

The First Thousand Days

AN EVIDENCE PAPER

September 2017

The First Thousand Days: An Evidence Paper

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Strong Foundations is an initiative of the Australian Research Alliance for Children & Youth, Bupa Australia, the Bupa Health Foundation, the Murdoch Children's Research Institute, and PwC Australia.

Strong Foundations has been generously supported by the Bupa Health Foundation.

Suggested citation: Moore, T.G., Arefadib, N., Deery, A., & West, S. (2017). *The First Thousand Days: An Evidence Paper*. Parkville, Victoria; Centre for Community Child Health, Murdoch Children's Research Institute.

The writing team acknowledges the valuable commentaries by the following expert reviewers:

Associate Professor Jill Sewell, Associate Professor Stephanie Brown, Associate Professor Jeffrey Craig, and Dr Michael Little. Feedback on the section on Aboriginal children and families was provided by Ms Claire Stacey and Mr John Burton from SNAICC - National Voice for our Children.

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Glossary

Aboriginal: Aboriginal and Torres Strait Islander Australians

Aboriginal Health: Refers to physical, social, emotional and cultural well-being of the whole Community in which each individual is able to achieve their full potential as a human being, thereby bringing about the total well-being of their Community. Connection to land, spirituality and ancestry, kinship networks, and cultural continuity are commonly identified by Aboriginal people as important health-protecting factors.

Adverse childhood experiences (ACEs): Adverse childhood experiences (ACEs) are potentially traumatic events that can have negative, lasting effects on health and well-being. These experiences range from physical, emotional, or sexual abuse to parental divorce or the incarceration of a parent or guardian.

Allergy: Occurs when a person's immune system reacts to substances in the environment that are harmless for most people. These substances are known as allergens and are found in dust mites, pets, pollen, insects, ticks, moulds, foods and some medicines.

Antenatal (also known as prenatal): Period before childbirth

Biological embedding: The process whereby environmental and social experiences influence human biological and developmental processes and influence health, well-being, learning, or behaviour over the life course.

Central nervous system: The brain, brainstem, and spinal cord.

Child abuse: Refers to the four different types of child abuse: physical, sexual, and emotional abuse, and neglect.

Colonisation: The forming of a settlement or colony by a group of people who seek to take control of territories or countries. In Australia, the British authorities maintained that the land was terra nullius ('no one's land') and believed they were legally entitled to occupy the land.

Complex childhood trauma: Exposure to multiple or prolonged traumatic events and the impact of this exposure on development.

Co-morbidity: When two disorders or illnesses occur in the same person, simultaneously or sequentially, they are described as comorbid. Comorbidity also implies interactions between the illnesses that affect the course and prognosis of both.

Council of Australian Governments (COAG): The peak intergovernmental forum in Australia. Chaired by the Prime Minister, COAG's role is to promote policy reforms that are of national significance, or which need co-ordinated action by all Australian governments.

Developmental domains: Refers to the five key developmental domains which are identified by the Australian Early Development Census: physical health and wellbeing; social competence; emotional maturity; language and cognitive skills; and communication skills and general knowledge.

Developmental plasticity: The capacity to express specific adaptive responses to environmental conditions. These can be immediate, short-term or long-term changes in physiology and behaviour.

Differential susceptibility: The association between certain genetic profiles and increased susceptibility to environmental conditions. This means that some children are more influenced by their environmental conditions than others as a function of the presence or absence of specific genetic characteristics.

Dysbiosis: Disturbances in the composition of the microbial communities either in or on the body, usually associated with adverse health conditions.

Early childhood trauma: The traumatic experiences that occur to children aged 0-6. These traumas can be the result of intentional

violence—such as child physical or sexual abuse, or domestic violence—or the result of natural disaster, accidents, or war. Young children also may experience traumatic stress in response to painful medical procedures or the sudden loss of a parent/caregiver. (See also *Adverse childhood experiences*).

Endocrine system: Collection of glands in the body that produce hormones and release them into the bloodstream. The endocrine system works with the nervous system and the immune system to help the body cope with different events and stresses.

Epigenetic changes: DNA modifications that do not change the DNA sequence, but can modify gene activity by helping determine whether genes are turned on or off. Epigenetic change is a regular and natural occurrence but can also be influenced by several factors including age, the environment/lifestyle, and disease state.

Family and domestic violence: The intentional use of violence, threats, force or intimidation to control or manipulate a family member, partner or former partner.

Foetus: An unborn offspring, from the embryo stage (the end of the eighth week after conception, when the major structures have formed) until birth.

Foetal alcohol spectrum disorder (FASD): The effects of alcohol on the embryo or foetus produce a spectrum of disorders that impact physical, learning and behavioural outcomes. The range of effects is collectively termed 'foetal alcohol spectrum disorder'.

Genome: An organism's complete set of DNA, including all of its genes.

Genotype: The genetic makeup of an organism.

Global climate change: A change in the typical or average weather of a region or city (e.g. change in a region's average annual rainfall, or a city's average temperature for a given season). It also refers to a change in earth's overall climate (e.g. the earth's average temperature, or typical precipitation patterns).

Gut-brain-immune axis. Bidirectional connections between gut microbiota, brain and immune system which act as an interconnected network.

Harm: Any detrimental effect of a significant nature on the child's physical, psychological or emotional wellbeing and development.

Health: A state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity. (Health is defined differently by Western and Aboriginal people - see *Aboriginal health*).

Homelessness: There is no one definition of homelessness. However, in this report, homelessness refers to when a family does not have a sense of security, stability, privacy, safety, and the ability to control living space. It also refers to persons or families with no place of usual residence who move frequently between various types of accommodations (including dwellings, shelters and institutions for the homeless or other living quarters).

Homeostatic: The body's tendency to maintain a condition of balance within its internal environment, even when faced with external changes. These relate to steady levels of internal balance with things such as temperature and other vital conditions such as the water and contents of the blood.

Human Rights: Human rights are rights inherent to all human beings, whatever our nationality, place of residence, sex, gender, national or ethnic origin, colour, religion, language, or any other status. These rights are all interrelated, interdependent and indivisible.

Illicit drug: A drug that is prohibited from manufacture, sale or possession in Australia.

Immune system: Made up of a network of cells, tissues and organs in the body, designed to protect from, or get rid of, infection. (See also *Gut-brain-immune axis*.)

Intergenerational trauma: Trauma that is passed down through generations of families as a result of traumatic experiences such as loss and grief.

Low-grade inflammation: The body's immune system response which serves to initiate the elimination of toxic agents and the repair of damaged tissue. A chronic inflammatory response is likely to be involved in the early stages of a range of chronic conditions.

Matthew effects: Refers to the pattern of increasing advantage or disadvantage following early advantage or disadvantage where the gap between the advantaged and disadvantaged expands with time.

Mental health: A state of well-being in which every individual realizes his or her own potential, can cope with the normal stresses of life, can work productively and fruitfully, and is able to make a contribution to her or his community.

Mental illness: A health problem that significantly affects how a person feels, thinks, behaves, and interacts with other people. It is diagnosed according to standardised criteria. The term 'mental disorder' is also used to refer to these health problems.

Microbiome: The communities of bacteria, viruses, fungi and other symbiotic organisms that live in and on the human body.

Neglect: Neglect is the most common form of abuse and occurs when a parent or caregiver does not give a child the care he or she needs according to its age. Neglect can mean not giving food, clothing, and shelter, medical attention, exposing a child to dangerous environments, not responding to the child's emotional needs, poor supervision of a child, and abandoning the child.

Neuron: A nerve cell that processes and transmits information.

Neuroplasticity: The biological capacity of the central nervous system to change structurally and functionally in response to experience, and adapt to the environment

Non-communicable diseases: Also known as chronic diseases, they tend to be of long duration and are the result of a combination of genetic, physiological, environmental and behavioural factors.

Pathogen: A bacterium, virus, or other microorganism that can invade the body and produce disease.

Peripheral nervous system: This is our second nervous system and contains all the nerves in the body that lie outside of the spinal cord and brain (the nerves that go from the skin, muscle, and organs to the spinal cord and eventually the brain). It conducts information to and from the central nervous system.

Phenotype: The outcome of the interaction between the genotype and the environment, and is the organism's actual physical form and behaviour

Placenta: An endocrine organ that develops in the uterus during pregnancy and provides oxygen and nutrients to the foetus, while removing waste products from its blood.

Postnatal: Period after childbirth

Poverty: When a family's income fails to meet basic necessities such as adequate food, shelter and clothing, and impacts a family's lifestyle. Poverty often has a federally established threshold that differs across countries. Australia does not have an established poverty threshold. Typically poverty is measured with respect to families and not the individual, and is adjusted for the number of persons in a family. Poverty is associated with the undermining of a range of key human attributes, including health and wellbeing.

Psychoactive substance: Substances that, when taken in or administered into one's system, affect mental processes, e.g. cognition or affect. Refers to the whole class of substances, licit and illicit.

Racism: The avoidable and unfair phenomena that lead to inequalities in power, resources and opportunities across racial or ethnic groups. It can be expressed through beliefs and stereotypes, prejudices and discrimination, and occurs at many social levels, including interpersonally and systemically, and as internalised racism.

Social determinants of health: The social, economic and environmental conditions into which we are conceived, born, grow, live, and age.

Social gradient of health: The phenomenon where the higher a person's socioeconomic position, the healthier they are likely to be. That is, at any given point along the socioeconomic continuum, one is likely to experience inferior health outcomes to those above them.

Social supports: In this report, the terms 'social supports', 'social connections' and 'social relationships' are used interchangeably and refer to three categories of family support: practical; emotional; and advice and information.

Socioeconomic status: The social standing or class of an individual or group. It is often measured as a combination of education, income and occupation.

Synapse: Junction between two neurons which allows for information to be carried from one neuron to another

Telomeres: The caps at the end of chromosomes which stop DNA from unravelling during cell division, and shorten as we age.

Temperament: Distinct patterns of feelings and behaviours which shape affective, attentional and motor responses in various situations. Individual differences in the regulation of experience emerge early in life and remain moderately stable across development.

Temperamental bias: A bias towards certain temperamental characteristics which is often (but not always) genetic.

Trauma: An emotional response to a terrible event like an accident, violence against an individual, or natural disaster.

Triple hit effects: The theory that there are three conditions or events that are required to disturb development: an existing predisposition or vulnerability, a critical period of brain development, and exposure to environmental stressors.

United Nations: An international organisation founded in 1945. It is currently made up of 193 Member States. The mission and work of the United Nations are guided by the purposes and principles contained in its founding Charter.

United Nations Convention on the Rights of the Child: The Committee on the Rights of the Child is the body of 18 Independent experts that monitors implementation of the Convention on the Rights of the Child by its State parties. It also monitors implementation of two Optional Protocols to the Convention, on involvement of children in armed conflict and on sale of children, child prostitution and child pornography.

World Health Organisation: A specialised agency of the United Nations which directs and coordinates international health in six primary areas: Health systems; promoting health through the life-course; non-communicable diseases; communicable diseases; corporate services; preparedness, surveillance and response.

1. Introduction

1.1 The first 1000 days

The focus of this paper is on the earliest stages of child development, the period from conception to the end of the child's second year. This period has become known as the first 1000 days, a catchphrase that has become the rallying point for a number of Australian and international initiatives. While some of these have a general focus, such as the work of a cross-parliamentary group in the UK Parliament (Leadsom, Field, Burstow & Lucas, 2013; WAVE Trust, 2013, 2015), others are more narrowly focused on issues such as nutrition (Save the Children, 2012; Thousand Days, 2016) or on specific populations such as Aboriginal children (Arabena, Howell-Muers, Ritte, & Munro-Harrison, 2015; Arabena, Ritte & Panozzo, 2016).

The reason for focusing on this specific period is the growing body of evidence which shows that experiences during this period can have life-long consequences for health and wellbeing. Thus, as noted in the report of the World Health Organisation's Commission on Social Determinants of Health (2008),

Many challenges in adult society have their roots in the early years of life, including major public health problems such as obesity, heart disease, and mental health problems. Experiences in early childhood are also related to criminality, problems in literacy and numeracy, and economic participation. This paper seeks to summarise what is known about the biological processes and environmental characteristics that shape development during the first 1000 days, and what impact these have over the life span.

While there have already been many reviews of the literature on early development, all concluding that this period of life is critical in shaping health and wellbeing over the life course, there are several reasons why a new review of the evidence is needed, and why this paper differs from previous reviews.

First, research in this area is rapidly advancing, and our understanding of the specific mechanisms that impact upon development is becoming more and more detailed and nuanced. Keeping up with the exponential growth in research is an ongoing challenge, and regular updates such as this one are needed.

Second, the new research has revealed whole aspects of biological functioning that were not previously recognised as playing a role in development, such as telomere effects and the role of the microbiome. This review is the most comprehensive attempt yet to incorporate all known sources of influence on development, and even those well read in this area will learn from the paper.

Third, the focus of the paper is on the first 1000 days, rather than the early years in general as in most previous reviews. This is on the grounds that the first 1000 days is the period of maximum developmental plasticity, and therefore the period with the greatest potential to affect health and wellbeing over the life course.

1.2 Evolving ideas regarding the early years

Although the notion of critical / sensitive periods in development has been around for many years (e.g. Bailey, Bruer, Symons & Lichtman, 2001), the full extent of early developmental plasticity has not become evident until recently. Moreover, in Australia at least, the general public's perception of how young children develop and learn is often based on the perception that children are passive absorbers of knowledge who do not show signs of genuine learning until they are older (Bales & Kendall-Taylor, 2014; Kendall-Taylor & Lindland, 2013).

The recent interest in the early years (Allen, 2011; Field, 2010; Shonkoff & Phillips, 2000; Social Research Unit at Dartington, 2013) has been prompted by growing awareness that what happens during this period of development has lifelong consequences for children's health and wellbeing (Center on the Developing Child at Harvard University, 2010; Fox, Levitt & Nelson, 2010; Moore, 2014a, National Scientific Council on the Developing Child, 2007; Shonkoff, Garner, Committee on Early Childhood, Adoption, and Dependent Care, & Section on Developmental and Behavioral Pediatrics, 2012). It has also been prompted by a growing understanding of how early disparities in children's functioning can develop and the problems that this can create for future education, employment and opportunities (Brinkman et al., 2012, 2013; Centre for Community Child Health, 2008; Woolfenden et al., 2013).

This heightened awareness of the importance of the early years has led to many government initiatives, nationally and internationally. In Australia, this includes the National Early Childhood Development Strategy. However, many of these initiatives have primarily focused on the 3-5 year period and ensuring school 'readiness' (e.g. the Council of Australian Governments National Partnership Agreement on Early Childhood Education).

New evidence is now leading to a focus on the earliest stages of development, including the prenatal period (Barouki et al., 2012; Paul, 2010; Prescott, 2015; Shonkoff, Richter, van der Gaag & Bhutta, 2012). There are currently three key concepts that are supported by this growing body of evidence:

- Developmental plasticity and the developmental origins of health and disease (DOHaD) hypothesis
- Social climate change and the 'mismatch' hypothesis
- Ecological impacts on development and the social determinants of health and disease.

Collectively, evidence relating to these key concepts transforms our understanding of how children develop and highlights the critical role of the very earliest stages of development - the first 1000 days.

1.3 Scope of paper

This paper examines the impact of early experiences on all aspects of development and functioning, including physical health and wellbeing, mental health, social functioning and cognitive development. This is in keeping with the broader definition of health adopted by the World Health Organization (WHO) which defines health as 'a dynamic state of complete physical, mental, spiritual and social wellbeing and not merely the absence of disease or infirmity' (WHO, 1998). In Australia, Aboriginal and Torres Strait Islander people take this position even further, viewing health as 'not just the physical wellbeing of the individual but the social, emotional and cultural wellbeing of the whole community' (National Aboriginal Health Strategy Working Party, 1989). Connection to land, spirituality and ancestry, kinship networks, and cultural continuity are commonly identified by Aboriginal people as important health-protecting factors (Zubrick et al., 2014).

This paper also recognises that, as enshrined in the United Nations Convention on the Rights of the Child (1990), children have the fundamental human right to a high standard of health and wellbeing. The Committee on the Rights of the Child, charged by the United Nations with promoting and monitoring progress towards world-wide implementation of the Convention, has adopted a number of General Comments to guide governments in fulfilling their obligations under the Convention (Committee on the Rights of the Child, 2005, 2006, 2013). General Comment No. 7 (2005) - on implementing child rights in early childhood - stresses that young children have rights from the beginning of their lives. It acknowledges the special vulnerability of the very young to poverty, discrimination and other adversities that can compromise their rights and undermine their capacities and well-being. General Comment 13 (2013) - on the right of the child to the enjoyment of the highest attainable standard of health - interprets children's right to health as

an inclusive right, extending not only to timely and appropriate prevention, health promotion, curative, rehabilitative and palliative services, but also to a right to grow and develop to their full potential and live in conditions that enable them to attain the highest standard of health through the implementation of programmes that address the underlying determinants of health.

This paper seeks to identify these underlying determinants of health, understood in the broad terms noted earlier, so that children's rights to high standards of health and wellbeing may be better observed.

2. Biological processes shaping health and development

This section provides a synthesis of the evidence regarding the three key concepts that have led to the current focus on the earliest stages of development. Before considering this evidence, we need to understand the relationship between mind, brain and body.

2.1 The relationship between mind, brain and body

In seeking to understand early development, there has been a tendency to focus on neurological development at the expense of other aspects of development. Thus, efforts to disseminate new research knowledge have used the metaphor of 'brain architecture' to convey the sense of the importance of early neurological development (National Scientific Council on the Developing Child, 2007), and discussed how positive early experiences build neuronal connections and adverse experiences disrupt them (National Scientific Council on the Developing Child, 2004, 2005). This way of framing early development reflects an underlying belief in the importance of the brain as the seat of personhood and learning.

However, as Moore (2014a) has noted, 'framing brain development in terms of building neuronal connections and brain architecture fails to capture the fact that brain functioning is not purely cognitive, that 'learning' is not purely conscious, that the brain is not purely skull-based, and that the brain is closely linked with other key bodily systems.'

First, the brain is not purely cognitive, but is also profoundly emotional (Davidson & Begley, 2012). Thus, our emotions directly influence the functions of the entire brain and body, from physiological regulation to abstract reasoning. In fact, emotion serves as a central organising process within the brain, and our ability to organise our emotions directly shapes the ability of the mind to integrate experience and adapt to future stress (Siegel, 2012).

Second, learning is not a purely conscious process. Much of our most important emotional and interpersonal learning during the first few years occurs before we have developed the neurological capacities for conscious awareness and memory (Cozolino, 2016; Siegel, 2012). Thus, many of the most important aspects of our lives are controlled by reflexes, behaviours, and emotions learned and organised outside our awareness.

Third, the brain is not just skull-based, but 'embodied', being shaped by messages from all over the body via the central and peripheral nervous systems.¹ This embodied brain shapes and is shaped by both its external and internal environments (Barrett, 2011; Beilock, 2015; Claxton, 2015; Craig, 2015; Edelman, 2006; Johnson, 2006; Varela, Thompson & Rosch, 1991).

Finally, the brain is not a stand-alone bodily system, but is intricately connected to other major bodily systems, including the immune, endocrinal, metabolic, gastrointestinal, cardiovascular, enteric and musculoskeletal systems (Barrett, 2011; Beilock, 2015; Claxton, 2015; Damasio & Damasio, 2006; McFarlane, 2017; Mayer, 2016). These systems shape and are shaped by each other, and function as an integrated mind-brain-body system. This means that what is 'learned' in the prenatal and first two to three years of life affects not only the neurological system but also the other bodily systems to which the brain is connected, with potentially profound consequences over the life course (Moore, 2014a).

1 This is our second nervous system and contains all the nerves in the body that lie outside of the spinal cord and brain (the nerves that go from the skin, muscle, and organs to the spinal cord and eventually the brain). It conducts information to and from the central nervous system.

With this understanding in mind, we will now examine the evidence regarding the three key concepts mentioned earlier:

- Developmental plasticity and the developmental origins of health and disease (DOHaD) hypothesis
- Social climate change and the 'mismatch' hypothesis
- Ecological impacts on development and the social determinants of health and disease.

