Children face unique vulnerabilities to environmental hazards at every stage of life



Fragile Beginnings



Children today face a new set of challenges that were unimaginable just a generation ago. Across the world, climate change and environmental degradation are threatening child survival, health and well-being. Given children's unique metabolism, physiology and developmental needs, no group is more vulnerable to environmental harm. Exposure can impact children early and have a lifelong effect.

The Fragile Beginnings series examines the growing body of research on the unique vulnerabilities of children: in utero and at birth, during infancy and childhood and throughout adolescence. Its purpose is to enable evidence-based advocacy and action on children's environmental health.

The series is comprised of three briefs

- 1. Pregnant women and the developing fetuses' unique vulnerabilities to environmental hazards
- 2. Children's unique vulnerabilities to environmental hazards
- 3. Adolescents' unique vulnerabilities to environmental hazards

Each brief is divided into three parts (a) key messages, (b) examples of environmental hazards affecting children, and (c) a technical summary based on a review of the scientific literature.

Videos exploring children's unique vulnerabilities



<u>How climate change</u> <u>impacted Aisha's childhood</u>



This is why extreme heat is life-threatening for children



How the unsound management of chemicals poisons children



1. Pregnant women and the developing fetuses' unique vulnerabilities to environmental hazards



This section focuses on the impact of environmental hazards on developing fetuses. The specific effects of environmental hazards on a pregnant woman's own health are critical but beyond the scope of this document.

Key messages

- Every child has the right to a healthy start in life.
 But environmental hazards can cause harm before a child is born or even conceived.
- Environmental hazards can damage the egg or sperm cells long before conception takes place, and the effects can be passed down from generation to generation.
- The fetal period is critical as it shapes a child's lifelong health and development.
- There are critical windows of vulnerability to environmental hazards, particularly during the first trimester, when women might not be aware of their pregnancies.
- Adverse birth outcomes such as miscarriages, stillbirth, preterm births, birth defects and low birthweight have been linked to exposure to environmental hazards.
- A fetus's developing cells, organs and tissues are uniquely vulnerable to damage from environmental hazards.
- The consequences can last a lifetime, including health conditions like obesity, cognitive impairment, neurological disorders, lung disease and cancer which can arise in infancy into late adulthood.
- While all women are at risk, the heaviest burden falls upon women in low- and middle-income countries, especially those living in fragile and conflict-affected situations, deepening inequities between groups.
- Preventing exposure to environmental hazards can ensure that future generations live to their full potential. It can also prevent economic loss to countries, including to health systems.

Examples of environmental hazards and how they affect pregnancy

Climate-related hazards

Malaria and Zika

Malaria infection rates in the first trimester of pregnancy are especially high. The parasites that cause malaria can cause severe anaemia in the pregnant woman, pregnancy loss and maternal death. Malaria can also cause babies to be born prematurely and have low birthweight. Children born after antenatal exposure to malaria may have impaired cognitive function.

While pregnant women are no more likely to acquire Zika virus than other women, the virus can seriously harm the fetus's developing nervous system.

Zika virus infection during pregnancy can be devastating, causing pregnancy loss or resulting in babies being born prematurely. Babies who survive may suffer from congenital Zika syndrome, which includes malformation of the head and eyes and significant developmental delays.

Even babies who do not have obvious defects at birth can still present with cognitive and language neurodevelopmental delay in childhood.

Cholera

Cholera is a severe diarrhoeal infection caused by ingesting food or water contaminated with the bacterium *Vibrio cholerae* and can cause dehydration. When a woman is pregnant, severe dehydration can cause problems for the pregnant woman and fetus, including compromised blood flow to the placenta and harmful levels of acid in the amniotic fluid, which can lead to pregnancy loss.

Extreme heat

During pregnancy, a pregnant woman's body adapts to many metabolic changes and maintains a steady and appropriate temperature. But in conditions of extreme heat, a pregnant woman's ability to regulate a healthy temperature can be overwhelmed.

Extreme heat can contribute to dehydration, which can reduce levels of amniotic fluid that protect the fetus.

Extreme heat is associated with preterm birth. It has also been linked to low birthweight, congenital anomalies and gestational diabetes.

Food insecurity

A pregnant woman is especially vulnerable to food insecurity because she needs more nutritious food (both in terms of quantity and quality) so her body can both support the growing fetus and continue to function properly. Climate hazards can reduce the amounts of nutritious food produced, the availability and the cost.

Undernutrition during pregnancy is linked to babies being born too early and too small. It is also linked to neural tube defects, including spina bifida, where the spine and spinal cord do not form properly. Children born to mothers who are experiencing undernutrition because of food insecurity have been shown to have impaired cognitive development.



Pollution

Air pollution

A pregnant woman can be exposed to higher levels of air pollution than a non-pregnant woman due to changes that occur in a woman's breathing system during pregnancy.

Chemicals in air pollution can directly affect a pregnant woman's lungs and create inflammation in her body. They can also enter a pregnant woman's circulation and then cross into the placenta.

Air pollution can affect the health of the pregnant woman (e.g., diabetes in pregnancy, high blood pressure), the ability for her to carry to term and the weight of the baby. It can cause pregnancy loss early on (miscarriage) or later in the pregnancy (stillbirth).

If a child is born after being exposed to air pollution in the womb, that child is at risk of developing neurodevelopmental disorders or asthma, the most common chronic childhood disease.





Lead

There is no safe level of lead exposure.

A fetus's developing brain is particularly vulnerable to lead.

Lead can compete for entry into a pregnant woman's body with important nutrients such as calcium and iron, which are needed at higher levels during pregnancy to support the growth and development of the fetus. Lead can also be released into the pregnant woman's bloodstream from her bones, which store lead from past exposures.

Antenatal exposure to lead is linked to reduced cognitive and behavioural development in children.

Pesticides

Exposure to pesticides can be harmful to a pregnancy, even if a woman is exposed before she gets pregnant.

Pesticides can contribute to impairment of DNA, which can harm the developing fetus. Antenatal exposure to pesticides is linked to birth defects and childhood cancers including neuroblastoma and leukaemia, the most common childhood cancer. Antenatal pesticide exposure can also negatively affect the development of a child's brain.

Arsenic

Arsenic is a toxic heavy metal that can contaminate drinking water and foods. Arsenic can accumulate at levels up to three times higher in the placenta than in a pregnant woman's body.

Arsenic exposure during pregnancy can cause pregnancy loss or premature birth. It is also associated with cognitive deficits in childhood.

Tobacco smoke

When a pregnant woman smokes or is around someone who smokes, her fetus can be exposed to over 7,000 toxic chemicals. There is no safe level of tobacco smoke.

Tobacco smoke can alter blood flow to the fetus, disrupting the delivery of oxygen and nutrients. Babies exposed to tobacco smoke may be born prematurely, have birth defects or die from sudden infant death syndrome. The effects of antenatal smoke exposure can also contribute to many chronic diseases in childhood.



Environmental exposures before and during pregnancy shape a child's lifelong health and well-being

Technical brief

Critical windows of vulnerability

During preconception, conception and all the months of pregnancy, there are critical windows of time where certain cells, organs and tissues are most susceptible to environmental hazards. Understanding these windows can shed light on why environmental hazards can devastate a pregnancy or cause effects on health and development decades later. Protecting pregnant women from environmental hazards is an important step to ensure the health of future generations.

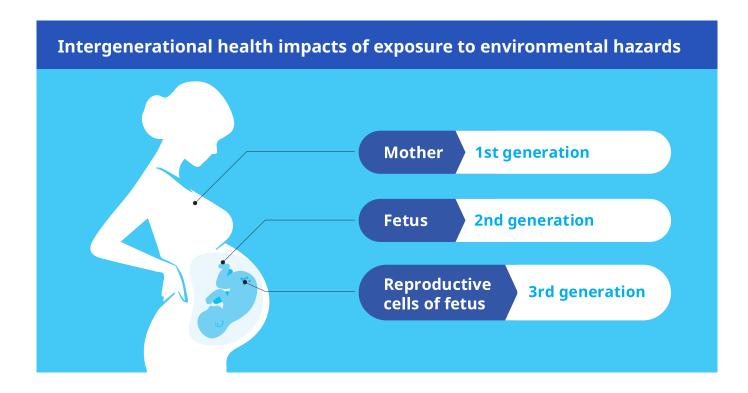
Preconception: The first exposure window

The health of both parents matters. The health of a sperm and egg which combine to create an embryo is foundational to the health of a pregnancy, the fetus and the baby after birth.² The weeks just before conception are particularly important.³ But the impact of environmental hazards can be traced back several generations, and can occur even before a future parent is born.

The damage from direct exposure to environmental hazards to the reproductive cells of both men and women can happen decades before they conceive a child.⁴ This is because a person's reproductive cells are already present when they are in the womb. It has been shown, for instance, that even when a woman does not smoke during pregnancy, her child is at increased risk of asthma if her own mother (the grandmother) smoked while pregnant with her.⁵

Damage can occur anytime, including before conception, continuing in utero, and persist through childhood and adolescence.⁶ Egg and sperm cells also develop differently, which can affect when the windows of sensitivity to environmental hazards occur and, ultimately, fertility and health impacts.⁷

Changes to DNA in the reproductive cells can also be passed on to generations not directly exposed.⁸ This means that a great-grandchild who did not have any direct exposure to an environmental hazard can still develop an environmentally related health condition, such as obesity.⁹





A closer look: How environmental hazards can change DNA instructions in the body

DNA is the genetic material that contains the instructions for a person to develop, survive and reproduce. Environmental hazards can change both the structure and function of DNA. Changes to the structure of DNA are called mutations. Changes to the function of DNA, where genes can be turned on or off, are more common and are called epigenetic changes.¹⁰

Mutations and epigenetic changes can occur in all cells in a body, including reproductive cells. If a mutation or epigenetic change caused by an environmental hazard takes place in a reproductive cell, the damage can be passed on to the next generation.

Epigenetic changes can occur at any point in a person's life but there are certain windows of increased vulnerability, such as during sperm and egg development, fertilization and the formation of an embryo.¹¹

Pregnancy: The second exposure window

During pregnancy, a woman's body undergoes many changes to accommodate the metabolic and nutritional needs of the growing fetus and prepare for breastfeeding. Some of these changes can increase the vulnerability of a pregnant woman and fetus to environmental hazards. They can also alter key processes in a pregnant woman's body and change the way her body responds to physical stresses, such as extreme heat, putting both the woman and the fetus at risk of harm.

1. Increased food and fluid intake

A pregnant woman needs more food and fluids than a non-pregnant woman. This increases a pregnant woman's exposure to toxicants in contaminated food and drinking water. For instance, arsenic, a common contaminant of drinking water,¹² is linked to miscarriage, preterm birth and stillbirth,¹³ as well as cognitive deficits in childhood.¹⁴

An increased need for fluids during pregnancy also puts a woman at risk of dehydration. This can cause low amniotic fluid levels, which limits fetal movement and creates pressure on the fetus from the umbilical cord. Diseases like cholera, which make people lose fluids, make pregnant women especially susceptible to dehydration. Severe dehydration can compromise blood flow to the placenta and cause harmful levels of acid in the amniotic fluid, leading to fetal death. Dehydration during heat waves may also contribute to growth restriction and low birthweight.

Pregnant women have increased caloric and nutrient needs. This means they are more at risk of complications from food insecurity. Food insecurity is associated with a variety of adverse outcomes, including preterm birth, low birthweight and neural tube defects including spina bifida.¹⁸ Food insecurity in pregnant women is also associated with impaired cognitive development in children.¹⁹

Consuming foods contaminated with fumonisins (mycotoxins found on corn and in corn flour) is associated with neural tube defects.²⁰ Climate change will increase the number of tropical regions susceptible to fungal growth, and this will likely lead to increased food contamination with fumonisins.

2. Changes to the respiratory system

Hormonal changes during pregnancy drive key changes in a pregnant woman's respiratory system, which can affect the body's response to environmental hazards such as air pollution.²¹ Pregnant women inhale more air per minute, which means they will breathe in more air pollutants. Air pollutants can have a duel negative effect because they can both harm the fetus directly and act systemically by creating inflammation and reactive oxygen species, which are unstable molecules that can damage DNA.²²

Antenatal air pollution exposure is associated with miscarriage,²³ preterm birth,²⁴ low birthweight²⁵ and stillbirth.²⁶ It is also linked to common chronic childhood conditions, including neurodevelopmental disorders²⁷ and asthma.²⁸ Exposure to wildfire smoke is linked to preterm birth and low birthweight.²⁹

Exposure to second-hand smoke, which can contaminate children's environments when tobacco products are burned or when a smoker exhales smoke, contains over 7,000 chemicals, including approximately 70 that can

cause cancer.³⁰ Prenatal tobacco smoke exposure can alter blood flow and metabolism in the fetus and cause accumulation of chemicals both in the mother and the fetus.³¹ Maternal smoking or second-hand smoke is linked to stillbirths, preterm births, birth defects and infant deaths.³² The effects of prenatal smoke exposure continue through childhood and adolescence and include obesity, attention-deficit/hyperactivity disorder, mood disorders and childhood cancers.³³

Increased demand for oxygen

Increased intake of oxygen necessary to meet the demands of the fetus can increase the production of reactive oxygen species, which are unstable molecules that can damage DNA.³⁴ Pregnancy alters the body's ability to detoxify these reactive oxygen species, and environmental factors can increase their production.³⁵ When there is an imbalance between the production and detoxification of these species, oxidative stress in cells occur.³⁶

Increased oxidative stress in pregnancy is linked to adverse effects such as pre-eclampsia. It also is linked to the risk of developing cancer during childhood, and metabolic syndrome, cardiovascular disease and neurological diseases later in life.³⁷

3. Changes in movement of substances in the body

The movement of substances, including chemicals, in a body can change during pregnancy. For example, a pregnant woman's bones will remodel to release calcium for the fetus, which can release lead into the bloodstream at the same time.

Other changes include the movement of fats in the body. Early in pregnancy, the storage of fat increases in a woman's body. Later in pregnancy, fat is broken down to provide energy for the fetus.³⁸ 'Fat-loving' (i.e., lipophilic) toxicants can be stored in this fat and then released into a woman's bloodstream, which can potentially reach the fetus.³⁹ Lipophilic chemicals include an array of harmful persistent pollutants, such as DDT, dioxins and heavy metals.⁴⁰

4. Changes in metabolism

Other metabolic shifts to support fetal growth and development include changes in the metabolism of glucose and proteins. These changes are hormonally controlled and susceptible to endocrine-disrupting chemicals (EDCs) and other toxicants. When metabolism during pregnancy is negatively affected by EDCs, diseases such as gestational diabetes, hypertensive disorders or restricted fetal growth can occur. Miscarriage can also happen.⁴¹

5. Changes in the maternal heart, blood and circulation

During pregnancy, the heart increases the amount of blood it pumps per minute. The total volume of blood, including the plasma (i.e., the liquid portion of blood) is also increased. ⁴² Increased plasma volume is critical to maintain blood flow to the uterus. Low plasma volume is associated with gestational hypertension and other pregnancy complications. ⁴³

Red blood cell production also increases in pregnancy. As iron is a key component of these cells, pregnant women require more iron.⁴⁴ Conditions such as food insecurity, which may reduce availability of iron from dietary sources, can lead to iron deficiency and anaemia.

6. Changes in heat regulation

Changes in heat regulation to cope with the metabolic changes related to pregnancy include increased blood volume, dilation of blood vessels in the skin and increased sweating.⁴⁵ If a pregnant woman is exposed to extreme heat, she may not be able to transfer enough heat to the external environment. If this happens, her health and the fetus's health can be affected, with consequences for newborn and child health if the pregnancy goes to term.⁴⁶ Extreme heat experienced during pregnancy is associated with preterm birth and has been linked to gestational diabetes, low birthweight and congenital anomalies.⁴⁷

7. Changes in immune function

During pregnancy, a woman's immune system will effectively suppress itself to enable the fetus to grow.⁴⁸ This increases a woman's susceptibility to certain infectious diseases and the severity of illness.⁴⁹ It also increases the risk of fetal death.⁵⁰ Research shows that pregnant women have an increased incidence of many types of infections, including some mosquito-borne illnesses that are increasing due to climate change, such as Zika virus infection and malaria.⁵¹

Malaria has significant impacts on the health of pregnant women and fetuses. Infections in the first trimester are especially high. The parasites that cause malaria can infect red blood cells and accumulate in the placenta, contributing to adverse pregnancy outcomes, including severe anaemia and death in the mother, miscarriage, stillbirth, preterm birth and low birthweight. Malaria during pregnancy has also been shown to be associated with impaired cognitive development in children.⁵²

While Zika virus symptoms during pregnancy are usually mild and similar to those seen in non-pregnant people, Zika virus infection during pregnancy can cause miscarriage, stillbirth and preterm birth.⁵³ Zika virus can affect stem cells in the fetal nervous system.⁵⁴ Babies who survive may suffer from congenital Zika syndrome, which includes having a severely small head, brain and eye anomalies, congenital contractures, intrauterine growth restriction, seizures and neurodevelopmental delay.⁵⁵ Babies who are born without observable defects at birth can still present with cognitive and language neurodevelopmental delay in childhood.⁵⁶ Zika virus can also be transmitted sexually.



The placenta's dual role: Protector and pathway for pollutants and infections

The placenta is a remarkable organ. It takes on critical functions during pregnancy, acting like the lungs, gastrointestinal tract, kidneys and endocrine glands of the developing fetus.⁵⁷ A healthy placenta is essential to a healthy pregnancy.

While the main role of the placenta is to supply the fetus with oxygen and nutrients, the placenta also controls the adaptions a woman's body makes during pregnancy and acts like a barrier to minimize fetal exposures to toxicants and infectious agents.⁵⁸ Unfortunately, many harmful substances, such as lead, mercury, pesticides and other chemicals, can penetrate the placenta barrier and cross through it, harming the fetus and causing potentially lifelong effects to a child.⁵⁹ Some chemicals also concentrate in the placenta at higher levels than in the pregnant woman. For instance, arsenic can accumulate at levels up to three times higher in the placenta compared to the pregnant woman.⁶⁰

Infectious agents, such as bacteria, parasites and viruses, can also cross the placenta and cause serious congenital infections, preterm birth and stillbirth.⁶¹ For example, the Zika virus and a group of antenatal infections known by the acronym TORCH (*Toxoplasma gondii*, other, rubella virus, cytomegalovirus, herpes simplex virus) can devastate the health of the pregnancy and fetus.

A closer look: How environmental hazards affect placenta development and function

Researchers have recently begun to explore how environmental hazards might affect the development of a normally functioning placenta. The placenta has a large number of hormone receptors, which makes it vulnerable to chemicals that can disrupt the endocrine system, including heavy metals, some pesticides and per- and polyfluoroalkyl substances (PFAS).⁶² Endocrine disruption in the placenta can cause oxidative stress and altered epigenetic programming, gene expression and placental function.⁶³

Importantly, abnormal placenta development and function is associated with pre-eclampsia, a condition that includes high blood pressure and kidney damage in a pregnant woman. Pre-eclampsia is one of the most severe complications of pregnancy and can lead to maternal morbidity as well preterm birth and perinatal death.⁶⁴ In children who survive, it can also lead to neurodevelopmental delay and cardiovascular and metabolic disease later in life.⁶⁵ Antenatal air pollution exposure has been shown to be associated with pre-eclampsia.⁶⁶

Exposure during the three pregnancy trimesters

The first trimester (weeks 1 to 13)

The first two weeks after fertilization: Even before a missed period would signal that a woman is pregnant, environmental hazards can interfere with implantation of the embryo into the womb and cause miscarriage.⁶⁷ Also during this period, the patterns of chemical tags on DNA, which can alter the function of DNA if affected by toxicants, are established.⁶⁸ By the time the embryo is implanted, the majority of DNA methylation tagging patterns are established.⁶⁹

Weeks 4 to 8: Organs begin to form. By the eighth week of pregnancy, all major external and internal structures are formed, and organ systems have started to develop. During this time, the embryo's tissues and organs are rapidly developing, which makes them especially vulnerable to environmental hazards. As a result, weeks 4 to 8 is the window of time when major birth defects can occur.⁷⁰

From week 9: This starts the fetal period. Until birth, organs continue to develop and mature and remain

vulnerable to environmental hazards. Critical windows of development correspond to the periods of time that cells are dividing and changing into different types and tissues and the times that organs take on their shape.

