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REVIEW ARTICLE

A Review of Per- and Polyfluoroalkyl Substances (PFAS) and Age of Menarche among Adolescent Girls

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ABSTRACT: Per- and polyfluoroalkyl substances (PFAS) represent a group of widespread environmental contaminants that have been linked to several adverse health outcomes related to endocrine disruption, such as the age of menarche. Age of menarche represents an important milestone in pubertal development, and alterations are related to adverse health outcomes. Recent data have shown that the age of menarche has significantly changed over the last few decades, which may be due in part to PFAS exposure; however, the data are mixed. We aimed to investigate the relationship between PFAS exposure and the age of menarche, and associated effects on the endocrine system. We performed an extensive literature review using PubMed and Google Scholar databases and evaluated 30 studies published after the year 2000. Of these, 20 (≈65%) reported a statistically significant association between higher PFAS exposure and altered timing of menarche, most commonly a delay, while 11 (≈35%) reported no clear association. Several longitudinal cohorts reported effect sizes such as hazard ratios for delayed menarche with PFOS exposure (HR = 0.75; 95% CI 0.59-0.96) and increased odds for menarche with PFDA exposure (OR = 2.70; 95% CI 1.32-5.51). This review highlighted current data that demonstrates PFAS adverse events on menstruation and endocrine development. Future studies should focus on clarifying causality and exposure-response relationships to inform public health regulation.

KEYWORDS: Transitional Medical Sciences, Environmental Science, Disease Prevention, PFAS, Menarche, Endocrine Disruption, Adolescence, Puberty.

Introduction

Per- and polyfluoroalkyl substances Per- and polyfluoroalkyl substances, most commonly referred to as "PFAS," represent a group of thousands of man-made chemicals that consist of carbon chains with a characteristic carbon-fluorine bond.[1] Of note, the carbon-fluorine bond is the strongest covalent bond in nature, which confers its durability and resistance to microbial degradation. Their resistance to degradation has made them useful in several industries to make insulation materials, non-stick cookware, cosmetics, and stain-resistant clothing.[2] Despite their utility, their resistance to degradation means that PFAS, once released, can persist for decades in the environment and have the ability to bioaccumulate in animals and humans. PFAS exposure has been suspected of being harmful to living organisms, and in humans, exposure is believed to negatively affect the endocrine system (the hormones in our body). Studies have shown that PFAS negatively affect the thyroid, adrenal glands, pancreas, and gonads, which can result in disruption of the endocrine system. Additionally, PFAS is detectable in the blood and urine of over 80 percent of people, which highlights the importance of understanding the effects of PFAS on individuals and populations.[3] Since PFAS can act as endocrine disruptors, the human body's hormonal systems can be affected. An important clinical indicator and health consequence of endocrine disruption in the general population is the effect of PFAS on menarche in adolescent girls.[1,4,5]

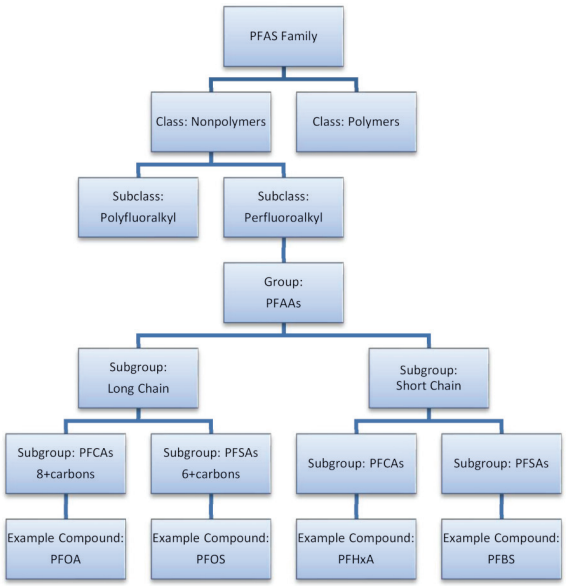

Figure 1: Overview of PFAS classification.[6]

Menarche:

Menarche, defined as the first menstrual period in a biologically female adolescent, is one of the first signs of healthy pubertal development. It occurs between the ages of 10-16, and is based on a 28-day cycle in which, around the 7th day, the female menstruates. Menstruation is the shedding of the uterine lining, and a number of factors determine the age of menarche, including heredity, nutritional intake, physical well-being, and