2.2 Developmental plasticity and the developmental origins of health and disease

2.2.1 Developmental plasticity

One of the most significant features of human biology is our capacity to adapt to different social and physical environments. This capacity is known as *developmental plasticity* (Bateson & Gluckman, 2012; Gluckman & Hanson, 2006; Gluckman et al., 2009; Gluckman, Hanson & Buklijas, 2010; Gluckman, Hanson & Low, 2011; Hanson et al., 2011; Low, Gluckman & Hanson, 2012; Padmanabhan, Cardoso & Puttabyatappa, 2016; West-Eberhard, 2003, 2005). While we retain some capacity to adapt throughout our lives, developmental plasticity is at its greatest in the first 1000 days or so of life (Barker, 2012; Gluckman et al., 2010), and it plays an important role in development from the moment of conception (Lane, Robker & Robertson, 2014).

Adapting to the immediate environment is the major developmental goal or activity during the first 1000 days and this developmental focus makes the influence of the environment particularly critical over this time. During development, there are brief *critical periods* during which a system or organ has to mature (Barker, 2012). These occur at different times for different systems, and they occur in utero for most systems. After birth, only the brain, liver and immune system remain plastic. Thus, much of human biological development is completed during the first 1000 days (Barker, 2012).

In the brain or central nervous system, it is more accurate to talk about *sensitive periods*, time windows during which the effect of experiences on brain development is unusually profound and can strongly shape the neural circuits (Ismail, Fatemi & Johnston, 2017). This is another instance of developmental plasticity, known as *neuroplasticity*, and refers to the biological capacity of the central nervous system to change structurally and functionally in response to experience, and adapt to the environment (Ismail, Fatemi, & Johnston, 2017). Neuroplasticity is greatest during pre- and postnatal brain development: the young brain has a repertoire of neuroplasticity responses that are not evident in adults, and which allow the young brain to develop appropriately and adapt constantly to environmental experiences and exposures.

This capacity to adapt makes the human species both versatile and vulnerable at the same time: the changes made might be adaptive for the immediate environment, but they can come with long-term costs, both psychologically and physically (Blair & Raver, 2012; Gluckman et al., 2009; Thompson, 2014). For instance, in the early development of the brain, neuroplasticity can lead to significant maladaptive outcomes depending on factors such as the nature and extent of adverse exposures, and the stage of neurodevelopment during which they occur. Patterns of abnormal neuroplasticity have been identified as core features of many paediatric disorders of the central nervous system, including cerebral palsy, intellectual disabilities, autism spectrum disorders, and neuropsychiatric disorders such as attention deficit hyperactivity disorder (Ismail, Fatemi, & Johnston, 2017).

2.2.2 Biological embedding

Adaptation involves a process variously known as *biological embedding* (Gluckman et al., 2010; Hertzman, 1999; Hertzman & Boyce, 2010; Nelson, 2013; Social Research Unit at Dartington, 2013), *developmental programming* (Barker, 2012; Lucas, 1991, 1998; Social Research Unit at Dartington, 2013; Thornburg, 2015; West-Eberhard, 2003) or *conditioning* (Hanson & Gluckman, 2014). It is through this process of biological embedding that the foetus (and infant), in response to cues such as nutrition or hormones, adapt their phenotype to their particular environment in ways that have life-long consequences.

Two central mechanisms underlie this adaptation process: *epigenetics* (whereby the 'genes listen to the environment') and *synaptic pruning* (whereby the 'brain listens to the environment') (Keating, 2016). In both cases, developmental experiences and the social context in which they occur have the capacity to become biologically embedded with lifelong impacts on health and other outcomes (Keating, 2016).

2.2.3 Epigenetic effects

The first key mechanism underpinning biological embedding and developmental programming is *epigenetic change* (Bilbo & Schwarz, 2012; Burris, Baccarelli, Wright, & Wright, 2016; Denburg & Daneman, 2010; Hertzman & Boyce, 2010; Keating, 2016; Kundakovic & Champagne, 2015; Moore, 2015; Szyf, McGowan, & Meaney, 2008; Szyf, Weaver, & Meaney, 2007). Contrary to common understanding, genes do not single-handedly determine any of our characteristics (Moore, 2015). Instead, development is a dynamic process that involves interplay between genes and the environment (Kundakovic & Champagne, 2014). Neither genes nor the environment have a direct and independent impact on development or functioning. Thus, a child may have a combination of genes that predisposes them to a particular condition or behaviour, but never develop the condition or behaviour because they were never exposed to the particular environment needed to trigger this condition – and the gene thus remains 'dormant'.

Similarly, a child may be exposed to a particular triggering environment but lack the genes that would predispose them to respond adversely to that environment. When genes and environment do interact, they result in epigenetic changes. These involve changes in how the genes function but do not alter the genes' DNA sequence – in effect, epigenetic changes determine whether genes are expressed or otherwise (turned on or off) (Carey, 2011; Duncan, Gluckman & Dearden, 2014; Francis, 2011; Lester, Conrard & Marsit, 2016; Moore, 2015).² This means that, rather than being born with a fixed genome³, we are born with a *developing genome* that changes in response to environmental context (Moore, 2015).

In humans, the epigenetic system is most sensitive to environmental influences during the period of developmental plasticity (i.e. the first 1000 days) because this is the time when epigenetic marks undergo critical modifications. Once a tissue or system is fully developed, while still somewhat plastic, it is less sensitive to alterations by environmental stimuli (Barouki et al., 2012).

Epigenetic changes have been implicated in the development of a wide range of disorders, from cardiovascular disease (Thornburg, 2015) to autism spectrum disorders (Loke, Hannan & Craig, 2015; Schanen, 2006) and cognitive disorders (Graff & Mansuy, 2009), and may be triggered by a wide range of environmental exposures and experiences (Coe & Lubach, 2008; Guyer et al., 2009; Gluckman & Hanson, 2005; Hertzman, 2010; Hertzman & Wiens, 1996; Keating, 2016; Martin & Dombrowski, 2008; Meaney et al., 2007; Robinson, 2013; Shonkoff, 2010).

² Moore (2015) suggests that the analogy of a dimmer switch more accurately captures this effect: DNA can be turned on 'a little bit, a moderate amount, a lot, full blast, or any amount in between'.

³ The genome is an organism's complete set of DNA, including all of its genes. Each genome contains all of the information needed to build and maintain that organism. In humans, a copy of the entire genome is contained in all cells that have a nucleus.

The pre- and peri-conceptual periods (before and just after fertilization) are highly sensitive periods (Chavatte-Palmer et al., 2016). Epigenetic changes can start to occur shortly after conception, and continue to occur during both the prenatal and postnatal periods. At conception, the egg and sperm combine their genetic material to form an embryo, whose set of genes reflects both the age and the environmental exposures of both parents (Lane et al., 2014). During fertilisation and the first cell divisions, the embryo is highly sensitive to signals from the mother's reproductive tract: the fluid that surrounds the embryo during its passage to the womb varies according to the mother's nutritional, metabolic, and inflammatory states, reflecting the particular world in which she lives (Leese et al., 2008). The embryo has a high degree of developmental plasticity, and responds to these environmental cues by modulating its metabolism, gene expression, and rate of cell division. In this way, the maternal tract and the embryo collaborate to generate a developmental trajectory adapted to suit the anticipated external environment, to maximize survival and fitness of the organism (Hochberg et al., 2011; Lane et al., 2014). But if the resulting phenotype is a poor match for conditions after birth, or if adaptation constrains capacity to withstand later challenges, offspring are at risk (Godfrey, Gluckman & Hanson, 2010).

Factors at conception that can affect the development of the embryo include maternal nutrition and infections. Poor maternal nutrition at conception can have a major impact on the developmental program, altering the rate of cell division and subsequent prenatal and postnatal nutritional development (Chavatte-Palmer et al., 2016; Lane et al., 2014). Maternal infections during this very early stages of development can also produce epigenetic changes, altering the immune functioning of the child (Lane et al., 2014).

Epigenetic changes can occur throughout pregnancy. Hormones and nutrients that cross the placenta can be affected by the mother's body composition, metabolism, and long-term lifestyle (Gluckman et al., 2009). The foetus is sensitive to hormonal and other physiological indicators of maternal stress, and heightened exposure to stress in the womb is associated with greater reactivity to stress after birth, as well as longer-term problems with emotional and cognitive functioning. In general, prenatal stress exposure makes children more reactive to challenge and threat (Thompson, 2014). In addition, maternal stress and toxin exposure during pregnancy, and maternal-infant interactions after birth have been linked to changes in the offspring's epigenetic state (Champagne, 2008, 2011; Skinner, Manikkam, & Guerrero-Bosagna, 2010; Thompson, 2014). Even natural variations in the quality or quantity of maternal care can have a long-term impact the offspring's brain and behaviour (Champagne, 2011). The prevalence of these effects suggests that epigenetic effects are a central mechanism by which environmental experiences, both positive and negative, become biologically embedded and 'get under the skin' (Keating, 2016).

Epigenetic changes can also occur during the postnatal period. For instance, stressors such as poverty in early childhood can alter the programming of the immune system (McDade, 2012; Miller et al., 2009, 2011; Miller & Chen, 2013; Raposa et al., 2014; Ziol-Guest, Duncan, Kalil & Boyce, 2012). Because the immunological system is developing during this time, changes get embedded in a manner that persists across the lifespan and makes the person more susceptible to diseases. The mechanism involved is the epigenetic modification of genes expressed in the brain that shape neuroendocrine and behavioural stress responsivity throughout life (Weaver, 2009). A harsh family climate - characterised by conflict, a lack of warmth, inadequate parenting, and household chaos - alters key immune cells and produces a chronic inflammatory state in the body (Miller & Chen, 2010; Miller et al., 2011a, 2011b; Raposa et al., 2014). Hormonally, early stress confers altered patterns of endocrine and autonomic discharge. Acting together with other exposures and genetic liabilities, the resulting inflammation promotes other pathogenic mechanisms that ultimately foster chronic disease (Miller et al., 2011).

There is now also evidence that epigenetic changes can be inherited. This means that the experiences of parents and even grandparents can be transmitted across generations and contribute to non-genomic transmission of disease risk across generations (Gapp & Bohacek, 2017; Gluckman, Hanson, & Buklijas, 2010; Gluckman, Hanson, & Low, 2011; Hansen et al., 2011; Heindel, 2015; Kundakovic & Champagne, 2015; Lane et al., 2014; Low, Gluckman, & Hanson, 2012; Moore, 2015; Wang, Liu & Sun, 2017). When parents have been exposed to adverse experiences (including nutrition, environmental toxins, abusive behaviour, and social stress) that have produced changes to their epigenome, these changes can sometimes be passed on to their children, with powerful effects on their physiological, metabolic and cellular functions (Gapp & Bohacek, 2017; Wang et al., 2017). While this is not a universal effect, when it does occur children receive genes that are in an active or 'switched on' state rather than a dormant or latent state. Thus, the long-term consequences of adverse environmental conditions during the first 1000 days may not be limited to one generation, but may lead to poor health in the generations to follow, even if these individuals develop in optimal conditions themselves (Roseboom & Watson, 2012; Wang et al., 2017).

Perhaps most damaging is when parents have experienced trauma, as a result of childhood abuse, family violence, war and so on. The impact of this intergenerational trauma on development during the first 1000 days is significant. The adverse impact of parental trauma can be transferred to the child through a range of influences, including: epigenetic changes resulting from the trauma exposure itself, the caregiver's mental health as a result of the trauma (Schwerdtfeger, Werner, Peters, & Oliver, 2013), and the co-morbidities (e.g. drug and alcohol abuse) that often result from trauma (Cohen, Hien & Batchelder, 2008). For example, parents of infants who have experienced sustained trauma are more likely to (amongst other things): use punitive, aggressive, and physical forms of discipline (Gara, Allen, Herzog, & Woolfolk, 2000; Appleyard & Osofsy, 2003); while children of parents who have experienced sustained trauma are more likely to (amongst other things) have insecure and disorganised attachment during infancy (which is also associated with depressive symptoms in childhood and later life) (Lee & Hankin, 2009; Hankin, 2005; Bosquet et al., 2014).

In addition to this, research has shown that long-term behavioural responses to stress and epigenetic alterations in adult children of traumatised parents can be facilitated by in utero effects (Boersma et al., 2014), variations in early postnatal care, and/or other early life experiences that are influenced by parental exposure to trauma (Yehuda et al., 2015; Champagne & Meaney, 2006). In a recent study of holocaust survivors, Yehuda and colleagues (2015) found that genetic modifications as a result of trauma are capable of being passed onto children, affecting subsequent generations.

Recently, another form of biological embedding - *telomere effects* - has been identified. This involves biologically embedding at a cellular level.

2.2.4 Telomere effects

Telomeres are the caps at the end of each of our chromosomes, and can be likened to the plastic tips at the end of shoelaces (Blackburn & Epel, 2012, 2017; Blackburn, Epel & Lin, 2015; Prescott, 2015) (See figure 1).

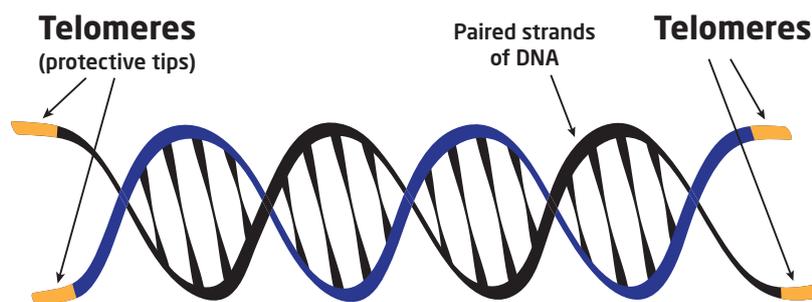


Figure 1. Telomeres (Telomerase Activation Sciences, 2011). <http://www.tasciences.com/what-is-a-telomere/>

Telomeres play a vital role in determining our health and longevity. Over the course of our lives, our cells divide numerous times, and the function of the telomeres is to stop the DNA from unravelling during this process. Our telomeres shorten with each division of our cells, and when they become too short, the cells stop dividing altogether, which causes our tissues to degenerate and ultimately die (Jaskelioff et al, 2011). These senescent cells (cells that no longer divide and grow) can also leak pro-inflammatory substances that make us more vulnerable to pain and chronic illness as we age. Thus, shortened telomeres not only shape our *health-span* (how long we live a healthy life), but also our *disease-span* (how long we live with disease that interferes with our quality of life) (Blackburn & Epel, 2017).

Telomeres are shaped by our genes, but also respond to how we live - the foods we eat, our responses to emotional challenges, the amount of exercise we get, whether we were exposed to childhood stress, and even the level of trust and safety in the neighbourhood (Blackburn & Epel, 2017). Chronic stress is known to be associated with shortened telomeres in adults, and evidence is accumulating that this is also true of such exposures early in life, and that the effect is dose-dependent (i.e. the more severe and sustained the stress, the shorter the telomeres) (Brown et al., 2009; Oliveira et al., 2016; Price et al., 2013). In a study of Romanian children, Drury and colleagues (2011) found that those who had been placed in institutional care for long periods before the age of five had significantly shorter telomeres than other children their age.

Cellular aging begins in the womb. Telomere length can be directly transmitted from mother to child at the point of conception: if the mother's telomeres are short throughout her body (including those in the egg) when she contributes the egg, the baby's telomeres will also be short - from the moment the baby starts developing (Blackburn & Epel, 2017). The developing child's telomeres can be further shaped by the mother's nutrition and stress levels during the pregnancy. Fathers can also transmit shortened telomeres, although not to the same extent as mothers (Blackburn & Epel, 2017).

There is a sense in which parents can not only transmit both epigenetic changes such as shortened telomeres, but also the environments that produced these changes:

Organisms always develop in specific contexts, and organisms that survive and reproduce will normally conceive the next generation in contexts like those in which they developed. In this way, individuals of the next generation are typically born into environments that are, in many ways, much like the environments their parents were born into; as a result, they then have similar experiences. And having inherited their parents' DNA *and* (in a sense) their parents' developmental environments, the offspring then typically develop the characteristics that helped their parents survive, doing so in the same way their parents did.

(Moore, 2015)

As Blackburn & Epel (2017) point out, this means that social disadvantage can be transmitted across generations: if the parents' telomeres were shortened by chronic stress, poverty, unsafe neighbourhoods, or chemical exposures, they can pass these shortened telomeres on directly to their children. As these children grow, they are likely to be exposed to poverty and stress, which will erode their telomeres further. They will pass these on to their own children, so that each new generation of babies has shorter telomeres than the previous one. Thus, Blackburn and Epel (2017) argue, 'From the first moments of birth, telomeres may be a measure of social and health inequalities.'

Fortunately, as we will see later, telomere shortening can be slowed, prevented or even reversed through exposure to positive environments, so the impact of early adverse experiences or inheritance can be counteracted.

2.2.5 Synaptic pruning

The second mechanism whereby environmental experiences become biologically embedded is *synaptic pruning* (Keating, 2016; Webb et al., 2001). While a baby is born with billions of brain neurons, it has relatively few synapses⁴. The initial surge in synaptic connections between brain neurons occurs after birth as the child goes through a process of rapid learning. During this period billions of neurons in the brain send electrical signals to communicate with each other, and it is these connections that become the foundation of brain development. Connections are strengthened through recurrent use, and our experiences and environment determine which connections are used most. Connections that are used more become stronger and enduring, while those that are not used become weak and eventually fade away through a process called *synaptic pruning* (Shonkoff & Phillips, 2000; Center on the Developing Child at Harvard University (CDCHU), 2016).

Well used circuits create pathways for strong connections in and between areas of the brain that are responsible for motor skills, sight, emotions, behavioural regulation, logic, language, and memory during the early critical period of development. Although different areas of the brain are responsible for each different function, they are all interrelated and one form of skill cannot completely develop without support from others. In other words, what comes first forms a foundation for everything that comes later (Sweatt, 2009; CDCHU, 2016). Building more advanced language, cognitive, social, and emotional skills on a weak foundation is significantly more challenging with age, even if a conducive environment is restored in later life (CDCHU, 2016). As circuits develop successively, different experiences are critical at different ages and if one stage is not developed appropriately, this will inevitably undermine the appropriate development of the next stage and so on (CDCHU, 2016).

The primary way that these brain connections are reinforced and strengthened is through the child's interaction with his/her care giver(s) through a process of 'serve and return' (CDCHU, 2016). Children pursue interactions through facial expressions, gestures, babbling, and words, and adults who are responsive 'return' these 'serves' with similar vocalising, gestures, and emotional engagement (CDCHU, 2016). However, if the caregiver's response is unreliable, inappropriate, or absent, the developing brain's architecture can be disrupted as a result of this under-stimulation. This then adversely impacts later stages of development, learning, behaviour and health outcomes (Reis, Collins, & Berscheid, 2000; Meaney, 2001; Champagne, Francis, Mar & Meaney, 2003; CDCHU, 2016).

2.2.6 Developmental origins of health and disease

What makes this new knowledge about developmental plasticity and programming so important is the evidence that experiences during the first 1000 days can have lifelong effects. This is the Developmental Origins of Health and Disease (DOHaD) hypothesis (Barouki et al., 2012; Gluckman & Hanson, 2004; Gluckman, Hanson & Beedle, 2007; Halfon et al., 2014; Heindel, 2007; Heindel & Vandenberg, 2015; Prescott, 2015; Rosenfeld, 2015; Rubin, 2016). This hypothesis maintains that environmental exposures to stress, undernutrition or environmental toxins during critical periods of development can have long-term effects on health and wellbeing by 'programming' organs, tissues, or body system structures or functions in ways that increase the risk of metabolic, cardiovascular, immunological, and neurobehavioral disorders, and even cancer.

⁴ A synapse is what allows information to flow from one brain cell (neuron) to another.

Our development from single cells to highly complex multi-celled organisms is an extraordinarily delicate and complex process, and can be disrupted by numerous factors, such as disease or environmental toxins (Davies, 2014; Prescott, 2015). As Prescott (2015) describes it,

During the months before our birth, most of our major structures and body functions are put in place. Small or subtle happenings in this period can have slow ripple effects that may not be revealed for many years. Key organs, such as the brain, the heart, the kidneys and the lungs are all formed in this period. In many cases the full quota of cells in these organs is fixed at birth (in the heart and kidney) or soon after (the brain and lungs). Even unnoticed, early adverse events might reduce the quota of heart-muscle cells, or the number of functional kidney units (nephrons), or the lung capacity that we are born with. And much of that is set for life. Once these organs are formed, we can't grow new heart muscle or nephrons, although stem-cell research is trying hard to overcome these biological limitations.