For brain development, the critical period is from weeks 3 to 16. But brain development can continue to be affected by environmental hazards after this time as the brain will continue to grow and develop.

The second trimester (weeks 14 to 27)

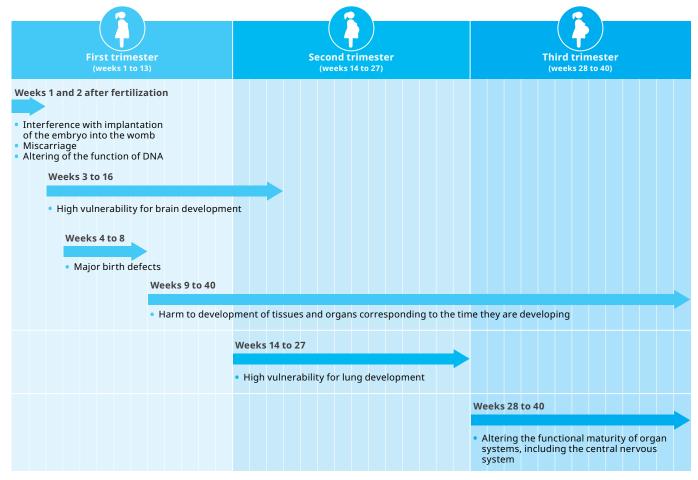
This is a time of rapid growth and development.

During the second trimester, lungs are most vulnerable to impairment from air pollution.⁷¹

The third trimester (weeks 28 to 40)

This is when fetal weight increases and organs continue to mature so they can be ready to function at birth.⁷² During this time, environmental exposures can alter the functional maturity of organ systems, including the central nervous system.⁷³

Timeline of health impacts from exposure to environmental hazards during trimesters



Note: An array of pregnancy-related disorders, including those caused by environmental hazards, can lead to the death of the embryo (miscarriage).

All women are at risk of environmental hazards during pregnancy – but some women are more at risk than others

Women in low- and middle-income countries, including fragile and humanitarian contexts

Women living in poverty and in low- and middle-income countries are more likely to suffer ill effects from environmental hazards. A woman's health at the start of pregnancy and her genetic make-up can also play a role. Those particularly at risk include women who live in regions where unclean fuels are still used to cook, heat and light homes, as this generates high levels of household air pollution. Others particularly at risk are those in regions where contamination of food crops with fumonisins (a group of mycotoxins) is common. Women working in dangerous jobs and without adequate workplace regulations, such as informal e-waste recycling, artisanal small-scale gold mining and in agricultural roles where pesticides are used, are also at great risk

as they are regularly exposed to harmful chemicals.⁷⁶ Women can also be exposed to hazardous chemicals in female-dominated workplaces such as hairdressers and nail salons, or in daily living from cosmetics and household products (such as cleaning agents).⁷⁷

The increased risk to health for pregnant women in urban slums

Living in an urban slum significantly affects the health of a pregnant woman and her developing fetus. Overcrowded and unsanitary conditions increase the prevalence of infectious diseases. Pregnant women in urban slums often experience inadequate nutrition and lack access to quality health care services. Air and water pollution are common, as slums are often located near industrial sites or waste dumps, exposing pregnant women to harmful chemicals.⁷⁸



How environmental hazards affect fetal, newborn and child health

Health outcome	Causes
Numerous health conditions in a pregnant woman can affect the pregnancy	The health status of a pregnant woman is critical to the health of the developing fetus and pregnancy. High blood pressure, gestational diabetes and obesity can contribute to adverse pregnancy outcomes and can be affected by environmental chemicals. For example, phthalates (chemicals added to plastics and found in many personal care and household products), PFAS, polychlorinated biphenyls (PCBs, a persistent organic pollutant formerly used in the electricity industry) and some flame retardants (such as polybrominated diphenyl ethers or PBDEs) significantly increase the risk of gestational diabetes. ⁷⁹ Air pollution is also associated with gestational diabetes and high blood pressure. ⁸⁰
Death	Exposure to environmental hazards is associated with an array of pregnancy-related disorders that can lead to miscarriage, stillbirth and sudden infant death syndrome. ⁸¹
Preterm birth	Many environmental hazards are linked to preterm birth (i.e., being born before 37 weeks of gestation).82 Some examples include air pollution, lead, some pesticides, PFAS and phthalates.83 Complications from preterm births are the leading cause of death among children under 5 years of age. Being born premature is also associated with many developmental problems in childhood.84
Low birthweight	The World Health Organization defines low birthweight as weight at birth less than 2,500 grams (5.5 pounds). Low birthweight can be caused by intrauterine growth restriction as well as prematurity. It is associated with fetal and neonatal morbidity and mortality as well as impaired growth and cognitive development in childhood. ⁸⁵ It is also associated with chronic disease in adults, such cardiovascular disease, type 2 diabetes and metabolic syndrome. ⁸⁶ Many environmental hazards are associated with low birthweight, including air pollution, tobacco smoke and pesticides. ⁸⁷
Birth defects	Air pollution, consumption of foods contaminated with fumonisins, extreme heat, hazardous waste and solvents are just some of the exposures that have been shown to be linked to various birth defects. These defects include malformation of the central nervous system, heart, abdominal organs, limbs and other structures. ⁸⁸
Numerous chronic conditions in childhood and adulthood	Beyond birth, diseases related to antenatal exposures can develop in childhood, adolescence and adulthood. Antenatal exposure to air pollution is associated with chronic childhood conditions, such as asthma, allergic disease, neurodevelopmental disorders, obesity and high blood pressure. It is also linked to adulthood diseases such as cardiovascular disease and chronic obstructive pulmonary disease, the third-leading cause of death worldwide. Antenatal pesticide exposure is associated with neurodevelopmental disorders and childhood cancers including neuroblastoma and leukaemia, the most common childhood cancer. Exposure to mercury during pregnancy can lead to devastating neurological outcomes in a child. 91

2. Children's unique vulnerabilities to environmental hazards

Children's specific metabolism, physiology and developmental needs, makes them more vulnerable to environmental harm. This section focuses on the specific effects of environmental hazards in infancy and throughout childhood. Health effects that emerge during childhood as a result of antenatal exposures are not reviewed.

Key messages

- As soon as a newborn breathes for the first time, environmental hazards can begin to affect development due to the unique vulnerabilities of children.
- Children eat, drink and breathe relatively more than adults, meaning they also take in more harmful contaminants. In addition, they are less able to break down and expel toxicants.
- Children have more contact with environmental hazards. They are closer to the ground where toxicants like soil and dust settle. Their exploratory hand-to-mouth and object-to-mouth behaviours make them more likely to ingest harmful substances.
- Children especially infants grow rapidly, making them more at risk of malnutrition, which can be increased by environmental hazards that cause ood and water scarcity.
- Moreover, children's limited diets can lead to greater exposure to food contaminants, like pesticides or microorganisms. Breast milk can pass harmful environmental exposures from mother to child.
- Infants and young children are more prone to dangerous heat loss as well as overheating. Even into later childhood, children are less able to adjust to rises in ambient temperature than adults.
- Anatomical differences can make children more vulnerable to environmental hazards, from developing airways to more absorbent skin and gastrointestinal tracts, more penetrable blood-brain barriers and, in the case of infants, even different haemoglobin.
- These vulnerabilities come at a time when a child's organs and body systems are rapidly developing from their antenatal form to the form the individual will carry into adulthood.
- Children's longer lifespans mean that exposures to harmful substances during childhood are more likely to result in disease or other adverse health effects later in life.
- Children are unable to protect themselves during this period of high vulnerability. They are entirely reliant on the adults in their lives to protect them from harm.

Examples of different environmental hazards and how they affect children





Climate-related hazards

Malaria

Children under 5 years of age account for around 80 per cent of malaria deaths in the WHO Africa Region (which has 95 per cent of global deaths). A young child's immune system clears the infection differently than that of adults, which can lead to different syndromes, including severe anaemia. Malnutrition, which growing children are more vulnerable to due to their increased nutritional needs, also contributes to poor immune response and deaths from malaria.

Extreme temperatures

Newborns, especially small and premature infants, regulate body temperature much less efficiently and thus are at risk of both hypothermia and hyperthermia. Hypothermia occurs when body heat loss goes unchecked, and can cause newborns to succumb to other illnesses. Hyperthermia can result from common situations such as overbundling or being left in direct sunlight, a hot car or around heaters or fires.

Infants and very young children are also vulnerable to extreme temperatures, with infants under 1 year of age being more prone to heat-related deaths. Older children can take longer to adjust to heat and thus may be more vulnerable when outdoor temperatures suddenly rise (particularly when playing sports).

Food insecurity

Particularly in the first 5 years of life, children's growing bodies require more calories and nutrients relative to body weight than adults, making them more vulnerable to situations where nutritious food is not available. Malnutrition can negatively affect growth and development with lifelong lasting consequences. Malnutrition can also affect children's developing immune systems, and make them more likely to die of common infections such as diarrhoea, pneumonia and malaria.

Contaminated food and water

Growing children eat and drink more relative to their body weight than adults. When food and water are contaminated with chemicals such as heavy metals or pesticides, children's developing organs can be harmed. Each chemical can do different kinds of damage due to different age-specific patterns of absorption or metabolism in the gut. For example, exposure to nitrates – common contaminants in drinking water – puts infants at risk of 'blue baby syndrome' (low oxygen levels) due to their particular metabolic activity. Hazardous chemicals, such as bisphenols (e.g., BPA), can also leach from plastic packaging and food contact materials and enter into children's food and drinks.

Contamination of food and water with pathogens can lead to bacterial, viral and parasitic infections which can be associated with both short- and long-term health problems including diarrhoea and malnutrition. Polio can also be transmitted through contaminated food and water.

Pollution

Air pollution

Young children have faster breathing rates and so breathe in more pollutants relative to their body weight than adults. Exposure to air pollution, whether outdoors or in the household from use of polluting fuels for heating, cooking and lighting, can harm many systems of a child's body and have lifelong effects.

Air pollution can negatively affect the development of children's lungs, which sets the trajectory for their adult lung capacity. It is also associated with infectious diseases, such as upper respiratory tract infections and pneumonia, and chronic lung diseases, including asthma.

Air pollution can harm children's developing brains and immune systems. It can also affect sleep quality, which is important for brain development. Some adult medical problems such as high blood pressure, chronic obstructive pulmonary disease and lung cancer are associated with childhood exposure to air pollution.

Lead

There is no safe level of lead. Young children, particularly toddlers, have increased exposure to lead because they put their hands and objects in their mouths, thereby ingesting lead-contaminated dust, soil and other substances. They also are far more likely than adults to absorb the ingested lead into their bloodstreams. Once in the body, lead can have significant and lasting impact on the developing brain, affecting a child's IQ, attention and school performance.

Pesticides

Young children breathe, drink and eat more relative to body weight than adults, giving them higher exposure to pesticides in contaminated air, water and food (food being the most common source of chronic pesticide exposure). Children are closer to the ground and put their hands and objects in their mouths, which can expose them to pesticides in household dust and soil. Children are also at risk of unintentional poisonings which can be fatal, especially when pesticides are improperly stored, such as in soft drink bottles and food containers.

Pesticides can affect the enzyme systems in developing brains which are necessary for normal nervous system development and function. They can also damage cells which are rapidly dividing in growing tissue. Pesticides are associated with childhood cancers, including brain cancer and leukaemia, a type of blood cancer that is the most common cancer in children.

Plastics

Children have widespread exposure to plastics in everyday items, including products designed for infants and children, such as bottles and toys. Young children put objects in their mouths as part of normal exploratory behaviour, which can increase exposure to harmful chemicals used in plastics, as well as microplastics and nanoplastics that are shed when plastics break down.

Numerous chemicals that migrate out of plastics can disrupt the body's hormone system, impacting processes such as metabolism, brain development, growth and reproductive development during critical periods. Some of these chemicals are linked to health problems in children, including obesity and early puberty.

Second-hand smoke

Second-hand smoke can contain over 7,000 chemicals that harm children's developing respiratory systems and other organs. No amount of second-hand smoke is safe. Second-hand smoke is linked to ear infections, pneumonia and asthma, the most common chronic disease in childhood. It is also associated with sudden infant death syndrome.

Carbon monoxide poisoning

Carbon monoxide is a colourless, odourless toxic gas released by use of polluting fuels in the home, inadequate ventilation and poorly functioning stoves and furnaces. Carbon monoxide poisoning can be fatal. Newborns and infants are more vulnerable to carbon monoxide poisoning than adults.

Haemoglobin – the substance in the blood that carries oxygen – is different in infants than it is in adults. The haemoglobin in infants binds more readily with carbon monoxide, which results in lower capacity to carry oxygen to tissues that already have a high demand in growing infants.



The factors which make children uniquely vulnerable to environmental hazards

Technical brief

Environmental hazards affect infants and children in different ways than adolescents and adults due to dynamic physiology and metabolism, unique and different exposures, cognitive immaturity and longer life expectancy.

Dynamic physiology

1. Increased intake

Children eat, drink and breathe more per kilogram of body weight relative to adults because they are growing. ⁹³ This difference is even greater in infants compared to older children. This can lead to higher rates of intake of harmful substances per kilogram of body weight when there are contaminants in food, water and air. ⁹⁴ Hazardous chemicals, such as bisphenols (e.g., BPA), can also leach from plastic packaging and food contact materials and enter into children's food and drinks. ⁹⁵ Microbial contamination of food and water can lead to bacterial, viral and parasitic infections which can be associated with an array of short- and long-term health effects including diarrhoea and malnutrition. Polio can also be transmitted through contaminated food and water. ⁹⁶

Growth rate and nutritional needs

Children's nutritional needs are different from those of adults, and vary with age. Infants, especially those aged 0-6 months, have the highest relative rate of weight gain.⁹⁷ Full-term infants double their birthweight in 4–5 months and triple it by 1 year of age.98 Weight gain continues after infancy, although at a slower rate, and then accelerates again during adolescence.99 Increased growth is accompanied by increased caloric intake, making infancy the period of relatively highest intake. When intake is inadequate, children can develop malnutrition, which can cause wasting (i.e., too thin for height) and subsequently lead to increased risk of death or stunting (i.e., too short for age), a condition which prevents children from reaching their physical and cognitive potential.¹⁰⁰ In 2022, an estimated 149 million children under 5 were stunted and 45 million were wasted. 101

Children who are malnourished are also at a higher risk of death from infections such as diarrhoeal illnesses, pneumonia and malaria. Malnutrition is seen as an important risk factor for cholera, a diarrhoeal disease for which children under 5 bear the greatest burden in endemic areas. 103

Environmental exposures can affect children's growth, with the most sensitive period extending from conception until age 2 years. ¹⁰⁴ For example, children exposed to household air pollution have been shown to have reduced linear growth and increased stunting compared to children who do not live in homes where polluting fuels are used. ¹⁰⁵

Increased fluid needs

The amount of fluids that children need per kilogram of body weight per day is highest from birth to 6 months, 106 with the ratio gradually decreasing until they are adults. This makes younger children more susceptible to dehydration when there is inadequate access to fluids, or when there are increased fluid losses from conditions such as diarrhoea or exposure to extreme heat.

Increased respiratory rates

Infants and young children also have a higher resting metabolic rate and rate of oxygen consumption per kilogram of body weight than adults. 107 Respiratory rates in early infancy are around 2.5–3.3 times higher than the respiratory rates of adults, making young infants particularly vulnerable to exposure to air pollution. Respiratory rates gradually decrease but remain higher than those of adults until early adolescence.



2. Increased absorption of toxicants

Increased absorption from the gastointestinal tract

As children have different nutritional needs, the small intestine can respond by increasing the absorption of certain nutrients. For instance, calcium absorption in infants is around five times the rate of that in adults. Some environmental toxicants, such as lead, can compete with nutrients and also be absorbed at higher rates. For example, infants and children absorb 40–50 per cent of ingested lead, compared to 3–10 per cent in adults.¹⁰⁸

Increased absorption through the skin

The ratio of a newborn's skin surface area to body weight is three times greater than that of adults, meaning their skin can absorb more of a harmful substance per unit of body weight than that of an adult.¹⁰⁹ In addition, the outermost protective layer of the skin is 20–30 per cent thinner – and thus more absorptive – in children aged 3–24 months compared to adults.¹¹⁰ Water loss through the skin is also higher in infants and children and decreases with age.¹¹¹ A small recent study showed that the skin's protective layer reaches adult thickness by age 6.¹¹²

Age	Respiratory rate (breaths/min)
Premature infant	40-70
0–3 months	40-70
3-6 months	30-60
6-12 months	25-40
1–3 years	20–30
3–6 years	20-25
6–12 years	14-22
Over 12 years	12-18

Source: Johns Hopkins Hospital.¹¹³

3. Dynamic and different metabolism

Children's ability to metabolize, or break down, harmful substances that enter the body changes with age.

Take, for example, organophosphate pesticides, which can cause both acute poisonings and chronic low dose exposures and are known to affect cognitive development. The body has an enzyme called PON1 which detoxifies organophosphate pesticides. Measured activity of PON1 is lower in children up to at least 7 years of age, creating a period of increased vulnerability to these pesticides. 114

A closer look at the unique metabolism of infants: Nitrates and blue baby syndrome

Nitrates and nitrites, substances commonly found in foods and drinking water, can cause 'blue baby syndrome' that results from reduced oxygen levels in the blood. When infants ingest these substances, several metabolic factors lead to an increased formation of methaemoglobin, which, unlike normal haemoglobin, cannot bind and carry oxygen. Some of these factors include:

- The acid balance in an infant's gut makes it more favourable to bacteria that convert nitrates to nitrites. Nitrites can change the iron in haemoglobin, causing conversion to methaemoglobin.
- Infants have fetal haemoglobin, which is more readily converted to methaemoglobin.
- Infants have a reduced ability to convert
 methaemoglobin back to normal haemoglobin
 because, compared to adults, infants have only
 about half the level of methaemoglobin reductase,
 the enzyme which performs the conversion.

Source: Agency for Toxic Substances and Disease Registry, and American Academy of Pediatrics Council on Environmental Health.¹¹⁵

4. Differences in excretion

The body eliminates waste through the kidneys via urine, the gastrointestinal tract via faeces and the lungs via exhaled air. The kidneys are the main route of excretion. At birth, the filtration rate of the kidneys is about one third of adult values, increasing to adult levels by age 8–12 months. This means that infants clear substances excreted by the kidneys at a slower rate than adults.

5. Differences in structure and function of respiratory system

In addition to having increased respiratory rates, there are structural and functional differences between the airways of infants and children compared to adults. Infants up to age 2–6 months breathe primarily through their nose which makes them more vulnerable to conditions which block their nasal passages, such as upper respiratory infections, which are associated with exposure to air pollution.¹¹⁷

The size and shape of the airway between the larynx and trachea is also different, which means that even small amounts of oedema can significantly reduce the diameter of the paediatric airway, decreasing airflow and making breathing more difficult.¹¹⁸ This difference makes children far more vulnerable to infections, including the uncommon but potentially fatal respiratory infection called bacterial tracheitis.