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exercise.[7] Menarche is a pivotal point in the development of biological females, as it shows the body has matured to a point of fertility. Over time, studies have shown that the age of menarche has significantly fluctuated over the years, and this fluctuation is believed to be due in part to exposure to environmental toxicants. One such important group of environmental toxicants, PFAS, may be implicated, and current investigations are underway to evaluate the effects of PFAS on reproductive health in women. [7-9]

Per- and polyfluoroalkyl substances and menarche:

While several recent studies have implicated PFAS in hormonal disruption (e.g., estrogen receptor inhibition, steroid hormone impairment, and dysregulation), the relationship between PFAS and women's menstrual health is currently under investigation. Recently, the focus of PFAS in the realm of women's health has come into view, especially because PFAS contaminates several women's beauty products. For example, numerous articles have shown that PFAS are readily quantified in sanitary products such as tampons and pads. Some of the companies with products known to contain PFAS in significant quantities are "Procter & Gamble" and "Kimberly-Clark." P&G owns both "Tampax", a brand of tampons, and "Always", a brand of sanitary napkins, and Kimberly Clark owns "Kotex", a brand of both pads and tampons. [10, 11] This finding is concerning since PFAS has been seen to affect many parts of the human body, and its presence in sanitary products means that there is direct contact with skin and the possibility of internal absorption. In this systematic review, the authors set out to summarize and critique the current body of literature that investigates the effects of PFAS on the menstrual cycle and hormonal balance.

Proposed mechanisms include activation of peroxisome proliferator-activated receptors (PPARs), interference with estrogen and androgen receptor signaling, altered steroidogenesis, and disruption of the hypothalamic-pituitary-gonadal axis feedback. These molecular pathways could shift the timing of puberty and menarche.[4, 10]

Shifts in age at menarche have population-level consequences: early menarche is also associated with higher risks of breast cancer,[12] type 2 diabetes,[13] and obesity,[14] while delayed menarche can signal endocrine dysfunction and impact fertility. Understanding environmental drivers such as PFAS is thus critical for reproductive and long-term health. Despite growing awareness of PFAS as endocrine disruptors, no recent review has synthesised evidence specifically on PFAS exposure and the timing of menarche in adolescent girls. This paper addresses the gap by systemically summarising and evaluating the available literature.

Methods

We performed an extensive literature review using PubMed and Google Scholar databases. Our initial search terms included combinations of words including "menarche," "PFAS," "Per- and polyfluoroalkyl substances," "adolescence," "puberty," and "menstruation," which returned 572 papers. From each included article, we extracted: publication year, study design (cross-sectional, longitudinal, case-control), sample size, geographical location, PFAS compounds assessed, exposure pathways studies (e.g., water, food, cosmetics, methods of PFAS measurement, reproductive or endocrine endpoints evaluated (e.g., age at menarche, estradiol, DHEAS), and whether the study reported a statistically significant association between PFAS exposure and menarche timing. This resulted in a final number of thirty-one papers evaluated. All thirty-one articles meeting these inclusion criteria were analysed; in the Results, we highlight studies that directly assessed PFAS exposure and age at menarche while also summarizing additional papers that examine related endocrine and pubertal outcomes to provide context.

Results

Below, we summarise the main findings from the 31 included studies, grouped by exposure pathways and reported effects on pubertal development.

PFAS exposure and common pathways in women:

The widespread use of PFAS and their long half-lives contribute to their long-term toxicity and overall health impacts. Many studies show that up to 80% of people have detectable levels of PFAS.[2] The most environmentally ubiquitous PFAS of biological importance are PFOS, PFOA, PFHxS, PFNA, and PFDE.[15, 16] PFAS exposure patterns vary by several factors, including geography, age, race/ethnicity, occupation, and socioeconomic status.[17] In some countries, mean PFAS exposures exceed 2 ng/mL, which is above the threshold set by the EPA for adverse effects. Most commonly, women are exposed to PFAS through contaminated drinking water, but also through contaminated seafood, air pollution, cooking with non-stick pans, and female beauty products. Lifetime exposure to PFAS begins as early as *in utero*, where PFAS are able to cross the placental barrier and be absorbed by the fetus. In early development, it has been shown that PFAS is transmitted through breast milk from mothers to infants, and these early exposures can have long-term effects on women as they develop into puberty.[17, 18]

