The Unique Vulnerabilities of Children to Environmental Hazards



Karen English, Colleen Lau, and Paul Jagals

1 Introduction

Young children are at extraordinarily high risk of adverse environmental health outcomes because biological and behavioural factors make them more vulnerable to environmental hazards than older children (teens) and adults [1-3]. The most vulnerable times for very young children are the first 1000 days of their lives, from conception up to the end of their second year after birth [4]. The special vulnerabilities of very young children during the first 1000 days are largely due to their environmental settings and their exposures that range widely, depending on their environmental interaction. Their vulnerabilities can be categorised in terms of timing (phases), environmental settings, and the hazards within those settings that characterises their environmental exposures.

The timing categories are (1) of the father and mother and even previous generations up to the point of conception, (2) during pregnancy, and (3) after birth, up to day 1000 [1, 3–6]. Environments range from: (1) environments of the parents-to-be and earlier generations that can influence the young child even before conception and range from domestic, workplace, transport, institutional (healthcare facilities), commercial (shopping), recreational environments (swimming pools, parks), and more—these multiple environments in which we live every day, the potential exposures and hazards vary in intensity and type; (2) in the intra-uterine environment, exposures of the mother determines indirect exposures of the unborn

K. English \cdot P. Jagals (\boxtimes)

Children's Health and Environment Program, Child Health Research Centre, The University of Queensland, Brisbane, QLD, Australia e-mail: p.jagals@uq.edu.au

C. Lau

Department of Global Health, College of Health and Medicine, Australia National University, Canberra, ACT, Australia

[©] Springer Nature Singapore Pte Ltd. 2020

Y. Xia (ed.), Early-life Environmental Exposure and Disease, https://doi.org/10.1007/978-981-15-3797-4_6



Fig. 1 The outline of the chapter

child; and (3) after birth, the child will be directly exposed to mainly domestic environments but will be interspersed with transport environments, healthcare facilities, and more [1, 4, 7].

We also need to consider the different types of hazards, which can be physical, chemical, or biological, and their specific modes of exposure which are ingestion, sublingual contact, inhalation, and dermal contact [8]. Exposure to hazardous substance found in these environments and during these three timing periods can occur through multiple sources and pathways. These include through the diet (e.g. pesticides and food-borne pathogens entering the food chain at various stages of production and consumption and from contact with chemicals leaching from packaging), particles in dust, natural hazards (e.g. pollens, animals, radiation, infectious agents), air pollution (e.g. traffic-related air pollution), and direct contact with products containing chemicals and microbes [1, 9, 10].

The purpose of this chapter is to inform on phases of exposures to environments and their hazards a child is directly and/or indirectly exposed to in the first 1000 days of life, as conceptualised in Fig. 1.

2 Environmental Hazards

We generally have a good understanding of single and mixtures of exogenous environmental chemical, physical and infectious hazards in the different environments that a child and their parents spend time in [4]. In recent times, rapidly intensifying socio-economic, environmental and technological drivers are exacerbating complex exposures to well-known as well as emerging hazards. Parental behaviour-mostly determined by their socio-economic status and (paradoxically) often in efforts to protect the child—may also lead to increasing early life exposure to environmental hazards. The way we set ourselves up in society—relying on both ecological services (greenness, water, air) and our engineered services (urban environments, healthcare facilities, food and water supply, as well as waste management)—can increase hazard potential if not optimally managed [4, 7, 11-14]. These services are also under pressure from broader regional and global environmental and demographic drivers such as climate change, population growth, urbanisation, antimicrobial resistance, prolific production use of chemicals, emerging infectious diseases, and pollution caused by inadequate waste management. These drivers are rapidly enhancing our environmental hazard potential and thus increasing exogenous pressures on the body's cellular toxicity pathways (internal exposure). Ultimately, these pressures may lead to adverse health outcomes—especially in the window of vulnerability in a child's early life-and could also continue to have negative impacts in later life [1, 12, 15]. More about specific health outcomes can be found in other chapters in this book. This section is about external environmental hazards mostly associated with multiple exposures to multiple hazards over time.