The middle ear

The anatomy of the middle ear is different in young children compared to adults. In children, the eustachian tubes are smaller and more level, making it more difficult for fluid to drain out of the ear and contributing to increased incidence of middle ear infections in children. Tobacco smoke is a well-documented risk factor for middle ear infections, and recent evidence suggests ambient air pollution may also be a risk factor. Globally, middle ear infections affect over 80 per cent of children below the age of 3 years and can lead to hearing loss, language delay and impaired cognitive development. 120

6. Differences in components that make up blood

Haemoglobin, the protein in red blood cells that carries oxygen, is different in infants compared to adults. At birth, newborns have 65–90 per cent fetal haemoglobin, which is present in utero. Levels of fetal haemoglobin decrease by 6–12 months of age, when only 2 per cent of total haemoglobin is in the fetal form.¹²¹

The presence of fetal haemoglobin makes infants more susceptible to carbon monoxide poisoning, because fetal haemoglobin is more likely to bind with carbon monoxide than adult haemoglobin. ¹²² Carbon monoxide is a colourless, odourless toxic gas. ¹²³ Burning low-grade solid fuel and biofuels in a small stove or fireplace can generate high levels of carbon monoxide which can be deadly without appropriate ventilation. Burning high-grade fuels such as natural gas, butane or propane can also cause carbon monoxide poisoning if devices are not properly maintained or vented. ¹²⁴

7. Differences in thermoregulation

Infants and young children regulate temperature differently than adults which makes them more vulnerable to extreme temperatures, both low and high. Their ratio of body surface area to mass is greater than that of adults which permits greater heat transfer between their bodies and the environment. In addition, they have higher metabolic rates and heart rates, they spend more time outdoors and in vigorous activities, and they cannot remove themselves from environments with unsafe temperatures. Children under 1 year of age are especially vulnerable to heat-related deaths. Extreme temperatures are increasingly more likely due to global climate change.

Extreme temperatures: Risks to children with special health-care needs

While all children are vulnerable to extreme temperatures, children with special health-care needs may have increased loss of water through the skin and lungs that can't be measured, which can put them at increased risk of dehydration.

Source: Mangus and Canares. 126

Children also take longer to acclimatize to a warmer environment than adults, which means they are slower to

make necessary physiologic changes such as increasing sweat production and blood flow during exercise. ¹²⁷ This is particularly important for young athletes when ambient temperatures change quickly.

8. Immature immune systems

Innate immune system

The innate immune system is the first line of defence and is present at birth. While it was previously thought that the innate immune system of neonates was simply immature, scientists are beginning to understand its complexity. There are many cells in the innate immune system that are not fully functional at birth – such as neutrophils, which kill bacteria – which puts newborns, and in particular preterm infants, at higher risk of bacterial and viral infections. 129

Adaptive immune system

Adaptive immunity is not present at birth but is developed over time. It involves specialized immune cells and antibodies that attack and destroy foreign invaders and are able to prevent future diseases by remembering what those substances look like and mounting a new immune response.¹³⁰ A newborn's adaptive immune system functions differently, making them more susceptible to respiratory infectious diseases and reducing their response to vaccination.¹³¹



Antibodies passed from mother to fetus during pregnancy can provide protection against many infections, but these antibody levels generally wane by 6 months of age. Breastfeeding infants also can benefit from passive immunity from antibodies transferred through breast milk.

Microbiome

At birth, as newborns go from a sterile in-utero environment to an external world laden with microbes, they need to be able to quickly respond to some foreign pathogens while tolerating other microbes. Some of these microbes will become part of the microbiome, the community of microorganisms that live on the skin, in the gut and in other parts of the body.¹³²

Deadly combination: Children's immature immune systems face increased challenges from vector-borne diseases in a changing climate

The incidence of vector-borne diseases is expected to rise in the context of a changing climate. While people are at risk of these diseases, children are at higher risk of mortality due to immature immune and other body systems.

Malaria

In 2022, children under 5 years of age accounted for about 78 per cent of all malaria deaths in the WHO Africa Region, where 94 per cent of all malaria cases and 95 per cent of deaths occurred. The effect of malaria on organs in the body changes with age and may influence how often different malaria syndromes in children and adults occur. In areas with high malaria transmission, severe anaemia is especially noted during the first and second years of life, partly due to the way the spleen clears malaria-infected red blood cells in young children. Children's brains may also be particularly vulnerable to malaria.

Dengue virus

Dengue virus infection can manifest as a benign syndrome, dengue fever, or a severe syndrome with haemorrhagic fever and shock. In severe dengue, clinical symptoms are more significantly associated with death in infants compared with older children. Infants born to mothers with immunity to dengue can develop severe dengue the first time they are infected with the dengue virus. This occurs because maternal antibodies initially protect infants from dengue infection, then break down during the course of infancy, creating a period of enhanced infection where severe dengue can develop.

Source: Moxon et al., World Health Organization, and Jain and Chaturvedi. 133

The microbiome is increasingly being recognized for its role in health and disease across the lifespan.¹³⁴ It contributes to metabolic functions, protects against pathogens and educates the immune system.¹³⁵ Breakdown products of the microbiome in the gut can also affect maturation of the nervous system.¹³⁶ While early research suggested that the gut microbiome reaches adult composition by age 3, recent studies suggest it may continue to evolve during childhood. Environmental hazards including air pollution, tobacco smoke, pesticides and extreme heat can affect the microbiome.¹³⁷

9. Altered permeability of the blood-brain barrier

The blood-brain barrier is a network of blood vessels and tissue comprised of closely spaced cells that allows transport of vital molecules like oxygen into the brain while limiting harmful substances and microbes from reaching the brain. Although the barrier is fully functional at birth, activity of transporters and enzymes at the barrier differs from adults to meet the needs of the developing brain. This means that movement of harmful substances can differ, making infants more vulnerable to chemicals. Some harmful substances, such as lead and cadmium, may cause oxidative stress leading to a weakening of the blood-brain barrier and allowing transmission of these substances into the brain.



Unique and different exposures

1. Behavioural changes

Children in LMICs may be at particular risk of increased exposures related to hand-to-mouth behaviours. For example, in these countries, homes are more likely to have earthen floors, increasing risk of soil ingestion. A recent study done in Bangladesh showed that children's ingestion of soil was higher than that of children in high-income countries. Infants aged 6–23 months had the highest rates of soil ingestion, with crawling children touching soil more than walking children.

Source: Kwong et al.142

Hand-to-mouth and object-to-mouth behaviours

As children grow and their brains and bodies develop, they engage with the world in unique ways. The milestones that children meet as part of the healthy acquisition of physical, cognitive and social skills can, however, put them at increased risk of environmental exposures. For example, the 6-month cognitive milestone of putting things in their mouths to explore them and the 9-month motor milestone of crawling can increase exposures to soil, dust and toxicants on floors and objects. 144



During different developmental stages, children use bottles and various toys. Some are designed to be put in the mouth, while others may be put in the mouth as part of normal exploratory behaviour. Mouthing plastic objects can expose children to chemicals such as plasticizers (like BPA, phthalates and others) and flame retardants, as well as microplastics and nanoplastics. A 2024 umbrella review found that major classes of plastic-associated chemicals are associated with health effects in children including obesity, adverse neurodevelopment and early puberty.

Source: Aurisano et al., and Symeonides et al. 145



The composition of house dust can be significantly affected by activities near the home. For instance, the application of pesticides within 4 kilometres of a home has been shown to be a significant determinant of indoor contamination, putting young children at risk of pesticide exposure. Toddlers in agricultural communities have also been shown to have higher levels of pesticides in their urine compared to adults. The significant of the significantly affected by activities near the home. For instance, the application of pesticides within 4 kilometres of a home has been shown to be a significant determinant of indoor contamination, putting young children at risk of pesticides in the significant determinant of indoor contamination, putting young children at risk of pesticides in the significant determinant of indoor contamination, putting young children at risk of pesticide exposure.

A perfect storm: How children's unique vulnerabilities put them at risk of lead poisoning

Globally, 800 million children have been shown to have lead poisoning. The majority of these children live in LMICs. There is no safe level of exposure to lead, which is found in many items including lead-based paint, lead pipes, contaminated waste sites, some toys and jewellery, traditional cosmetics, lead-based ceramic glazes, certain spices and others. Young children's hand-to-mouth and object-to-mouth behaviours, crawling, higher gut absorption and developing brain put children at high risk of negative effects of lead poisoning. Lead levels typically peak between 18 and 30 months of age. Children living in a town in Zambia with historical mining of lead, which resulted in polluted soils and homes, had higher blood lead levels than adults living under similar conditions, with peak levels around 2 years of age.

Source: United Nations Children's Fund and Pure Earth, Lanphear et al., and Yabe et al. 148

A closer look: The microenvironment of the school bus

Children who travel to school on a bus with a diesel engine can be exposed to high concentrations of pollutants during their commutes or at loading and unloading zones. Diesel exhaust is a toxic mixture containing fine particulate matter, sulfur dioxides, heavy metals, polyaromatic hydrocarbons, volatile organic compounds and other toxicants. While all children are vulnerable to diesel exhaust, children with asthma may be at particular risk. Buses fueled with cleaner fuels (such as ultralow sulfur diesel) have been shown to improve lung function and reduce school absenteeism, especially in children with asthma.

Source: Adar et al., Pandya et al., and Behrentz et al. 149

Different microenvironments of exposure

Children have different microenvironments of exposure. Depending on age and mobility, children spend their time closer to the floor than adults, putting them at increased risk of exposure to chemicals which settle closer to or on the ground. For example, mercury vapour, aerosolized pesticides and radon are heavier than air, so concentrations are highest near the floor. Radon is a naturally occurring radioactive gas and carcinogen that can leak through cracks and gaps into homes.

Time spent indoors and outdoors

Young children also require more sleep and naps which may lead to more time spent in the house, which can be problematic in the presence of household air pollution (such as emissions from unclean fuels burned for cooking, heating and lighting), volatile organic compounds and particles from plastics, mould and dust. Home based informal e-waste recycling activities can also present toxic hazards for children.

As children enter preschool and school, they also begin to spend more time outside, which is essential for their physical, cognitive and mental health. Outdoor play has been shown to be associated with lower obesity rates, improved mood, increased attention and better learning outcomes. When outdoor environments are unhealthy and lack safe open play spaces, however, children can be put at risk of exposure to air pollution, pesticides and other toxic chemicals, climate hazards such as heat waves and floods, and contaminants of war (such as heavy metals).

2. Unique diets

Breastfeeding

Breast milk is the ideal food for newborns and infants. It has been shown to have nutritional, metabolic, immune and neurological benefits.¹⁵⁴ Both the World Health Organization (WHO) and UNICEF recommend exclusive breastfeeding for the first 6 months of life, and the introduction of nutritionally adequate and safe complementary foods at 6 months together with continued breastfeeding up to 2 years of age or beyond.¹⁵⁵

Breast milk can also unfortunately be a significant source of environmental chemical exposures. ¹⁵⁶ Breast milk has a higher fat content than blood, so chemicals which concentrate in fat may be present in higher levels in breast milk. Many chemicals have been found in breast milk, including heavy metals, flame retardants, plasticizers, sunscreens and various persistent organic pollutants. ¹⁵⁷ In 2017, WHO conducted a global monitoring study of human milk in 52 countries, analysing levels of many persistent pollutants including dioxins, furans, polychlorinated biphenyls (PCBs) and DDT. The highest pollutant levels in milk were seen mostly in areas with industrial activity. The highest levels of DDT and its metabolites were almost exclusively associated with countries where malaria is still endemic. ¹⁵⁸

Contaminants in milk can also come from substances stored within a mother's body. For example, releasing calcium from bones during lactation can also release stored lead that can then be excreted into breast milk. 159 Chemicals stored in fat can also be excreted into breast milk, meaning maternal weight loss may lead to increased organic pollutants in breast milk. 160

Despite potential risks from environmental exposures in breast milk, it remains widely agreed that breast milk is still best given its well-documented benefits.¹⁶¹ Efforts should be focused on reducing environmental contamination in the first place in order to reduce maternal exposures and protect breast milk.

Limited diets

Children's diets differ from those of adults. They consume more milk, fruits and vegetables than adults. They may eat a diet that is less varied than that of adults, putting them at higher risk of ingesting environmental contaminants in favoured or commonly eaten foods. For example, because inorganic arsenic can be found in rice, infants who eat rice cereal are at increased risk of arsenic exposure. In some countries rice cereal is one of the first solid foods given to infants and is a significant part of their diet. 163

Cognitive immaturity and dependence on adults

Children do not yet have the cognitive maturity necessary to protect themselves from harm in both natural and built environments. Children are curious but lack judgement and the ability to read warning labels or instructions. Children also may be reluctant to admit they have ingested a substance or be unable to communicate details of what happened.¹⁶⁴

Common household items including medications, cosmetics, personal care products, household chemicals including pesticides, and (in LMICs) kerosene are among the most common causes of unintentional childhood poisonings. Age and developmental stage can affect access to substances – infants, for example, have increased risk of exposure to harmful substances at ground level. Poisoning rates increase around the age of 2 years as children become more mobile and have more access to harmful substances. 166

Children under the age of 1 year have the highest rate of fatal poisonings, especially in LMICs.¹⁶⁷ Poisoning mortality rates are generally highest in infants and decrease until age 14, then increase again after age 15, potentially due to substance use, unintentional or undetermined drug overdoses or entry into the workplace.

Household products that look and feel like toys or candy can be particularly dangerous to children. For example, laundry detergent pods have been shown to be especially attractive to children because of their candy-like appearance. These pods have been shown to cause more severe symptoms and adverse health outcomes than exposures from non-pod laundry detergent. In one US-based study, 94 per cent of laundry pod exposures involved children under 5 years of age.

Source: United States Centers for Disease Control and Prevention. 168



Longer life expectancy

When children are exposed to environmental hazards early in life, some health outcomes may not appear for decades. It is now widely accepted that early life exposures are associated with many non-communicable diseases later in life including cancer, obesity, diabetes, high blood pressure, heart disease and lung disease. 169

During childhood, cells and tissues are rapidly dividing, which make them prime targets for carcinogens that can cause mutations in dividing DNA.¹⁷⁰ Early exposure to carcinogens is a risk factor for cancer later in life.¹⁷¹ Because young people have many expected years of life,

cancers which have long latency periods can affect them more than if the same exposure happened to an older person who will not live enough years for the cancer develop. For example, it is well established that early childhood sunburns are a risk factor for malignant melanoma, the deadliest form of skin cancer, later in life. Other childhood exposures that are associated with cancers in adulthood include household air pollution, ambient air pollution and diesel exhaust (lung cancer); asbestos (mesothelioma); arsenic (lung, urinary and non-melanoma skin cancer); and aflatoxins (liver cancer).¹⁷²



Period of extreme vulnerability: The neonatal period

A healthy start in life begins in the antenatal period, which is the most vulnerable time for environmental exposures. The next most vulnerable period is after birth, from the first day of life through day 28.

Transition to life outside the womb

The transition to life outside the womb is a time of dramatic and unparalleled changes in an infant's organs.¹⁷³ The lungs take their first breath and begin gas exchange, triggering radical shifts in cardiovascular blood flow and function so that the heart can take over the work of the placenta and umbilical vessels. Feeding via the gastrointestinal tract begins. Red blood cell counts, which are relatively higher in utero, begin to fall to postnatal levels and in the process can cause transient jaundice.¹⁷⁴

During and after the immediate transition, vulnerability is heightened. While organs are functional, function can be substantially different from that of older children and adults. For instance, a neonate's immune responses are inefficient, relying on innate rather than adaptive or specific immunity, which contributes to their susceptibility to infections. The immune cell response in neonatal lungs is also deficient, putting them at risk of pneumonia. Additionally, neonates have a limited capacity to regulate their body temperature, making them vulnerable to heat loss as well as overheating.

A period of increased mortality

Newborns are, in fact, the most vulnerable population in the world; the risk of dying in the first week of life is higher than in any other period in the human lifespan.¹⁷⁸ Every year, 30 million newborns require special or intensive newborn care in a hospital.¹⁷⁹ In 2022, almost one half of all deaths in children under 5 years of age occurred in the newborn period.¹⁸⁰ Maternal exposures to environmental hazards during pregnancy can also play a role, as they may be associated with conditions such as low birthweight, prematurity and birth defects that increase vulnerability to diseases and death, especially in the neonatal period.¹⁸¹

Special considerations for premature infants

The neonatal period presents more difficult challenges for babies born prematurely, i.e., before 37 weeks of gestation. Unlike full-term infants, the organ systems of premature babies are not prepared to support life outside the womb, especially in infants born severely premature who require more interventions to survive. Short-term complications of prematurity include respiratory distress, unstable circulation, feeding difficulties, infections, brain injury, eye damage and anaemia. 183

What happens when newborns are faced with environmental challenges

While the newborn baby is adjusting to their first critical month outside the womb, environmental challenges may be especially problematic. Newborns – especially premature infants – can become hypothermic if they are not in a sufficiently warm environment, which can contribute to mortality from other neonatal illnesses.¹⁸⁴ A naked newborn exposed to an environmental temperature of 23°C suffers the same heat loss as a naked adult in 0°C.¹⁸⁵

Newborns also are particularly susceptible to hyperthermia, which can result from overbundling, especially in hot, humid climates; being left in direct sunlight or in a parked car in hot weather; or being placed too close to a fire, heater or hot water bottle. Hyperthermia can lead to severe dehydration and potentially death.

Other environmental challenges include air pollution and lack of adequate water, sanitation and hygiene (WASH), which can increase the risk of infections, including pneumonia.¹⁸⁷

With environmental challenges that can put already vulnerable newborns at increased risk, appropriate care can be critical. Access to special care (which includes access to WASH, electricity, oxygen supply, specialized nursing staff and other requirements) can help prevent neonatal deaths. Nearly all neonatal deaths (98 per cent) occur in low- and middle-income countries (LMICs), with the highest neonatal mortality rates being found in countries with humanitarian crises. 189

The first week of life: The highest risk of death, intensified by environmental hazards Changing heart and lung functions to adapt to life outside the womb Risk of dying in first week of life is higher than in any other period in the human lifespan Inefficient regulation of body temperature increases risk of hypothermia/hyperthermia Inefficient immune system increases risk of infections

Developing organ systems in childhood and environmental hazards

1. Brain and central nervous system

Early childhood is a period of significant and dynamic brain development. Brain plasticity (which is at its maximum during the first years of life) allows a child to learn and develop through their experiences. It also, however, makes the brain susceptible to environmental hazards which can alter the trajectory of brain development.

Children are born with most of their neurons, the nerve cells that send and receive signals that allow us to do everything from breathing to talking, eating, walking and thinking.¹⁹⁰ There is some evidence to suggest that neurons also continue to form throughout life.

Neurons undergo many phases of development that are critical for building the networks that make up the complex architecture of the brain. After neurons proliferate, they migrate to their final location in the brain. Most migration takes place in utero but recent evidence suggests that in some areas of the brain, migration can continue for several months after birth.¹⁹¹ Connections between neurons, called synapses, start to develop in utero. In the first few years of life, more than 1 million new synapses form every second.¹⁹²

Different parts of the brain reach peak synapse production at different times, affecting the plasticity of each region. Synapses are also pruned, a process driven by experiences. The overproduction of synapses followed by pruning and the interconnection of neurons to form neural networks are essential to a child's learning. Finally, neurons complete development through myelination, the wrapping of fatty cells around the axon of the neuron which helps the neuron send signals faster.¹⁹³

Environmental hazards can affect all these crucial steps in the building of the brain's architecture, with implications on future cognitive function. For instance, lead can interfere with the normal function of important chemical messengers in the brain, which in turn affects the formation and sculpting of neural networks as well as myelination.¹⁹⁴ Even at low levels of exposure, lead is associated with decreased academic achievement, lower IQ and attention problems.¹⁹⁵ Exposure to air pollution and hazardous chemicals in plastics in early childhood also can affect neurodevelopment.¹⁹⁶ Air pollution can also affect sleep quality in young children which is important because insufficient sleep in early years is associated with cognitive impairments.¹⁹⁷

Exposure to various environmental hazards, including heavy metals and other pollutants, can also cause epigenetic changes (or changes to the way a gene is expressed) in the developing brain, particularly in early childhood.¹⁹⁸

2. Respiratory system

Lungs and respiratory tract

After birth, the lungs continue to develop. The alveoli, the tiny air sacs where oxygen and carbon dioxide are exchanged, grow in number. The greatest increase happens during the first 18–24 months of life and continues to age 8.¹⁹⁹ The blood vessels in the lungs also develop during the first 2–3 years of life.²⁰⁰ Lung function develops throughout childhood and continues until late adolescence.²⁰¹ Lung function tracks along percentiles, which means that the lung function an infant has at birth largely determines lung function throughout life.²⁰²

Environmental hazards can negatively affect lung development, especially during the period when

alveoli are rapidly increasing. Adverse exposures can also affect the epithelium, which is a protective lining in the airways that also has multiple immune functions. Early life exposures to environmental hazards such as fine particulate matter in air pollution can affect the structural and functional integrity of the epithelium and contribute to the development of respiratory diseases, including asthma.²⁰³ Exposure to air pollution in childhood is also associated with pneumonia with complicated course and poor health outcomes.²⁰⁴ Second-hand smoke, which can contaminate children's environments when tobacco products are burned or when a smoker exhales smoke, contains over 7,000 chemicals, including approximately 70 that can cause cancer.²⁰⁵ Second-hand smoke is linked to respiratory infections and asthma attacks.²⁰⁶ It is also linked to sudden infant death syndrome.