As previously mentioned, drinking water is a primary exposure pathway to PFAS. There have been several studies highlighting drinking water contamination with PFAS. A 2019 study showed that from a sample from the Mid-Ohio Valley, a mean of 3.55 ng/ml of PFOA was reported.[5] Another study shows that the guideline calculated for immunotoxicity in pediatrics in PFOS is 1.3 ng/ml, and PFOA is 0.3 ng/ml.[1] Health officials and regulatory agencies estimate that 98 percent of the United States population has detectable PFAS levels in their blood, and 200 million people have been drinking contaminated water.[2]

Another common exposure pathway of PFAS is through food contamination. A study in 2009 showed that in a study of 20 different food items, multiple types of PFAS, such as PFOS, PFOA, and PFHxA, were detected, and PFOS had the highest level of detection.[19, 20] PFOA was detected in 8 of the items, and PFHxA was found in raw veal, sausages, chicken nuggets, and packaged lettuce. A study in Tarragona County,

Spain, found PFOA and PFHpA in food samples and 7 out of the 13 PFAS in the blood samples of the residents.[19] Another study conducted in 2023 by Han *et al*. stated that PFAS accumulated in oil crops, such as soybean, sesame, and peanuts, during their growth stage, and animal-based oil raw materials, like butter and fish oil, which also means the meat and dairy products used for the production are contaminated.[21]

Across the epidemiologic literature, studies using serum PFAS concentrations[17] and longitudinal follow-ups[21] were more likely to detect significant associations than small cross-sectional studies relying on single-time-point urine measures. Geographic differences may also explain mixed findings, as exposure levels in the U.S. and European cohorts often exceed those in Asian studies.

Study Findings and Quantitative Summary:

This section synthesises quantitative epidemiologic evidence on PFAS exposure and pubertal timing, with emphasis on menarche.

Overall, of the 31 studies reviewed, 20 reported a statistically significant association between at least one PFAS and altered menarche timing (commonly delayed menarche), while 11 found no significant relationship. The most frequently studied compounds were PFOS and PFOA, followed by PFNA and PFDA. Reported effect estimates include odds ratios up to 2.70 (PFDA) for early menarche and hazard ratios as low as 0.75 for delayed menarche with PFOS exposure. Sample sizes ranged from fewer than 100 participants in some biomonitoring studies to more than 800 in large longitudinal cohorts.

Several other studies reinforced these findings. One study reported that higher serum PFOS and PFNA were associated with reduced testosterone and IGF-1 levels, which can delay pubertal onset and progression.[21] Others observed that girls in the highest quartile of PFOS exposure experienced menarche later than peers in the lowest quartile,[17] and that lower sex hormone-binding globulin (SHBG) among adolescents with greater PFAS exposure, a hormonal change associated with pubertal timing.[22] Together, these studies strengthen the evidence that PFAS may disrupt endocrine signaling critical to menarche.

A study done in 2024, analyzing 231 cosmetic products from 80 brands purchased between 2019 and 2021. The total mass of PFAS in cosmetics sold in California was estimated to be 1.1 to 1.9 tonnes per year. The results of the analysis showed that 6-8% of the products tested contained at least 1 PFAS at a concentration greater than 100 ppm. The highest concentrations were found in foundation, measuring up to 10500 ppm, concealers, and mascaras, of up to 8200 ppm.[23]

There have been several studies that have investigated the relationship between PFAS exposure and pubertal development in women, though the data are mixed. A longitudinal study found that exposure to some of the most commonly found PFAS (e.g., perfluorooctanoate, perfluorooctane sulfonate, and perfluorooctanoate) led to significantly altered levels of important steroid hormones that regulate the menstrual cycle in girls. For example, girls in the 4th quartile of PFDA and PFUnDA exposure had a higher odds of early menarche, compared to those in the 1st quartile. (OR = 2.70 for PFDA and OR = 2.39 for PFUnDA. PFAS was positively associated with dehydroepiandrosterone sulfate (DHEAS) in 435 girls. A sensitivity analysis conducted evaluating only girls without hormonal contraceptives and adjusting for BMI showed PFOS (B= 1.65, 95% C.I. [0.16, 3.14]), PFNA (B= 0.60, 95% C.I. [0.07,1.14]), and PFDA (B= 1.32, 95% C.I. [0.27, 2.36]) were positively associated with DHEAS.[18]