Hazards considered environmental in origin include contaminants that occur naturally, those that occur naturally but are driven to extremes (heat and other environmental disasters), contaminants in goods manufactured for a specific purpose, or by-products and wastes from the way we produce and use those goods to support the way we live. The built environment and social factors may also contribute to the risks and impacts on health in early life [12, 14, 16, 17].

Examples of naturally present hazards include radioactive materials such as rock and soil that emit radon; metals such as arsenic that contaminate drinking water; particulate matter that results from fires, wind, and erosion; pollens and allergens from plants and animals; infectious pathogens from animals (zoonotic diseases) and in water and soil; and dust mites that can be allergenic [9, 13, 18].

Contaminants and products manufactured and transferred through human environments include pesticides used on food crops and chemicals in services such as disinfection by-products in water supplies. More specifically in children, phthalates in toys and specially prepared early-life environments such as baby rooms furnished with goods and textiles that contain flame retardants can be toxic [8, 11].

Hazardous exposures through shortcomings in services and production processes include inadequate safe water supply, airborne particulates and gases from combustion processes derived from generating energy (cooking and heating), inadequate sanitation and waste management [13, 19–21].

Humans are now travelling more than ever before, both in their day-to-day commuting as well as long-distance international travel. Transport environments, such as those that involve cars, trains, buses, planes, ships, as well as their service points such as fuelling and transfer stations and ports, result in exposures to considerably higher levels of emissions from exhaust particles, gases and fuel fumes [14, 20, 22], as well as chemicals used in the manufacturing of vehicles and planes, including plastics and flame retardants [23]. Crowding during transport and at stations also increases the risk of exposure to infectious agents [24]. Very young children are often part of this environment—being taken from and to health care, childcare, and other journeys even at a very young age.

Environmental hazard potential is also increased by low socioeconomic status of the parents. These include the domestic location (distance from the source of pollution), whether the home (domestic) environment is based in an urban, suburban, or rural location, as well as the design, condition, and age of the dwelling, and also the materials of which the goods used in the home are made of [17, 25]. Services such as quality of nutritional and safe food and drinking water supply, condition of indoor air, and efficient waste management all will play a role in moderating environmental exposures in early life [3, 10, 21].

3 Exposure Environments

Why do we differentiate between specific environments? Exposure to chemicals and infectious agents in our environments is ubiquitous and worldwide and especially relevant to vulnerable young children. In the past decades, the manufacture and use of chemicals in our homes, institutions, industries, care facilities, and urban services and utilities have increased substantially [7, 17, 26]. The presence and concentration of these anthropogenic hazards, as well as natural hazards, vary within specific environments. It is important to understand that human exposure to environmental hazards is determined by multiple factors, including time spent in these environments, as well as by physiology and behaviours that modify exposure [27]. Understanding environmental interaction during preconception, pregnancy, and in early infancy is therefore critical to our understanding of how very young children are especially vulnerable to detrimental health outcomes when exposed to hazards in these different environments [28].

3.1 Intrauterine Environment

Preconception and prenatal exposures are especially important because exposure to many and varied synthetic chemicals and infectious agents has now become the norm around the world—not just in the industrialised countries. Maternal exposure to chemicals and infectious agents during pregnancy is known to increase the risk of

foetal exposure because of potential transfer across the placenta to the foetus as well as transfer during birth [1, 7]. Maternal exposures during pregnancy can also have multigenerational effects, including direct toxic effects on developing oocytes in female offspring, but also via other mechanisms. Among several proposed mechanisms, epigenetic changes, changes that alter gene function without altering DNA structure, transmitted through generations have been implicated in preconception and multigenerational exposure effects on health [1, 4, 6, 29].

Maternal health status can determine the extent to which chemicals cross the placenta. For example, high blood pressure and maternal drug and alcohol use have been associated with relatively greater concentration of chemicals—notably lead—compared to maternal concentrations, while other factors appear to reduce the degree of transfer [4, 30]. The specific structure, chemical composition, and relative persistence of xenobiotic chemicals also determine the pattern of placental transfer. Persistent chemicals (PBDE and PFC) are lipophilic or are bound to proteins that enter the hepatic bloodstream circulation and directly expose to the foetus. Some chemicals (e.g. methyl mercury) bioaccumulate in the foetus to levels higher than those measured in the mother and can affect the developing brain of the foetus during the first 1000 days and beyond while at the same time having mild adverse effects on the mother [31].