Effects from air pollution on the developing respiratory system can last throughout life. For example, chronic obstructive pulmonary disease (COPD) is an adult condition that has links to early exposures to air pollution.²⁰⁷



3. Kidneys and urinary system

Children's kidneys are more vulnerable to environmental hazards than those of adults. Although the generation of nephrons – the functional units of the kidneys – is complete by birth, the filtration function of the kidneys matures during infancy, making it a period of vulnerability of the kidneys to environmental hazards such as heavy metals.²⁰⁸ Altered kidney filtration is an established risk factor for chronic kidney disease²⁰⁹ as kidneys cannot generate new nephrons to compensate for altered function of existing nephrons.²¹⁰

4. Immune system

The immune system and the microbiome develop in early postnatal life through complex processes that aim to meet the challenge of protecting against foreign pathogens while not attacking the body's own tissue, which would lead to autoimmune diseases. These developing processes are more or at least differently susceptible to environmental hazards than their adult counterparts. For example, dioxins – persistent organic pollutants – are harmful to children at significantly lower doses than those needed to produce effects in adults.²¹¹ Effects of early exposure to environmental hazards can be long lasting or appear long after exposure.

The developing innate and adaptive immunities are vulnerable to negative effects from environmental hazards such as air pollution, which can disrupt immune function in the respiratory tract and contribute to the development of allergic rhinitis and asthma.²¹²

5. Endocrine and reproductive systems

Reproductive cells

Children are born with the cells that will become eggs and sperm. Damage to these cells can occur anytime, including during childhood and adolescence, all the way up to conception.²¹³ Egg and sperm cells also develop differently, which can affect the windows of sensitivity

to environmental hazards and, ultimately, fertility and health impacts.²¹⁴

'Mini-puberty'

After birth, there are two periods of activation of the system that controls reproductive and sexual development, i.e., the hypothalamic-pituitary-gonad axis which connects the brain with the testicles and ovaries. The first period is called 'mini-puberty' and takes place from birth to around age 6 months in boys and from birth to potentially age 2–4 years in girls. The second period is puberty, which takes place during adolescence.

'Mini-puberty' is less well known than its adolescent counterpart but can have lasting impacts on a child's reproductive and sexual health. After birth, the drop in placental hormones in the newborn's circulation leads to a surge in activity in the hypothalamic-pituitary-gonad axis that causes the release of hormones. In boys, this affects the development of the testes, penis and prostate gland while in girls the effects are less well understood. 'Mini-puberty' is a critical window for exposure to endocrine-disrupting chemicals, including those found in plastics (BPA, phthalates, flame retardants), polychlorinated biphenyls (banned worldwide but still widely found in the environment)²¹⁵ and DDT. These chemicals have a wide array of effects, including premature breast development and both early and late puberty.²¹⁶

6. Haematologic system

The production of blood cells in children takes place in the bone marrow and involves high rates of cell division and growth. The rapid division of blood cells makes them vulnerable to environmental hazards. Leukaemia is a cancer of the white blood cells, which help fight infections, and is the most common type of cancer in children. Several environmental hazards are associated with leukaemia, including ionizing radiation, benzene and pesticides.²¹⁷





Environmental exposures during critical windows of vulnerability in childhood can have lasting negative effects on a child's health

Brain and central nervous system

Rapid growth of the brain's architecture and function means damage from environmental hazards during early development can have lifelong impacts.

Lungs

Rapid growth of alveoli from birth to age 2 and up to age 8 means early exposures can lead to issues like pneumonia, childhood asthma and chronic lung disease in adulthood.

Kidneys

Maturation of kidney filtration occurs in infancy. Harmful exposures during this period increase the risk of chronic kidney disease.

Immune system

Early development of the immune system and microbiome is sensitive to exposures, raising risks of infections, asthma and allergies.

Haematologic system

Rapid blood cell division during childhood makes this period sensitive to exposures linked to blood cancers like leukaemia.

Reproductive system

Reproductive cells, present from birth, can be damaged by harmful exposures. The 'mini-puberty' phase is critical for organ development and is vulnerable to endocrine-disrupting chemicals.

3. Adolescents' unique vulnerabilities to environmental hazards

While adolescence (10-19 years old) is often considered the healthiest stage of life, dynamic changes in adolescent bodies and brains make them uniquely vulnerable to environmental hazards. This section focuses on environmental exposures that take place during adolescence. Health effects that emerge during adolescence as a result of antenatal or early childhood exposures are not reviewed.

Key messages

- Puberty causes virtually every system in the adolescent body to go through profound transformation of both physiology and function. From the brain to the lungs, bones, immune system and reproductive system, these dramatic changes are vulnerable to disruption by pollutants, disease, nutritional deficiency and extreme weather events driven by climate change.
- The brain development that occurs during adolescence is second only to infancy in terms of extent and significance. Neurological changes shape a wide range of cognitive functions, from thinking to emotional processing, memory and motivation. These changes are vulnerable to environmental risks.
- Adolescents are at high risk of developing mental health conditions. Navigating these changes is more difficult when community support and social infrastructure are disrupted by climate change-related disasters.
- Certain changes that occur in the brain during adolescence can lead to poor self-regulation and an increased tendency towards risk-seeking behaviours.
- The elevated nutritional needs of adolescents make them susceptible to harm from poor nutrition that can affect their health for the rest of their lives.
- Lung function during adolescence transforms to reach adult capacity. Environmental hazards such as air pollution can harm this transition, affecting lifelong respiratory health and increasing the future risk of respiratory diseases.
- Adolescents face unique risks of communicable diseases beyond those specific to childhood. These include an increased risk of tuberculosis, as well as the hazards posed by HIV and other sexually transmitted infections.
- Chemicals found in plastics and pesticides can increase the prevalence of adolescent obesity, which has quadrupled since 1990. Adolescents are at risk of obesity due to their increased energy and nutritional needs – risk which is further heightened by unhealthy diets and insufficient physical activity. Adolescent obesity can lead to many serious health problems in adult life.
- Environmental hazards are increased for adolescents who are involved in child labour, particularly in hazardous work in informal e-waste recycling, plastic waste recycling, artisanal and small-scale gold mining, and agriculture, where the likelihood of exposure to toxicants is high and the knowledge and prevalence of protections from these exposures is low. Informal work around the home such as cooking with polluting fuels can also expose adolescents to harmful household air pollution.

Examples of environmental hazards and how they affect adolescents





Climate-related hazards

Extreme weather

Climate change can affect the physical, social and emotional development of adolescents. Adolescents are at risk of vector-borne diseases such as malaria, which will become more prevalent in a changing climate. Extreme heat can affect sleep, learning and school attendance. It can also cause heat illness in adolescents playing outdoor sports or working in outdoor occupations. Floodwaters pose risk to adolescents, who have increased risk-taking behaviours that can lead to injuries and drowning.

Climate change also threatens most aspects of adolescent mental health. Climate-related disasters can shatter the social and community infrastructure that adolescents need as they navigate the complexities of peer relationships, self-esteem and identity development, and put them at risk for mental health disorders such as post-traumatic stress disorder, depression and anxiety.

Food insecurity

Environmental hazards such as climate changerelated floods and droughts can reduce food security, affecting adolescents, who have increased caloric and nutritional needs to feed the rapid growth of their bodies and brains. Food insecurity and poor nutrition can affect the accumulation of bone, muscle and fat and alter the maturation of biological systems as well as the timing of puberty. Appropriate levels of bone development are important, as not achieving healthy bone mass in adolescence is associated with osteoporosis (i.e., weak or brittle bones) in adults. Poor nutrition can also contribute to iron deficiency – a mineral important to growing adolescents – and related anaemia, and can affect the ongoing development of the brain and immune system.

Infectious diseases

Adolescents are at risk from many common diseases that affect children - such as gastrointestinal infections, lower respiratory tract infections and malaria – and also have increased risk of infections that emerge in adolescence. Adolescents are at higher risk of active tuberculosis (TB) than children, possibly due to changes in immunity during puberty but also due to expanded social networks and exposures in schools and transit situations. Adolescence also marks a time for risk of sexually transmitted infections (STIs), including HIV as well as syphilis, gonorrhoea, chlamydia and human papillomavirus (HPV). Many factors contribute to STIs in adolescents, including lack of access to services for reproductive and sexual health, inconsistent condom use and increased risktaking behaviours.

Pollution

Air pollution

Air pollution, both ambient and household, affects many systems in an adolescent's body. It can disrupt developing lung function, which can lead to reduced lung function and lung disease later in life. It can also trigger flares of asthma, a chronic disease which can be challenging to manage for adolescents gaining autonomy. Air pollution has negative effects on the developing brain and is associated with disrupted sleep. Poor outdoor air quality may interfere with physical activity, which can in turn affect adolescents' physical fitness. Air pollution can also contain toxic chemicals called obesogens, which can promote obesity.

Pesticides

Adolescents who work in child labour in agriculture are at risk from pesticide exposure which can affect their developing brains and cause neurological symptoms. Pesticides can also affect other developing tissues. For example, although no longer used in

agriculture, exposure to DDT during puberty in girls has been linked to increased risk of breast cancer in adulthood.

Endocrine-disrupting chemicals

Adolescents are exposed to a wide array of chemicals in the air they breathe, the foods they eat, the water they drink and the products they use. Many chemicals have been shown to disrupt the function of hormones in the body which control vital processes such as growth, metabolism, reproductive and sexual development, and immune function. There is some research that shows certain endocrine-disrupting chemicals (EDCs) may affect timing of puberty; ongoing research is needed to define sensitive time windows for exposure. EDCs such as plasticizers, pesticides, perand polyfluoroalkyl substances (PFAS) and polycyclic aromatic hydrocarbons (PAHs) can also promote obesity - an important consideration given the increasing rates of obesity in adolescents globally and the lifelong impacts of adolescent obesity, including future heart disease.



The factors which make adolescents uniquely vulnerable to environmental hazards

Technical brief

Dynamic physiology

1. Puberty

The beginning of adolescence is marked by the onset of puberty, which triggers a period of rapid growth and development of the body and brain, including the attainment of sexual and reproductive maturity.²¹⁸ After 'mini-puberty' in infancy, which ends around age 6 months in boys and age 2–4 years in girls, there is a reactivation of the systems that control reproductive development, i.e., the hypothalamic-pituitary-gonad axis which connects the brain to the testicles or ovaries.²¹⁹ The age at which puberty starts differs amongst individuals, typically around ages 8–12 in girls and 9–14 in boys.²²⁰

There are numerous physiologic changes that happen during puberty. The growth spurt associated with puberty is the most dramatic change. This growth spurt starts earlier in girls than in boys; after menses start, girls generally grow an additional 2.5 cm. Boys continue to grow slowly after puberty ends and reach their adult height around age 18. Bone mass continues to accrue, not reaching its peak until a person reaches their early 20s. The cardiovascular, renal and immune systems are also developing. Sex hormones produced during puberty can affect the regulation of the immune system, possibly contributing to the increased risk of autoimmune disease in adolescent girls.²²¹ Blood pressure and heart rate make a transition to adult values along with height and weight.²²²

The steady growth in height, the accumulation of bone, muscle and fat mass, and the maturation²²³ of various biological systems that occur during puberty can be affected by nutrition, making puberty a nutrition-sensitive window to promote healthy growth. Extended undernutrition in adolescents, as shown in patients with anorexia nervosa, can result in reduced grey matter (nerve cells) and white matter (extensions of nerve cells) in the brain, especially in networks which control higher executive functions.²²⁴ Inadequate intake of protein and vitamins B12, C and D can also impair most immune functions.²²⁵



Timing of puberty

In the past 30 years, the onset of puberty has occurred earlier than before in both boys and girls.²²⁶ Genetics largely determine the onset of puberty, but other factors such as nutritional status also play a role. For example, chronic energy insufficiency can delay puberty, while chronic energy surplus is related to earlier puberty start.²²⁷ Environmental factors may also be important. For instance, animal data suggests that exposure to EDCs may play a role in shifting the onset of puberty, although overall human research has been inconclusive and precise windows of exposure have not been clearly defined.²²⁸ Nevertheless, a 2024 umbrella review of meta-analyses that evaluated associations between human health and exposure to major classes of plastic-associated chemicals did find that childhood exposure to some chemicals, such as the phthalate DEHP, was significantly associated with precocious puberty in girls.²²⁹

Timing of puberty is important beyond adolescence as it is associated with adult health outcomes. For example, early onset of puberty is associated with increased risk of type 2 diabetes, obesity, heart disease and some cancers, including breast, endometrial (i.e., the lining of the uterus) and prostate cancers.²³⁰

2. Developing brains

Over the past 20 years, research has shown that the extent and significance of the development of neural systems that occurs during adolescence is second only to those that occur infancy.²³¹ During this time, there are rapid changes in the cerebral cortex, which is the outermost layer of the brain that controls key cognitive functions such as thinking, learning, memory and problem solving.

The development of the cerebral cortex includes maturation of neurotransmitter systems which help brain cells communicate, and significant increases in the sizes of certain regions of the brain, including the amygdala, an almond-shaped region of the brain that helps process emotions, and the hippocampus, which is involved in learning and memory.²³² Connections between nerve cells, or synapses, continue to be pruned during this time, which facilitates brain plasticity and is a hallmark of adolescent brain development.²³³ There is also a surge in connectivity and communication between different lobes of the brain.²³⁴ Synapses which transmit dopamine - which is closely associated with how the brain handles reward seeking, motivation and impulsivity - are modified in the adolescent brain, and may be related to tendency to risky decision making.²³⁵

Brain development during adolescence can be affected by many environmental hazards. For example, animal studies have shown that exposure to air pollution can disrupt the pruning of synapses and the development of neural circuitry.²³⁶ Studies which evaluate the structure of human brains using MRI have shown that both fine particle pollution (i.e., PM_{2.5}) and gaseous pollutants such as ozone and nitrogen oxides have been linked to changes in brain structure and function such as cortical thickness, surface area and volume, the microstructure of grey matter (nerve cells) and white matter (extensions of nerve cells), cerebral blood flow, brain metabolites and functional connectivity.²³⁷

The significant and complex development of neural circuits in adolescence raises concern that the adolescent brain is vulnerable to environmental hazards during this period. Emergence of health effects from antenatal and childhood exposures can compound health effects from exposures during adolescence, which can complicate research seeking to evaluate links between exposures and specific health effects. Exposure to mercury during adolescence, for instance, may be associated with behaviour changes, although more studies are needed²³⁸ as antenatal and childhood exposures are also common.

Clear evidence that the developing adolescent brain may be uniquely vulnerable to toxic exposures comes from the study of the effects of tobacco and other substances such as marijuana. The timing of drug exposure can be important. For instance, exposure during adolescence to THC, the main psychoactive ingredient in the cannabis plant, can increase risk of substance abuse later in life, and exposures to nicotine during early adolescence may increase risk of dependence.²³⁹

Healthy environments during adolescence, a critical period of brain development, are vital to healthy development. In addition, programs and strategies that help adolescents build resiliency and manage environmental challenges can promote adolescent and lifelong health.²⁴⁰

3. Psychological and emotional development and mental health

Adolescence is a critical time for emotional development. The myriad of changes that occur in the brain and body of an adolescent are concurrent with social changes involving peers, schools, workplaces and communities, which adolescents need to learn to navigate.²⁴¹ The onset of puberty drives changes in the brain's limbic system, which regulates emotions and behaviours. Adolescents learn to identify and regulate emotions and learn how they affect thoughts and behaviours. In early adolescence,²⁴² self-esteem is often at its lowest point





and tends to improve in middle to late adolescence.²⁴³ Adolescents have increased interest in engaging socially with peers and potential romantic partners.

During early adolescence, individuals are susceptible to peer pressure, have a limited focus on long-term consequences, and are unable to effectively estimate risk, all of which can lead to increased risk-taking behaviours and poor self-regulation.²⁴⁴ Adolescents are also at high risk of developing mental health conditions such as major depression, eating disorders, substance use and anxiety disorders.²⁴⁵ Supportive homes, schools and communities can help adolescents handle stress effectively.

Climate change threatens adolescents' emotional well-being. At a time when they are vulnerable to anxiety, adolescents feel the threat that climate change brings.²⁴⁶ Stressors associated with climate change such as social and economic disruptors could increase harmful substance use in a vulnerable adolescent population.²⁴⁷ Climate change-related events such as flooding and wildfires can present unique mental health challenges to adolescents, forcing them to navigate the complexities of their social and emotional development while dealing with complex stressors such as migration and displacement.²⁴⁸ For example, climate change-related disasters are associated with post-traumatic stress disorder in adolescents, mainly those exposed to disaster-related injury, death and loss while simultaneously lacking social and family support.²⁴⁹ Overall, while not specific to adolescents, climate change events and conditions are also related to stress, anxiety, depression and mood.²⁵⁰ Further research is needed to delineate specific mental health outcomes for adolescents as the climate crisis continues.