Another longitudinal study, from 2023,[17] focused on school-age girls in Greater Cincinnati and the San Francisco Bay Area, evaluating age of thelarche (the start of adult breast development), pubarche (the initial appearance of pubic hair), and menarche, all a pivoting point for female reproductive capacity. Several PFAS were associated with adverse changes in the age of menarche. For example, girls in the 3rd quartile of PFOS exposure had a statistically significant increase in age of menarche compared to those in the 1st quartile of exposure (HR= 0.75, 95% C.I. [0.59,0.96]). Additionally, increasing levels of PFOA among girls were associated with a delay in menarche (HR= 0.04, 95% C.I. [0.01,0.25]).[17]

C **Age of Menarche**

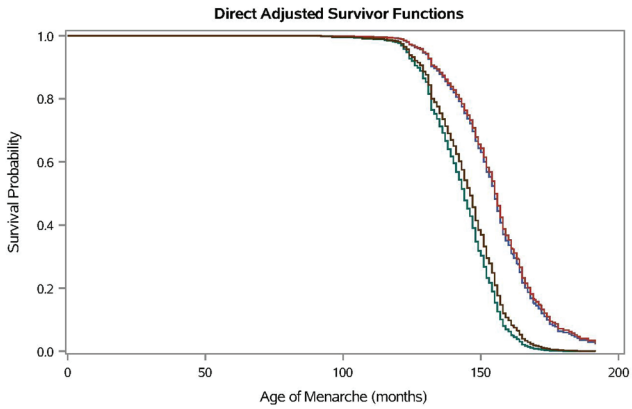

Figure 2: Impact of PFOA Exposure on Age at Menarche: Survival Analysis[17] Girls with higher PFOA exposure (red and brown lines) experienced a delay in menarche compared to those with lower PFOA levels (green and blue lines). The high PFOA group showed survival probabilities above 50% beyond 150 months of age, suggesting that PFAS exposure may disrupt the normal pubertal timing by interfering with hormonal signals necessary for initiating menstruation.

Some studies have shown a positive correlation between reducing PFAS exposure and improvement in reproductive outcomes. One study investigated the relationship between the reduction of PFAS levels in drinking water and fertility outcomes in Minnesota. The study compared areas that had water filtration facilities installed and areas that did not. The area that had not had filters installed showed significantly lower average birth weight (OR = 1.36, 95% C.I. [1.25, 1.48]), preterm births (OR = 1.14, 95% C.I. [1.09, 1.19]), and gestational age. The average reproductive rate was also significantly lower when PFAS exposures were high (Incident rate ratio = 0.73, 95% C.I. [0.69, 0.77]).[24]

Other cohort studies have evaluated the association between PFAS, reproductive hormones, and the age of menarche in large, prospective designs. The authors measured the relationship between PFAS and estradiol in children ages 6-9 who

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were living near a chemical plant in Ohio. It was found that median concentrations of PFHxS, PFOA, PFOS, and PFNA were 7, 30, 21, and 1.7 ng/mL among girls. Regression analyses showed that PFOS and PFNA were associated with lower serum percent levels of testosterone (-6.6%, 95% C.I. [-10.1, -2.8%]) and Insulin Growth Factor-1 (IGF-1) (-3.8%, 95% C.I. [-6.4,-1.2]), respectively, which serve as important hormones for proper pubertal development.[21] In another study, authors found that mean values of sex hormone-binding globulin (SHBG) decreased significantly among participants who were in the greater than 50th percentile exposure levels of PFOA, PFOS, and PFUA compared to individuals in the <50th percentile exposure groups.[22] In totality, these studies have shown moderate to strong relationships between population-level PFAS exposures and adverse effects on sex hormone levels, pubertal development, and subsequent age of menarche.

■ Discussion

Our review shows that nearly two-thirds of the available studies report a measurable association between PFAS exposure and altered timing of menarche, suggesting that endocrine disruption from PFAS may have meaningful developmental consequences. Importantly, this evidence base has expanded rapidly in the last five years, highlighting an emerging but still methodologically heterogeneous field. Variability in exposure metrics, geographical differences, and small sample sizes likely contribute to conflicting findings.