Protection for the foetus is provided by two protective barriers, the blood-brain barrier in the foetus itself and the placental barrier (Wong, Wais & Crawford, (2015). Both barriers develop during early pregnancy and act as filters to regulate the flow of specific nutrients and substances. The blood-brain barrier acts as a barrier to protect the development and function of neurons. These develop early: in the developing human brain, the growth of neurons begins in the embryonic period at about 6 weeks' gestation, peaks at 14 weeks, and is largely complete by 25 weeks. Meanwhile, the placenta acts as a selective filter for potentially harmful substances circulating in the maternal blood (Wong et al., 2015). Until relatively recently, it was thought that the foetus was completely protected from the mother's physical and emotional environment by these twin barriers (Gluckman et al., 2007; Paul, 2010). However, we now know that, while the placenta provides some protection against infection and maternal cortisol, there is free exchange between the embryonic and maternal blood systems, and the placental wall (which is thinnest in the first trimester when the foetus is developing most rapidly) does not protect the foetus against drugs, alcohol, smoking, environmental toxins or severe maternal stress (Coe & Lubach, 2008; Wong et al., 2015).⁵ As Donald (1979) memorably stated, 'The first 38 weeks of life spent in the allegedly protected environment of the amniotic sac are medically more eventful and more fraught with danger and accident than the next 38 years in the life span of most human individuals.'

⁵ This exchange between the embryonic and maternal blood systems provides opportunities for the prevention of disease – eg. adding folic acid during the early conception period to prevent spina bifida.

The DOHaD hypothesis maintains that the foetus not only registers these changes in the intrauterine environment, but also uses this information as a kind of 'weather forecast' from their mothers that prepares them for the type of world in which they will have to live, and alters its phenotype⁶ accordingly (Barker, 2004; Coe & Lubach, 2008; Gluckman & Hanson, 2005; Gluckman et al., 2009; Robinson, 2013). This process of *predictive adaptation* works in the interests of the foetus and infant when the antenatal and postnatal environments are both optimal and stable, as this ensures that any changes to the phenotype do not compromise later health and development. However, when these environments are less than optimal and when the prenatal and postnatal environments do not match, *predictive mismatches* (discussed below) can occur (Bateson, Gluckman & Hanson, 2014; Gluckman & Hanson, 2005; Prescott, 2015).⁷ Adult conditions such as coronary heart disease, stroke, diabetes, and cancer that once were regarded solely as products of adult behaviour and lifestyles are now seen as being linked to processes and experiences occurring in pregnancy or infancy (Heindel, 2007; Moore, 2014a).

By now, the evidence underpinning the DOHaD hypothesis is sufficiently robust for it to be considered a paradigm rather than a hypothesis (Hanson et al., 2011; Heindel & Vandenberg, 2015; Heindel et al., 2015; Prescott, Millstein, Katzman & Logan, 2016; Rubin, 2016). There is a flourishing field of research focusing on the developmental origins of health and disease, with its own professional associations internationally (International Society for Developmental Origins of Health and Disease) and nationally (DOHaD Society of Australia and New Zealand) (Prescott et al., 2016).

2.3 Summary

The brain is not a stand-alone bodily system, but is intricately connected to other major bodily systems, (including the immune, endocrinal, metabolic, cardiovascular, enteric and musculoskeletal systems) which shape and are shaped by each other. What is 'learned' in the prenatal and first two to three years of life affects not only the neurological system but also the other bodily systems to which the brain is connected, with profound consequences over the life course.

One key concept which captures how early life shapes lifelong health and development is *developmental plasticity*, and refers to our capacity to adapt to different social and physical environments. This capacity is at its greatest in the first 1000 days. During development, there are brief critical periods during which a system or organ has to mature. While most systems mature in utero, brain development occurs mostly in the first two years after birth and is strongly shaped by a child's social and physical experiences. This is a form of developmental plasticity, known as *neuroplasticity*, and refers to the biological capacity of the central nervous system to change structurally and functionally in response to experience, and adapt to the environment. While changes made might be adaptive for the immediate environment, they can come with long-term costs, both psychologically and physically.

Children are able to adapt to their environments because the foetus (and infant) respond to cues such as nutrition or hormones, by adapting their phenotype to their particular environment. This is called *biological embedding*. Two central mechanisms underlie this adaptation process: *epigenetic effects*, which refers to changes in the function of genes as a result of environmental factors; and *synaptic pruning*, which refers to the process of removing underused synapses in the brain, affecting synaptic connections in and between areas of the brain that are responsible for cognitive, social and emotional development.

6 Where the genotype represents the genetic makeup of an organism, the phenotype is the outcome of the interaction between the genotype and the environment, and is the organism's actual physical form and behaviour.

7 Other models of how the foetus adapts to changes in the intrauterine environment include the thrifty phenome hypothesis (Hales & Barker, 1992; Wells, 2011) and the maternal capital hypothesis (Wells, 2010, 2012). Regardless of the exact form that adaptation takes, the end results are the same – long term effects on developmental health and wellbeing.

Telomere effects are another way in which biological embedding occurs. Telomeres are the caps at the end of each chromosome and they play a vital role in determining our health and longevity. Over the course of our lives, our cells divide numerous times, and the function of the telomeres is to stop our DNA from unravelling during this process. Telomeres are shaped by our genes, but also respond to how we live and when they become too short, because of toxic stress or other adverse life factors, our cells stop dividing and can leak pro-inflammatory substances that make us more vulnerable to pain and chronic illness as we age. Telomere length can be directly transmitted from mother to child but can also be further shaped by the mother's nutrition and stress levels during the pregnancy.

What makes this new knowledge about developmental plasticity and programming so important is the evidence that experiences during the first 1000 days can have lifelong effects. This is the Developmental Origins of Health and Disease (DOHaD) hypothesis, which maintains that environmental exposures such as stress or undernutrition / overnutrition during critical periods of development can have long-term effects on health and wellbeing by 'programming' organs, tissues, or body system structures or functions. The DOHaD hypothesis maintains that this starts even before birth, where the foetus uses the intrauterine environment to 'predict' the type of world it will be born into, and alters its phenotype accordingly. This *predictive adaptation* is in the interests of the foetus and infant when the antenatal and postnatal environments are both optimal, however, when these environments are less than optimal and when the prenatal and postnatal environments do not match, *predictive mismatches* (discussed in section 3) can occur.

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The second major body of evidence reshaping our understanding of early development relates to the social and environmental changes that have occurred over the past half century or so, and the impact of these on health and wellbeing.

3. Global factors influencing health and development

3.1 Social climate change and its impact on families

Over the last several decades we have experienced a series of social, economic, demographic and technological changes that are unprecedented in their rapidity and scale. Dubbed the 'Great Acceleration' (McNeil & Engelke, 2015; Steffen et al., 2015), these changes have dramatically altered the conditions under which we are living (Friedman, 2016; Keeley, 2015; Li, McMurray & Stanley, 2008; Putnam, 2015; Silbereisen & Che, 2010; Trask, 2010; Wells, 2009) and the social and physical health problems we are experiencing (Kearns, Beaty & Barnett, 2007; Li et al., 2008; Palfrey, Tonniges, Green & Richmond, 2005).

These changes arise from the same fundamental factors that have contributed to global climate change and constitute a form of *social climate change* (Moore, 2014a) that has resulted in changes to the conditions under which families raise their children, as well as changes in families themselves (Bauman, 2011; Cassells, Toohey, Keegan, & Mohanty, 2013; Giddens, 2002; Golombok, 2015; Hayes, Weston, Qu & Gray, 2010; Parke, 2013; Richardson & Prior, 2005; Trask, 2010; Wells, 2009). For example, there have been dramatic changes in employment opportunities and conditions for families (Richardson & Prior, 2005), with more parents working full-time, in shift work, doing non-standard hours, working longer hours, more unemployed families, and more children being raised in poverty (Hayes et al., 2010; Richardson & Prior, 2005). Moreover, the search for cheap housing (Zhu, 2014) and secure employment has led to families moving away from the communities in which they were raised, leaving many families isolated and lacking supportive personal networks (extended family, friends or other families of young children).

Australian governments of all political persuasions have done (and continue to do) much to protect families from the adverse effects of these social and economic changes. Despite this, the problems persist. One of the main reasons that tackling this widening gap has proven to be so challenging is that the nature of the problems facing society and governments have altered - they are now more likely to be 'wicked' problems (Australian Public Services Commission, 2007; Head & Alford, 2008; Moore & Fry, 2011; Rittel and Webber, 1973; Weber & Khademian, 2008). Wicked problems are complex and intractable and cannot be resolved using traditional governance and leadership models, nor by service-driven approaches (Grint, 2010; Moore & Fry, 2011). Examples of wicked problems include child protection, family violence, Aboriginal disadvantage, social exclusion, health inequalities, entrenched poverty, and obesity. Some wicked problems (e.g. poverty and child abuse) while not new, have become more of a concern because of increased awareness regarding their adverse consequences on child development, and the complex nature of their underlying causes.

3.2 The mismatch hypothesis

Evidence is now accumulating that some of the physical and mental health problems that are now prevalent arise from, a mismatch between human evolutionary capacities and modern environments (Kearns et al., 2007; Lieberman, 2013a).

There are two forms of mismatch that can result in disease: predictive mismatch and evolutionary mismatch. As discussed in the previous section, *predictive mismatch* occurs when our bodies make adaptations based on predictions regarding the kind of environments we are going to be living in, and the environments do not match the predictions. *Evolutionary mismatch*, however, occurs when our bodies encounter conditions for which they were not evolutionarily adapted (Gluckman and Hanson, 2006; Gibson, 2009; Hanson and Gluckman, 2014; Lieberman, 2013). While many of the recent social and environmental changes that have occurred have been beneficial (e.g. greater availability of food, improved sanitation, and scientific medicine, leading to lower infant mortality and increased longevity), other changes have created conditions for which our bodies were not designed, with damaging effects upon our physical and mental health. Lieberman (2013) argues that the net effect of these changes has been to reduce or eliminate a number of the normal sources of stress which human bodies require for healthy development - removing extremes of heat and cold, feast and famine, exercise and rest. When we do not experience these normal sources of stress during development, then our bodily systems - including our metabolic, immune, endocrinal, cardiovascular and muscular-skeletal systems - fail to develop properly, leading to the emergence of mismatch diseases in adult life, if not before.

This mismatch between our evolutionary capacities and our modern living environments has led to a major change in the nature of the physical and mental health problems that people experience. Such issues are now far more likely to be chronic rather than acute conditions, and are known as *non-communicable diseases* (NCDs) (Prescott, 2015) or *mismatch diseases* (Lieberman, 2013). Over the last half century or so, there has been a huge growth in the incidence of these conditions. Nearly two thirds of deaths worldwide are attributable to NCDs (Bloom et al., 2011), and vast numbers of people are living with disabilities caused by mismatch diseases. According to the Australian Institute of Health and Welfare (2015a), about half of all Australians have a chronic disease, and around 20 per cent have at least two.⁸ Mismatch diseases account for the bulk of health care spending throughout the world (Lieberman, 2013), and are now seen as a major global threat to humanity, not only to our health, but to the social and economic advancement of all nations (Prescott, 2015; United Nations, 2011).

Mismatch environmental influences during the first 1000 days have been shown to affect our susceptibility to a wide range of the non-communicable diseases and conditions in later life (Balbus et al., 2013; Barouki et al., 2012; Gibson, 2009; Heindel et al., 2015; Lieberman, 2013; Prescott, 2015). They include allergies, immune and autoimmune diseases, neurodevelopmental and neurodegenerative diseases/dysfunctions, arthritis and osteoporosis, inflammatory bowel disease, some cancer types, infertility, changes in timing of puberty, depression, and psychiatric disorders such as schizophrenia (Barouki et al., 2012; Heindel et al., 2015; Prescott, 2015).

Other evidence suggests that mismatch diseases can also result from the impact that changed living conditions have had on the human microbiome.

⁸ These calculations are based on the incidence of eight chronic diseases: arthritis, asthma, back problems, cancer, chronic obstructive pulmonary disease, cardiovascular disease, diabetes and mental health conditions.

3.2.1 The role of the microbiome

Vast numbers of bacteria, viruses, and fungi (collectively known as the *microbiome*) live in and on the human body and play an important role in maintaining our health and wellbeing (Haahtela, et al., 2013; Blaser, 2014a; Collen, 2015; Dietert, 2016; Mayer, 2016; Relman, 2012). Microbes outnumber our human cells and therefore the genes that are transmitted from parents to infant are predominantly microbial⁹. It has been estimated that only 1 per cent of genetic transfer is human (Mayer, 2016), with the genes of microbes - the 'second human genome' (Relman, 2012) - making up the rest. Thus, rather than being a single stand-alone species, humans are more properly understood to be superorganisms made up of thousands of biologically diverse species (Dietert, 2016). The microbiome contributes significantly to individual differences between us: while humans are relatively homogeneous in their genetic makeup, we vary greatly in the composition of microbiomes, with only a third of the microbiome's constituent genes found in a majority of healthy individuals (Human Microbiome Project Consortium, 2012; Lloyd-Price, Abu-Ali, & Huttenhower, 2016).

The diverse ecology of microbes that make up the microbiome has coevolved with our species over millennia (Haahtela et al., 2013; Logan, Jacka & Prescott, 2016). These microbes provide us with essential services in exchange for being housed and fed. In particular, it is the bacteria in our gut that play a critical role in our physical and even our mental health (Blaser, 2014a; Haahtela, et al., 2013; Mayer, 2016). The beneficial functions they perform include helping digest food components that our guts cannot process (including essential elements in breastmilk), regulating our bodies' metabolisms, producing hormones, detoxifying dangerous chemicals we ingest with our food, training and regulating the immune system, and preventing the invasion and growth of dangerous pathogens (Mayer, 2016).

By virtue of its ability to confer an extensive set of protective and functional benefits to its human host, the gut microbiome can be considered a microbial or metabolic 'organ', and maintaining the proper health and functionality of this 'organ' is of significant importance (Huang et al., 2013). In short, it is the microbiome that helps keep us healthy (Blaser, 2014a). The brain, the gut, and the microbiome are in constant close communication, and function as parts of a single integrated system - the *brain-gut-microbiome axis* (Mayer, 2016). The first 1000 days are particularly crucial in shaping the architecture of this axis: both the brain and the microbiome are still developing, and changes during this period tend to persist for life. The consequences may not emerge until later in life, when the diversity and resilience of the gut microbiome decreases, making us vulnerable to degenerative diseases such as Alzheimer's or Parkinson's disease (Mayer, 2016).

Any change in the abundance, or composition or diversity of these micro-organisms can have significant health consequences. For instance, it may lead to failures to regulate and restore appropriate immune and inflammatory responses (Haahtela et al., 2013; Huang et al., 2013; Weng & Walker, 2013), which can contribute to chronic inflammatory conditions such as inflammatory bowel disease and asthma, and may even play a role in the development of conditions such as autism spectrum disorder, psychiatric disorders such as depression, and neurodegenerative conditions such as Parkinson's disease (Dash, Clarke, Berk & Jacka, 2015; Haahtela et al., 2013; Huang et al., 2013; Mayer, 2016; Vuong & Hsiao, 2017; Zheng et al., 2016). Since the traffic on the brain-gut-microbiome axis is two-way, our mental states can shape the composition of our gut bacteria. For instance, one study found that the infants of mothers who experience cumulative stress during pregnancy show marked disturbances in the composition of their gut bacteria, and subsequently have more health problems, such as infant gastrointestinal symptoms and allergic reactions (Zijlmans et al., 2015).

Disturbances of the composition of the microbiome - known as *dysbiosis* - can take several forms: a loss of beneficial microbes, an expansion of harmful microbes, or a loss of overall microbial diversity (Logan, 2015; Logan et al., 2016; Petersen & Round, 2014). Logan (2015) suggests that the conditions promoting dysbiosis are unequally distributed across society, with those living in socioeconomically deprived conditions where grey space (as opposed to green space) is the dominant environmental feature being more likely to be experiencing dysbiosis.

⁹ Recent estimates (Sender, Fuchs & Milo, 2016a, 2016b) suggest that, rather than vastly outnumbering the cells in the human body, the ratio of bacteria in the human microbiome to cells in the body is roughly 1:1. However, this does not include other microbes such as viruses, fungi and parasites.

The two sources of microbial exposure that are important for human health and development - environmental and human microbiota - have both become less diverse as a result of modern lifestyle changes. In addition, environmental changes such as urbanisation, higher exposure to chemicals and less exposure to green spaces, have reduced our exposure to a diverse range of plant, animal and microbial life. This has been linked with a range of mismatch diseases, including allergies, and Type 1 diabetes (Haahtela et al., 2013) and asthma (Huang & Boushey, 2015; Noval Rivas, Crother & Arditi, 2016). Our developmental health is also at risk because parts of our ancestral microbiome are disappearing (Blaser, 2014b). This is due to a range of factors, including overuse of antibiotics (in treating humans and in promoting the growth of the animals we eat) (Anderson et al., 2017; Hersh & Kronman, 2017), overuse of caesarean section births when not strictly necessary, the widespread use of sanitisers and antiseptics, and the shift to a Westernized high-fat high-carbohydrate high-fructose diet (Albenberg & Wu, 2014; Huang et al., 2013; Sonnenburg & Sonnenburg, 2014). As Mayer (2016) notes, it is easier to reduce gut microbial diversity in adults than it is to increase it above the level established in the first 1000 days.

Although the womb was thought to provide a sterile environment for the foetus, we now know that some bacteria are able to cross the placenta (Rodriguez et al., 2015), although we know very little about the nature and impact of microbes that do so. What we do know is that, from birth onwards, infants are rapidly colonised by a remarkably wide diversity of bacteria. In the case of the colonisation of the gut, the composition of gut microbiota in infants is markedly different from those in adults, but becomes progressively more adult-like as the infant acquires more microbes from the people around them, and reaches an adult-like form by the age of three (Yatsunenکو et al., 2012). Thus, the transition from no microbiota to an adult-like microbiome is all accomplished during the first 1000 days or so of life (Blaser, 2014a; Logan et al., 2016).

Just as the human epigenome is developmentally programmed by the early environment, so too is the human microbiome (Logan et al., 2016). In the postnatal period, microbial colonisation is influenced by factors such as gestational age, antibiotic exposure, delivery mode (caesarean section delays and later the establishment of the gut microbiome), breastfeeding, formula milks, timing and types of solid foods, and genetic factors (Logan et al., 2016; Rodriguez et al., 2015). The importance of acquiring a full complement of microbiota in the early years is captured in the *self-completion hypothesis* - which maintains that the single, most pivotal sign in distinguishing a life course of health versus that filled with disease is a successful and timely 'seeding' with an optimal complement of microbiota (Dietert, 2014; Dietert & Dietert, 2012; Dietert, 2016). There appears to be a narrow developmental window for effective seeding surrounding birth, and the completion of the full microbiome over the next two and a half to three years shapes their gut microbiome for a lifetime (Mayer, 2016; Wopereis et al., 2014). The immune dysregulation created by missing gut microbes during key periods of immune maturation can remain into adulthood (Dietert, 2014) and act as a biomarker of specific health risks (Dietert, 2014).

The microbiome evidence is another form of mismatch. The developing immune system appears to be particularly susceptible to modern environmental change, with the most common and earliest developing non-communicable diseases being immune-related conditions such as allergies (Prescott, 2013) and obesity (Segovia, Vickers & Reynolds, 2017).

3.2.2 Allergies

Australia has one of the highest rates of allergic diseases in the world (Prescott, 2015), with the latest generation of infants experiencing an epidemic of potentially life-threatening food allergies which were uncommon in their parents and rare in their grandparents. In just ten years there has been a five-fold rise in serious (anaphylactic) food allergies in pre-schoolers (Mullins, 2007). The 'Healthnuts' study (Osborne et al., 2011) of over 2,000 Melbourne infants found that more than 10 per cent of one year olds now have a food allergy. Even more have other allergic conditions, such as eczema. There also appears to be more severe disease, earlier onset and delayed resolution. Common food allergies, such as egg and milk allergy, which were previously transient in early childhood, are becoming increasingly persistent (Osborne et al., 2011).