Effects of climate change on learning

As adolescents are developing physically, cognitively and socially, schools can play an important role in promoting adolescent health.²⁵¹ Climate change-related weather events can result in school closures. A 2024 report from the World Bank found that in low-income countries, students on average lost 18 days of school annually, compared with 2.4 days in wealthier countries.²⁵² Furthermore, extreme heat itself negatively affects learning.²⁵³

4. Developing bones

Around one half of bone mass is accrued during adolescence, making it a critical window for bone health as reduced peak bone mass is a risk factor for osteoporosis, which can have significant health impacts in older adults.²⁵⁴ Environmental factors such as poor nutrition (particularly, reduced intake of calcium and vitamin D), lack of weight-bearing activity, and smoking can negatively affect bone mass.²⁵⁵

5. Metabolic changes

Adolescents have increased energy and nutrient requirements to meet the needs of their rapidly growing bodies.²⁵⁶ It can be a challenge to meet these needs while maintaining a healthy weight. Maintaining a healthy weight during adolescence is important because obesity during adolescence is associated with an array of medical problems including prediabetes, type 2 diabetes, fatty liver disease, abnormal levels of fats in the blood, polycystic ovarian syndrome, obstructive sleep apnoea and mental health disorders.²⁶⁷ Obesity during adolescence is also associated with adult health problems including heart disease and death from any cause.²⁵⁸

Since 1990, the number of obese adolescents globally has quadrupled.²⁵⁹ Many factors contribute to obesity including unhealthy diets, low levels of physical activity and sedentary lifestyles.²⁶⁰ Additional environmental factors can play a role, including exposure to obesogens, which are chemicals that can affect hormones that control hunger and satiety, and can disrupt functions in cells related to metabolism and inflammation.²⁶¹ These chemicals are ubiquitous and include the plasticizers bisphenol A and phthalates, as well as dioxins, the pesticide atrazine, PFAS, flame retardants and PAHs.²⁶²

6. Cardiovascular development

The cardiovascular system continues to develop in adolescence and sets the stage for cardiovascular health in adulthood. For example, high blood pressure in adolescence is a risk factor for high blood pressure and cardiovascular disease in adults.²⁶³ Air pollution has been shown to be associated with increased blood pressure in adolescents.²⁶⁴ There is also suggestive evidence that early atherosclerosis can begin in adolescence and be affected by exposure to ambient air pollution, which in adults has been shown to be a modifiable risk factor for heart disease.²⁶⁵

7. Lung development

Lung function continues to mature in adolescence, developing rapidly around ages 11–15 years in both boys and girls. Lung function growth then slows but continues in girls until the late teen years, and in boys until the early twenties. As adolescents transition to adult status, their lung function can affect the likelihood of having respiratory diseases later in life.²⁶⁶

Air pollution has been shown to have significant impact on the development of lung function in adolescents. In a well-known study done in Los Angeles in the United States, improved outdoor air quality achieved over decades was associated with a decreased proportion of adolescents with low lung function at age 15 years. The finding was seen in adolescents both with and without asthma.²⁶⁷

8. Vulnerability to certain infectious diseases

Although globally there have been significant reductions in the burden of and death from communicable diseases among children under 5 years of age, the decline in mortality rate has been less pronounced in adolescents. Research that specifically looks at global communicable disease burden in adolescents has historically been lacking. In 2021, a major global report showed that while gastrointestinal infections, lower respiratory tract infections and malaria were major causes of communicable disease in both children and adolescents, TB and HIV emerged as important causes specifically during adolescence.²⁶⁸

Tuberculosis

Global attention on TB has been focused on cases. in adults, with the true burden of TB in adolescents remaining uncertain. Age-related trends have, however, been observed. For example, the risk of progression from latent TB to active TB is lowest in children aged 5–9 years, then increases through adolescence, possibly due to changes in immunity during puberty. TB disease type also shifts during adolescence to a more transmissible form typically seen in adults. There are also differences in susceptibility to TB by sex: The disease affects younger girls and boy equally, but the risk for females increases around the time of menarche, resulting in a higher incidence of TB and disease progression compared to boys of the same age. Social and environmental factors that contribute to TB in adolescents include expanded social networks, overcrowded housing, greater exposure in schools and on transit, and air pollution.²⁶⁹

HIV

Overall, new infections and HIV deaths are decreasing in adolescents as in all age groups globally.²⁷⁰ In 2022, adolescents aged 10–19 years accounted for 4 per cent of people living with HIV but carried 10 per cent of new infections, with 71 per cent of those new infections occurring in girls. Sub-Saharan Africa carries the majority of the burden of new HIV infections in adolescents, with only 33 per cent of new cases occurring outside the region.²⁷¹

Increasing global investment in preventing TB and HIV in adolescents is needed.²⁷² TB services that address the needs of adolescents are required, such as integration of peer support and mental health services to address the social isolation and disruption of education that may be caused by TB and its treatment.²⁷³





Other sexually transmitted infections

Other STIs including syphilis, chlamydia, gonorrhoea, trichomoniasis and genital herpes (related to HPV) are prevalent in adolescence. In the 2019 Global Burden of Disease analysis which evaluated the incidence of other STIs from 1990 to 2019 in people ages 10–24 years, adolescents aged 10–14 were the only age group to have an increase in the number of cases.²⁷⁴ Inconsistent condom usage contributes to STIs.²⁷⁵

Adolescent girls are especially vulnerable to STIs such as chlamydia and HPV due to differences in the structure of cells and mucous in the cervix, which is the lower part of the uterus that connects with the vagina.²⁷⁶ Certain strains of HPV are associated with cervical cancers.²⁷⁷ HPV is also associated with other cancers, including anal and penile cancer.²⁷⁸

Understanding complex environmental factors such as neighbourhood influences related to local structural barriers to accessing knowledge and services related to sexual and reproductive health is critical, as is reducing the access gap that exists between rural and urban areas.²⁷⁹

9. Physical fitness

Adolescence is an important time to lay the groundwork to develop and maintain physical fitness that can affect lifelong health. There is strong evidence that higher levels of cardiorespiratory fitness and healthy body composition in adolescence are associated with better cardiovascular health later in life.²⁸⁰ Despite the importance of adolescence as a key time to maintain physical fitness, global data suggests that around 80 per cent of adolescents aged 11–17 years do not meet current physical activity guidelines.²⁸¹

Unhealthy and unsafe built environments, air pollution and disruptions caused by climate change-related disasters can all create barriers to adolescents being physically active, which can affect their physical fitness and cardiovascular health.

10. Changes in sleep patterns

Sleep plays an important role in physical and mental health, immune function and performance in school. In adolescents, the processes in the body that control sleep regulation undergo dramatic changes that lead to adolescents having different sleep patterns. Adolescents generally have later sleep-onset time which makes it difficult to fall asleep early in the evening and get up in the morning. Adolescents 10–12 years old are recommended to get 9–12 hours of sleep while older adolescents are recommended to get 8–10 hours of sleep.²⁸²

Many social factors can affect sleep hygiene including school start times, homework load and job responsibilities. Although not specific to adolescents, environmental hazards, including both indoor and outdoor air pollution and extreme heat, can lead to sleep disruption.²⁸³



Evolving cognitive maturity

Potential for risk-taking behaviours

As adolescents develop, the first changes that occur in the brain spark greater sensitivity to rewards, threats, novelty and peers, whereas the parts of the brain related to cognitive control and self-regulation take longer to mature. This trajectory may bias adolescents' decision making and sensation seeking during the period wherein they are engaging in new and unsupervised activities.²⁸⁴

Unintentional injuries such as road traffic accidents and drowning are the leading cause of death and disability among adolescents.²⁸⁵ In particular, they are a major cause of disability with lifelong consequences, such as acquired brain injury. Adolescents may not recognize the hazard posed by some environmental conditions such as floodwaters or contaminated bodies of water.²⁸⁶ Adolescents are more likely to engage in risky behaviour around water, including consuming alcohol, which can contribute to risk of drowning.²⁸⁷

Climate change increases flooding, which increases risk of drowning, particularly in low- and middle-income countries. Heat events may also increase this risk, as adolescents may seek relief from elevated temperatures. Drowning accidents of children and adolescents aged 5–14 are more likely to occur in open waters, such as rivers, lakes, ponds, reservoirs and seas.²⁸⁸

Challenges in managing chronic diseases

Adolescence is a challenging period in which to manage illnesses effectively, including chronic diseases. Adolescents need to balance their developing need for autonomy and peer relationships with the constant challenge of adhering to treatment plans and/or avoiding triggers for diseases. For example, asthma management is challenging for adolescents, and smoking or exposure to air pollution can trigger flares.²⁸⁹ Type 1 diabetes is also difficult to manage in adolescence, as control and monitoring of blood sugar levels is time intensive and can impact self-esteem and body image at a time when these traits are just developing.²⁹⁰



Special considerations for adolescent girls and young women

Menstruation

Adolescent girls need environments and services that allow them to manage menstruation safely, hygienically and without embarrassment, all of which are fundamental to their sexual and reproductive health and overall well-being.²⁹¹ Poor or inadequate water, sanitation and hygiene (WASH) facilities contribute to infections and anxiety and potential physical or sexual violence.²⁹² Climate change events can also reduce access to WASH and sanitary products.²⁹³

Pregnancy during adolescence

In 2022, an estimated 13 per cent of adolescent girls and young women globally gave birth before age 18, with rates differing widely by global region. Adolescent birth rates are lowest in countries in Europe and North Amer-

ica, and highest in countries in sub-Saharan Africa.²⁹⁴ Pregnancy during adolescence presents unique physical, social and emotional challenges during an already dynamic period and can affect a girl's education, livelihood and health, and subject her to social stigma, rejection and violence.

Many social and environmental factors contribute to adolescent pregnancy, including limited education and low economic status; childhood marriage; lack of access to contraceptives and/or lack of knowledge on how to use them; and sexual abuse.²⁹⁵ Substance use and peer pressure can also play a role.²⁹⁶ Adolescent pregnancy is associated with problems during pregnancy such as pre-eclampsia and maternal anaemia, and with adverse pregnancy outcomes such as low birthweight, prematurity and stillbirth. Adolescents who are pregnant have a higher risk of anaemia due to higher iron needs.²⁹⁷



Unique and different exposures

Occupational

Adolescents who enter the workplace may encounter unique hazards in occupational settings, both in environments with adult-specific safeguards and in informal settings where safeguards are not in place.²⁹⁸ Adolescents may engage in hazardous work in informal e-waste recycling, plastic waste recycling, artisanal and small-scale gold mining, and agriculture. Restrictions and laws around adolescent involvement in various work settings may differ from country to country. The participation of adolescents above the minimum working age in work that does not disrupt their health and personal development or interfere with their schooling is generally regarded as something positive; this can include activities such as assisting in a family business or earning money outside school hours and during school holidays.²⁹⁹ The term 'child labour', on the other hand, refers to work that deprives children of their childhood, potential and dignity, and that is harmful to physical and mental development. Child labour also interferes with schooling.300 According to the International Labour Organization, adolescent boys are more likely than girls to be engaged in child labour.³⁰¹

Adolescents can encounter many hazards in various labour situations including exposure to pesticides and other dangerous agrochemicals. Pesticides are known to affect developing nervous systems, although research on adolescent-specific exposures is lacking. A 2008 study done in Egypt evaluated males 9-18 years of age hired as seasonal workers to spray pesticides in cotton fields with backpack applicators.³⁰² Children and adolescents who applied pesticides had lower activity levels of an enzyme that works at the intersection of nerve cells and is blocked by certain pesticides. They also performed less well on neurobehavioural tests such as those that evaluated their memory and attention span, and reported more neurological symptoms including blurred vision, dizziness, headache and difficulty concentrating. Adolescents may also be engaged in physically strenuous tasks such as carrying heavy loads, standing, stooping and bending for long periods, and repetitive and forceful movements in awkward body positions. Additionally, they may be exposed to extreme temperatures, use dangerous cutting tools, or work around farm vehicles and heavy farm machinery.303



Adolescent girls may have responsibilities such as cooking in the home, which can expose them to harmful household air pollution when polluting fuels and technologies are used. Assessments of household air pollution exposure in adolescent girls are limited, however, despite the fact that they are known to assist with household tasks like cooking and garbage burning.³⁰⁴

Finally, in some contexts, climate change may affect adolescents' entry into the labour market. Extreme weather events may threaten family livelihoods, creating economic pressure that may cause families to pull adolescents from education to supplement income.³⁰⁵

Long life expectancies

At different ages during adolescence, certain cells and tissues of various organs are rapidly dividing, making them prime targets for carcinogens that can cause mutations in replicating DNA.³⁰⁶ Exposure to carcinogens is a risk factor for cancer later in life. Because young people have many expected years of life, cancers which have long latency periods can affect them more than an older person with the same carcinogenic exposure, who will not live enough years to have the cancer develop. For example, one of the sensitive periods for the human breast is during adolescence, when exposure to DDT has been shown to be associated with breast cancer in adult women.³⁰⁷

References

- Etzel, Ruth A., and Philip J. Landrigan, 'Children's Exquisite Vulnerability to Environmental Exposures', ch. 2 in *Textbook of Children's Environmental Health*, edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 18–27.
- Yao, Xiaoxi, et al., 'Fertility Loss: Negative effects of environmental toxicants on oogenesis', Frontiers in Physiology, vol. 14, 4 August 2023, e.1219045; Greeson, Katherine W., et al., 'Inheritance of Paternal Lifestyles and Exposures Through Sperm DNA Methylation', Nature Reviews Urology, vol. 20, no. 6, 18 January 2023, pp. 356–370.
- 3 Stephenson, Judith, et al., 'Before the Beginning: Nutrition and lifestyle in the preconception period and its importance for future health', Lancet, vol. 391, no. 10132, 5 May 2018, pp. 1830–1841.
- 4 Perera, Frederica, and Julie Herbstman, 'Prenatal Environmental Exposures, Epigenetics, and Disease', *Reproductive Toxicology*, vol. 31, no. 3, 12 September 2011, pp. 363–373.
- 5 Ibid.; Li, Yu-Fen, et al., 'Maternal and Grandmaternal Smoking Patterns are Associated with Early Childhood Asthma', Chest, vol. 127, no. 4, April 2005, pp. 1232–1241.
- 6 Agency for Toxic Substances and Disease Registry, 'How Can Parents' Preconception Exposures and In Utero Exposures Affect a Developing Child?'https://archive.cdc.gov/www_atsdr_cdc_gov/csem/pediatric-environmental-health/preconception.html, accessed 5 December 2024.
- 7 Yao et al., 'Fertility Loss'; Greeson et al., 'Inheritance of Paternal Lifestyles and Exposures'; Moore, Keith L., T. Vidhya Persaud and Mark G. Torchia, The Developing Human: Clinically oriented embryology, 11th ed., Elsevier, London, 2020.
- 8 Nilsson, Eric E., Ingrid Sadler-Riggleman and Michael K. Skinner, 'Environmentally Induced Epigenetic Transgenerational Inheritance of Disease', Environmental Epigenetics, vol. 4, no. 2., April 2018.
- 9 Skinner, Michael K., et al., 'Ancestral Dichlorodiphenyltrichloroethane (DDT) Exposure Promotes Epigenetic Transgenerational Inheritance of Obesity', BMC Medicine, vol. 11, no. 228, October 2013.
- 10 United States Centers for Disease Control and Prevention, 'Epigenetics, Health and Disease', 13 November 2024, <www.cdc.gov/genomicsand-health/epigenetics/index.html>, accessed 5 December 2024.
- 11 Perera and Herbstman, 'Prenatal Environmental Exposures'.
- 12 World Health Organization, 'Arsenic', 7 December 2022, <www.who.int/ news-room/fact-sheets/detail/arsenic>, accessed 11 September 2024.
- 13 Ortiz-Garcia, Nancy Y., et al., 'Maternal Exposure to Arsenic and its Impact on Maternal and Fetal Health: A review', The Cureus Journal of Medical Science, vol. 15, no. 11, 21 November 2023, e49177.
- Tolins, Molly, Mathuros Ruchirawat and Philip Landrigan, 'The Developmental Neurotoxicity of Arsenic: Cognitive and behavioral consequences of early life exposure', Annals of Global Health, vol. 80, no. 4, July 2014, pp. 303–314.
- Song, Yongye, et al., 'A Study of the Fluid Intake, Hydration Status, and Health Effects among Pregnant Women in Their Second Trimester in China: A cross-sectional study', *Nutrients*, vol. 15, no. 7, 2 April 2023, e1739.
- Tran, Nguyen-Toan, et al., 'Cholera in Pregnancy: A systematic review and meta-analysis of fetal, neonatal, and maternal mortality', PLOS One, vol. 10, no. 7, 15 July 2015, e0132920; El Hayek, Pamala, et al., 'Cholera Infection Risks and Cholera Vaccine Safety in Pregnancy', Infectious Diseases in Obstetrics and Gynecology, 22 May 2023, e4563797.
- Bonell, Ana, et al., 'Effect of Heat Stress in the First 1000 Days of Life on Fetal and Infant Growth: A secondary analysis of the ENID randomised controlled trial', Lancet Planetary Health, vol. 8, no. 10, October 2024, pp. e734–e743.

- Augusto, Ana Lucia Pires, et al., 'Household Food Insecurity Associated with Gestacional and Neonatal Outcomes: A systematic review', BMC Pregnancy and Childbirth, vol. 20, no. 229, 17 April 2020; Carmichael, Suzan L., et al., 'Maternal Food Insecurity is Associated with Increased Risk of Certain Birth Defects', Journal of Nutrition, vol. 137, no. 9, September 2007, pp. 2087–2092; Richterman, Aaron M., et al., 'Food Insecurity as a Risk Factor for Preterm Birth: A prospective facility-based cohort study in rural Haiti', BMJ Global Health, vol. 5, no. 7, July 2020, e002341.
- 19 Augusto et al., 'Household Food Insecurity'.
- 20 Du, Rebecca Y., et al., 'Systematic Review of Clinician Awareness of Mycotoxin Impact in Neural Tube Defects and Best Practices for Pediatric Neurosurgeons: Implications for public health and policy', Child's Nervous System, vol. 35, no. 4, April 2019, pp. 637–644; American Academy of Pediatrics Council on Environmental Health, 'Birth Defects and Other Adverse Developmental Outcomes', in Pediatric Environmental Health, 4th ed., edited by Ruth A. Etzel and Sophie J. Balk, American Academy of Pediatrics, Itasca, Ill., 2019, p. 862.
- 21 LoMauro, Antonella, and Andrea Aliverti, 'Respiratory Physiology of Pregnancy: Physiology masterclass', *Breathe*, vol. 11, no. 4, December 2015, pp. 297–301.
- Saenen, Nelly D., et al., 'Air Pollution-Induced Placental Alterations: An interplay of oxidative stress, epigenetics, and the aging phenotype?', Clinical Epigenetics, vol. 11, no. 124, 17 September 2019; Johnson, Natalie M., et al., 'Air Pollution and Children's Health: A review of adverse effects associated with prenatal exposure from fine to ultrafine particulate matter', Environmental Health and Preventative Medicine, vol. 26, no. 72, 12 July 2021.
- 23 Nyadanu, Sylvester D., et al., 'Prenatal Exposure to Ambient Air Pollution and Adverse Birth Outcomes: An umbrella review of 36 systematic reviews and meta-analyses', *Environmental Pollution*, vol. 306, 1 August 2022, e119465.
- 24 Ghosh, Rakesh, et al., 'Ambient and Household PM2.5 Pollution and Adverse Perinatal Outcomes: A meta-regression and analysis of attributable global burden for 204 countries and territories', PLOS Medicine, vol. 18, no. 9, 28 September 2021, e1003718.
- 25 Nyadanu et al., 'Prenatal Exposure to Ambient Air Pollution'.
- 26 Zhang, Huanhuan, et al., 'Ambient Air Pollution and Stillbirth: An updated systematic review and meta-analysis of epidemiological studies', *Environmental Pollution*, vol. 278, 1 June 2021, e116752.
- 27 Lin, Li-Zi, et al., The Epidemiological Evidence Linking Exposure to Ambient Particulate Matter with Neurodevelopmental Disorders: A systematic review and meta-analysis', *Environmental Research*, vol. 209, June 2022, e112876.
- 28 Hazlehurst, Marnie F., et al., 'Maternal Exposure to PM(2.5) During Pregnancy and Asthma Risk in Early Childhood: Consideration of phases of fetal lung development', *Environmental Epidemiology*, vol. 5, no. 2, 2 March 2021, e130.
- Perera, Frederica, and Kari Nadeau, 'Climate Change, Fossil-Fuel Pollution, and Children's Health', New England Journal of Medicine, vol. 386, no. 24, 15 June 2022, pp. 2303–2314.
- 30 United States Centers for Disease Control and Prevention, 'About Secondhand Smoke', 15 May 2024, <www.cdc.gov/tobacco/secondhand-smoke/index.html>, accessed 28 November 2024.
- 31 Zhou, Sherry, et al., 'Physical, Behavioral, and Cognitive Effects of Prenatal Tobacco and Postnatal Secondhand Smoke Exposure', Current Problems in Pediatric and Adolescent Health Care, vol. 44, no. 8, September 2014, pp. 219–241.