Many infectious diseases during pregnancy are known to adversely affect the foetus [32], and the transmission of many of these diseases is strongly driven by environmental factors. For example, malaria and Zika (both mosquito-borne diseases) are associated with poor health in the pregnant woman as well as a high risk of birth complications and foetal abnormalities [33, 34]. Preconception immunisation is therefore important for protecting the foetus against intrauterine infections, especially pathogens that are strongly associated with congenital abnormalities (e.g. rubella) [35].

3.2 Domestic Environment

Most of us, including pregnant women and young children, spend the majority of our time at home. Yet many households are quite unaware of the high hazard potential of the home environment [4, 14]. Environmental hazards at home will therefore pose the highest risk simply because of the combination of time spent in this environment, and ignorance about how to manage household goods and services that are potentially hazardous.

During pregnancy, an unborn child's exposure to what are in their environments is largely determined by how the mother interacts with the domestic and also other environments. As we have seen with the intra-uterine environment, the modern-day unborn child is not as well protected against the environment as was once thought. After birth, a child generally spends most of the rest of its time during the first 1000 days in its domestic environment [14, 36].

3.3 The Global Environment and Infectious Agents

While environments closer to the very young child are the focus of this chapter, it is worthwhile to consider this much broader context of environment, as it has direct bearing on the condition of the child's direct daily environments. With globalisation, the unprecedented movement of people, animals, and goods around the world has resulted in an increasingly global environment for both infectious and non-infectious diseases [37]. In addition to air, food, water, and soil, exposure to infectious agents can also occur through direct contact with other people and animals (zoonotic diseases) and through insects (e.g. mosquito-borne disease). Globalisation and our increasingly connected world mean that outbreaks and epidemics can spread more rapidly than ever around the world. The emergence and global spread of Zika virus and the devastating consequences of congenital Zika syndrome provide a striking example of the potential impact of infectious disease outbreaks on child health [38].

4 Other Special Vulnerabilities

4.1 Preconception Parental Exposures

The preconception environments of the parents-to-be, and even earlier generations, can influence the very young child. For example, paternal preconception exposures to environmental hazards—which could be encountered in many environments—have been shown to induce epigenetic changes in sperm and are associated with health effects in the child [39]. Similarly, maternal grandmother smoking has been associated with childhood asthma [40]. More about this can be found in other chapters of this book.

4.2 Post-birth Exposures

Infants breathe, drink, and eat more relative to older children, teens, and adults. They are thus subject to proportionally greater exposure to environmental hazards once they are born [4].

In recent years, research has increased our understanding of the reasons environmental contaminants have a different effect on children compared to adults. While children generally have smaller body mass than adults, their exposures (relative to body mass to pollutants) can be much larger, and thus children respond very differently when exposed to environmental hazards than adults [1, 4, 18].

4.3 Breast Milk

For some chemical hazards, the most significant exposure during early life is through breast milk [31]. Ubiquitous hazardous chemicals include persistent organic pollutants, like organochlorine pesticides, polychlorinated biphenyls, brominated flame retardants, dioxins, and perfluorinated alkyl substances, which are transferable to the young child though breast milk and milk formula [41]. However, at this time, health scientists believe the benefits of breastfeeding far outweigh any risks [4].

4.4 Dermal Exposure

Infants have a higher skin surface area to bodyweight ratio and greater contact with their surroundings than adults. An infant may absorb through the skin a larger dose of a chemical agent on a body weight basis than would an older child or adult. Newborns, especially those who are preterm, experience increased absorption of some compounds through the skin [4, 17].

4.5 Children Have Unique Behaviours

Young children develop rapidly through the first 1000 days. Their activity patterns and behaviours change. Newborns typically spend prolonged periods of time in a single environment such as at home and are thus exposed for relatively longer periods to any hazardous element that might be in the vicinity [17]. They become more mobile and begin to spend more time on the ground as well as in the 'dust' zone of a home, which is approximately up to 1.5 m from the floor level [11]. Behaviours such as pica (tendency to ingest non-dietary substances), crawling, and the tendency to explore items by mouth increase ingestion of contaminants on surfaces. Very young children are also more exposed to pesticide residues from home applications, dust and contaminants carried by dust, volatile organic chemicals from carpets, and chemicals such as chromated copper arsenate from wooden playground equipment [4, 7].