Research shows that the very early postnatal period is a critical time for immune development and allergy prevention (Prescott, 2011). In the days following birth, there is an enormous influx of bacteria in an infant's gut, which stimulates the local immune system and the processes that prevent bacteria from 'infecting' or 'invading' (Prescott, 2011). A perfect balance develops where 'friendly' bacteria have a home and are fed by the infant; and in return, they ensure that he/she has a healthy immune system and that less friendly bacteria are kept away (Prescott, 2011).

Progressive modernisation and cleaner living appears to have altered the balance between humans and their friendly gut microbes (Prescott, 2011). This has been a strong element in the 'hygiene hypothesis', which proposes that there may be an association between the change in exposure to microbes and the increased incidence of allergies (Warner, 2003). Infants who go on to develop allergic disease are known to have lower levels of 'friendly' bacteria in the first week of life (Björkstén et al., 2001; Kalliomäki et al., 2001), and higher levels of disease-producing bacteria (Böttcher et al., 2000). Newer studies also show reduced diversity of the gut bacteria in infants who go on to develop allergic disease (Sjögren et al., 2009). Collectively, this evidence strongly suggests that the pattern of colonisation of bacteria in the first few weeks of life may influence the patterns of immune development in later life.

3.2.3 Obesity

A second major mismatch condition is obesity. Like diabetes and other non-communicable diseases, obesity is characterised by chronic low-grade inflammation, which in turn contributes to further disease progression, in part by changing homeostatic set points (such as insulin sensitivity or blood pressure) (Medzhitov, 2008). Obesity is a complex condition with multiple causes (Campbell, 2016). While not a disease in itself, obesity is a major risk factor for the development of Type 2 diabetes (Franks & McCarthy, 2016) and other adverse health outcomes. Childhood obesity is of significant concern given that it has been described as reaching epidemic proportions in recent times (Segovia et al., 2017; Young, Johnson, & Krebs, 2012), and because patterns of weight gain, metabolism, and even the total numbers of fat cells in our bodies are determined in early life (Prescott, 2015).

Some of the key prenatal influences on the development of childhood obesity include the mother's smoking habits during pregnancy (Oken, Levitan & Gillman, 2008); the mother's weight gain during pregnancy (Oken et al., 2007; Segovia et al., 2017); and the mother's blood sugar levels during pregnancy, particularly, whether she develops pregnancy-related (gestational) diabetes (Hillier et al., 2007; Harvard School of Public Health, 2016). However, environmental influences do not stop with birth. Instead, they simply shift from a small, confined space largely controlled by the mother's genes, lifestyle, and physiology, to an unbounded environment with equally influential effects (Harvard School of Public Health, 2016). There are three variable postnatal factors during infancy that impact weight in later life: how rapidly an infant gains weight, initiation and length of breastfeeding, and the duration of infant sleep. The first two of these factors (rapidity of infant weight gain and initiation and length of breastfeeding) are discussed in section 6.1.2 (Nutrition in infancy) of this report.

Research shows an association between restricted sleep and weight gain in adults (Singhal, 2007), and there is now reason to believe that a similar association may hold true for infants (Harvard University School of Public Health, 2016). In a study of 915 children, infants who slept fewer than 12 hours a day were twice as likely to be overweight at age 3, compared with infants who slept more than 12 hours a day (Demerath et al., 2009). Influences associated with shorter infant sleep duration include maternal depression during pregnancy, early introduction of solid foods (before 4 months), and infant TV viewing (Karaolis-Danckert et al., 2006). The mechanisms underlying the association between sleep duration and obesity are unclear (Taheri, 2006). However, studies in adults have shown that sleep restriction can lead to changes in the levels of certain hormones responsible for the control of hunger and appetite (Spiegel, Tasali, Penev & Van Cauter, 2004).

Several studies suggest that childhood obesity increases the risk of food allergies (Castro-Rodriguez et al., 2001; Gold, Damokosh, Dockery, & Berkey, 2003), while others suggest that allergic inflammation may impact metabolism (and increase the likelihood of obesity). In other words, the relationship between allergy and obesity may operate in both directions (Prescott, 2015). The connection between metabolism and the immune system occurs at many levels; hormonal interactions, sensing of nutrients by immune cells, and in the gut bacteria (Prescott, 2015).

Early life holds the keys to how and why allergies and obesity develop, and is the best opportunity to reverse the allergy epidemic (Prescott, 2011). Thus, in considering environmental factors that may be driving the rise in allergic disease, we must particularly consider their effects in pregnancy and the early postnatal period (Prescott, 2011). On the basis of the proposed interplay between our modern dietary patterns and cleaner environments, prebiotic and probiotic bacteria have both been used to prevent allergy in early life (Prescott, 2015).

3.3 Summary

Social and environmental changes over the past half century or so have resulted in a *social climate change* that has transformed the conditions under which families raise their children, as well as changes in families themselves. Dramatic changes to things such as employment and housing have significantly contributed to a rise in the conditions that are known to adversely impact child health and wellbeing, such as poverty and social isolation. To that end, it is becoming increasingly evident that the complex and entwined nature of these 'wicked' problems is such that traditional governance and service-driven approaches are no longer effective in driving change.

The drastic rise in some of today's most commonly occurring health problems is believed to be influenced by this social climate change, which has resulted in a mismatch between human evolutionary capacities and modern environments. This is referred to as the *mismatch hypothesis* and can be broken into two categories: *predictive mismatch* (which occurs when our bodies make adaptations based on predictions regarding the kind of environments we are going to be living in, and the environments do not match the predictions) and *evolutionary mismatch* (which occurs when our bodies encounter conditions for which they were not evolutionarily adapted or designed). This mismatch between our evolutionary capacities and our modern living environments has given rise to chronic physical and mental health conditions that are known as non-communicable diseases and conditions.

Mismatch diseases can also result from the impact that changed living conditions have had on our microbiome (the collection of bacteria, viruses, and fungi that live in and on our body). Our microbiome is critical to our health and is in constant communication with our gut and brain, all three of which function as an integrated system. Changes in the abundance, composition, or diversity of our microbiome can result in significant health consequences. The two sources of microbial exposure that are important for human health and development - environmental and human microbiota - have both become less diverse as a result of modern lifestyle changes, such as urbanisation and less exposure to green spaces.

Acquiring a full complement of microbiota in the first 1000 days is central to optimal health throughout the lifespan. This is known as *the self-completion hypothesis*. There appears to be a narrow developmental window for effective 'seeding' surrounding birth, and the completion of the full microbiome over the next two and a half to three years of life, which shapes the gut microbiome for a lifetime. Two of the most common and earliest developing non-communicable diseases/conditions are inflammatory conditions such as allergies and obesity.

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The third body of evidence that is reshaping our understanding of early development concerns the social determinants of health.

4. Social determinants of health

Our health and broader life outcomes are not exclusively based on our genetic or biological disposition (Hertzman et al., 2010; Bambra et al., 2010; Goldfeld et al., 2012). Rather, they are strongly shaped by the social, economic and environmental conditions into which we are born, grow, live, and age (WHO Commission on the Social Determinants of Health, 2008). These social conditions, known as the *social determinants of health*, ultimately work through biological pathways to shape our health and wellbeing. Key social determinants include (but are not limited to): socioeconomic status, educational attainment, employment status, poverty, geographic location, disability, gender, and social connectivity.

Our social determinants have the power to shape our lives for better or worse (Halfon, Larson & Russ, 2010; Marmot Review, 2010), unilaterally assigning us to a social standing that impacts our economic resources, status and autonomy (Moore, McDonald & McHugh-Dillon, 2014a; VicHealth, 2013). Social determinants play a critical role in the first 1000 days (Bronfenbrenner, 1979; Barker, 1994; Wadsworth, 1997; Hertzman, 2000; Shonkoff & Philips, 2000), as it is during this period that a number of vital skills and abilities develop (Moore, McDonald & McHugh-Dillon, 2015; Dyson et al., 2010; Hertzman, 2010; Strategic Review of Health Inequalities in England post-2010 Committee, 2010; Shonkoff, 2012).

Social determinants are often interrelated and/or co-existing. For example, we know that families who experience adversity often do so as a result of multiple determinants and in multiple areas of their life (Bromfield, Lamont, Parker, & Horsfall, 2010; Oroyemi et al., 2009). As such, social determinants should be understood as a set of correlating and interactive factors that may or may not lead to adversity within the family (Olsson & Hwang, 2003).

Social determinants are also transmitted across generations. Several researchers in different fields have observed that parents not only transmit genetic and epigenetic characteristics, but also the environments that produce those characteristics (Blackburn & Eppel, 2017; Lieberman, 2013; Moore, 2015). According to Blackburn and Eppel (2017), the evidence from telomere syndrome families suggests that it is possible for the effects of social disadvantage to accumulate over the generations. Social disadvantage is associated with poverty, worse health and shorter telomeres, and parents whose telomeres are shortened by this disadvantage may directly transmit those shorter telomeres to their babies in utero. Those children will be born with telomeres shortened by their parents' life circumstances. As these children grow up, they are also exposed to poverty and stress, and their telomeres will erode even further. In a downward spiral, each generation directly transmits its ever-shortening telomeres to the next.

Lieberman (2013) makes a similar argument about the spread of mismatch diseases, describing an insidious feedback loop that promotes the spread of these diseases. We get sick from non-infectious mismatch diseases caused by our bodies being poorly or inadequately adapted to the novel environments we have created, then we fail to address the novel environmental factors responsible for the mismatch, which then allows the disease to remain prevalent or sometimes to become more common or severe. Lieberman calls this phenomenon *dys evolution* and argues that this is not a form of biological evolution, because it does not involve a direct transfer of mismatch diseases from one generation to the next. Instead, it is a form of cultural evolution, because it involves a transfer of behaviours and environments that promote mismatch diseases in the next generation, that is, our children (Jablonka & Lamb, 2014; Lieberman, 2013).

4.1 Social gradient effects in health and wellbeing

Research shows that the lower one's social standing in life (e.g. persistent unemployment or chronic homelessness), the worse the long-term health and wellbeing outcomes are likely to be (Adler & Stewart, 2010; WHO, 2010). This global phenomenon is referred to as the *social gradient* in health and is evident from the very top of the socioeconomic spectrum to the very bottom. Social gradients represent more than just disparities between the poor and the wealthy, they also represent disparities within similar cohorts. That is, at any given point along the socioeconomic continuum, one is likely to experience inferior health outcomes to those above them (Shepherd, Li & Zubrick, 2012; Marmot & Wilkinson, 2006). For example, the Australian Socio-Economic Index of Disadvantage for Areas (SEIFA) illustrates a notable discrepancy between health outcomes according to level of disadvantage – with outcomes becoming progressively worse with increased disadvantage (ABS, 2010).

It is impossible to effectively address existing social gradients in health without first addressing the social determinants of health. One of the most significant social determinants of child health and wellbeing is poverty.

4.2 Poverty¹⁰

Research by the Australian Institute of Health and Welfare (AIHW) found that in 2013-14, approximately 70,000 Australian children received support from homelessness services (AIHW, 2010).

A significant body of evidence highlights the strong correlation between poverty in the first 1000 days and adverse health and wellbeing outcomes in later life (Goldfeld & West, 2014; Kruk, 2013; Hertzman et al., 2010; Marmot Review, 2010; Khanam et al., 2009). Evidence is now emerging of the effects of poverty on brain development (Barch et al., 2016; Hair, Hanson, Wolfe & Pollak; 2015; Luby et al., 2013; Luby 2015). One of these studies (Barch et al., 2016) found that areas of the brain responsible for learning, memory, and regulation of stress and emotions were connected to other parts of the brain in a 'weaker' way in children from low income families, as compared to children from higher income families. The level of this weakness varied according to the degree and length of poverty the child had been exposed to (Barch et al., 2016). These findings indicate there is a clear and coherent risk trajectory in which caregiving nurturance shapes the development of key brain regions in the context of poverty and that the development of these brain structures in turn shapes academic outcomes (Luby, 2015). Moreover, research shows that while children from high income families with developmental delays are likely to catch up to their peers in later life, children of low income families are much less likely to do so and in fact, the gap between them and their more affluent counterparts is likely to grow exponentially (Feinstein, 2003).

It is worth noting that while persistent poverty in the first 1000 days has a cumulative negative impact on development, prolonged poverty during later stages of life is less likely to have a significant impact on future life outcomes (Dickerson & Popli, 2012). Equally, relieving poverty (particularly in the first 1000 days) has been shown to increase birth weight and other outcomes, which can reduce the likelihood of negative outcomes in later life (Strully et al., 2010). For example, Costello and colleagues (2003) found that even a minor increase in income amongst families experiencing poverty resulted in decreased rates of childhood mental ill health (Costello, Compton, Keeler & Angold, 2003).

¹⁰ Here, the terms 'poverty', 'low income' and 'economic hardship' will be used interchangeably. Furthermore, the term 'financial distress' will be used to refer to the behavioural and emotional response that parents experience when facing economic hardship (Starkey et al., 2013).

4.2.1 Poverty in pregnancy

Starting from pregnancy, the experience of poverty begins to shape the unborn child's life. Women in the most economically disadvantaged areas of Australia are less likely to receive antenatal care in their first trimester of pregnancy, compared to women in the most economically advantaged areas (55 per cent versus 68 per cent) (AIHW, 2015a). Moreover, poverty in pregnancy is associated with increased use of tobacco, alcohol and other drugs; and poor nutrition and obesity – all of which have been shown to increase the likelihood of health and developmental vulnerabilities in children (Penman-Aguilar, Carter, Snead & Kourtis, 2013; Bailey, 2006; Weis et al., 2004; Conde-Agudelo, Rosas-Bermundez & Kafury-Goeta, 2006).

Poverty is also likely to increase a mother's exposure to psychological stressors, such as domestic violence and homelessness, which impacts the body's normal regulation of hormones during pregnancy and increases the likelihood of foetal growth delay and preterm birth (Weinstock 2005). Research shows that pre-term babies face an increased risk of various short and long-term health and well-being adversities (Luciana, 2003; Platt, 2014), such as depression, infectious and non-infectious respiratory problems, neonatal jaundice, epilepsy, cerebral palsy, visual impairments, cognitive impairment and developmental coordination disorder (Moore et al., 2015; Patton et al., 2004).

4.2.2 Poverty in infancy

The existing correlation between poverty in infancy and adverse outcomes in later life is not a random occurrence. Research into the influence of poverty on child development has primarily been guided by three key theories:

1. *That economic hardship can contribute greatly to psychological distress in parents and hence negatively impact their care-giving capacity (Conger & Conger, 2002).*

The relationship between financial distress and depression / anxiety is strongly supported by research (Green et al., 2005; Prawitz et al. 2006; Zimmerman & Katon, 2005; Coiro, 2001). A UK study by Green and colleagues (2005) found that families in the lowest income quintile were three times as likely than those in the highest income quintile to experience mental ill health (including depression and anxiety) (Green et al., 2005), and nine times more likely to experience a psychiatric disorder (Marmot, 2010).

Parental psychological distress is the primary connecting factor between economic hardship and less responsive and/or hostile parenting behaviours (Mistry et al., 2002; Solantaus et al., 2004; Parke et al., 2004; Gershoff et al., 2007). In a meta-analysis of 46 studies, Lovejoy and colleagues (2000) found a strong correlation between depressive symptoms in mothers and disengaged (e.g. withdrawn, ignoring) and hostile (e.g. mean and angry) parenting (Lovejoy, Graczyk, O'Hare & Neuman, 2000). Findings also showed that the effects of parental depression were strongest amongst low income mothers of infants.

When infants and young children interact with a disengaged or irritable caregiver, the anxiety that is created in them can cause their body to increase production of potentially harmful stress hormones (Dawson & Ashman, 2000; NSCDC, 2014). If this bodily reaction occurs often and over a sustained period of time, it can affect the child's brain development and interfere with his/her ability to learn and increase the likelihood of mental ill health in later life (NSCDC, 2005). A 20 year longitudinal study by Weissman and colleagues (2006) found that children of parents with depression were three times more likely to experience anxiety disorders, major depression, and substance dependence in later life (Weissman et al., 2006).

2. *That families with greater financial resources have the capacity to make greater investments in the development of their child, where disadvantaged families may only be able to invest in the child's more immediate needs (Bradley & Corwyn, 2002; Evans, 2004; Corcoran & Adams, 1997; Duncan & Magnuson, 2003; Linver et al., 2002).*

Investments in child development relate to degree of family support, access to learning materials available in the home and the family's standard of living (adequate food, housing, clothing, medical care, etc.). Research shows that children who experience poverty are less likely to live in cognitively stimulating environments, have less access to books, fewer age-appropriate toys, fewer informal learning settings, fewer educational materials, and spent more time in front of the television (Bradley & Corwyn, 2002; Duncan & Brooks-Gunn, 1997; Evans, 2004).

Linver and colleagues (2002) found that the correlation between family income and child cognitive development is noticeably reduced when investments (e.g. language stimulation, providing books and other learning materials, and exposing the child to learning experiences outside the home) are introduced to the child's everyday environment. Moreover, the level of parental investment was shown to significantly dominate the association between income and child behaviour problems at 3 and 5 years of age (Linver, Brooks-Gunn & Kohen, 2002).

3. *That children who are born into (and have a prolonged experience of) poverty are more likely to experience prolonged stress (Evans & Kim, 2012).*

Research shows that children who experience persistent poverty in the first 1000 days are more likely to display symptoms that are consistent with anxiety (higher blood pressure; irregular cortisol production; irregular metabolic activity; and poorer immune functioning) (Evans & Kim, 2012; Blair et al., 2011; Miller, Chen & Parker, 2011a). This is because children who live in poverty are more likely to have co-occurring exposure to family distress and separation, maternal depression, family and domestic violence, reduced parental responsiveness, and increased use of physical discipline (Bradley & Corwyn, 2002; Conger & Donnellan, 2007; Grant et al., 2003). They are also more likely to live in homes that are overcrowded; in neighbourhoods that are less connected and have less social supports; and be exposed to more toxins, crime and traffic (Evans, 2004; Evans & Kim, 2012; Evans & Kim, 2010; Sameroff, Seifer, & McDonough, 2004).

During the first 1000 days, the neural circuits responsible for managing stress are particularly malleable (NSCDC, 2014). A child's early experiences determine how these circuits are activated and controlled in the future. Prolonged and excessive toxic stress during this period can impact the developing brain circuits and hormonal systems in a way that leads to poorly controlled stress response systems; ones that are overly reactive or slow to shut down when faced with challenges throughout the lifespan (NSCDC, 2014; Loman & Gunner, 2010). For example, research shows that children who experienced persistent poverty in early life are more likely to exhibit non-adaptive coping strategies (disengagement and avoidance) in later life (Evans & Kim, 2012). Non-adaptive strategies are associated with higher levels of internalising (depression and anxiety) and externalising (aggression and impulsive) behaviours (Wadsworth & Achenbach, 2005; Compas et al., 2001).

Because children depend on their caregivers to meet their every need, the experience of poverty impacts them in multiple and concurrent ways, during the most critical period of their life. The quality of care that a child receives, the number and quality of learning opportunities it has, and the level and duration of stress that it experiences, are all significantly influenced by the experience and duration of poverty.

There is evidence for each of these theories, and the link between poverty in infancy and adverse outcomes in later life may be a product of all three pathways. While much has been learned, we have yet to fully understand the relative strength of these different influences or how they fit together. Further research is needed to settle these questions.

Aboriginal children experience poverty at significantly higher rates than their non-Aboriginal counterparts (ACOSS, 2014), and this is only one of a number of social determinants that inequitably affect Aboriginal people.

4.3 Social determinants and Aboriginal¹¹ health

Provided the necessary social conditions are in place, Aboriginal culture is a protective force for Aboriginal children, families and communities. Aboriginal parents have strong cultural practices in family life and child rearing, and know how to keep their children safe and to raise them to be active contributors to family and community life (Lohar, Butera & Kennedy, 2014). However, the effects of intergenerational trauma, cultural disconnection and family disruption among many Aboriginal communities, are increasingly being recognised as factors which can have significant adverse outcomes for some Aboriginal children (Council of Australian Governments [COAG], 2009).