- 32 World Health Organization, Tobacco Control to Improve Child Health and Development: Thematic brief', WHO, Geneva, 2021.
- 33 Maciag, Michelle C., Aroub Yousuf and Marissa Hauptman, 'Impact of Prenatal Exposure to Smoking on Child Health', Clinical Obstetrics and Gynecology, vol. 65, no. 2, June 2022, pp. 388–396.
- 34 Hussain, Tarique, et al., The Role of Oxidative Stress and Antioxidant Balance in Pregnancy', *Mediators of Inflammation*, 27 September 2021, e9962860.
- 35 Gitto, Eloisa, et al., 'Oxidative Stress of the Newborn in the Pre- and Postnatal Period and the Clinical Utility of Melatonin', *Journal of Pineal Research*, vol. 46, no. 2, March 2009, pp. 128–139.
- 36 Mathiesen, Line, et al., 'Fetal Exposure to Environmental Chemicals; Insights from placental perfusion studies', *Placenta*, vol. 106, March 2021, pp. 58–66; Dai, Yifeng, et al., 'Early-Life Exposure to Widespread Environmental Toxicants and Maternal-Fetal Health Risk: A focus on metabolomic biomarkers', *Science of the Total Environment*, vol. 739, 15 October 2020, e139626.
- 37 Gitto et al., 'Oxidative Stress of the Newborn'.
- 38 Trivett, Cara, Zoe J. Lees and Dilys J. Freeman, 'Adipose Tissue Function in Healthy Pregnancy, Gestational Diabetes Mellitus and Pre-eclampsia', European Journal of Clinical Nutrition, vol. 75, no. 12, 15 June 2021, pp. 1745–1756.
- 39 Kapraun, Dustin F., et al., 'A Generic Pharmacokinetic Model for Quantifying Mother-to-Offspring Transfer of Lipophilic Persistent Environmental Chemicals', *Toxicological Sciences*, vol. 189, no. 2, 24 September 2022, pp. 155–174.
- 40 Mustieles, Vicente, and Juan P. Arrebola, 'How Polluted is Your Fat? What the study of adipose tissue can contribute to environmental epidemiology', *Journal of Epidemiology and Community Health*, vol. 74, no. 5, May 2020, pp. 401–407.
- 41 Merrill, Alyssa K., Marisia Sobolewski and Martha Susiarjo, 'Exposure to Endocrine Disrupting Chemicals Impacts Immunological and Metabolic Status of Women During Pregnancy', Molecular and Cellular Endocrinology, vol. 577, 1 November 2023, e.112031.
- 42 Sanghavi, Monika, and John D. Rutherford, 'Cardiovascular Physiology of Pregnancy', *Circulation*, vol. 130, no. 12, 16 September 2014, pp. 1003–1008.
- 43 Aguree, Sixtus, and Alison D. Gernand, 'Plasma Volume Expansion Across Healthy Pregnancy: A systematic review and meta-analysis of longitudinal studies', BMC Pregnancy and Childbirth, vol. 19, no. 508, 2019.
- 44 Camaschella, Clara, 'Iron Deficiency', *Blood*, vol. 133, no. 1, 3 January 2019, pp. 30–39.
- Wyrwoll, Catilin S., 'Rising Stars: The heat is on How does heat exposure cause pregnancy complications?', *Journal of Endocrinology*, vol. 259, no. 1, 25 August 2023, e230030.
- 46 Ibid.
- 47 Lakhoo, Darshnika P., et al., 'A Systematic Review and Meta-analysis of Heat Exposure Impacts on Maternal, Fetal and Neonatal Health', Nature Medicine, 5 November 2024, print publication forthcoming.
- 48 Chan, Monica Y., and Mary Alice Smith, 'Infections in Pregnancy', vol. 5 ch. 16 in *Comprehensive Toxicology*, 3rd ed., edited by Charlene A. McQueen, Elsevier, Amsterdam, 2018, pp. 232–249.
- 49 Ibid.
- 50 Ibid.
- 51 Ibid.
- 52 Rogerson, Steven J., and Holger W. Unger, 'Pregnancy and Malaria: The perfect storm', *Current Opinion in Infectious Diseases*, vol. 35, no. 5, October 2022, pp. 410–416.

- 53 Zorrilla, Carmen D., et al., 'Zika Virus Infection in Pregnancy: Maternal, fetal, and neonatal considerations', Journal of Infectious Diseases, vol. 216, suppl. 10, 15 December 2017, pp. S891–S896; World Health Organization, 'Zika Virus', 8 December 2022, <www.who.int/news-room/fact-sheets/detail/zika-virus>, accessed 5 December 2024.
- 54 Dahiya, Nisha, et al., 'ZIKV: Epidemiology, infection mechanism and current therapeutics', Frontiers in Tropical Diseases, vol. 3, 23 January 2023, 1059283.
- 55 Krauer, Fabienne, et al., 'Zika Virus Infection as a Cause of Congenital Brain Abnormalities and Guillain-Barré Syndrome: Systematic review', PLOS Medicine, vol. 14, no. 1, 3 January 2017, e1002203; Musso, Didier, Albert I. Ko and David Baud, 'Zika Virus Infection: After the pandemic', New England Journal of Medicine, vol. 381, no. 15, 4 October 2019, pp. 1444–1457.
- Marbán-Castro, Elana, et al., 'Zika Virus Infection in Pregnant Women and Their Children: A review', European Journal of Obstetrics & Gynecology and Reproductive Biology, vol. 265, October 2021, pp. 162–168.
- 57 Burton, Graham J., and Abigail L. Fowden, 'The Placenta: A multifaceted, transient organ', *Philosophical Transactions of the Royal Society of London*, vol. 370, no. 1663, 5 March 2015, e20140066.
- 58 Khorami-Sarvestani, Sara, et al., 'Placenta: An old organ with new functions', *Frontiers in Immunology*, vol. 15, 19 April 2024, e1385762.
- 59 Mathiesen et al., 'Fetal Exposure to Environmental Chemicals'.;
 Dai et al., 'Early-Life Exposure to Widespread Environmental Toxicants'.
- 60 Ortiz-Garcia et al., 'Maternal Exposure to Arsenic'.
- 61 Megli, Christina J., and Carolyn B. Coyne, 'Infections at the Maternal–Fetal Interface: An overview of pathogenesis and defence', *Nature Reviews Microbiology*, vol. 20, no. 2, February 2022, pp. 67–82.
- 62 Gingrich, Jeremy, Elvis Ticiani and Alumdena Veiga-Lopez, 'Placenta Disrupted: Endocrine disrupting chemicals and pregnancy', *Trends in Endocrinology & Metabolism*, vol. 31, no. 7, July 2020, pp. 508–524.
- 63 Bloom, Michael, Meghana Varde and Roger Newman, 'Environmental Toxicants and Placental Function', *Best Practice and Research in Clinical Obstetrics and Gynaecology*, vol. 85, part B, December 2022, pp. 105–120.
- 64 Dimitriadis, Evdokia, et al., 'Pre-eclampsia', Nature Reviews Disease Primers, vol. 9, art. 8, 16 February 2023.
- 65 Ibid.
- 66 Mazumder, Hoimonty, et al., 'Maternal Health Outcomes Associated with Ambient Air Pollution: An umbrella review of systematic reviews and meta-analyses', Science of the Total Environment, vol. 914, 1 March 2024, 169792
- 67 Moore, Persaud and Torchia, *The Developing Human*.
- 68 Breton-Larrivée, Mélanie, Elizabeth Elder and Serge McGraw, 'DNA Methylation, Environmental Exposures and Early Embryo Development', Animal Reproduction, vol. 16, no. 3, 23 October 2019, pp. 465–474.
- 69 Isaevska, Elena, et al., 'Exposure to Ambient Air Pollution in the First 1000 Days of Life and Alterations in the DNA Methylome and Telomere Length in Children: A systematic review', Environmental Research, vol. 193, February 2021, e110504.
- 70 Moore, Persaud and Torchia, *The Developing Human*.
- 71 Decrue, Fabienne, et al., 'Increased Impact of Air Pollution on Lung Function in Preterm versus Term Infants: The BILD study', *American Journal of Respiratory and Critical Care Medicine*, vol. 205, no. 1, 1 January 2022, pp. 99–107.
- 72 American College of Obstetricians and Gynecologists, 'How Your Fetus Grows During Pregnancy', January 2024, <www.acog.org/womens-health/faqs/how-your-fetus-grows-during-pregnancy>, accessed 23 August 2024.

- 73 Moore, Persaud and Torchia, *The Developing Human*.
- Di Renzo, Gian C., et al., 'International Federation of Gynecology and Obstetrics Opinion on Reproductive Health Impacts of Exposure to Toxic Environmental Chemicals', International Journal of Gynecology & Obstetrics, vol. 131, no. 3, December 2015, pp. 219-225.
- World Health Organization, 'Household Air Pollution', 16 October 2024, <www.who.int/news-room/fact-sheets/detail/household-air-pollution-</p> and-health>, accessed 5 December 2024.
- World Health Organization, Children and Digital Dumpsites: E-waste exposure and child health, WHO, Geneva, 2021; Perks, Rachel, 'Let's Support Women in Artisanal and Small-Scale Mining', World Bank Blogs, Washington, D.C., 8 March 2024, https://blogs.worldbank.org/en/ energy/lets-support-women-artisanal-and-small-scale-mining>, accessed 12 December 2024; Food and Agriculture Organization of the United Nations and Rotterdam Convention, Addressing Gender Issues in Pesticide Management, FAO, Rome, 2022.
- Gender and Hazardous Substances: Report of the Special Rapporteur on the implications for human rights of the environmentally sound management and disposal of hazardous substances and wastes, Marcos Orellana, A/79/163, United Nations, New York, 16 July 2024; Strategic Approach to International Chemicals Management and the International Pollutants Elimination Network, Women, Chemicals and the SDGs, IPEN, Geneva, 2022.
- McNab, Shanon, and Lynn Freedman, Maternal Newborn Health and the Urban Poor: A global scoping, Columbia University Mailman School of Public Health, New York, 2016.
- Yao, Xiaodie, et al., 'Environmental Pollutants Exposure and Gestational Diabetes Mellitus: Evidence from epidemiological and experimental studies', Chemosphere, vol. 332, August 2023, e138866.
- 80 Mazumder et al., 'Maternal Health Outcomes'.
- Nyadanu, et al., 'Prenatal Exposure to Ambient Air Pollution'; Zhang et al., 'Ambient Air Pollution and Stillbirth'.; Anderson, Mark E., Daniel C. Johnson and Holly A. Batal, 'Sudden Infant Death Syndrome and Prenatal Maternal Smoking: Rising attributed risk in the Back to Sleep era', BMC Medicine, vol. 3, no. 1, art. 4, 11 January 2005.
- Etzel, Ruth A., 'Is the Environment Associated With Preterm Birth?', JAMA Network Open, vol. 3, no. 4, 2020, e202239.
- 83
- World Health Organization, 'Preterm Birth', 10 May 2023, <www.who.int/ news-room/fact-sheets/detail/preterm-birth>, accessed 26 May 2023.
- World Health Organization, 'Low Birth Weight', https://www.who.int/ data/nutrition/nlis/info/low-birth-weight>, accessed 5 December 2024.
- Smith, C. J., et al., 'The Impact of Birth Weight on Cardiovascular Disease Risk in the Women's Health Initiative', Nutrition, Metabolism & Cardiovascu-Diseases, vol. 26, no. 3, March 2016, pp. 239-245.
 - Triche, Elizabeth W., and Nazli Hossain, 'Environmental Factors
- Implicated in the Causation of Adverse Pregnancy Outcome', Seminars in Perinatology, vol. 31, no. 4, August 2007, pp. 240-242.
- 'Birth Defects and Other Adverse Developmental Outcomes', p. 865.
- Núñez-Sánchez, María Á., et al., 'Inherited Epigenetic Hallmarks of Childhood Obesity Derived from Prenatal Exposure to Obesogens', International Journal of Environmental Research and Public Health, vol. 20, no. 6, 7 March 2023, e4711; Hazlehurst et al., 'Maternal Exposure to PM(2.5) During Pregnancy'; Brumberg, Heather L., et al., 'Ambient Air Pollution: Health hazards to children', Pediatrics, vol. 147, no. 6, June 2021, e2021051484; Tingskov Pedersen, Casper E., et al.,

- 'Prenatal Exposure to Ambient Air Pollution is Associated with Early Life Immune Perturbations', Journal of Allergy and Clinical Immunology, vol. 151, no. 1, January 2023, pp. 212-221; Kim, Juyong B., et al., 'Cumulative Lifetime Burden of Cardiovascular Disease From Early Exposure to Air Pollution', Journal of the American Heart Association, vol. 9, no. 6, 17 March 2020, e014944; Deolmi, Michela, et al., 'Early Origins of Chronic Obstructive Pulmonary Disease: Prenatal and early life risk factors', International Journal of Environmental Research and Public Health, vol. 20, no. 3, February 2023, e2294.
- Hertz-Picciotto, Irva, et al., 'Organophosphate Exposures During Pregnancy and Child Neurodevelopment: Recommendations for essential policy reforms', PLOS Medicine, vol. 15, no. 10, October 2018, e1002671; Van Maele-Fabry, Geneviève, Laurence Gamet-Payrastre and Dominique Lison, 'Household Exposure to Pesticides and Risk of Leukemia in Children and Adolescents: Updated systematic review and meta-analysis', International Journal of Hygiene and Environmental Health, vol. 222, no. 1, January 2019, pp. 49–67; Khan, Aiza, Joseph Feulefack and Consolato M. Sergi, 'Pre-conceptional and Prenatal Exposure to Pesticides and Pediatric Neuroblastoma: A meta-analysis of nine studies', Environmental Toxicology and Pharmacology, vol. 90, February 2022, 103790.
- World Health Organization, 'Preventing Disease Through Health Environments: Exposure to mercury - A major public health concern', 2nd ed., WHO, Geneva, 8 April 2021.
- Center on the Developing Child, 'InBrief: Connecting the brain to the rest of the body', Harvard University, Cambridge, Mass., 2020.
- Etzel, Ruth A., and Philip J. Landrigan, 'Children's Exquisite Vulnerability to Environmental Exposures', ch. 2 in Textbook of Children's Environmental Health, 2nd ed., edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 23-37.
- Agency for Toxic Substances and Disease Registry, 'The Child as Susceptible Host: A Developmental Approach to Pediatric Environmental Medicine', part of the Principles of Pediatric Environmental Health Case Studies in Environmental Medicine, United States Centers for Disease Control and Prevention, Atlanta, Ga., 2012.
- Landrigan, Philip J., et al., 'The Minderoo-Monaco Commission on Plastics and Human Health', Annals of Global Health, vol. 89, no. 1, art. 23, 2023.
- World Health Organization, 'Drinking-Water', 13 September 2023, <www.who.int/news-room/fact-sheets/detail/drinking-water>, accessed 10 December 2024; World Health Organization, 'Food Safety', 4 October 2024, <www.who.int/news-room/fact-sheets/detail/food-safety>, accessed 10 December 2024.
- United States National Research Council, Pesticides in Diets of Infants and Children, National Academies Press, Washington, D.C., 1993.
- La Charite, Jamie, 'Nutrition and Growth', ch. 21 in The Harriet Lane Handbook, 22nd ed., edited by Keith Kleinman, Lauren McDaniel and Matthew Molloy, Elsevier, Philadelphia, Pa., 2021.
- 99 Pesticides in Diets.
- World Health Organization, 'Malnutrition', 1 March 2024, <www.who.int/ news-room/fact-sheets/detail/malnutrition>, accessed 9 December 2024.
- 101 Ibid.
- 102 Caulfield, Laura E., et al., 'Undernutrition as an Underlying Cause of Child Deaths Associated with Diarrhea, Pneumonia, Malaria, and Measles', American Journal of Clinical Nutrition, vol. 80, no. 1, July 2004, pp. 193-198.
- 103 Deen, Jacqueline L., et al., 'The High Burden of Cholera in Children: Comparison of incidence from endemic areas in Asia and Africa', PLOS Neglected Tropical Diseases, vol. 2, no. 2, art. e173, February 2008.
- 104 Sinharoy, Sheela S., Thomas Clasen and Reynaldo Martorell, 'Air Pollution and Stunting: A missing link?', Lancet Global Health, vol. 8, no. 4, April 2020, pp. e472-e475.
- 105 Mulat, Elias, Dessalegn Tamiru and Kalkidan Hassen Abate, 'Impact of Indoor Air Pollution on the Linear Growth of Children in Jimma, Ethiopia', BMC Public Health, vol. 24, art. 488, 2024.

- 106 United States Environmental Protection Agency, Exposure Factors Handbook: 2011 edition, National Center for Environmental Assessment, Washington, D.C., 2011.
- 107 World Health Organization, Environmental Health Criteria 59: Principles for evaluating risks from chemicals during infancy and early childhood - The need for a special approach, WHO, Geneva, 1986.
- 108 Johns Hopkins Hospital, The Harriet Lane Handbook, edited by Keith Kleinman, Lauren McDaniel and Matthew Molloy, Elsevier, Phildelphia, Pa., 2021.
- 109 Agency for Toxic Substances and Disease Registry, Toxicological Profile for Lead, United States Department of Health and Human Services, Atlanta, Ga., August 2020, p. 281.
- 110 'The Child as Susceptible Host'.
- 111 Stamatas, Georgios N., et al., 'Skin Maturation from Birth to 10 Years of Age: Structure, function, composition and microbiome', *Experimental Dermatology*, vol. 32, no. 9, September 2023, pp. 1420–1429.
- 112 Ibid.
- 113 Ibid.
- 114 Huen, Karen, et al., 'Developmental Changes in PON1 Enzyme Activity in Young Children and Effects of PON1 Polymorphisms', Environmental Health Perspectives, vol. 117, no. 10, October 2009, pp. 1632–1638.
- 115 Agency for Toxic Substances and Disease Registry, Toxicological Profile for Nitrate and Nitrite, United States Department of Health and Human Services, Washington, D.C., July 2017; American Academy of Pediatrics Council on Environmental Health, 'Nitrates and Nitrites in Water', ch. 34 in Pediatric Environmental Health, 4th ed., edited by Ruth A. Etzel and Sophie J. Balk, American Academy of Pediatrics, Itasca, Ill., 2019, pp. 601–610.
- 116 European Food Safety Authority Scientific Committee, et al., 'Guidance on the Risk Assessment of Substances Present in Food Intended for Infants below 16 Weeks of Age', EFSA Journal, vol. 15, no. 5, art. e04849, May 2017.
- 117 Ziou, Miriam, et al., 'Outdoor Particulate Matter Exposure and Upper Respiratory Tract Infections in Children and Adolescents: A systematic review and meta-analysis', Environmental Research, vol. 210, art. 112969, July 2022.
- 118 Rajan, Sujatha, and Sunil K. Sood, 'Bacterial Tracheitis', Medscape, 1 November 2023, https://emedicine.medscape.com/article/961647-overview, accessed 10 December 2024.
- 119 National Institute on Deafness and Other Communication Disorders, 'Ear Infections in Children', United States National Institutes of Health, 16 March 2022, <www.nidcd.nih.gov/health/ear-infections-children#:~: text=through%20the%20mouth.-,Why%20are%20children%20more%20 likely%20than%20adults%20to%20get%20ear,tubes%20and%20the%20 middle%20ear.>, accessed 10 December 2024.
- 120 Bowatte, Gayan, et al., 'Air Pollution and Otitis Media in Children: A systematic review of literature', *International Journal of Environmental Research and Public Health*, vol. 15, no. 2, art. 257, February 2018.
- 121 Nyangasa, Salama, et al., 'The Rate and Pattern of Fetal Hemoglobin Decline Adjusted to Sickle Cell Status of Newborns in Dar es Salaam, Tanzania: A prospective cohort study', American Journal of Hematology, vol. 98, no. 9, September 2023, pp. e241–e243.
- 122 'The Child as Susceptible Host'.
- 123 World Health Organization, WHO Guidelines for Indoor Air Quality: Selected pollutants, WHO, Copenhagen, 2010.
- 124 Ibid
- 125 Xu, Zhiwei, et al., 'Impact of Ambient Temperature on Children's Health: A systematic review', Environmental Research, vol. 117, August 2012, pp. 120–131.
- 126 Mangus, Courtney W., and Therese L. Canares, 'Heat-Related Illness in Children in an Era of Extreme Temperatures', *Pediatrics in Review*, vol. 40, no. 3, March 2019, pp. 97–107.
- 127 Ibid.

