalamic anomalies in children with idiopathic precocious puberty (where no clear cause is identified), aiding diagnosis and treatment.

Medical and Maternal Influences

Maternal Environment

The prenatal environment critically shapes the development of the fetal endocrine system. Several maternal factors have been linked to altered programming of the child's hormonal axis, influencing the timing of puberty:

- i. Maternal Obesity: Excess maternal adiposity increases circulating leptin and inflammatory markers, which may cross the placenta and affect fetal neuroendocrine development, predisposing children to earlier puberty.
- ii. High Fat or High Sugar Diet: Diets rich in unhealthy fats and sugars during pregnancy can lead to metabolic alterations in the fetus, including insulin resistance and altered adipokine signaling, which in turn influence puberty.
- iii. Smoking and Alcohol Use: Both are known teratogens that disrupt fetal brain and endocrine development, increasing risk for earlier pubertal onset.
- iv. Chronic Stress: Maternal stress elevates cortisol levels, which can cross the placental barrier and program the fetal hypothalamic-pituitary-adrenal (HPA) axis, potentially altering later stress responses and puberty timing.
- v. Gestational Diabetes: Elevated maternal glucose affects fetal insulin and growth factor pathways, which are important regulators of growth and development, including puberty.

While some researchers have hypothesized that repeated prenatal ultrasound exposure might influence fetal hormone milieu, current evidence is insufficient to confirm a causal relationship.

Pharmaceuticals and Medical Devices

Children exposed to certain medications or medical devices over prolonged periods may experience altered pubertal timing due to endocrine-disrupting effects:

- i. **Hormonal Medications:** Long-term use of medications containing estrogens, progestins, or androgens can artificially modify the timing of puberty.
- ii. **Steroid-Containing Creams:** Topically applied corticosteroids can be absorbed systemically, especially with frequent or large-area application, potentially suppressing or altering endogenous hormone production.
- iii. **Phthalate-Containing IV Tubing:** Phthalates are known endocrine disruptors that can leach from medical tubing into the body during intravenous therapy, interfering with hormone synthesis and signaling.
- iv. **Certain Antiepileptic Drugs:** Some anticonvulsants can alter liver metabolism of sex steroids or influence neuroendocrine regulation, thereby affecting pubertal onset.

Careful monitoring is warranted for children on these treatments to detect any changes in pubertal development.

Health Implications of Precocious Puberty

Early puberty causes premature closure of growth plates (epiphyseal fusion) in bones, leading to a shorter window for height gain and consequently reduced adult stature. Early hormonal changes can alter fat distribution and insulin sensitivity, increasing the risk of obesity and metabolic syndrome. Insulin resistance associated with early puberty predisposes individuals to type 2 diabetes later in life.

Early exposure to estrogens and androgens may disrupt ovarian function, increasing the likelihood of PCOS, which involves hormonal imbalance and fertility issues. Early menarche extends the duration of estrogen exposure over a woman's lifetime, increasing the risk of hormone-dependent cancers like breast cancer. Early physical maturation often mismatches emotional and cognitive development, leading to psychological challenges, social difficulties, and increased risk for anxiety and depression.

Children undergoing early puberty may engage sooner in behaviors such as substance use, sexual activity, and delinquency, due to social pressures and hormonal influences.

Is it possible to reverse early puberty

In some cases, early puberty can be reversed, but the approach depends on the underlying cause. If early puberty is caused by an underlying medical condition, such as a hormonal imbalance or tumor, treating the underlying condition can help reverse the symptoms of early puberty.

For instance, if a tumor is producing excess hormones that are causing early puberty, removing the tumor can help normalize hormone levels and reverse the symptoms of early puberty. Similarly, if a hormonal imbalance is causing early puberty, hormone therapy or medications can help regulate hormone levels and reverse the symptoms.

In some cases, early puberty may be caused by lifestyle factors, such as being overweight or obese, which can be reversed with weight loss and a healthy diet. Additionally, avoiding environmental factors that can contribute to early puberty, such as exposure to endocrine-disrupting chemicals, can also help reverse the symptoms.

It's important to note that not all cases of early puberty can be reversed and some may require long-term management. If your child is experiencing symptoms of early puberty, it's important to seek medical evaluation to determine the underlying cause and determine the best course of treatment.

Reference:

- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4608358/>
- <https://www.aafp.org/afp/2010/0701/p79.html>
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Management, Prevention, and Lifestyle Interventions

Medical Therapies

The gold standard treatment for central precocious puberty involves administering synthetic GnRH analogues, which desensitize the pituitary gland and temporarily halt puberty progression. This treatment allows children to maintain normal growth rates and improves final adult height. It is reversible and well-tolerated when carefully managed.

Lifestyle and Environmental Measures

Since prevention is always better than cure, therefore it is highly recommended to adopt following strategies:

- i. **Reducing Exposure to Plastics, Pesticides, and Cosmetic Chemicals:** Many everyday products contain endocrine-disrupting chemicals (EDCs) such as phthalates, parabens, and BPA. Limiting the use of plastic containers, avoiding pesticides on foods, and choosing EDC-free cosmetics can reduce the chemical burden that may hasten puberty.
- ii. **Improving Diet Quality with Whole, Unprocessed Foods:** Diets rich in fruits, vegetables, whole grains, and lean proteins support healthy metabolism and reduce exposure to additives and excess sugars that can disrupt endocrine function.
- iii. **Promoting Physical Activity:** Regular exercise helps regulate body weight, insulin sensitivity, and hormonal balance, thereby supporting normal pubertal timing.
- iv. **Regulating Sleep and Limiting Screen Exposure:** Proper sleep hygiene and reduced exposure to artificial light, especially blue light from screens, maintain normal melatonin rhythms, which play a role in delaying puberty onset.

- v. **Creating Emotionally Stable Home Environments:** Reducing chronic stress through supportive parenting and stable family dynamics can normalize cortisol and oxytocin levels, reducing the risk of early puberty.

Public Health and Policy-Level Actions

- i. **Stricter Regulation of Endocrine Disrupting Chemicals (EDCs):** Governments can enforce bans or limits on harmful chemicals in consumer products, improving overall population health.
- ii. **Improved Food Labeling:** Clear information about pesticide residues and chemical additives empowers consumers to make safer dietary choices.
- iii. **Urban Air Quality Measures:** Reducing air pollution can lower children's exposure to airborne endocrine disruptors.

Early Screening in High-Risk Populations: Identifying children at risk for precocious puberty through medical history or environmental exposures enables timely intervention and reduces adverse outcomes.

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2908954/>

- <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4549171/>
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iv.

Conclusion

Precocious puberty arises from an intricate web of neuroendocrine, metabolic, environmental, psychosocial, and circadian influences. The KNDy neuronal network acts as a biological integrator of these signals, and the limbic system plays a crucial role in emotional modulation of pubertal timing. Interdisciplinary strategies combining medical, environmental, nutritional, and psychosocial interventions are essential to address this growing public health concern.

References