Faced with all the evidence regarding the challenges confronting Aboriginal populations and the poor health and other outcomes they experience, it is easy to lose sight of the strengths of Aboriginal culture and the resilience of many Aboriginal communities and people. This may partly reflect a tendency to focus unduly on the negatives. As Nguyen and Cairney (2013) note, current government frameworks that collect data about Aboriginal people often focus on deficits, disadvantage and dysfunction. These frameworks gather statistical information for the purposes of policy analysis and program development and therefore use indicators that are important to policy, but lack a wellbeing perspective that recognises the strengths and resilience of Aboriginal and Torres Strait Islander people or reflects their worldviews, perspectives and values. The definition of wellbeing developed by the Social Health Reference Group for the National Aboriginal and Torres Strait Islander Health Council and National Mental Health Working Group (2004) highlights the importance of connection to land, culture, spirituality, ancestry, family and community, and how these affect the individual. As stated by Zubrick et al. (2014),

There are unique aspects of Aboriginal culture that can have a significant influence on Aboriginal health and that enables Aboriginal people to maintain spirituality central to the Indigenous notion of health. Connection to land, spirituality and ancestry, kinship networks, and cultural continuity are commonly identified by Aboriginal people as important health-protecting factors. These are said to serve as sources of resilience and as a unique reservoir of strength and recovery when faced with adversity, and can compensate for, and mitigate against, the impact of stressful circumstances on the social and emotional wellbeing of individuals, families and communities.

In the case of Aboriginal populations and children, the social determinants explanation of inequities in health outcomes is more complicated.

11 In this section, the term "Aboriginal" refers to Aboriginal and Torres Strait Islander Australians. Based on 2011 Census data, around 3 per cent of the Australian population (approximately 670 000 people) were estimated as being Aboriginal (Steering Committee for the Review of Government Service Provision, 2014). According to Boulton (2016), these fall into two demographic layers: approximately 80 per cent live in urban centres on the east coast, while the remaining 20 per cent live in small towns and remote communities across the tropical north of the country and through the Central and Western deserts. The health and developmental outcomes for these two populations can differ significantly.

The social determinants of Aboriginal health include (but are not limited to) social status, employment, poverty, housing, education, the experience of racism, and intergenerational trauma¹² (Gee et al., 2014; Muid, 2006). The available data indicates that all of these factors have a disproportionately large impact on the health and wellbeing of the Aboriginal population (ABS, 2011, 2012a, 2012b; AIHW, 2015b, 2015c; Gracey & King, 2009). Additionally, a growing body of evidence highlights a significant relationship between a robust affinity with traditional cultures and improved health and wellbeing outcomes amongst Aboriginal peoples (Colquhoun & Dockery, 2012; Wexler, 2009; Fleming & Ledogar, 2008). For example, applying data from the 2002 National Aboriginal and Torres Strait Islander Survey (NATSISS), Dockery (2009, 2010) found that greater attachment to traditional culture is associated with a range of improved outcomes for Aboriginal peoples (e.g. self-assessed health, educational attainment, employment status, incarceration rates and alcohol abuse).

Aboriginal children experience poverty at significantly higher rates than their non-Aboriginal counterparts (ACOSS, 2014). Furthermore, Aboriginal mothers are more likely to experience higher-risk pregnancies (including teenage pregnancy), with maternal mortality for Aboriginal women being double that of other Australian women (Humphrey et al., 2015; Chandler et al., 2003). Despite these alarming rates, only around half of Aboriginal women will receive antenatal care in their first trimester of pregnancy – compared with the national average of two-thirds (Harris & Wells, 2016). Aboriginal women are also more likely to experience domestic and family violence during pregnancy (Taft, Watson, & Lee, 2004) and use alcohol and tobacco during pregnancy (ABS, 2016). In 2015, almost four in ten Aboriginal children aged 0-3 years had a birth mother who had smoked or chewed tobacco during pregnancy, and about one in ten had a birth mother who drank alcohol during pregnancy (ABS, 2016). Aboriginal children continue to be over-represented in the out-of-home-care system: they are seven times more likely than non-Aboriginal children to have involvement with child protection services (AIHW, 2016) and eleven times more likely to be placed in out-of-home care (often with non-Aboriginal families outside of their community) (AIHW, 2015b). Evidence shows that these numbers are steadily increasing (AIHW, 2016).

Given the above, it is not surprising that Aboriginal children have some of the poorest health and developmental outcomes in Australia. On average, Aboriginal children have poorer outcomes than non-Aboriginal children on almost all standard indicators of wellbeing (Harris & Wells, 2016; Productivity Commission, 2016). Infants of Aboriginal mothers continue to die at almost double the rate of infants born to non-Aboriginal mothers and are twice as likely to be born with low birth weight (AIHW, 2015b; Holland, 2015).

In addition to this, Aboriginal children overall have lower levels of participation in Early Childhood Education and Care (ECEC) services, which has been shown to improve children's lifelong outcomes across all areas of health and wellbeing (Sims, 2011). However, despite the fact that research has identified a range of complex and co-occurring reasons for lower Aboriginal engagement with ECE (in particular, a dearth in the availability of culturally appropriate services and supports), the educational experiences of Aboriginal Australians is often framed from a 'deficit' viewpoint (where a lack of Aboriginal engagement with mainstream educational institutions is seen as the 'problem') (Krakouer, 2016).

Not surprisingly, Aboriginal children are twice as likely as their non-Aboriginal counterparts to be developmentally vulnerable across all developmental domains (Australian Government, 2015). Vulnerabilities at school entry track through to poor literacy and numeracy outcomes across all schooling years (Australian Curriculum Assessment and Reporting Authority, 2012; Falster et al., 2016). In older Aboriginal Australians, this trajectory of disadvantage is likely to lead to poor educational attainment and life opportunities, unemployment, poor health and premature mortality, and over-representation in the criminal justice system and out-of-home-care (Falster et al., 2016; Thomson, De Bortoli & Buckley, 2013; Weatherburn, 2014).

While we know that the 'traditional' social gradients in health contribute significantly to the disparities between Aboriginal and non-Aboriginal children, there are additional factors (unique to the experience of Aboriginal Australians) that also play a contributing role. The following sections will explore these in further detail.

¹² Resulting as an aftermath of colonisation (i.e. the stolen generation, disposition of land, culture and language, displacement and more).

Social gradients and Aboriginal health

As discussed, inequities in health outcomes are often reflective of inequities in social, economic and political status. However, the association between ‘traditional’ social determinants (e.g. education, housing, income etc.) and health are likely to be more complex for Aboriginal children for a number of reasons. One is that marginalisation and discrimination are often deeply embedded in the lives of Aboriginal people, limiting the likelihood of health benefits that are typically associated with improved income, education, etc. (Gracey & King, 2009). Another reason is that the generational marginalisation of Aboriginal people can impact optimal development in a number of ways, placing some Aboriginal children at a greater disadvantage from the beginning of life and limiting the acquisition of skills that can be drawn upon for the benefits of health at all levels of the gradient (Shepherd, 2012). Finally, kinship, spirituality, connection to traditional lands and cultural continuity play a central role in the health and wellbeing of Aboriginal Australians (Poroch et al., 2009). However, these factors are not captured in our ‘traditional’ understanding of the social determinants of health (Boulton, 2016; Currie, 2009; Muntaner, Eaton, Miech & O’Campo, 2004; Shepherd, 2012).

Boulton (2016) argues that understanding the origins of Aboriginal child ill-health requires a wider time frame. We need to understand that the immediate consequences of disadvantage – the social determinants of ill-health such as lack of education, unemployment, overcrowding and poverty – are far down the pathway of causality. In other words, these social determinants are themselves the result of far-distant historical factors. The historical and ongoing circumstances of colonisation and the profound and sustained marginalisation of Aboriginal peoples are credible explanations for a much less consistent social gradient within Aboriginal populations (Shepherd et al., 2012). Racism¹³, materialised in the marginalisation of a group of people, is acknowledged as having a detrimental impact on the health of Aboriginal (and other minority groups) throughout the world (Priest et al., 2012; Priest et al., 2011). Racism can impact health in a number of ways. Specifically: it can reduced access to optimal health determinants such as quality education and employment; diminish a positive self-identity; increase stress, substance misuse and self-harm; decrease access to social supports; and have detrimental effects on cultural identity (Paradies et al., 2009). Moreover, the experience of racism can restrict access to cultural activities, which are found to be protective factors for Aboriginal people’s overall health and wellbeing (Bals, Turi, Skre & Kvernmo, 2011).

The range of human rights violations that Aboriginal Australians have experienced (and continue to experience) as a result of colonisation is extensive. These include the effects of policies such as separation of Aboriginal children from their families (Australian Human Rights Commission, 1997). Unequivocal evidence supports that this has resulted in the experience of first hand and second hand trauma amongst Aboriginal families (Nutton & Fast, 2015; Royal Commission into Aboriginal Deaths in Custody, 1991; Waxler, 2009; Wesley-Esquimaux & Smolewski, 2004; Wilczynski et al., 2007). Ongoing firsthand (e.g. experiences of the child protection system) and secondary exposure to traumatic experiences induces intergenerational trauma (Atkinson, Nelson & Atkinson, 2010), which Muid (2006) defines as: “the subjective experiencing and remembering of events in the mind of an individual or the life of a community, passed from adults to children in cyclic processes as collective emotional and psychological injury over the life span and across generations” (Muid, 2006, p. 36). Much like culture, trauma (and its symptoms) can become ‘normalised’ in a population and transformed into a way of life (Duran & Duran, 1995). Intergenerational trauma closely correlates with social gradients, as the impact of trauma greatly imprints on all parts of a person’s life (and that of their children).

The interruption between family and community, characterised by breakdown of social norms (evident for example, in the increase of family violence and drug and alcohol abuse), is particularly related to adverse health outcomes amongst Aboriginal families and children (Stanley, Tomison & Pocock, 2003). The Aboriginal and Torres Strait Islander Social Justice Commissioner (ATSISJC) summarises this as ‘internalised colonialism’ or ‘lateral violence’ (ATSISJC, 2011), where rather than directing its anger toward its oppressor, the oppressed directs its anger internally – toward its own cultural group (Osborne, Baum & Brown, 2013).

13 Racism is conceptualised as comprising avoidable and unfair phenomena that lead to inequalities in power, resources and opportunities across racial or ethnic groups. It can be expressed through beliefs and stereotypes, prejudices and discrimination, and occurs at many social levels, including interpersonally and systemically, and as internalised racism (Berman & Paradis, 2010).

The social determinants of health, particularly relating to poverty and the systematic discrimination of Aboriginal Australians, help us to understand the foundations upon which disparities in health and wellbeing outcomes of Aboriginal and non-Aboriginal children are formed. While there is limited evidence which directly relates to the social determinants of Aboriginal children health (particularly in the first 1000 days), the fact that Aboriginal children are significantly more likely to be born with low birthweight, amplifies the early start and consequences of socioeconomic disadvantage.

Much work is underway to redress inequities between Aboriginal and non-Aboriginal children. For example, *The First 1000 Days Australia* model, led by Professor Kerry Arabena at the University of Melbourne, is building a coordinated, comprehensive, culturally informed strategy to strengthen Aboriginal families to address their children's needs. This model incorporates a research program that is premised on culture being the main protective factor in ensuring the health and wellbeing of Aboriginal families (Arabena, Howell-Muers, Ritte, & Munro-Harrison, 2015; Arabena, Ritte & Panozzo, 2016).

4.4 Summary

Our health and broader lifelong outcomes are strongly shaped by the social, economic and environmental conditions into which we are born, grow, live, and age. These conditions, which are almost always interrelated and co-occurring, are referred to as *the social determinant of health*. Starting in the first 1000 days, our social determinants have the power to shape our lives for better or worse, assigning us to a social standing that impacts our economic resources, status and autonomy.

The lower a child's social standing in life, the worse their health and wellbeing outcomes are likely to be. This is known as *the social gradient effect in health and wellbeing* and occurs from the very top of the socioeconomic spectrum to the very bottom. Social gradients represent more than just disparities between the poor and the wealthy, they also represent disparities within similar cohorts. It is impossible to effectively address existing social gradients in health without first addressing the social determinants of health. Poverty is perhaps the most powerful social determinant of child health and wellbeing.

A prolonged experience of poverty in the first 1000 days has been shown to adversely impact health and wellbeing outcomes over an entire lifespan. Poverty impacts a child's development in three primary ways:

1. It places significant psychological distress on a child's caregiver(s) and hence negatively impacts their care-giving capacity.
2. It makes it challenging for caregivers to make greater investments in the development of their child. Families who live in poverty are less likely to be able to afford or access learning materials, and their standard of living (adequate food, housing, clothing, medical care, etc.) is also more likely to be low.
3. It means that children are more likely to be exposed to ongoing traumatic experiences (such as homelessness, domestic violence and reduced parental responsiveness). Prolonged toxic stress during this period can impact the developing brain and hormonal systems, with lifelong consequences.

Children from low income families with developmental delays are less likely to catch up to their peers in later life. However, relieving poverty has been shown to increase birth weight and have other positive impacts on child health and development.

Aboriginal children are significantly more likely to experience sustained poverty than their non-Aboriginal counterparts, and have significantly higher adverse health and development outcomes. Vulnerabilities at school entry track through to poor literacy and numeracy outcomes across all schooling years, and eventually this trajectory of disadvantage is likely to lead to poor educational attainment and life opportunities, unemployment, poor health and premature mortality.

The association between 'traditional' social determinants (e.g. education, income etc.) and health are likely to be more complex for Aboriginal children due to a number of reasons, including marginalisation and the experiences of racism that are often deeply embedded in the lives of Aboriginal Australians. These experiences place Aboriginal children at a greater disadvantage from the beginning of life, and limit health and wellbeing outcomes at all levels of the gradient. Additionally, our 'traditional' understanding of the social determinants of health do not capture things such as kinship and connection to traditional lands and cultural continuity, which play a central role in the health and wellbeing of Aboriginal Australians.

The historical circumstances of colonisation and the profound and sustained marginalisation of Aboriginal peoples has resulted in the experience of first hand and intergenerational trauma amongst Aboriginal families. Much like culture, trauma (and its symptoms) can become 'normalised' in a population and transformed into a way of life. The interruption between family and community, characterised by breakdown of social norms (evident for example, in the increase of family violence), is particularly related to adverse health outcomes amongst Aboriginal children.

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What we have described so far has pertained to the biological and social mechanisms of development and change. However, these do not relate to the actual early life experiences that influence health and wellbeing outcomes. These are the subject of the next section.

5. Child, family, community and environmental factors shaping health and development

This section reviews some of the primary child, family, community and environmental factors that influence development in the first 1000 days. These include: child characteristics (such as temperament), the relational environment (such as parent engagement), and the physical environment (such as housing).

5.1 Child characteristics

5.1.1 Temperament

Temperament refers to individual differences in the regulation of experience which emerge early in life and remain moderately stable across development (Kagan, 2012; Lewis & Olsson, 2011; Rothbart, 1989, 2012). These distinct patterns of feelings and behaviours shape the child's affective, attentional and motor responses in various situations. For example, temperament can affect young children's mood and emotions, how they approach and react to situations, and their level of fear, frustration, sadness and discomfort (Encyclopedia on Early Childhood Development, June 2012; Rothbart, 1989).

The influence of temperament on developmental pathways and outcomes are now widely recognised (Rothbart, 2011; Rothbart & Bates, 2006). For example, Lewis and Olsson (2011) found that stressful family environments experienced in the infant's first year of life and high reactive, avoidant, and impulsive temperament styles directly and independently contribute to anxiety and depressive symptoms in children at 4 years of age. Children's temperamental traits show only modest stability during infancy and toddlerhood and show a rather large increase in stability by around 3 years of age (B. W. Roberts & DeVecchio, 2000), as executive attention develops further.

Biological foundation of temperament

The biological foundation of a temperamental bias (a bias towards certain temperamental characteristics) is usually, but not always, genetic (Kagan, 2012). In some cases it is the result of severe stress or infection in the pregnant mother which affects the foetus.

The behaviours in infants and young children that are most often attributed to a temperamental bias are unusually high or low levels of irritability, motor activity, smiling, ease of regulating these responses, and a consistent tendency to approach or to avoid unfamiliar people, objects, and places (Kagan, 2012).

Temperamental bias does not determine a particular future trait because life experiences affect the bias and create a pool of possible personality traits. By the second year of a child's life, the child's temperamental biases and the products of experience have integrated. This integration makes it difficult to detect the early temperamental biases of most children, because the same behaviour could be the partial result of a temperamental bias or the product of experience alone. For example, not all shy children inherit a temperamental bias favouring that quality (Kagan, 2012).

Interplay between temperament and environment

There is evidence to suggest that children differ in the extent to which they are influenced—for better and for worse—by their environmental experiences, due to ‘vulnerability’ in their make-up. This vulnerability may be behavioural (e.g. difficult temperament) or genetic (Bakermans-Kranenburg & van IJzendoorn, 2007, 2010, 2011; Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2007; Ellis et al., 2011). Children with such ‘risk’ characteristics are thought to be disproportionately affected by poor rearing influences, while more likely to benefit from highly supportive environments (Boyce & Ellis, 2005).

Hartman and Belsky (2015) found that temperamental and genetic characteristics played a role in distinguishing children that were either more or less susceptible to parental influences. In particular, two temperamental patterns have emerged as being indicators of heightened susceptibility to rearing: negative emotionality, and a highly sensitive personality (Hartman & Belsky, 2015). By contrast, less susceptible individuals are less affected by rearing conditions, whether they are positive or negative. For example, Belsky (2005) observed that highly negatively emotional (low adaptability, high activity, and low emotional regulation) infants were disproportionately more susceptible to rearing. This was also a quality that was identified as moderating the impact of early experiences. For example, quality of maternal discipline (gentle guidance versus forceful control) has been shown to influence substantially more variance in the self-control of infants and toddlers who display greater negative emotionality (Kochanska, 1993).

5.1.2 Differential susceptibility

The association between certain genetic profiles and increased susceptibility to environmental conditions (GXE) reveal that some children are more influenced by their environmental conditions than others as a function of the presence or absence of specific genetic characteristics (Caspi & Shiner, 2006). Specifically, “susceptibility genes” have been identified and shown to influence the risk for problematic developmental outcomes in the presence of adverse environmental conditions. For example, one study found that certain genetic characteristics moderate the relationship between child maltreatment and antisocial behaviour (Cicchetti, Rogosch, & Thibodeau, 2012), while another found this to be the case between maternal prenatal depression on child emotional dysregulation (Babineau et al., 2015).

In addition to moderating the effects of stressful environmental conditions, differential susceptibility has also been shown to moderate development in supportive environments. That is, children who are disproportionately vulnerable to adversity are also disproportionately more likely to benefit from highly supportive environments (Belsky et al., 2007; Boyce & Ellis, 2005). For example, in a longitudinal study of infants, maternal insensitivity when children were 10 months of age predicted externalising problems more than 2 years later, but only for children carrying the ‘risk’ gene (Bakermans-Kranenburg & van IJzendoorn, 2006). Moreover, while children carrying the “risk” gene displayed the most externalising behaviour of all children when mothers were judged insensitive, they also manifested the least externalising behaviour when mothers were judged as highly sensitive.

As we have seen, a child’s temperamental bias, coupled with early life experiences, can influence their development and lifelong outcomes for better or worse. While early life experiences are hugely significant for all children, for those who are disproportionately influenced by their environment, due to ‘risk’ characteristics, the nature and quality of their interpersonal relationships play a particularly vital role in determining their lifelong health and wellbeing outcomes.

The following section reviews the evidence relating to the role of family and caregivers in the development and wellbeing of a child, beginning in the first 1000 days, with continued effects across the lifespan.

5.2 Parental and family characteristics

As noted earlier, families have become much more diverse in their structure over the past decades, and many new family forms have emerged: lesbian mother families; gay father families; families headed by single mothers by choice; and families created by assisted reproductive technologies such as in vitro fertilisation, egg donation, sperm donation, embryo donation and surrogacy (Golombok, 2015; Parke, 2013). Concerns have been expressed about the possible impact of these new family structures on child development, but these have proven to be unfounded.

The research evidence shows that children in these various new family forms are just as likely as children in 'traditional' families to do well or have problems. What matters is not how the family is constituted but the quality of parenting, the child's own personal characteristics and, importantly, the social and physical environment in which they are raised (including societal attitudes). Research shows that the quality of family relationships and the wider social environment are more influential in a child's psychological development than are the number, gender, sexual orientation, or biological relatedness of their parents or the method of their conception (Golombok, 2015). While it is true that the psychological adjustment of children in single-mother families are more at risk of psychological problems than are children from homes where the father is present, this difference is largely accounted for by factors that often accompany single parenthood, such as economic hardship, maternal depression and lack of social support, as well as factors that preceded the transition to a single-parent home, such as parental conflict (Golombok, 2015).