- 128 Yu, Jack C., et al., 'Innate Immunity of Neonates and Infants', *Frontiers in Immunology*, vol. 9, art. 1759, 30 July 2018.
- 129 Simon, A. Katharina, Georg A. Hollander and Andrew McMichael, 'Evolution of the Immune System in Humans from Infancy to Old Age', Proceedings of the Royal Society B: Biological sciences, vol. 282, no. 1821, art. 20143085, 22 December 2015.
- 130 National Cancer Institute, 'Adaptive immunity', United States National Institutes of Health, <www.cancer.gov/publications/dictionaries/cancer-terms/def/adaptive-immunity>, accessed 10 December 2024.
- 131 Pieren, Daan K. J., Mardi C. Boer and Jelle de Wit, 'The Adaptive Immune System in Early Life: The shift makes it count', Frontiers in Immunology, vol. 13, art. 1031924, 17 November 2022.
- 132 Yu et al., 'Innate Immunity'.
- 133 Moxon, Christopher A., et al., 'New Insights into Malaria Pathogenesis', Annual Review of Pathology: Mechanisms of Disease, vol. 15, 2020, pp. 315–343; World Health Organization, 'Malaria', 4 December 2023, www.who.int/news-room/fact-sheets/detail/malaria, accessed 10 December 2024; Jain, Amita, and Umesh C. Chaturvedi, 'Dengue in Infants: An overview', FEMS Immunology & Medical Microbiology, vol. 59, no. 2, July 2010, pp. 119–130.
- 134 Derrien, Muriel, Anne-Sophie Alvarez and Willem M. de Vos, 'The Gut Microbiota in the First Decade of Life', *Trends in Microbiology*, vol. 27, no. 12, December 2019, pp. 997–1010.
- 135 Ahn, Jiyoung, and Richard B. Hayes, 'Environmental Influences on the Human Microbiome and Implications for Noncommunicable Disease', Annual Review of Public Health, vol. 42, 2021, pp. 277–292.
- 136 Gama, Jessica, Bianca Neves and Antonio Pereira, 'Chronic Effects of Dietary Pesticides on the Gut Microbiome and Neurodevelopment', Frontiers in Microbiology, vol. 13, art. 931440, 30 June 2022.
- 137 Ahn and Hayes, 'Environmental Influences on the Human Microbiome'; ibid.
- 138 National Cancer Institute, 'Blood-Brain Barrier', United States National Institutes of Health, www.cancer.gov/publications/dictionaries/cancer-terms/def/blood-brain-barrier>, accessed 10 December 2024.
- 139 'Guidance on the Risk Assessment of Substances Present in Food'.
- 140 Schmitt, Georg, et al., 'The Great Barrier Belief: The blood-brain barrier and considerations for juvenile toxicity studies', *Reproductive Toxicology*, vol. 72, September 2017, pp. 129–135.
- 141 Tobwala, Shakila, et al., 'Effects of Lead and Cadmium on Brain Endothelial Cell Survival, Monolayer Permeability, and Crucial Oxidative Stress Markers in an In Vitro Model of the Blood-Brain Barrier', *Toxics*, vol. 2, no. 2, June 2014, pp. 258–275.
- 142 Kwong, Laura H., et al., 'Soil Ingestion among Young Children in Rural Bangladesh', *Journal of Exposure Science & Environmental Epidemiology*, vol. 31, no. 1, January 2021, pp. 82–93.
- 143 Etzel and Landrigan, 'Children's Exquisite Vulnerability'.
- 144 UNICEF Parenting, 'Your Baby's Developmental Milestones', UNICEF, https://www.unicef.org/parenting/child-development/your-babys-developmental-milestones>, accessed 10 December 2024.
- 145 Aurisano, Nicolò, et al., 'Estimating Mouthing Exposure to Chemicals in Children's Products', Journal of Exposure Science & Environmental Epidemiology, vol. 32, no. 1, January 2022, pp. 94–102; Symeonides, Christos, et al., 'An Umbrella Review of Meta-analyses Evaluating Associations between Human Health and Exposure to Major Classes of Plastic-Associated Chemicals', Annals of Global Health, vol. 90, no. 1, art. 52, 19 August 2024.
- 146 Madrigal, Jessica M., et al., 'Contributions of Nearby Agricultural Insecticide Applications to Indoor Residential Exposures', Environment International, vol. 171, art. 107657, January 2023.
- 147 Hyland, Carly, and Ouahiba Laribi, 'Review of Take-Home Pesticide Exposure Pathway in Children Living in Agricultural Areas', Environmental Research, vol. 156, July 2017, pp. 559–570.

- 148 United Nations Children's Fund and Pure Earth, The Toxic Truth: Children's exposure to lead pollution undermines a generation of future potential, UNICEF, New York, July 2020; Lanphear, Bruce P., et al., 'Environmental Lead Exposure during Early Childhood', Journal of Pediatrics, vol. 140 no. 1, January 2002, pp. 40–47; Yabe, John, et al., 'Current Trends of Blood Lead Levels, Distribution Patterns and Exposure Variations among Household Members in Kabwe, Zambia', Chemosphere, vol. 243, art. 125412, March 2020.
- 149 Adar, Sara D., et al., 'Adopting Clean Fuels and Technologies on School Buses: Pollution and health impacts in children', American Journal of Respiratory and Critical Care Medicine, vol. 191, no. 12, 15 June 2015, pp. 1413–1421; Pandya, Robert J., et al., 'Diesel Exhaust and Asthma: Hypotheses and molecular mechanisms of action', Environmental Health Perspectives, vol. 110, suppl. 1, February 2002, pp. 103–112; Behrentz, Eduardo, et al., 'Relative Importance of School Bus-Related Microenvironments to Children's Pollutant Exposure', Journal of the Air & Waste Management Association, vol. 55, no. 10, 2005, pp. 1418–1430.
- 150 'The Child as Susceptible Host'.
- 151 United States Environmental Protection Agency, 'What is Radon?', EPA, 20 November 2024, www.epa.gov/radon/what-radon, accessed 10 December 2024.
- 152 UNICEF Parenting, 'Baby Sleep', UNICEF, <www.unicef.org/parenting/ child-care/baby-sleep>, accessed 10 December 2024.
- 153 Glassy, Danette, and Pooja Tandon, 'Playing Outside: Why it's important for kids', American Academy of Pediatrics, 13 May 2024, <www.healthy-children.org/English/family-life/power-of-play/Pages/playing-outside-why-its-important-for-kids.aspx>, accessed 10 December 2024.
- 154 Serreau, Raphaël, Yasmine Terbeche and Virginie Rigourd, 'Pollutants in Breast Milk: A scoping review of the most recent data in 2024', Healthcare, vol. 12, no. 6, art. 680, March 2024.
- 155 World Health Organization, Infant and Young Child Feeding', 20 December 2023, www.who.int/news-room/fact-sheets/detail/infant-and-young-child-feeding, accessed 10 December 2024.
- 156 Fan, Winnie, et al., 'The Intrauterine Environment and Early Infancy', ch. 14 in *Textbook of Children's Environmental Health*, 2nd ed., edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 175–185.
- 157 American Academy of Pediatrics Council on Environmental Health, 'Human Milk', ch. 15 in *Pediatric Environmental Health*, 4th ed., edited by Ruth A. Etzel and Sophie J. Balk, American Academy of Pediatrics, Itasca, Ill., 2019, pp. 237–239.
- 158 van den Berg, Martin, et al., 'WHO/UNEP Global Surveys of PCDDs, PCDFs, PCBs and DDTs in Human Milk and Benefit-Risk Evaluation of Breastfeeding', Archives of Toxicology, vol. 91, no. 1, January 2017, pp. 83–96.
- 159 World Health Organization, WHO Guideline for the Clinical Management of Exposure to Lead, WHO, Geneva, 2021.
- 160 Lignell, Sanna, et al., 'Environmental Organic Pollutants in Human Milk before and after Weight Loss', Chemosphere, vol. 159, September 2016, pp. 96–102.
- 161 'Human Milk'.
- 162 Etzel and Landrigan, 'Children's Exquisite Vulnerability'.
- 163 United States Food and Drug Administration, 'Supporting Document for Action Level for Inorganic Arsenic in Rice Cereals for Infants', FDA, Silver Spring, Md., August 2020.
- 164 World Health Organization and United Nations Children's Fund, World Report on Child Injury Prevention, WHO, Geneva, 2008.
- 165 World Health Organization, Children and Chemicals: Training for health care providers, 3rd ed., WHO, Geneva, 2023.
- 166 World Report on Child Injury Prevention.
- 167 Ibid.
- 168 United States Centers for Disease Control and Prevention, 'Health Hazards Associated with Laundry Detergent Pods United States, May–June 2012', Morbidity and Mortality Weekly Report, vol. 61, no. 41, 19 October 2012, pp. 825–829.

- 169 Barouki, Robert, et al., 'Developmental Origins of Non-communicable Disease: Implications for research and public health', Environmental Health, vol. 11, art. 42, 2012.
- 170 Carpenter, David O., and Sheila Bushkin-Bedient, 'Exposure to Chemicals and Radiation During Childhood and Risk for Cancer Later in Life', Journal of Adolescent Health, vol. 52, no. 5, suppl., May 2013, pp. S21–S29.
- 171 Oliveria, S. A., et al., 'Sun Exposure and Risk of Melanoma', *Archives of Disease in Childhood*, vol. 91, no. 2, February 2006, pp. 131–138.
- 172 Straif, Kurt, 'Environmental Carcinogens and Childhood Cancer', ch. 42 in *Textbook of Children's Environmental Health*, 2nd ed., edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 554–572.
- 173 Anthony, Ross, et al., 'Adaptation for Life after Birth: A review of neonatal physiology', *Anaesthesia & Intensive Care Medicine*, vol. 21, no. 2, pp. 71–79, February 2020.
- 174 Ibid
- 175 European Food Safety Authority, et al., 'The 2021 European Union Report on Pesticide Residues in Food', EFSA Journal, vol. 21, no. 4, art. e07939, April 2023.
- 176 Hooven, Thomas A., and Richard A. Polin, 'Pneumonia', Seminars in Fetal and Neonatal Medicine, vol. 22, no. 4, August 2017, pp. 206–213.
- 177 Lunze, K., and D. H. Hamer, 'Thermal Protection of the Newborn in Resource-Limited Environments', *Journal of Perinatology*, vol. 32, no. 5, May 2012, pp. 317–324.
- 178 Oza, Shefali, Simon N. Cousens and Joy E. Lawn, 'Estimation of Daily Risk of Neonatal Death, Including the Day of Birth, in 186 Countries in 2013: A vital-registration and modelling-based study', *Lancet Global Health*, vol. 2, no. 11, November 2014, pp. e635–e644.
- 179 World Health Organization, Survive and Thrive: Transforming care for every small and sick newborn, WHO, Geneva, 2019.
- 180 World Health Organization, 'Newborn Mortality', 14 March 2024, www.who.int/news-room/fact-sheets/detail/newborn-mortality, accessed 9 December 2024.
- 181 Sakali, Anastasia-Konstantina, et al., 'Environmental Factors Affecting Pregnancy Outcomes', Endocrine, vol. 80, no. 3, 2023, pp. 459–469.
- 182 Institute of Medicine (US) Committee on Understanding Premature Birth and Assuring Healthy Outcomes, 'Mortality and Acute Complications in Preterm Infants', ch. 10 in *Preterm Birth: Causes, consequences, and prevention*, edited by Richard E. Behrman and Adrienne Stith Butler, National Academies Press, Washington, D.C., 2007.
- 183 Ibid.
- 184 Antony et al., 'Adaptation for Life after Birth'.
- 185 Maternal and Newborn Health/Safe Motherhood Unit and the Division of Reproductive Health, Thermal Protection of the Newborn: A practical guide, WHO. Geneva. 1997.
- 186 Ibid.
- 187 Downey, L. Corbin, P. Brian Smith and Daniel K. Benjamin Jr., 'Risk Factors and Prevention of Late-Onset Sepsis in Premature Infants', Early Human Development, vol. 86, no. 1, suppl., July 2010, pp. 7–12; Lin, Li-Zi, et al., 'Ambient Air Pollution and Infant Health: A narrative review', eBioMedicine, vol. 93, art. 104609, July 2023.
- 188 World Health Organization, Human Resource Strategies to Improve Newborn Care in Health Facilities in Low- and Middle-Income Countries, WHO, Geneva, 2020.
- 189 Ibid.
- 190 National Institute of Neurological Disorders and Stroke, 'Brain Basics: The life and death of a neuron', United States National Institutes of Health, 29 November 2024, <www.ninds.nih.gov/health-information/ public-education/brain-basics/brain-basics-life-and-death-neuron>, accessed 10 December 2024.
- 191 Paredes, Mercedes F., et al., 'Extensive Migration of Young Neurons into the Infant Human Frontal Lobe', *Science*, vol. 354, no. 6308, art. aaf7073, 7 October 2016.

- 192 Center on the Developing Child, 'InBrief: The science of early childhood development', Harvard University, Cambridge, Mass., 2007.
- 193 Tierney, Adrienne L., and Charles A. Nelson III, 'Brain Development and the Role of Experience in the Early Years', *Zero to Three*, vol. 30, no. 2, November 2009, pp. 9–13.
- 194 National Scientific Council on the Developing Child, Early Exposure to Toxic Substances Damages Brain Architecture: Working Paper No. 4, Center for the Developing Child at Harvard University, Cambridge, Mass., 2006; Virgolini, Miriam Beatriz, and Michael Aschner, Molecular mechanisms of lead neurotoxicity, in Advances in Neurotoxicology, M. Aschner and L.G. Costa, Editors. 2021, Academic Press. p. 159-213.
- 195 Council On Environmental Health, et al., 'Prevention of Childhood Lead Toxicity', *Pediatrics*, vol. 138, no. 1, art. e20161493, July 2016.
- 196 Landrigan et al., 'Minderoo-Monaco Commission'; Brumberg, Heather L., et al., 'Ambient Air Pollution: Health hazards to children', *Pediatrics*, vol. 147, no. 6, art. e2021051484, June 2021.
- 197 Liu, Jianghong, et al., 'Air Pollution Exposure and Adverse Sleep Health across the Life Course: A systematic review', *Environmental Pollution*, vol. 262, art. 114263, July 2020; Pittner, Katharina, et al., 'Sleep across the First Year of Life is Prospectively Associated with Brain Volume in 12-Months Old Infants', Neurobiology of Sleep and Circadian Rhythms, vol. 14, art. 100091, May 2023.
- 198 National Scientific Council on the Developing Child, Early Experiences Can Alter Gene Expression and Affect Long-Term Development: Working Paper No. 10, Center on the Developing Child at Harvard University, Cambridge, Mass., May 2010.
- 199 Calogero, C., and Peter D. Sly, 'Developmental Physiology: Lung function during growth and development from birth to old age', ch. 1 in Paediatric Lung Function, edited by U. Frey and P. J. F. M. Merkus, European Respiratory Society, Lausanne, March 2010, pp. 1–15.
- 200 Ibid.
- 201 Gauderman, W. James, et al., 'The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age', New England Journal of Medicine, vol. 351, no. 11, 9 September 2004, pp. 1057–1067.
- 202 Vilcins, Dwan, and Peter D. Sly, 'Asthma, Allergy, and the Environment', ch. 48 in Textbook of Children's Environmental Health, 2nd ed., edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 650–660.
- 203 Xu-Chen, Xilei, et al., 'The Airway Epithelium during Infancy and Childhood: A complex multicellular immune barrier – Basic review for clinicians', Paediatric Respiratory Reviews, vol. 38, June 2021, pp. 9–15.
- 204 Nhung, Nguyen Thi Trang, et al., 'Short-Term Association between Ambient Air Pollution and Pneumonia in Children: A systematic review and meta-analysis of time-series and case-crossover studies', Environmental Pollution, vol. 230, November 2017, pp. 1000–1008; World Health Organization, Ambient Air Pollution: Training for health care providers, 3rd ed., WHO, Geneva, 2023.
- 205 United States Centers for Disease Control and Prevention, 'About Secondhand Smoke', CDC, 15 May 2024, <www.cdc.gov/tobacco/secondhand-smoke/index.html>, accessed 10 December 2024.
- 206 Ibid.
- 207 Deolmi, Michela, et al., 'Early Origins of Chronic Obstructive Pulmonary Disease: Prenatal and early life risk factors', International Journal of Environmental Research and Public Health, vol. 20, no. 3, art. 2294, February 2023.
- 208 Weidemann, Darcy K., Virginia M. Weaver and Jeffrey J. Fadrowski, 'Toxic Environmental Exposures and Kidney Health in Children', Pediatric Nephrology, vol. 31, no. 11, November 2016, pp. 2043–2054.
- 209 Sanders, Alison P., et al., 'Prenatal and Early Childhood Critical Windows for the Association of Nephrotoxic Metal and Metalloid Mixtures with Kidney Function', Environment International, vol. 166, art. 107361, August 2022.
- 210 Weidemann, Darcy K., Jeffrey J. Fadrowski and Virginia M. Weaver, The Environment and Kidney Disease in Children', ch. 53 in *Textbook of Children's Environmental Health*, 2nd ed., edited by Ruth A. Etzel and Philip J. Landrigan, Oxford University Press, New York, 2024, pp. 719–730.