Recap:

PFAS represent one of the most common groups of environmental toxicants that act as endocrine disruptors, and their effects from early exposure can have lifetime implications. This systematic review shows that PFAS exposure significantly alters the timing of menstruation, and this can, in turn, be related to adverse fertility and health outcomes among women.

Reducing:

PFAS exposure Given the strong evidence of PFAS toxicity, it's important to be mindful of ways to reduce PFAS exposure. Our findings suggest that mitigation efforts - such as water filtration and regulation of PFAS in cosmetics - may benefit reproductive health. However, global production of novel PFAS analogues continues, and regulatory actions often lag behind emerging evidence.

Studies have shown that the primary pathways of exposure to PFAS include contaminated drinking water and food, cosmetic use, clothing, and occupational exposures. A study showed that the highest PFAS concentrations within cosmetics were in foundation, concealer, and mascara.[23] Several current studies are investigating policies and social practices that are effective at lowering PFAS levels. For example, communities in Minnesota with filtered drinking water had lower water PFAS levels and improved fertility outcomes compared to similar surrounding areas without filtered water and higher PFAS levels.[24] Additionally, to combat PFAS contamination on a global scale, there have been several policies implemented over the last fifteen years, which have resulted in significantly lower PFAS levels. For example, the United States Environmental Protection Agency (U.S. EPA) implemented a task force in 2022 focused on destroying and removing PFAS-contaminated waste.[25] These practice changes and policy initiatives have shown promise in lowering PFAS levels and improving associated health outcomes. Future studies should continue to evaluate the effectiveness of these practices and ultimately implement them in various populations.

Other PFAS-related health effects:

In addition to perturbing the onset of menarche, numerous studies have found that PFAS is also associated with several other adverse health effects. For example, individuals who are exposed to PFAS and are at high risk for type 2 diabetes had elevated glycemic indicators (fasting proinsulin levels, glycated hemoglobin) and an overall higher risk of developing diabetes.[12] Additionally, PFAS exposure increases the risk of hypertension and obesity.[14,26] Breast cancer risk is multifactorial, with the possibility that PFAS exposure plays a contributing role.[14] These studies highlight the overall disease burden posed by PFAS exposure and add further impetus to finding ways to regulate our production and use of PFAS to create a healthier population.

Health economic burden:

The additional health effects related to PFAS exposure cause a significant strain on the healthcare system, not only in relation to disease burden, but also economic cost. For example, one study showed that $237.55 per capita of the total healthcare costs were attributable to childhood obesity, and a rough total of $13.62 billion and $49.02 billion of direct and indirect cost have been estimated to be spent annually by 2050.[27] Additionally, the healthcare costs related to fertility treatment has continued to rise, and as previously mentioned PFAS is strongly implicated in fertility issues.[28] Future studies should continue to evaluate the economic savings related to reducing PFAS exposure, as a means to lower healthcare costs and ultimately provide more resources to communities and individuals.

Future directions:

There have also been new legislation proposals to help regulate PFAS levels.[6,29] The EPA has imposed increasing regulation of PFAS in drinking water over the last few years in response to the overwhelming data showing adverse effects of PFAS on the environment and in humans. Despite the current literature, there still exists a need to continue research on the health effects of PFAS. Of note, many of the current studies only have toxicological data on several highly studied PFAS, whereas there remain hundreds of PFAS chemicals that have not been evaluated for their potential health effects on humans.[8] It is also important to continue funding research to evaluate the joint effects of exposures (e.g., metals, plastics, pesticides) on female health.

■ Conclusion

This review paper shows how PFAS remains a significant public health issue and is especially harmful to the health of

developing women. In response to the awareness of harmful environmental and biological effects of PFAS, several important regulatory policies and public health interventions have emerged. Future directions should employ harmonized PFAS measurement methods, larger multi-ethnic cohorts, and longitudinal hormone tracking to clarify dose-response effects. Limitations of current evidence include small sample size, cross-sectional designs, inconsistent adjustment for confounders such as BMI and socioeconomic status, and focus on a limited subset of PFAS while hundreds remain untested. Addressing these gaps will strengthen causal inference and guide policy.[30]

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