5.2.1 Neurobiology of interpersonal relationships

As noted in the section on *Interplay between temperament and environment*, temperament-related behaviour and parenting behaviour influence one another, and are independently associated with child socio-emotional development. Children's self-regulatory difficulties are more likely to lead to externalising problems when parents use inconsistent discipline strategies or are low in firm discipline (Bates, Pettit, Dodge, & Ridge, 1998; Lengua, Wolchik, Sandler, & West, 2000).

The notion that infants with difficult temperaments may be more susceptible to the effects of parenting than infants with less difficult temperaments is consistent with the larger differential susceptibility hypothesis, which proposes that children may differ in the degree to which parenting qualities affect aspects of child development (Stright, Gallagher, & Kelley, 2008). For example, children who have high levels of fearfulness are less likely to have internalizing and externalizing problems if their parents are high in warmth and in gentle discipline strategies (Kochanska, 1997; Sentse et al., 2009).

A child's development is significantly shaped by the nature of their relationship or attachment with their primary caregiver during infancy (Benoit, 2004). A consistently responsive and nurturing relationship between the child and its caregiver encourages a secure attachment and facilitates the development of future relationships throughout the child's life, while providing a safe foundation for learning.

5.2.2 Parent-child attachment and parenting style

To learn effectively, children need to feel calm, safe and protected (US Department of Health and Human Services, 2001). When this attachment process is interrupted, the child's brain places an emphasis on developing neuronal pathways that are associated with survival, before those that are essential to future learning and growth (US Department of Health and Human Services, 2001).

Attachment research during childhood demonstrates that infants are born with a range of attachment behaviours that seek proximity to and safety in supportive others (attachment figures). In this view, proximity and safety seeking is a way for the child to maintain or increase its positive feelings and minimise or regulate its stress feelings and defensive states (Bowlby, 1988).

Although there are various sub-categories of attachment styles, they can essentially be broken down into two overarching styles: secure attachment and insecure attachment.

Secure attachment is defined by a sense of attachment security, comfort with closeness and interdependence, and confidence in support seeking and other positive ways of managing stress. This occurs in infants whose caregiver(s) respond to their distress in a consistent, caring, and timely manner (e.g. picking up and comforting the infant). In this instance the infant can feel secure in their knowledge that they can express negative emotion and prompt a comforting response from the caregiver (Van Ijzendoorn, Schuengel & Bakermans-Kranenburg 1999). Here, the infant's strategy for dealing with distress is 'organised' and 'secure' – they seek proximity to and maintain contact with the caregiver until they feel safe (Benoit, 2004). A secure attachment is aligned with enhanced developmental outcomes in later life in areas such as self-reliance, self-efficacy, empathy, and social competence (Goldberg, 2000).

Insecure attachment arises when caregivers are unavailable, unresponsive or unpredictable in responding to the child's needs, proximity seeking fails to relieve distress, and alternative strategies for emotional regulation (other than proximity seeking) are developed (Mikulincer et al., 2003). Extended separation from the attachment figure leads to a sequence of reactions: an initial intense activation of the attachment system (including crying for and searching for the attachment figure); despair and apathy; and detachment (including avoidance of the caregiver, even on their return). However, despite this apparent lack of concern, infants with avoidant attachment patterns have been shown to have more physiological arousal than other infants, indicating that they have learned to suppress their distress (Goldberg, 2000). Insecure attachments are often associated with an increased likelihood of developing social and emotional maladjustment in later life (Benoit, 2004).

Parenting style in the first 1000 days is central to establishing the child's attachment style (Kochanska, Coy, Tjebkes, & Husarek, 1998), and therefore overall his/her health and wellbeing outcomes in later life (Waldfogel, 2006). Parenting style relates to the emotional context that caregivers create to communicate with the child (Rodrigo, Byrne & Rodriguez, 2014).

The three primary parenting styles – affection or warmth; behavioural control; and psychological control – have each been associated with varying child social and emotional outcomes (Zarra-Nezhad et al., 2014). For example, warm, responsive and supportive parenting has been shown to promote the development of children's emotion regulation and social skills (Hart et al., 2003). Also parental behavioural control (e.g. setting limits and showing consistency in discipline) has been associated with adaptive child development and low levels of externalising problem behaviour (Barber, 1996; Hart et al., 2003). However, negative parenting characteristics, including strictness, neglect, control, punishment, and lack of support will potentially lead to subsequent child behavioural and emotional problems in later life (Sadiq Sangawi, Adams & Reissland, 2015).

5.2.3 Contribution of fathers/male caregivers

While research on parenting often focuses primarily on the role of mothers or families in general, growing evidence supports the critical and unique role of fathers/male caregivers¹⁴ in early childhood development (Cabrera, Shannon, Tamis-LeMonda, 2007). A 2008 systematic review found that father engagement positively impacted social, behavioural, psychological and cognitive outcomes in children (Sarkadi, Kristiansson, Oberklaid & Bremberg, 2008). Specifically, greater father involvement has been linked to: greater levels of cognitive and social competence; increased capacity for empathy; positive self-control and self-esteem; better interactions with siblings; and better academic progress (Wilson & Prior, 2011).

14 In the remainder of this section, fathers will be used to mean fathers and male caregivers.

Whilst both parents play important roles in the early development of child security and attachment, research suggests that some influences are more distinct among fathers or mothers. For example, evidence supports that fathers play a more prominent role in facilitating play exploration which fosters emotional and behavioural self-regulation, whilst mothers are more likely to provide comfort in times of distress (Lamb, 2002). Moreover, research indicates that fathers play a distinct (as in different to mothers) role in children's socialisation. Specifically, fathers who model positive behaviours such as accessibility, engagement and responsibility contribute to: better psychosocial adjustment; better social competence and maturity; and more positive child/adolescent-father relationships (Wilson & Prior, 2011).

Conversely, poor father-child relationships and fathering behaviours can have a lasting effect on a child's social adjustment and relationships, associated with inferior adult social functioning; significantly reduced likelihood of secure adjustment; and a significantly greater risk of avoidant or dependent attachment styles (Goodwin & Styron, 2012). Moreover, poor quality early father-child relationships have been associated with an increased likelihood of mental health disorders such as depression, bipolar, anxiety disorders and phobias (regardless of socio-economic status and perceived quality of childhood maternal relationship) in later life (Goodwin & Styron, 2012).

It is evident that a child's interaction with their caregiver is perhaps the most powerful determinant of their future health and wellbeing. Regardless of the diversity of family structures, what is most important is the quality of care that the child receives. This is true for all children, but particularly so for children with temperaments that make them more susceptible to the effects of parenting. When a child is exposed to persistent trauma in the first 1000 days, regardless of their temperament, they are significantly more likely to experience a lifetime of poor health and wellbeing. The evidence for this is discussed in the section below.

5.3 Adverse interpersonal relationships and sustained trauma

While it is clear that positive health and developmental outcomes for children depend on caregiving that is responsive, warm and consistent, the evidence is equally as clear that unresponsive and harsh or punitive parenting in the early days is likely to result in adverse health and developmental outcomes throughout the life course (WHO, 2011; Hertzman & Boyce, 2010; Hart & Rubia, 2012; McLaughlin, Sheridan & Lamber, 2014).

Over the last decades overwhelming evidence has established a strong dose-response (response that varies based on levels of exposure) between exposure to adverse early experiences such as abuse and neglect, and increased likelihood of: cognitive and language difficulties, lower educational attainment, unemployment, poverty, homelessness, becoming victims or perpetrators of violence in later life, early mortality, heart disease, diabetes, liver disease, cancer, depression, anxiety, eating disorders, obesity, and suicide (Liu et al., 2013; Sethi et al., 2013; Fortin et al., 2014; Hart, Smith, Gruer & Watt, 2010).

5.3.1 Child abuse and neglect: an Australian snapshot

In 2014-15, 1 in 35 Australian children received child protection services, 73 per cent of whom were repeat clients with at least one previous child protection involvement (AIHW, 2016), highlighting the sustained nature of abuse and/or neglect in the overwhelming majority of cases. Infants (under the age of one) were the most likely age group to receive child protection services across Australia, while children who live in the most disadvantaged areas of Australia (an overwhelming majority of whom are Aboriginal) are also more likely to receive child protection services than any other cohort (AIHW, 2016).

There has been a consistent increase in the number of notifications and substantiations over the last 5 years, with a 6 per cent increase in the last 12 months alone (AIHW, 2016). Alarming, research shows that these figures are likely to under-represent the number of child maltreatment (including fatal) incidents (Schnitzer, Gulino, & Yuan, 2013; Sheldon-Sherman, Wilson, & Smith, 2013).

5.3.2 Adverse early life experiences and poor lifelong outcomes: the linking mechanisms

There are three key linking mechanisms through which sustained exposure to abuse and/or neglect increase the likelihood of ill (physical and mental) health and early mortality:

1. *It disrupts the progression of critical developmental processes (namely the stress response and brain development).*

The experience of toxic stress in the first 1000 days can result in significant harm to the body's stress response and cause the continuous production of stress hormones (Loman & Gunner, 2010). This ongoing 'wear and tear' on the body's stress response can seriously harm development, and ultimately health and well-being outcomes, in a number of ways (Centre on the Developing Child, 2012; Cicchetti & Rogosch, 2001; Andrews & Neises, 2012). Changes to the body's stress response can increase the risk for physical ill health, such as asthma, hypertension, heart disease and diabetes (Swanson, Entringer, Buss & Wadhwa, 2009), and has been linked to depression, anxiety, and disruptive behaviours in later life (Alink, Cicchetti, Kim, & Rogosch, 2012).

Similarly, chronic activation of the stress response can have concurrent adverse impacts on other regulatory systems, including the immune system. Studies have found that chronic childhood trauma is associated with excessive immune cell production, which can enhance the production of inflammatory cells, associated with increased feelings of anxiety (Reichenberg et al., 2001; VanZomeren-Dohm et al., 2013). High levels of inflammatory cells can also cause damage to the brain by decreasing brain growth, resulting in potential mental ill health and developmental delays (VanZomeren-Dohm et al., 2013; Andrews & Neises, 2007; Woods et al., 2005; Reichenberg et al., 2001).

Moreover, areas of the brain that are affected by high levels of immune cells are also involved in the regulation of anxiety and interpretation of fear responses, fear memories, and the recovery of traumatic memories (VanZomeren-Dohm et al., 2013). As such, disruptions to brain development during critical periods of development can lead to a disrupted fear response and ultimately behavioural dysregulation, common in traumatised children (VanZomeren-Dohm et al., 2013; Yehuda & LeDoux, 2007).

Trauma also impacts the child's developing brain. As we saw in the section on *Synaptic pruning*, a child's brain architecture can be dramatically disrupted as a result of under-stimulation (due to neglect) and also lead to epigenetic changes that interrupt the appropriate development of systems that manage the child's stress response in later life and can result in increased risk of adult ill health (Szyf, 2009; Bagot et al., 2009; CDCHU, 2016).

2. *It impacts the way in which children relate to and interpret the world around them.*

Prolonged exposure to neglect and/or abuse can cause a child to become more aware of and sensitive to stressors in their environment, where stressors become more prominent and/or threatening than they are likely to be (VanZomeren-Dohm et al., 2013). Research shows that children who are exposed to chronic abuse and/or neglect become hypervigilant toward perceived environmental threats and are more likely to interpret a neutral situation as hostile (Pollak & Kistler, 2002; Pollak, Cicchetti, Hornung & Reed, 2000). While the ability to detect a violent parent's anger will help the child know when to avoid contact with the parent, making it an adaptive tactic for survival, studies have shown that threat recognition comes at the expense of other emotions (Pollak & Kistler, 2002; VanZomeren-Dohm et al., 2013). This is because the disproportionate allocation of attention and cognitive resources used for threat detection leads to reduction in the ability to process and understand other emotional states in later life (Pollak et al., 2000).

The long-term impact of this is significant and includes an increase in the likelihood of engaging in disruptive social relationships and behaviours in later life (Kim-Spoon, Cicchetti & Rogosch, 2012; VanZomeren-Dohm et al., 2013). For example, abuse in early childhood has been shown to increase the likelihood of adult criminal behaviour by 28 per cent and violent crime by 30 per cent (Widom & Maxfield, 2001). This disruption in the child's response to stress is also likely to contribute to the development of adverse risk behaviours that lead to adverse health and well-being outcomes in later life (VanZomeren-Dohm et al., 2013).

3. *It is likely to result in the development of negative risk behaviours in later life that lead to an increase in the likelihood of risk factors that undermine health and well-being outcomes.*

The strong correlation between certain lifestyle factors that are known to significantly increase the likelihood of adult morbidity and mortality (known as health risk behaviours) has been widely established (Fortin et al., 2014; Hart, Smith, Gruer & Watt, 2010; Anda, Butchart, Felitti & Brown, 2010; Campbell, Rebekah, Walker & Egede, 2016). Such risk factors relate (but are not limited) to: smoking, obesity, high-risk sexual behaviours, unintended pregnancy, alcohol and drug abuse, and perpetration of violence (Fortin et al., 2014; The Center on the Developing Child, 2010).

A significant body of evidence demonstrates that a leading cause of engaging in such adverse health risk behaviours is sustained exposure to abuse and neglect in early childhood (The National Scientific Council on the Developing Child, 2010; Felitti et al., 1998; Campbell, Rebekah, Walker & Egede, 2016; Huang et al., 2015; Ng, Skorupski, Frey & Wolf-Wendel, 2013). In their landmark study, Felitti and colleagues (1998) found that risk behaviours such as alcohol and drug abuse were used for their immediate physiological or psychological benefits as a coping device and that when such behaviours were adapted as a coping mechanism, they tended to be used chronically, further enhancing their negative health impact (Felitti et al., 1998).

The correlation between abuse and neglect in early childhood and adverse health risk behaviours are echoed by other such studies (Mersky, Topitzes & Reynolds, 2013; Fortin et al., 2014; The National Scientific Council on the Developing Child, 2010) that have found a significant increase in the number of risk factors as a result of prolonged exposure to one or more adverse childhood experiences (ACEs). Evidence also shows that prolonged exposure to traumatic experiences in childhood is associated with increased risk for self-destructive behaviours such as self-mutilation and suicide attempts (Corcoran et al., 2006; Read et al., 2001; Widom, 1998).

Evidence surrounding the lifelong negative impacts of adverse early life experiences is significant. While traumatic early life experiences are often the result of abuse and neglect, one of the most influential factors that increase the likelihood of child abuse and neglect are family and domestic violence. This is discussed in the sections below.

5.4 Impact of family and domestic violence

Overwhelming evidence supports the correlation between children's exposure to family and domestic violence and the increased likelihood of adverse lifelong outcomes (Humphreys, 2008; Margolin & Gordis, 2000; Zeanah et al., 1999; Mathias, Mertin, & Murray, 1995; Cummings & Davies, 1994). Marshall and Watt (1999) found that interpersonal conflict was the strongest risk factor for behavioural problems, significantly associated with externalising and internalising behaviours and social and attention problems, when children were assessed at the age of five. They also found that more frequent and intense episodes of conflict increased the likelihood of childhood behavioural problems. But how early in a child's life is exposure to family conflict and violence likely to have an impact?

5.4.1 Domestic violence in pregnancy

Pregnancy is recognised as a period of high risk for the onset or worsening of domestic violence (Taft 2002; WHO 2000). Pregnant women who are victims of domestic violence are more likely to experience all forms of violence including physical assault (Catalano, 2013), sexual assault (WHO, 2011) and psychological aggression (Martin, et al., 2004). Violence during pregnancy can also be more extreme: women who experience domestic violence during pregnancy are likely to be hit in the abdomen (WHO, 2011); and likely to be hit more frequently (Martin et al., 2004), posing significant risk of harm to the mother and unborn child.

High levels of maternal stress can also result in an increase in the mother's cortisol production which can enter the foetus's brain via the placenta and the umbilical veins (Sandman et al., 1999). Although the placenta can inactivate a proportion of maternal cortisol, sustained stress adversely affects the growing brain. Maternal anxiety during the earlier part of the prenatal period is associated with lower birth weight, shorter gestational age and smaller infant head circumference at birth, suggesting a decrease in brain growth due to high levels of prenatal maternal stress (Lou et al., 1994).

Studies have also found a strong correlation between domestic violence during pregnancy and poor emotional regulation and academic outcomes in school (Durand, Schrailber, Franca-Junior & Barros, 2011); behavioural problems during infancy (Flach et al., 2011); poor maternal attachment (Quinlavin & Evans, 2005); an increase in internalising problems from as early as 24 months (McFarlane et al., 2014); and aggressive behaviours at school (Durand, Schrailber, Franca-Junior & Barros, 2011).

5.4.2 Domestic violence during infancy and early childhood

Research shows that abuse during pregnancy strongly predicts abuse immediately following birth (Huth-Bocks, Levendosky & Bogat, 2002). One study found that 95% of women who were subjected to violence during pregnancy were also subjected to violence within the first three months postpartum, with 52% requiring medical care for injuries sustained as a result of domestic violence (Stewart, 1994).

Witnessing violence can be extremely distressing even for infants. Threats to a caregiver is one of the most psychologically destructive traumas for children (Scheeringa & Zeanah, 1995). Infants who hear or witness anger and/or violence, or a parent being hurt can show symptoms of Posttraumatic Stress Disorder (PTSD), including eating problems, sleep disturbances, lack of typical responses to adults and loss of previously acquired developmental skills (Bogat et al., 2006, De Bellis & Thomas, 2003, Schore, 2001). Infants and toddlers who witness adult verbal conflict or violence against a family member, and/or whose mothers experience domestic violence during pregnancy are also more likely to demonstrate increased externalising behaviours (lower levels of social competence, difficulties with peer relationships, aggressiveness, and disruptive behaviour) in later life and greater child adjustment difficulties (Levendosky et al., 2006; McDonald et al., 2007).

The degree to which domestic violence affects parenting capacity is also likely to influence emotional outcomes in children exposed to domestic violence (Graham-Bermann & Levendosky, 1998). During times of distress, an infant will seek the protection and proximity of his caregiver by applying strategies that will gain the caregiver's attention. An infant's ability to self-regulate emotions during times of stress, anger or trauma can be significantly compromised in the long run if a caregiver does not appropriately respond to this need (Kaufman & Henrich, 2000).

There is overwhelming evidence that exposure to family and domestic violence during infancy and early childhood can negatively impact brain development, and ultimately other domains such as emotional regulation (Carpenter & Stacks 2009; Laing 2000; McIntosh 2003; Perry 2005). The impact of chronic stress and trauma on the developing brain has been discussed in the section on *Adverse interpersonal relationships and sustained trauma*.

As we have seen, the experiences that a child has within their familial environment, including the quality and nature of care they receive, and the people and things they are exposed to (as a direct result of their caregiver's actions and/or lifestyle) plays a fundamental role in shaping their future health and life outcomes. However, while quality of care is pivotal to the child's wellbeing, it is not the only factor that helps shape future outcomes. The community (including social supports) that a child grows up in and their physical environment (such as their housing conditions) are also extremely important in shaping their future health and wellbeing.

5.5 Community environments

Over the past few decades, communities in Australia and other developed nations have been steadily fragmenting, and people's sense of community has fragmented also (Barnes, Katz, Korbin & O'Brien, 2006; Hughes et al., 2007; Leigh, 2010). Nowadays, there may be little sense of community tied to locality, particularly in larger urban centres. There is also less trust and reciprocity, and more concerns about personal safety (Leigh, 2010).

There are many reasons for this fragmentation (Blau & Fingerman, 2009; Leigh, 2010). These include a partial erosion of traditional family and neighbourhood support networks, due to factors such as increased family mobility and the search for affordable housing. The continued population growth combined with the steady shift to cities is outstripping the capacity of cities to provide the basic physical and social infrastructure to support families adequately. But there are also factors such as increases in the speed and ease of transport and of communication methodologies that have enabled people to have contacts with much more widely spread social networks and reduced their reliance on people in their immediate neighbourhoods (Hughes et al., 2007; Leigh, 2010; Wellman, 2001).