4.6 Children Have Less Mature Immune Systems

Very young children are more susceptible to some infectious diseases (e.g. diarrhoea) and may develop more serious illnesses (e.g. whooping cough) compared to older children and adults. Also some vaccines are not effective in very young children, making it impossible to protect them through immunisation [42, 43].

4.7 Children Have a Longer Future and Are Generally More Sensitive

Children are in a rapidly developing stage of their early lives. From birth through the end of the first 1000 days and beyond, children differ from older people in their ability to absorb, metabolise, and excrete contaminants [4]. Detoxifying enzyme systems develop throughout childhood, so the ability to mitigate the effects of chemicals is age dependent and likely to be at its lowest protective capabilities during this phase [2]. Because children are physiologically immature and rapidly developing, environmental hazard exposure can result in long-term irreversible structural changes [44]. Diseases that develop through chronic exposures and/or have long latency periods are likely to have more serious impacts when exposure begins at an early age and continues over long periods of time. For instance, certain cancer outcomes such as melanoma are reported to be primed by exposure to environmental carcinogens early in life rather than total exposure throughout life [45].

5 Conclusions

Children—in the first 1000 days of their lives—are extremely vulnerable to environmental hazards. Their exposures to these hazards are determined by the phase in which the parents, the foetus, newborn, and infant find themselves. Exposures are varied because of the many different chemical, physical and infectious hazards that can be encountered in different environments of the child summarised as preconception, intrauterine, and after birth—predominantly in their domestic environments. This chapter aimed at raising the awareness of this complex exposure matrix of development phase, environmental hazard potential, and various environmental settings.

References

- 1. Landrigan PJ et al (2019) Pollution and children's health. Sci Total Environ 650:2389–2394
- Makri A et al (2004) Children's susceptibility to chemicals: a review by developmental stage. J Toxicol Environ Health Part B 7(6):417–435
- 3. Mattison DR (2010) Environmental exposures and development. Curr Opin Pediatr 22(2):208
- 4. Landrigan P, Etzel R (2014) Children's environmental health. Oxford University Press, New York
- 5. Robertson RC et al (2019) The human microbiome and child growth-first 1000 days and beyond. Trends Microbiol 27(2):131–147
- 6. Soubry A et al (2014) A paternal environmental legacy: evidence for epigenetic inheritance through the male germ line. BioEssays 36(4):359–371
- 7. Zota AR et al (2017) Reducing chemical exposures at home: opportunities for action. J Epidemiol Community Health 71(9):937–940