- 211 European Food Safety Authority, Luis Carrasco Cabrera and Paula Medina Pastor, 'The 2019 European Union Report on Pesticide Residues in Food', EFSA Journal, vol. 19, no. 4, art. e06491, April 2021.
- 212 Vilcins and Sly, 'Asthma, Allergy, and the Environment'.
- 213 Agency for Toxic Substances and Disease Registry, 'How Can Parents' Preconception Exposures and In Utero Exposures Affect a Developing Child?', United States Centers for Disease Control and Prevention, 25 May 2023, https://archive.cdc.gov/www_atsdr_cdc_gov/csem/pediatric-environmental-health/preconception.html, accessed 10 December 2024.
- 214 Yao, Xiaoxi, et al., 'Fertility Loss: Negative effects of environmental toxicants on oogenesis', Frontiers in Physiology, vol. 14, art. 1219045, 2023.
- 215 United Nations Environment Programme, 'PCBs: A forgotten legacy?', UNEP, <www.unep.org/topics/chemicals-and-pollution-action/pollution-and-health/persistent-organic-pollutants-pops/pcbs#:~:tex-t=Although%20no%20longer%20allowed%20to,environment%20and%20to%20human%20health.>, accessed 10 December 2024.
- 216 Lucaccioni, Laura, et al., 'Minipuberty: Looking back to understand moving forward', Frontiers in Pediatrics, vol. 8, art. 612235, 2020.
- 217 Wiemels, Joseph, 'Perspectives on the Causes of Childhood Leukemia', Chemico-Biological Interactions, vol. 196, no. 3, 5 April 2012, pp. 59–67.
- 218 Viner RM, Allen NB, and P. GC, Puberty, Developmental Processes, and Health Interventions., in Child and Adolescent Health and Development. 3rd edition., Bundy DAP, et al., Editors. 2017, The International Bank for Reconstruction and Development / The World Bank: Washington, DC.
- 219 Rohayem, Julia, et al., 'Mini-Puberty, Physiological and Disordered: Consequences, and potential for therapeutic replacement', Endocrine Reviews, vol. 45, no. 4, August 2024, pp. 460–492.
- 220 Abreu, Ana Paula, and Ursula B. Kaiser, 'Pubertal Development and Regulation', Lancet Diabetes & Endocrinology, vol. 4, no. 3, March 2016, pp. 254–264.
- 221 Yang, Qianfan, et al., 'Sex Hormone Influence on Female-Biased Autoimmune Diseases Hints at Puberty as an Important Factor in Pathogenesis', Frontiers in Pediatrics, vol. 11, art. 1051624, 2023.
- 222 Viner, Allen and Patton, 'Puberty, Developmental Processes, and Health Interventions'.
- 223 Norris, Shane A., et al., 'Nutrition in Adolescent Growth and Development', *Lancet*, vol. 399, no. 10320, 8–14 January 2022, pp. 172–184.
- 224 Ibid.
- 225 Ibid.
- 226 Lopez-Rodriguez, David, et al., 'Endocrine-Disrupting Chemicals and Their Effects on Puberty', Best Practice & Research Clinical Endocrinology & Metabolism, vol. 35, no. 5, art. 101579, September 2021.
- 227 Anderson, Greg M., et al., 'Metabolic Control of Puberty: 60 years in the footsteps of Kennedy and Mitra's seminal work', Nature Reviews Endocrinology, vol. 20, no. 2, February 2024, pp. 111–123.
- 227 Lopez-Rodriguez et al., 'Endocrine-Disrupting Chemicals and Their Effects on Puberty'; Uldbjerg, C. S., et al., 'Prenatal and Postnatal Exposures to Endocrine Disrupting Chemicals and Timing of Pubertal Onset in Girls and Boys: A systematic review and meta-analysis', Human Reproduction Update, vol. 28, no. 5, September–October 2022, pp. 687–716.
- 229 Symeonides, Christos, et al., 'An Umbrella Review of Meta-analyses Evaluating Associations between Human Health and Exposure to Major Classes of Plastic-Associated Chemicals', Annals of Global Health, vol. 90, no. 1, art. 52, 2024.
- 230 Anderson et al., 'Metabolic Control of Puberty'.
- 231 Patton, George C., et al., 'Our Future: A Lancet commission on adolescent health and wellbeing', Lancet, vol. 387, no. 10036, 11 June 2016, pp. 2423–2478.
- 232 Kendricks, Dalisa R., Steven R. Boomhower and M. Christopher Newland, 'Adolescence as a Sensitive Period for Neurotoxicity: Lifespan developmental effects of methylmercury', *Pharmacology Biochemistry* and Behavior, vol. 217, art. 173389, June 2022.
- 233 Spear, Linda Patia, 'Adolescent Neurodevelopment', Journal of Adolescent Health, vol. 52, no. 2, suppl. 2, February 2013, pp. S7–S13.
- 234 Kendricks, Boomhower and Newland, 'Adolescence as a Sensitive Period for Neurotoxicity'.
- 235 Ibid.

- 236 Herting, Megan M., Katherine L. Bottenhorn and Devyn L. Cotter, 'Outdoor Air Pollution and Brain Development in Childhood and Adolescence', Trends in Neurosciences, vol. 47, no. 8, August 2024, pp. 593–607.
- 237 Ibid
- 238 Kendricks, Boomhower and Newland, 'Adolescence as a Sensitive Period for Neurotoxicity'.
- 239 Kwan, Leslie Y., et al., 'This is Your Teen Brain on Drugs: In search of biological factors unique to dependence toxicity in adolescence', *Neurotoxicology and Teratology*, vol. 81, art. 106916, September–October 2020.
- 240 UNICEF Innocenti Global Office of Research and Foresight, The Adolescent Brain: A second window of opportunity – A compendium, United Nations Children's Fund, Florence, Italy, 2017.
- 241 Viner, Allen and Patton, 'Puberty, Developmental Processes, and Health Interventions'.
- 242 National Academies of Sciences, Engineering, and Medicine, et al., 'Normative Adolescent Development', ch. 2 in Promoting Positive Adolescent Health Behaviors and Outcomes: Thriving in the 21st century, edited by Robert Graham and Nicole F. Kahn, National Academies Press, Washington, D.C., 2020.
- 243 The Adolescent Brain.
- 244 Patton et al., 'Our Future: A Lancet commission'.
- 245 'Normative Adolescent Development'.
- 246 Romanello M, Di Napoli C, Drummond P, Green C, Kennard H, Lampard P, Scamman D. Arnell N. Aveb-Karlsson S. Ford LB. Belesova K. Bowen K. Cai W, Callaghan M, Campbell-Lendrum D, Chambers J, van Daalen KR, Dalin C, Dasandi N, Dasgupta S, Davies M, Dominguez-Salas P, Dubrow R, Ebi KL, Eckelman M, Ekins P, Escobar LE, Georgeson L, Graham H, Gunther SH, Hamilton I, Hang Y, Hänninen R, Hartinger S, He K, Hess JJ, Hsu SC, Jankin S, Jamart L, Jay O, Kelman I, Kiesewetter G, Kinney P, Kjellstrom T, Kniveton D, Lee JKW, Lemke B, Liu Y, Liu Z, Lott M, Batista ML, Lowe R, MacGuire F, Sewe MO, Martinez-Urtaza J, Maslin M, McAllister L, McGushin A, McMichael C, Mi Z, Milner J, Minor K, Minx JC, Mohajeri N, Moradi-Lakeh M, Morrissey K, Munzert S, Murray KA, Neville T, Nilsson M, Obradovich N, O'Hare MB, Oreszczyn T, Otto M, Owfi F, Pearman O, Rabbaniha M, Robinson EJZ, Rocklöv J, Salas RN, Semenza JC, Sherman JD, Shi L, Shumake-Guillemot J, Silbert G, Sofiev M, Springmann M, Stowell J, Tabatabaei M, Taylor J, Triñanes J, Wagner F, Wilkinson P, Winning M, Yglesias-González M, Zhang S, Gong P, Montgomery H, Costello A. The 2022 report of the Lancet Countdown on health and climate change: health at the mercy of fossil fuels. Lancet. 2022 Nov 5;400(10363): 1619-1654.
- 247 Vergunst, Francis, et al., 'Climate Change and Substance-Use Behaviors: A risk-pathways framework', *Perspectives on Psychological Science*, vol. 18, no. 4, July 2023, pp. 936–954.
- Scamman D, Arnell N, Ayeb-Karlsson S, Ford LB, Belesova K, Bowen K, Cai W, Callaghan M, Campbell-Lendrum D, Chambers J, van Daalen KR, Dalin C, Dasandi N, Dasgupta S, Davies M, Dominguez-Salas P, Dubrow R, Ebi KL, Eckelman M, Ekins P, Escobar LE, Georgeson L, Graham H, Gunther SH, Hamilton I, Hang Y, Hänninen R, Hartinger S, He K, Hess JJ, Hsu SC, Jankin S, Jamart L, Jay O, Kelman I, Kiesewetter G, Kinney P, Kjellstrom T, Kniveton D, Lee JKW, Lemke B, Liu Y, Liu Z, Lott M, Batista ML, Lowe R, MacGuire F, Sewe MO, Martinez-Urtaza J, Maslin M, McAllister L, McGushin A, McMichael C, Mi Z, Milner J, Minor K, Minx JC, Mohajeri N, Moradi-Lakeh M, Morrissey K, Munzert S, Murray KA, Neville T, Nilsson M, Obradovich N, O'Hare MB, Oreszczyn T, Otto M, Owfi F, Pearman O, Rabbaniha M, Robinson EJZ, Rocklöv J, Salas RN, Semenza JC, Sherman JD, Shi L, Shumake-Guillemot J, Silbert G, Sofiev M, Springmann M, Stowell J, Tabatabaei M, Taylor J, Triñanes J, Wagner F, Wilkinson P, Winning M, Yglesias-González M, Zhang S, Gong P, Montgomery H, Costello A. The 2022 report of the Lancet Countdown on health and climate change: health at the mercy of fossil fuels. Lancet. 2022 Nov 5;400(10363): 1619-1654.
- 249 Proulx, Kerrie, et al., 'Climate Change Impacts on Child and Adolescent Health and Well-Being: A narrative review', Journal of Global Health, vol. 14, art. 04061, 24 May 2024.
- 250 Cianconi, Paolo, Sophia Betrò and Luigi Janiri, 'The Impact of Climate Change on Mental Health: A systematic descriptive review', Frontiers in Psychiatry, vol. 11, art. 74, 2020.

- 251 United States Centers for Disease Control and Prevention, 'Schools are the Right Place for a Healthy Start for Adolescents', CDC, 21 September 2020, <www.cdc.gov/healthyyouth/about/why_schools.htm>, accessed December 12 2024.
- 252 Sabarwal, Shwetlena, et al., *Choosing Our Future: Education for climate action*, World Bank, Washington, D.C. 2024.
- 253 Park, R. Jisung, A. Patrick Behrer and Joshua Goodman, 'Learning is Inhibited by Heat Exposure, both Internationally and within the United States', Nature Human Behaviour, vol. 5, no. 1, January 2021, pp. 19–27.
- 254 GBD 2019 Adolescent Mortality Collaborators, 'Global, Regional, and National Mortality among Young People Aged 10–24 Years, 1950-2019: A systematic analysis for the Global Burden of Disease Study 2019', Lancet, vol. 398, no. 10311, 30 October 2021, pp. 1593–1618.
- 255 Gordon, Rebecca J., and Catherine M. Gordon, 'Adolescents and Bone Health', Clinical Obstetrics and Gynecology, vol. 63, no. 3, September 2020, pp. 504–511.
- 256 Soliman, Ashraf T., et al., 'Nutritional Interventions during Adolescence and Their Possible Effects', Acta Biomedica, vol. 93, no. 1, art. e2022087, February 2022.
- 257 Hannon, Tamara S., and Silva A. Arslanian, 'Obesity in Adolescents', New England Journal of Medicine, vol. 389, no. 3, 20 July 2023, pp. 251–261.
- 258 Ibid.
- 259 World Health Organization, 'Obesity and Overweight', 1 March 2024, www.who.int/news-room/fact-sheets/detail/obesity-and-overweight, accessed 14 December 2024.
- 260 Ibid.
- 261 Nicolaou, Marina, et al., 'Obesogens in Adolescence: Challenging aspects and prevention strategies', Children, vol. 11, no. 5, art. 602, May 2024.
- 262 Ibid.
- 263 Huang, Miao, et al., 'Effects of Ambient Air Pollution on Blood Pressure among Children and Adolescents: A systematic review and meta-analysis', Journal of the American Heart Association, vol. 10, no. 10, art. e017734, 18 May 2021.
- 264 Ibid.
- 265 Zhang, Kai, et al., 'Air Pollution, Built Environment, and Early Cardiovascular Disease', Circulation Research, vol. 132, no. 12, 9 June 2023, pp. 1707–1724.
- 266 Gauderman, W. James, et al., 'The Effect of Air Pollution on Lung Development from 10 to 18 Years of Age', New England Journal of Medicine, vol. 351, no. 11, 9 September 2004, pp. 1057–1067.
- 267 Gauderman, W. James, et al., 'Association of Improved Air Quality with Lung Development in Children', New England Journal of Medicine, vol. 372, no. 10, 5 March 2015, pp. 905–913.
- 268 GBD 2019 Child and Adolescent Communicable Disease Collaborators, 'The Unfinished Agenda of Communicable Diseases among Children and Adolescents before the COVID-19 Pandemic, 1990-2019: A systematic analysis of the Global Burden of Disease Study 2019', *Lancet*, vol. 402, no. 10398, 22 July 2023, pp. 313–335.
- 269 Laycock, Katherine M., Leslie A. Enane and Andrew P. Steenhoff, 'Tuberculosis in Adolescents and Young Adults: Emerging data on TB transmission and prevention among vulnerable young people', *Tropical Medicine and Infectious Disease*, vol. 6, no. 3, art. 148, September 2021.
- 270 Beckwith, Sam, Venkatraman Chandra-Mouli and Robert Wm. Blum, 'Trends in Adolescent Health: Successes and challenges from 2010 to the present', *Journal of Adolescent Health*, vol. 75, no. 4, suppl., October 2024, pp. S9–S19.
- 271 United Nations Children's Fund, 2023 Global Snapshot on HIV and AIDS: Progress and priorities for children, adolescents and pregnant women, UNICEF, New York, 2023.
- 272 Ferrand, Rashida Abbas, 'Communicable Diseases across the Entire Developmental Window of Childhood and Adolescence: An outstanding agenda', *Lancet*, vol. 402, no. 10398, 22 July 2023, pp. 269–271.
- 273 Laycock, Enane and Steenhoff, 'Tuberculosis in Adolescents and Young Adults'.

- 274 Zhang, Jing, et al., 'Global, Regional, and National Burdens of HIV and Other Sexually Transmitted Infections in Adolescents and Young Adults Aged 10–24 Years from 1990 to 2019: A trend analysis based on the Global Burden of Disease Study 2019', Lancet Child & Adolescent Health, vol. 6, no. 11, November 2022, pp. 763–776.
- 275 Tremblay, Frédérique, et al., 'A Systematic Review of the Association between History of Sexually Transmitted Infections and Subsequent Condom Use in Adolescents', BMC Public Health, vol. 24, art. 1000, 10 April 2024.
- 276 Shannon, Chelsea L., and Jeffrey D. Klausner, 'The Growing Epidemic of Sexually Transmitted Infections in Adolescents: A neglected population', Current Opinion in Pediatrics, vol. 30, no. 1, February 2018, pp. 137–143.
- 277 World Health Organization, 'Cervical Cancer', 5 March 2024, www.who.int/news-room/fact-sheets/detail/cervical-cancer, accessed 14 December 2024
- 278 United States Centers for Disease Control and Prevention, 'Basic Information about HPV and Cancer', 17 September 2024, www.cdc.gov/cancer/hpv/basic-information.html, accessed 14 December 2024.
- 279 Wiafe, Stephanie, Ariana Mihan and Colleen M. Davison, 'Neighborhood-Level Influences and Adolescent Health Risk Behaviors in Rural and Urban sub-Saharan Africa: A systematic review', International Journal of Environmental Research and Public Health, vol. 18, no. 14, art. 7637, July 2021.
- 280 Patton et al., 'Our Future: A Lancet commission'; Ruiz, J. R., et al., 'Predictive Validity of Health-Related Fitness in Youth: A systematic review', British Journal of Sports Medicine, vol. 43, no. 12, November 2009, pp. 909–923.
- 281 Guthold, Regina, et al., 'Global Trends in Insufficient Physical Activity among Adolescents: A pooled analysis of 298 population-based surveys with 1.6 million participants', Lancet Child & Adolescent Health, vol. 4, no. 1, January 2020, pp. 23–35.
- 282 Agostini, Alex, and Stephanie Centofanti, 'Normal Sleep in Children and Adolescence', *Child and Adolescent Psychiatric Clinics of North America*, vol. 30, no. 1, January 2021, pp. 1–14.
- 283 Rony, Moustaq Karim Khan, and Hasnat M. Alamgir, 'High Temperatures on Mental Health: Recognizing the association and the need for proactive strategies A perspective', Health Science Reports, vol. 6, no. 12, art. e1729, December 2023; Liu, Jianghong, et al., 'Air Pollution Exposure and Adverse Sleep Health across the Life Course: A systematic review', Environmental Pollution, vol. 262, art. 114263, July 2020.
- 284 'Normative Adolescent Development'.
- 285 World Health Organization, 'Adolescent and Young Adult Health', 26 November 2024, <www.who.int/news-room/fact-sheets/detail/ adolescents-health-risks-and-solutions>, accessed 14 December 2024.
- 286 Weirich, Chelsea A., and Todd R. Miller, 'Freshwater Harmful Algal Blooms: Toxins and children's health', *Current Problems in Pediatric and Adolescent Health Care*, vol. 44, no. 1, January 2014, pp. 2–24.
- 287 World Health Organization, Global Report on Drowning: Preventing a leading killer, WHO, Geneva, 2014.
- 288 Luo, Shi, et al., The Influence of Water Safety Knowledge on Adolescents' Drowning Risk Behaviors: A framework of risk-protect integrated and KAP theory', Frontiers in Public Health, vol. 12, art. 1354231, 2024.

- 289 Bitsko, Matthew J., Robin S. Everhart and Bruce K. Rubin, 'The Adolescent with Asthma', *Paediatric Respiratory Reviews*, vol. 15, no. 2, June 2014, pp. 146–153.
- 290 Corbett, Tracy, and Joanne Smith, 'Exploring the Effects of Being Diagnosed with Type 1 Diabetes in *Adolescence', Nursing Standard*, vol. 35, no. 7, 8 July 2020, pp. 77–82.
- 291 World Health Organization, Sanitation and Hygiene: Training for health care providers, 3rd ed., WHO, Geneva, 2023.
- 292 Ibid.
- 293 Alugnoa, Desmond N., Trevor Cousins and Mayumi Sato, 'Period Poverty and Menstrual Belonging: A matter of climate justice', Lancet Planetary Health, vol. 6, no. 7, July 2022, pp. e551–e552.
- 294 United Nations Children's Fund, 'Early Childbearing', November 2024, https://data.unicef.org/topic/child-health/adolescent-health/, accessed 18 November 2024.
- 295 World Health Organization, 'Adolescent Pregnancy', 10 April 2024, www.who.int/news-room/fact-sheets/detail/adolescent-pregnancy, accessed 18 November 2024.
- 296 Maheshwari, Marvi V., et al., 'Maternal and Neonatal Outcomes of Adolescent Pregnancy: A narrative review', *Cureus*, vol. 14, no. 6, art. e25921, 14 June 2022.
- 297 Ibid.
- 298 Golub, M. S., 'Adolescent Health and the Environment', *Environmental Health Perspectives*, vol. 108, no. 4, April 2000, pp. 355–362.
- 299 International Labour Organization, 'What is Child Labour', <www.ilo.org/international-programme-elimination-child-labour-ipec/ what-child-labour>, accessed 13 November 2024.
- 300 Ibid.
- 301 International Labour Office and United Nations Children's Fund, Child Labour: Global estimates 2020. trends and the road forward, ILO and UNICEF, New York, 2021.
- 302 Abdel Rasoul, Gaafar M., et al., 'Effects of Occupational Pesticide Exposure on Children Applying Pesticides', NeuroToxicology, vol. 29, no. 5, September 2008, pp. 833–838.
- 302 Child Labour: Global estimates 2020.
- 304 Kearns, Katherine A., et al., 'Estimating Personal Exposures to Household Air Pollution and Plastic Garbage Burning among Adolescent Girls in Jalapa, Guatemala', Chemosphere, vol. 348, art. 140705, January 2024.
- 805 Eskenazi, Brenda, et al., 'The International Society for Children's Health and the Environment Commits to Reduce Its Carbon Footprint to Safeguard Children's Health', Environmental Health Perspectives, vol. 128, no. 1, art. 014501, January 2020.
- 306 Carpenter, David O., and Sheila Bushkin-Bedient, 'Exposure to Chemicals and Radiation During Childhood and Risk for Cancer Later in Life', Journal of Adolescent Health, vol. 52, no. 5, suppl., May 2013, pp. S21–S29.
- 307 Cohn, Barbara A., et al., 'DDT and Breast Cancer in Young Women: New data on the significance of age at exposure', *Environmental Health Perspectives*, vol. 115, no. 10, October 2007, pp. 1406–1414.



Acknowledgements

Author: Maria Brown, with guidance from Abheet Solomon **UNICEF reviewers:** Maaike Arts, Alexandre Boon, Ulrike Gilbert, Katelyn Greer, Tomomi Kitamura, Desiree Narvaez, Chemba Raghavan, Eduardo Garcia Rolland, Kam Sripada

External reviewers: Prerna Banati, Ruth Etzel, Julia Gorman, Phil Landrigan, Frederica Perera, Skye Wheeler

The statements in this document should not be taken as representing the official position and policies of UNICEF.

The document will be revised as new information and contributions becomes available. Suggestions and comments are welcome and may be sent to ceh@unicef.org.

United Nations Children's Fund

3 United Nations Plaza New York, NY 10017, USA www.unicef.org