These changes in communities are important because both the social and physical environments of a community are known to have an impact on people's health and wellbeing (Barnes, Katz, Korbin & O'Brien, 2006; Edwards & Bromfield, 2009; Goldhagen, 2017; Pebley & Sastry, 2004; Pinker, 2015; Popkin, Acs & Smith, 2010; Sustainable Development Commission, 2009). There is evidence that our immediate social networks – those people we mix with on a regular basis – have a significant influence on our ideas, emotions, health, relationships, behaviour, and even our politics (Christakis & Fowler, 2009; US Department of Health and Human Services, 2011). Even 'consequential strangers' – people outside our circle of family and close friends, such as casual acquaintances – are important for personal and community wellbeing (Blau & Fingerman, 2009).

5.5.1 Social supports¹⁵

Social supports have a particularly significant role during periods of stress or major life transition.

Social supports during pregnancy

Pregnancy is a time of significant life change and requires significant psychological adjustment and support (Robles & Kiecolt-Glaser, 2003; Elsenbruch et al., 2007). The perception and experience of insufficient support has a visibly detrimental effect on not only maternal psychological wellbeing, but also adverse health and wellbeing outcomes for the child (Dibaba et al., 2013; Grote et al., 2010; Dunkel Schetter & Lobel, 2011; WHO, 2009).

15 The terms 'social supports', 'social connections' and 'social relationships' are used interchangeably and refer to three categories of family support: practical (having someone who can offer a lift, or help look after your child, etc.); emotional (having someone who will listen and provide emotional comfort and reassurance, particularly during a stressful situation, etc.); and advice and information (having someone to contact who can help and/or provide advice if the baby is not feeding/sleeping, etc) (McArthur & Winkworth, 2016).

Experiencing social support during pregnancy reduces the likelihood of maternal stress, depression and risk taking behaviours during and after pregnancy (Kawachi & Berkman, 2001; Rini et al., 2006; Robles & Kiecolt-Glaser, 2003;). Social supports during pregnancy can vary based on the mother's particular needs during her pregnancy, labour, delivery, and the postpartum period (Gjerdingenm, Froberg & Fontaine, 1991). For example, informational supports (such as prenatal classes) are associated with reduced physical pain and complications during labour and delivery (Firouzbakht, Nikpour, Khafri, 2014; Gjerdingenm et al., 1991), while support during labour and delivery is associated with the shorter duration of labour, less use of pain medications, and fewer caesarean sections and instrumental deliveries (Hodnett et al., 2011).

Moreover, studies have shown that the level of social supports during pregnancy can impact a woman's protective behaviours during this period: A German study by Elsenbruch and colleagues (2007) found that 33 per cent of women who had low social supports smoked during their first trimester of pregnancy compared to only 17 per cent of women who reported having high social supports (Elsenbruch et al., 2007).

Social supports in infancy

Parental social supports play a significant role in the health and wellbeing of children in a number of ways. It facilitates the child's contact with other caring adults and helps build positive attachment relationships, and plays a significant role in modelling relational skills for children (US Department of Health and Human Services, 2011). Social support also greatly affects parental care-giving capacity by promoting positive mental health and resilience during challenging periods (Green, Furrer, & McAllistar, 2007; Palamaro et al., 2012).

Importantly, positive social support reduces the likelihood of child maltreatment (Bishop & Leadbeater, 1999): the risk of child maltreatment increases when parents (particularly those who are experiencing concurrent vulnerabilities such as poverty, depression, unemployment etc.) have limited social supports (MacLeod & Nelson, 2000). Finally, social support helps families to access family and/or early intervention services (Kang, 2012). This is in part due to the notion that without adequate social networks, the opportunity to be 'introduced' to services may be limited (Winkworth, McArthur, Layton, & Thompson, 2010; Winkworth et al., 2010).

5.6 Physical environment¹⁶

Research continues to demonstrate the direct (cognitive, social, emotional, and biological outcomes) and indirect (parent's caregiving capacity) impact of physical environments on children's development (Evans, 2006; Evans & Hygge, 2007; Evans & Lepore, 1993).

5.6.1 Housing¹⁷

Access to stable and adequate housing is a basic human need (Maslow, 1948). It has a significant impact on the health and wellbeing of families and children as it provides a safe environment, autonomy, and security which is needed for full participation in social, educational, economic, and community life (AIHW, 2010; Vic DHS 2006; Wise 2003).

¹⁶ Physical environment refers to factors such as housing, access to parks and safe places for play, access to green spaces and natural environments and level of road traffic at the expense of pedestrian safety and comfort.

¹⁷ Housing refers to a dwelling that is safe, secure, affordable, and appropriate. It also encompasses issues relating to housing mobility, homelessness, neighbourhood characteristics, and over-crowding.

Young children are particularly vulnerable to inadequate housing as they are physiologically more susceptible to environmental hazards (e.g. mould, allergens and tobacco smoke); spend more time in the home and as such have more exposure to environmental hazards; are more susceptible to physical features of a home that can cause injury; and have limited communication abilities and control over their environment (AIHW, 2014).

Research has linked negative home environments during the first 1000 days with a host of developmental issues, including (but not limited to): inferior language development; behaviour problems; insufficient school readiness; aggression, anxiety and depression; and impaired cognitive development (Evans et al., 2010; Vernon-Feagans, Garrett-Peters, Willoughby & Mills-Koonce, 2011).

Longer-term effects have also been documented, including (but not limited to): decreased likelihood of high school graduation; increased likelihood of teen parenthood; increased likelihood of adult unemployment; decreased income; and higher rates of poverty (Duncan, Ziol-Guest & Kalil, 2010; Pungello et al., 2010).

Affordability

Cost of housing is often the largest and least flexible item in a family budget and low-income households are particularly vulnerable to housing stress, given that they tend to spend a greater proportion of their income on housing (AIHW, 2010). An Australian analysis of housing affordability stress (Stone & Reynolds, 2016) found that rates of stress have been increasing, especially among those living in rental accommodation. Focusing on children, this study found that 41 per cent of children from two-parent low-income families living in rented accommodation were experiencing housing affordability stress, a rate that jumps to 67 per cent with single parent families. While parents can buffer their children from the adverse effects of this source of stress, it is hard to sustain.

High housing costs can affect child wellbeing through the experience of family financial or material hardship (Harkness & Newman, 2005). Families who allocate a disproportionate amount of their income to housing have to cut back on other basic needs such as food, clothing, and heating (Lippman, 2005). Lack of affordable housing can also impact parenting capacity and mental health. Family stress can be related to housing affordability where housing costs are the main source of economic hardship and/or family conflict (Leventhal & Newman, 2010). Parents facing financial hardship face an increased risk of chronic stress, depression and partner conflict, which, in turn, correlates with more inconsistent, unsupportive, and punitive parenting styles (Leventhal & Newman, 2010). As we have seen, poor quality interactions between parent and child is related to greater likelihood of poor health and emotional and educational set-backs in later life (Harkness & Newman, 2005; Leventhal & Newman, 2010).

Homelessness

Homelessness may only occur once, may be episodic, or be chronic, however, on any dimension it is incompatible with a safe and nurturing environment for children (Nooe & Patterson, 2010). Homelessness is a complex problem that places children at increased risk of long-term poverty, homelessness in adulthood, unemployment, chronic ill-health, and other forms of disadvantage and social exclusion (AIHW, 2010).

While data on the health and development of homeless children are limited, studies in the United States show that homeless children face a greater risk of experiencing learning, developmental and behavioural problems. Infants and toddlers may experience delays in physical and mental development (Cooper, 2001; Horn & Jordan, 2007; Hicks-Coolick, Burnside-Eaton & Ardith, 2003). One Australian study of homeless pre-schoolers reported that about 50 per cent suffered significant emotional developmental delays (Dockery et al., 2010).

Compared with children from low-income households that have never been homeless, children from homeless families are twice as likely to be hospitalised and make significantly more visits to hospital emergency departments (Weinreb, Goldberg & Perloff, 1998). A higher incidence of asthma and other respiratory problems, infectious diseases, trauma related injuries, lead poisoning, chronic diarrhoea, visual and neurological deficits, delayed immunisations, tooth decay, ear and skin infections, conjunctivitis, and mental health problems and behavioural disorders have also been found (Cooper, 2001; Karim, Tischler, Gregory & Vostanis, 2006; Weinreb, Goldberg & Perloff, 1998; Yu, 2008).

Moreover, parents in homeless families are likely to suffer from depression and stress which may mean they are unable to give their children enough attention or affection (AIHW, 2010). The inability of parents to provide suitable housing can also lead to intervention by child protection agencies and the placement of children in foster care, which can cause further stress for these children (Dockery et al., 2010). Homeless parents typically have smaller social networks and higher levels of relationship conflict, accidents and violence (Nooe & Patterson, 2010). They are also at an increased risk for alcohol and drug dependence, depression, schizophrenia and suicide (Nooe & Patterson, 2010).

Homelessness also impacts a family's housing mobility (the frequency housing movement). The absence of a secure home can make it challenging for a child to establish a sense of identity and can result in difficulty for the child to establish bonds (e.g. friends, parks) that make them feel secure and develop healthy social habits (Roy, Maynard & Weiss, 2004). Continuous moving may also change social connections by eliminating a family's close social networks that provide emotional support and information about the community.

Research shows that renters, particularly those on low incomes, experience higher levels of mobility and that the negative effects of mobility are magnified with increased moves when changes in schools and residential mobility are combined (Leventhal & Newman, 2010). Jellyman & Spencer (2010) found an increased likelihood of behavioural problems in children where the total number of lifetime moves exceeded three (Jellyman & Spencer, 2008). Unaffordable housing can increase chronic mobility, particularly among low-income families, and homelessness can also eventuate from chronic mobility (Auh, Cook, Crull & Fletcher, 2006).

Dwelling characteristics

Children's development also depends on the environments in which they live and interact, including the quality of their environment and the interactions between the child and other people in the environment (AIHW, 2010).

Household income impacts the quality, type and size of housing a family can afford. Often the most affordable housing is also that of least quality, in terms of both the dwelling and neighbourhood (AIHW, 2010). The quality of housing can be compromised due to its age, inadequate maintenance, lack of basic amenities and poor design, and can lead to indoor air quality hazards including mould growth and the presence of toxic substances such as lead paint or asbestos (Cooper, 2001). Because younger children spend more time inside the home compared with adolescents, there may be a stronger association between housing quality and physical health for younger children (Leventhal & Newman 2010).

Exposure to environmental allergens also has strong and critical effects during infancy and early childhood and respiratory health may be determined by such exposure during the first year of life (Salam et al., 2004). Poor quality housing is usually situated in lower socioeconomic neighbourhoods and risk factors associated with these neighbourhoods also contribute to unfavourable child outcomes (Cooper, 2001; Dockery et al., 2010).

Overcrowding

Overcrowding refers to the minimum acceptable living area per person or the average number of people per dwelling. The adverse effects of overcrowding on children can persist throughout life, impacting future socioeconomic status and wellbeing; children are also at a greater risk of finding themselves in similar situations as their parents, leading to the intergenerational transmission of social disparity (Solari & Mare, 2007).

This is because the lack of space that children experience when living in crowded conditions can negatively impact their sense of autonomy, social behaviour, health, developmental outcomes, and school performance (Dockery et al., 2010). It can negatively impact their sleep patterns due to different schedules of household members that can lead to difficulty concentrating during the day and negatively affect mood and behaviour (Solari & Mare, 2007). Children in overcrowded houses are also less likely to have sufficient space for play and may also experience greater risk of abuse due to the greater difficulty they face in removing themselves from potentially volatile situations (Bartlett, 1997). Moreover, when parents have to cope with overcrowding it impacts their parenting behaviour and can lead to increased conflict between children and parents, as well as influence marital conflict (Dockery et al., 2010; Evans et al., 1998).

5.6.2 Built environments

Over 90 per cent of Australians live in urban environments (Easthope & Tice, 2011), and neighbourhoods are a key setting in which children begin their lives. To date, the evidence indicates that the way we design and build neighbourhoods have a range of personal and social benefits (Goldhagen, 2017), such as promoting healthier lifestyles and contributing to reducing the risk of non-communicable disease (Sallis, Floyd, Rodríguez & Saelens, 2012; Villanueva et al., 2016). Societal changes over decades have dramatically reduced the need for physical activity in daily life while creating ubiquitous barriers to physical activity (Sallis et al., 2012). Features of the built environment that promote healthier lifestyles include easy access to facilities, services, and social infrastructure, parks and recreational facilities, stores selling fresh produce (Ulmer, Chapman, Kershaw, & Campbell, 2014; Villanueva et al., 2016). On the other hand, a poorly designed neighbourhood has less connected street networks and limited access to shops and services, but an oversupply of fast food restaurants (Ulmer et al., 2014; Villanueva et al., 2016).

Most of the research on the relationship between the built environment and health has focused on children's physical activity and obesity, and the impact on child development is less well understood. Nevertheless, it is plausible that children's regular physical activity also benefits their cognitive, emotional and psychosocial development (Villanueva et al., 2016). Moreover, as Ulmer and colleagues (2014) note, at an individual level, the effect size of built environment interventions is likely to be small, but these benefits are important because they are experienced by many people, which creates a population-level exposure with sustainable public health benefits.

5.6.3 Natural environments

Access to nature and green space can have a significant impact on children's life-long development (Strife & Downey, 2009; Kellert, 2002; Grineski, 2006). Such access provides children with various cognitive, emotional, and physical benefits, including (but not limited to): better educational attainment, reduced stress and aggression, and lower risk of obesity (Faber Taylor & Kuo, 2006; Kellert, 2002; Louv, 2007; Stretesky & Lynch, 2002). Conversely, children's lack of exposure to nature is linked to a decline in their mental and physical health (Faber Taylor, Kuo, & Sullivan, 2001; Goldman & Koduru, 2000; Petty, Peacock, Sellens, & Griffin, 2005; Senier, Mayer, Brown, & Morello-Frosch, 2007).

Exposures to nature and environmental factors have a wide range of health and social benefits (Frumkin et al., 2017). They can even have an impact on a parent's care-giving capacity, by playing a significant role in coping with and recovering from stress and mental fatigue (Berto, 2014). Green spaces have also been shown to increase social interactions between families and children, promoting social trust and community perceptions of safety (Coley, Sullivan, & Kuo, 1997; Kuo, Bacaicoa, & Sullivan, 1998). Children living in poverty are more likely to lack access to natural environments as well as to be exposed to environmental hazards (Chakraborty & Armstrong, 2001; Dereziński, Lacy & Stretesky, 2003; Lester, Allen, & Hill, 2001).

The community and physical environments that children experience in the first 1000 days shape their later life outcomes, for better or worse. The level of social supports that a child's caregiver(s) has access to, drastically impacts their caregiving capacity; while a child's physical environments (including the quality and nature of their housing and the natural environments they can access) have the power to affect multiple and concurrent health and wellbeing outcomes throughout the lifespan.

Another significant aspect of a child's physical environment, which has significant consequences, beginning in the first 1000 days, is the level and nature of environmental toxins in the environment.

5.6.4 Environmental toxins and their effects

Environmental toxins in pregnancy

During the first 1000 days, the brains of infants and children are uniquely sensitive to environmental neurotoxicants at levels far below those that are known to harm adults (Heyer & Meredith, 2017; International Scientific Committee of the International Conference on Fetal Programming and Developmental Toxicity, 2007; Landrigan & Miodovnik, 2011; Miodovnik, 2011; National Research Council, 1993). Early life exposures to toxic chemicals are important causes of disease and neurodevelopmental disorders (Heyer & Meredith, 2017; Landrigan & Miodovnik, 2011; Landrigan et al., 2005; Martin & Dombrowski, 2008; Wong et al., 2015; Woodruff et al., 2004), with effects across the lifespan (Landrigan, 2016).

There are tens of thousands of synthetic chemicals currently used in developed nations, although not all of these are produced in significant quantities (Collaborative on Health and the Environment, 2007; Gray, 2008). For the majority of these chemicals, there is no toxicological screening data and there is little information on the potential effects on learning and development. While it is possible that some of these chemicals have beneficial effects, many do not. At least 200 industrially applied or produced chemicals have been associated with neurotoxicity in humans, and exposure to these modifying compounds, through consumer products or environmental pollution, poses serious threats to public health (Heyer & Meredith, 2017). Harmful exposures can start as early as in utero (Martin & Dombrowski, 2008; Schettler, 2010; WHO, 2017). Because of the omnipresence of chemicals in our daily life, pregnant women have continuous contact with chemicals in food, water, air, and consumer products (Wang et al., 2016). Despite the fact that the foetus is carried inside the mother's womb, the mother's chemical body burden is shared with her foetus; many substances easily cross the placenta and the foetal blood brain barrier to reach the developing brain (Grandjean and Landrigan, 2006; Wong et al., 2015). As a result, the next generations are born "pre-polluted" owing to these preconception and pre-birth exposures (Wang et al., 2016). Evidence of these exposures comes from analyses of the umbilical cords of newborn babies showing they contain an average of two hundred industrial chemicals (Murphy, 2010).

The brains of infants and children are uniquely sensitive to environmental neurotoxicants at levels far below those that are known to harm adults (Miodovnik, 2011). The foetus' small size and immature state of development mean that it is more vulnerable to environmental toxins during the prenatal period than at any other time in its life (Murphy, 2010). This is because they are exposed to larger doses relative to the body weight at a time when organ systems are being formed and rapid growth of neurological structures is occurring (International Scientific Committee of the International Conference on Foetal Programming and Developmental Toxicity, 2007; Martin & Dombrowski, 2008; Schettler, 2010; Wang, Padula, Sirota & Woodruff, 2016). Exposures to substances such as lead that have minimal or no discernible impacts in adults can permanently alter brain development and function in a child (Schettler, 2010).

These chemical exposures, especially during critical and sensitive windows of development such as pregnancy, can contribute to preterm birth (Burris, Baccarelli, Wright & Wright, 2016) and lead to a myriad of health consequences that can manifest across individuals' lifespans and potentially be transmitted to future generations (Wang et al., 2016). Four underlying mechanisms that produce these adverse outcomes have been proposed (Heyer & Meredith, 2017): oxidative stress, immune system dysregulation, altered neurotransmission and thyroid hormone disruption. Based on a review of the evidence regarding a range of environmental toxins, Heyer and Meredith (2017) estimated the period during which exposure to each toxicant was most likely to increase the risk of developing neurodevelopmental disorders such as ADHD, autism spectrum disorders, and schizophrenia. They found that the sensitive time-windows for the majority of toxicants occur between conception and birth.

Experimental evidence suggests that developmental exposure to persistent organic pollutants (POP) and to some non-persistent pesticides may disrupt metabolic regulation of glucose metabolism and insulin secretion, and thereby contribute to the current epidemic of obesity and metabolic disorders (Cummins, 2012; Debost-Légrand et al., 2016). These chemical pollutants are known as obesogens, and they can cause epigenetic changes in the embryo, thereby favouring the development of fat cells at the expense of other cell types (such as bone), making weight increase more likely (Cummins, 2012).

Some commonly encountered chemicals can disrupt the function of hormones and other chemical messengers that are vital to normal human development and function. Known as endocrine disruptors, these chemicals interfere with the body's key signalling pathways and can cause harm, especially during foetal and early life development (Gore et al., 2015; Schettler, 2010). The Endocrine Society's most recent Scientific Statement on Endocrine-Disrupting Chemicals (Gore et al., 2015) explains why such disruption can have long-term effects on development:

During embryonic development, organogenesis and tissue differentiation proceed through a series of tightly regulated and temporally coordinated events at the cellular, biochemical, and molecular levels, ultimately resulting in a functional, mature structure. Development is an Einbahnstrasse (one-way street), and thus natural substances such as hormones as well as environmental changes, including exposures to exogenous environmental chemicals, alter this unidirectional process. These latter perturbations may impart structural and functional changes that can profoundly deflect the developmental trajectory, often leading to lifelong phenotypic changes such as increased endocrine disease propensity.

Chemicals known to have endocrine disruption effects include bisphenol A, phthalates, atrazine, polychlorinated biphenyls and polybrominated diphenyl ethers (PCBs), and DDT. Other types of chemicals known to have adverse effects upon development include perfluoroalkyl substances, phenols, pesticides, and metals (Wang et al., 2016).