- Healy BF et al (2015) Bisphenol a exposure pathways in early childhood: reviewing the need for improved risk assessment models. J Expo Sci Environ Epidemiol 25(6):544–556
- 9. Miyake Y et al (2007) Home environment and suspected atopic eczema in Japanese infants: the Osaka maternal and child health study. Pediatr Allergy Immunol 18(5):425–432
- Gitterman BA, Bearer CF (2001) A developmental approach to pediatric environmental health. Pediatr Clin N Am 48(5):1071–1083
- 11. English K et al (2015) Assessing exposure of young children to common endocrine-disrupting chemicals in the home environment: a review and commentary of the questionnaire-based approach. Rev Environ Health 30(1):25–49
- 12. Hystad P et al (2014) Residential greenness and birth outcomes: evaluating the influence of spatially correlated built-environment factors. Environ Health Perspect 122(10):1095–1102
- Hotez PJ (2013) Pediatric tropical diseases and the World's children living in extreme poverty. J Appl Res Child Inform Pol Child Risk 4(2):10
- Goldizen FC, Sly PD, Knibbs LD (2016) Respiratory effects of air pollution on children. Pediatr Pulmonol 51(1):94–108
- Mill J, Petronis A (2008) Pre-and peri-natal environmental risks for attention-deficit hyperactivity disorder (ADHD): the potential role of epigenetic processes in mediating susceptibility. J Child Psychol Psychiatry 49(10):1020–1030
- 16. Jackson RJ (2011) Designing healthy communities. Wiley, San Francisco
- 17. Rovira J, Domingo JL (2019) Human health risks due to exposure to inorganic and organic chemicals from textiles: a review. Environ Res 168:62–69
- 18. Pronczuk-Garbino J (2005) Children's health and the environment: a global perspective: a resource manual for the health sector. World Health Organization, Geneva
- Kurmi OP, Lam KBH, Ayres JG (2012) Indoor air pollution and the lung in low-and mediumincome countries. Eur Respir Soc 4(1):239–254
- 20. Buonanno G et al (2013) Children exposure assessment to ultrafine particles and black carbon: the role of transport and cooking activities. Atmos Environ 79:53–58
- Brown J, Cairneross S, Ensink JH (2013) Water, sanitation, hygiene and enteric infections in children. Arch Dis Child 98(8):629–634
- 22. Shirinde J, Wichmann J, Voyi K (2015) Allergic rhinitis, rhinoconjunctivitis and hay fever symptoms among children are associated with frequency of truck traffic near residences: a cross sectional study. Environ Health 14(1):84
- 23. English K et al (2017) Polybrominated diphenyl ether flame retardant concentrations in faeces from young children in Queensland, Australia and associations with environmental and behavioural factors. Environ Res 158:669–676
- 24. Mohr O et al (2012) Evidence for airborne infectious disease transmission in public ground transport-a literature review. Eur Secur 17(35):20255
- 25. Leventhal T, Newman S (2010) Housing and child development. Child Youth Serv Rev 32 (9):1165–1174
- 26. World Health Organization (2017) Don't pollute my future! The impact of the environment on children's health. World Health Organization
- 27. Zartarian V et al (2017) Children's lead exposure: a multimedia modeling analysis to guide public health decision-making. Environ Health Perspect 125(9):097009
- Tamayo-Uria I et al (2019) The early-life exposome: description and patterns in six European countries. Environ Int 123:189–200
- Hanson MA, Skinner MK (2016) Developmental origins of epigenetic transgenerational inheritance. Environ Epigenet 2(1)
- 30. Müller MHB et al (2019) Prenatal exposure to persistent organic pollutants in northern Tanzania and their distribution between breast milk, maternal blood, placenta and cord blood. Environ Res 170:433–442
- Jacobson JL et al (1984) The transfer of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) across the human placenta and into maternal milk. Am J Public Health 74 (4):378–379

- 32. Zhao J et al (2013) Effect of intrauterine infection on brain development and injury. Int J Dev Neurosci 31(7):543–549
- 33. Honein MA et al (2017) Birth defects among fetuses and infants of US women with evidence of possible Zika virus infection during pregnancy. JAMA 317(1):59–68
- 34. Rogerson SJ et al (2018) Burden, pathology, and costs of malaria in pregnancy: new developments for an old problem. Lancet Infect Dis 18(4):e107–e118
- 35. Yawetz S, Barss V, Weller PF (2015, April) Immunizations during pregnancy. Up to date
- 36. Gopalan HN, Saksena S (1999) Domestic environment and health of women and children. In: Domestic environment and health of women and children. PNUMA, Delhi
- 37. De Cock KM et al (2013) The new global health. Emerg Infect Dis 19(8):1192
- 38. Rasmussen SA et al (2016) Zika virus and birth defects—reviewing the evidence for causality. N Engl J Med 374(20):1981–1987
- 39. Marcho C, Oluwayiose OA, Pilsner JR (2020) The preconception environment and sperm epigenetics. Andrology
- 40. Magnus P et al (2006) Cohort profile: the Norwegian mother and child cohort study (MoBa). Int J Epidemiol 35(5):1146–1150
- 41. Arnold C (2019) Baby steps forward: recommendations for better understanding environmental chemicals in breast milk and infant formula. Environ Health Perspect 127(6):064001
- 42. Demirjian A, Levy O (2009) Safety and efficacy of neonatal vaccination. Eur J Immunol 39 (1):36–46
- 43. Simon AK, Hollander GA, McMichael A (2015) Evolution of the immune system in humans from infancy to old age. Proc R Soc B Biol Sci 282(1821):20143085
- 44. World Health Organization (2005) Children's health and the environment: a global perspective: a resource manual for the health sector
- 45. Singh K et al (2013) Congenital malignant melanoma: a case report with cytogenetic studies. Am J Dermatopathol 35(8):e135–e138