Air pollution

Various types of air pollution are known to have an adverse impact on health, including combustion sources that include diesel- and gasoline-powered motor vehicles, coal-fired power plants, residential heating, cooking, and tobacco smoking (Bostrom et al., 2002). Research has tied exposure to traffic-related air pollution during pregnancy to a host of adverse birth outcomes, including premature delivery, low birth weight, and heart malformations (Currie & Walker, 2011; Perera et al., 2009; Trasande, Malecha & Attina, 2016). Exposure to high levels of air pollution during pregnancy – particularly during third trimester – can double a woman’s risk of having a child with autism, with the risk increasing proportionally with exposure to greater amounts of fine particulate matter in the air (Raz et al., 2015). Perera (2017) argues that the evidence is clear that developing children, and especially poor children, now bear a disproportionate burden of disease from both environmental pollution and climate change due to fossil fuel combustion. By sharply reducing our dependence on fossil fuels we would achieve highly significant health and economic benefits for our children, with both immediate and long-term benefits (Perera, 2017).

Polycyclic Aromatic Hydrocarbons

Polycyclic Aromatic Hydrocarbons (PAHs) are a group of over 100 different chemicals that are formed during the incomplete burning of coal, oil and gas, garbage, or other organic substances like tobacco or charbroiled meat. Perera et al. (2006) found that high prenatal exposure to PAHs was associated with lower mental development at age three, with implications for school performance. In a comparative study between Krakow (Poland), and New York (USA), Choi et al. (2006) found an adverse reproductive effect of relatively low PAH concentrations. Given foetal growth impairment has been linked to child developmental and health problems, the researchers concluded that substantial health benefits would result from global reduction of PAH emissions (Choi et al., 2006).

Other pollutants

Pollutants under scrutiny include not just PAHs, but other pollutants that are universally present such as carbon monoxide. One American study found that the infants and young children of women living in areas with severe air pollution had three times the incidence of heart malformations and valve defects (Ritz et al., 2002). Another study (Ritz, Wilhelm, Hoggatt, & Ghosh, 2007) found that pregnant women living in areas with high carbon monoxide or fine particle levels have a risk of preterm birth that is 10 to 25 per cent higher than those who live in neighbourhoods with cleaner air. It has also been established that rates of asthma are increased in children exposed to second-hand cigarette smoke and to fine particulate air pollution (Federal Interagency Forum on Child and Family Statistics, 2010; United States Environmental Protection Agency). Even more concerning is that risk of respiratory death is increased in infants exposed to fine particulate air pollution (Woodruff, Darrow, & Parker, 2008).

Environmental toxins in infancy

Infants are particularly vulnerable to environmental toxin exposure for several reasons (Heyer & Meredith, 2017; Miodovnik, 2011):

- Certain chemicals are shown to accumulate in maternal adipose tissue and breast milk and can be transmitted by breast feeding (Grandjean and Landrigan, 2006). Children may also undergo higher contamination levels from environmental pollutants. This is because they have relatively greater energy demands than adults due to development and therefore, relative to bodyweight they breathe air more rapidly and are reported to ingest a higher proportion of water, fruit, and vegetables in their diet than adults based on dietary analysis patterns (Heyer & Meredith, 2017).

- Children are inherently at an increased risk of exposure because they spend more time indoors and outdoors crawling and playing on the ground, thereby increasing levels of contact with potentially contaminated dust and soil or toxic residues on toys and other objects. As a result, levels of environmental toxicants in the blood and brains of babies and young children may be many times higher than those of adults living in the same environment (Heyer & Meredith, 2017; Miodovnik, 2011).
- Children's metabolic pathways are immature (National Research Council, 1993) and a child's ability to metabolise toxic chemicals is different to that of an adult (Atterberry, Burnett, & Chambers, 1997; Pastor, Sadd & Morello-Frosch, 2002). Children's early developmental processes are easily disrupted (National Research Council, 1993). They also have more time than adults to develop chronic diseases. Many diseases thought to be triggered by toxic chemicals, such as cancer and neurodegenerative diseases, are now understood to evolve through multistage, multiyear processes that may be initiated by exposures in infancy (Landrigan et al., 2006).

One of the sources of exposure to environmental toxins is through many new synthetic materials, such as food packaging, that infants and children come in contact with (Landrigan & Goldman, 2011).

Food packaging

Chemicals used in the packaging, storage, and processing of foodstuffs may harm health over the long term (Muncke, Myers, Scheringer, and Porta, 2014). This is because most of these substances are not inert and can leach into the foods we eat. People who eat packaged or processed foods are likely to be chronically exposed to low levels of these substances throughout their lives (Muncke et al., 2014).

Constant exposure to an estimated 4000 'food contact materials' in food packaging is common for most people in modern society (Cribb, 2014). These materials are a significant source of chemical food contamination, although current testing methods do not cover the risks which this low-level, lifelong exposure may cause, and therefore legally they are not considered as contaminants (Cribb, 2014). Understanding the immediate and long-term impacts of these exposures is essential to the prevention of chronic disease (Muncke, Myers, Scheringer, & Porta, 2014).

One of the conclusions to be drawn from this brief review of the evidence regarding the impact of environmental toxins on development concerns the importance of the timing of the exposures. As stated by the International Scientific Committee of the International Conference on Foetal Programming and Developmental Toxicity (2007),

Research into the environmental influence on developmental programming of health and disease has therefore led to a new paradigm of toxicologic understanding. The old paradigm, developed over four centuries ago by Paracelsus, was that "the dose makes the poison". However, for exposures sustained during early development, the most important issue is that "the timing makes the poison". This extended paradigm deserves wide attention to protect the foetus and child against preventable hazards.

This call for prevention has been echoed by others (Haugen, Schug, Collman & Heindel, 2015; Heyer & Meredith, 2017; Landrigan & Goldman, 2011). Given the mounting evidence of the effects of environmental exposures in the first 1000 days, Haugen and colleagues (2015) argue that the DOHaD paradigm should be expanded from its historical focus on nutrition to include environmental exposures to chemicals. The adverse effects of exposure to environmental toxins are preventable if the effects are recognised and appropriate action taken (Landrigan & Goldman, 2011). A notable example of this in Australia was the removal of lead from petrol.

5.7 Summary

A child's temperament, the quality of care they receive and the relationships they experience, the communities in which they reside, and the physical environments which they occupy, all have significant and lifelong consequences on the quality of their health and wellbeing.

The biological foundation of a temperamental bias (a bias towards certain temperamental characteristics) is usually, but not always, genetic (Kagan, 2012). In some cases it is the result of severe stress or infection in the pregnant mother which affects the foetus.

While a child's temperament is shaped by genetic susceptibilities, the development of temperamental traits are greatly influenced—for better and for worse—by their environmental experiences. However, some children are more influenced by their environmental conditions than others due to the presence (or absence) of specific genetic characteristics. This is known as differential susceptibility. Children with such 'risk' genes are thought to be disproportionately affected by poor rearing influences, while more likely to thrive in highly supportive environments.

The quality of a child's interpersonal relationships in the first 1000 days plays the most significant role in shaping a child's lifelong outcomes. While there is no evidence to suggest that the structure of a child's family (e.g. whether they are raised by same-sex parents) impacts future health and wellbeing, the quality of their relationships, and attachment with their caregiver is of utmost importance. Infants seek proximity to, and safety in their caregiver as a way of maintaining or increasing their positive feelings and minimising or regulating their stress states. An interrupted attachment process means that the child's brain places an emphasis on developing neuronal pathways that are associated with survival, before those that are essential to learning and growth.

Parenting style in the first 1000 days is central to a child's attachment style. The most positive parenting style is one that is characterised by responsiveness, warmth, sensitivity, acceptance, predictability, consistency and a lack of harsh, punitive forms of discipline.

Evidence also supports the unique role of male caregivers in early childhood development: associated with positive social, emotional and cognitive developmental outcomes. Conversely, poor father-child relationships and fathering behaviours can have lasting effects on areas such as social adjustment and increase likelihood of mental ill health in later life.

When positive and secure attachments are not created, and the child is exposed to significant and persistent adverse experiences, this is likely to result in poor health and development outcomes throughout the life course. There are three key reasons for this:

1. Trauma disrupts the progression of critical developmental processes (namely the stress response and brain development).
2. Trauma impacts the way in which children relate to and interpret the world around them.
3. Trauma is likely to result in the development of negative risk behaviours in later life that significantly increase the likelihood of adult morbidity and mortality.

A child's exposure to domestic violence is one primary way that trauma can take place. Domestic violence during pregnancy poses significant risk of harm to the mother and unborn child, while exposure after birth can be extremely distressing for an infant and negatively impact brain development and regulatory systems. Domestic violence also significantly impacts a parent's caregiving capacity.

A caregiver's sense of community and access to social supports can also impact child development, as they greatly impact a parent's caregiving capacity. Social support during pregnancy reduces the likelihood of maternal stress, depression and risk taking behaviours during and after pregnancy, and decreases the risk of child maltreatment after birth.

The child's physical environment is also significant to their lifelong health and wellbeing. The most obvious of these is housing, as children are particularly vulnerable to inadequate housing (the structural quality of the house, housing affordability, homelessness and continued housing mobility). For example, lack of appropriate housing impacts a caregiver's mental health, which in turn impacts their caregiving capacity; while overcrowding can negatively impact their sleep patterns and also put them at greater risk of abuse.

Access to nature and green space can also significantly impact a children's development, by providing (or denying) them the opportunity to access spaces that are conducive to thriving cognitive, emotional, and physical development. Children living in poverty are more likely to lack access to natural environments as well as to be exposed to environmental hazards.

The presence of environmental toxins during and after pregnancy grossly impact a child's health and wellbeing. Infants and children are more vulnerable to environmental toxins because they are exposed to larger doses relative to the body weight at a time when organ systems are being formed, and can permanently alter brain development and function in a child. Toxins such as air pollutants, carbon monoxide and even food packaging can be damaging to the child's developing organ systems.

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While we have thus far reviewed the evidence surrounding the biological, child, family, community and environmental factors that shape health and development in the first 1000 days, the following section examines some of the most influential individual level factors of child health and development.

6. Individual level factors influencing child health and development

6.1 Nutrition

There is now clear evidence that early life nutrition in the foetus, infant and young child can have profound effects on long-term health (Davies et al., 2016a, 2016b). From preconception to adulthood, our dietary intake has the ability to shape the individual or population health trajectories for better or worse (Gluckman et al., 2011; Langley-Evans & McMullen, 2010). Over the past decades, research has shown that good nutrition is central to optimal health and development outcomes and disease prevention. Conversely, excessive intake of energy or insufficient intake of protective nutrients, especially during critical periods of development, is associated with poor health and contributes to health disparities (Herman, Taylor Baer, Adams et al., 2014).

The presence or lack of good nutritional status of the mother and/or child is a critical factor in 'programming' the child for healthy development and positive long-term health and wellbeing outcomes. Foetal and early-life nutrition has been linked to setting the risk for conditions such as coronary heart disease, Type-2 diabetes, osteoporosis, asthma, lung disease and some forms of cancer (British Medical Association, 2009).

6.1.1 Nutrition in preconception and pregnancy

It is now well established that maternal nutrition can affect the offspring's epigenetic state and have lifelong effects on: the child's mental health (Sarris et al., 2015 & Jacka et al., 2013); food/flavour preferences (Gugusheff, Ong & Muhlhauser, 2011; Vucetic et al., 2010); satiety, muscle mass and insulin resistance (Low et al., 2012; Vaiserman, 2014). This is because the foetus uses nutritional input from its mother to anticipate the kind of nutritional world it will be born into, and adjusts its phenotype accordingly (Moore et al., 2014a). This was discussed in section 2.3.2 (the *Mismatch hypothesis*).

Research shows that the developmental induction of risks in an obesogenic environment (environmental influences that promote obesity) is considerably influenced by maternal nutritional status at conception, during pregnancy, and during weaning (Low et al., 2012). Women who are overweight or obese before pregnancy are at greater risk of developing hypertensive disorders such as pre-eclampsia during pregnancy, and giving birth to larger infants who are at increased risk of developing obesity in later life (World Health Organization, 2010). Overweight and obesity can also increase the risk of stillbirth, difficult delivery, haemorrhage and birth defects (Arabin & Stupin, 2014).

Weight gain during pregnancy can also affect the immediate and future health of infants: while excessive gestational weight gain can increase birth weight and postpartum weight retention, inadequate gestational weight gain can increase the likelihood of poor foetal development (Siega-Riz et al., 2009; ACOG Committee opinion, 2013).

Increasing evidence is also pointing toward a relationship between paternal obesity and adverse health outcomes in the offspring (McPherson, Fullston, Aitken & Lane, 2014). Paternal obesity impairs sex hormones, basic sperm function, and molecular composition (Ng et al., 2010; Sermondade et al., 2013) which results in disturbed embryo development and health, and an increased subsequent offspring disease burden (Fariello et al., 2012; Ribas-Maynou et al., 2012). However, some studies have shown that the reversibility of obesity-induced parental programming may be possible with diet and exercise interventions (Chen, Gong, & Xu, 2013; Ibrahim et al., 2012; Saez Lancellotti et al., 2013; Stephens & Polotsky, 2013).

6.1.2 Nutrition in infancy

The speed of postnatal growth is at its highest during infancy – a time when the infant is entirely dependent on others to meet its nutrient needs (Robinson, 2015). Inadequate nutrition and restriction of growth during this period can result in permanent stunting (Scientific Advisory Committee on Nutrition, 2011) in addition to having potential for lifelong deficits in neurological functioning (Innis, 2014). Excessive and rapid weight gain in infancy, however, is also a concern and has been linked to obesity in later life, as well as a number of risk factors for cardiovascular disease (Brands, Demmelmair & Koletzko, 2014 ; Weng et al., 2012).

Exclusive breastfeeding has been shown to at least modestly protect against excessive early infant gain and later obesity, an effect that may result from differences in composition of weight gain between breast-fed and formula-fed infants (Young et al., 2012). Moreover, the method of infant feeding (i.e. suckled directly at the breast or via a bottle) can also affect infant growth patterns. When feeding at the breast, the pace and volume of intake are controlled by the infant, but the caregiver maintains more control when bottle feeding (Crow, Fawcett, & Wright, 1980). Infants fed from a bottle (vs. fed at the breast) consume more milk, protein, and energy (Heinig et al, 1993; Isomura et al., 2011; Sievers, Oldigs, Santer, & Schaub, 2002), potentially resulting in greater weight gain (Dewey et al., 1993; Haisma et al., 2003; Heinig et al., 1993). Removing control from the infant may also impact the infant's ability to interpret satiety cues and self-regulate food intake accordingly (Dewey & Lonnerdal, 1986; Disantis, Collins, Fisher, & Davey, 2011; Li, Fein, & Grummer-Strawn, 2010; Matheny, Birch, & Picciano, 1990). These mechanisms are thought to occur regardless of what is in the bottle (i.e., breastmilk vs. formula).

Duration of breastfeeding

Research suggests that the initiation and duration of breastfeeding during infancy can also influence obesity in later life (Stettler, Zemel, Kumanyika, & Stallings, 2002). A longer duration of breastfeeding has been associated with decreased likelihood of obesity in later life (Harder, Bergmann, Kallischnigg, & Plagemann, 2005). Harder et al. (2005) found that the risk of obesity was reduced by 4 per cent for each month of breastfeeding, with this effect lasting up to a duration of breastfeeding of 9 months. Breast milk has also been found to decrease the likelihood of developing allergies in later life as it contains many immune factors (Prescott, 2011) and substances that promote favourable colonisation of the gut with friendly bacteria. (Prescott, 2011).

Introduction of complementary foods and drinks

Complementary feeding commences when breast milk alone is no longer sufficient to meet the nutritional requirements of infants, and other foods and liquids are needed, along with breast milk (WHO, 2016). The timely introduction of appropriate complementary foods are critical in ensuring optimal growth and wellbeing (UNICEF Innocenti Research Centre, 2005). If complementary foods or drinks are introduced too early or are not given safely in the correct quantity at the optimum time, growth rates can falter dramatically and lead to growth restriction and even stunting – associated with an under-developed brain (Michaelsen, Weaver, Branca & Robertson, 2010).

Complementary foods are often introduced at approximately 6 months of age, although some infants may need complementary foods earlier, but not before 4 months of age (Michaelsen et al., 2010). Complementary feeding should be a process of introducing foods, while maintaining breastfeeding. Highly salted foods should not be given during the complementary feeding period, nor should salt be added to food during this period (Michaelsen et al., 2010).

Evidence shows that obesity, diabetes, cardiovascular morbidity, and neuropsychiatric diseases can all be considered paediatric diseases. As such, disease prevention must start with improved nutrition (and reduced exposure to environmental chemicals) during development.

Davies and colleagues (2016) show that there are now significant data to support the hypothesis that early life nutrition in the foetus, infant and young child can have profound effects on long-term health. As the findings of further research become available, recommendations on optimizing early life nutrition should be formulated and made widely available as part of the preventative health policy agenda in Australia.

The other two primary individual level factors that significantly impact health and development in the first 1000 days, and beyond, are substance use and significant maternal stress. Evidence surrounding these are discussed in the below sections.

6.2 Substance use

6.2.1 Alcohol

Exposure to alcohol in the uterus is the leading cause of cognitive impairment and neurodevelopmental disorders (Centers for Disease Control and Prevention, 2002; Eustace, Kang, & Coombs, 2003), and the most common preventable cause of birth defects. Although the risk of birth defects increases with frequent maternal alcohol intake, alcohol exposure throughout pregnancy and before pregnancy is confirmed to have detrimental consequences for foetal brain development and growth and increases the likelihood of pre-term delivery and physical malformations (AIHW, 2016).

According to the 2013 National Drug Strategy Household Survey, 49 per cent of Australian women did not consume alcohol while pregnant, but over 50 per cent of pregnant women consumed alcohol before they knew they were pregnant, and 25 percent continued to drink even after they knew they were pregnant (AIHW, 2013).

Foetal alcohol spectrum disorder (FASD)

The effects of alcohol on the embryo or foetus produce a spectrum of disorders that affect physical, learning and behavioural outcomes (Abel, 2012; Burd, Cotsonas-Hassler, Marsoff, & Kerbeshian, 2003). The range of effects is collectively termed 'foetal alcohol spectrum disorder' (FASD) and can include abnormalities in the formation of the face, intellectual and learning disabilities, deficits in executive functioning, memory problems, speech and language delays, inattention, hyperactivity, internalising and externalising behavioural problems, and social impairments that remain apparent to varying degrees throughout life (Coles et al., 1997; Jacobson & Jacobson, 2002; Kingsbury & Tudehope, 2006). In adults, FASD is associated with high rates of mental health problems, alcohol and other drug misuse, and inappropriate sexual behaviour (Streissguth et al. 2004).

Studies show that overall, lower levels of alcohol are needed to produce behavioural anomalies than are needed to produce physical effects, and some brain regions are more susceptible to alcohol than other regions (Randall, 2001). Scanning of the brains of children with prenatal alcohol exposure has showed increased incidence of abnormalities in areas of the brain that are responsible for motor movement learning, behaviours, cognition and emotional regulation (Mattson & Riley, 1998; Mattson, Schoenfeld, & Riley, 2001). FASD is known to be under-recognised in Australia, and efforts have been made to develop criteria that can help with early identification (Watkins et al., 2014).

Threshold for risk

There is inconsistent data about the effect of social (i.e. 'light') alcohol consumption on pregnancy outcomes (Kalter, 2003). However, based on available research evidence, it cannot be stated that light drinking in pregnancy has been established to be safe. In particular, any messages implying that light drinking might be safe should not be disseminated without also providing a threshold or cut-off above which there is an increase in risk (Sayal, 2009). Safe recommendations should focus not just on the average amount of alcohol consumed, but also on patterns of consumption. Patterns more accurately reflect blood alcohol levels, as problems may develop even at low levels of alcohol consumption if binge drinking takes place and high blood alcohol levels are present in the critical phases of foetal development (Goransson et al., 2003).

6.2.2 Illicit drugs and other psychoactive substances

Illicit drugs and other psychoactive substances in pregnancy

The adverse effects of substance abuse can be considerable, and include premature birth, foetal distress, physical and/or mental retardation, birth defects and withdrawal symptoms upon birth (Russell 1995). Treatment resulting in separation could also have implications for infant-maternal attachment (Love & Tsantefski, 2006). In the longer term, the effects of in utero exposure to drugs and alcohol include (but are not limited to) increased risk of sudden infant death syndrome (SIDS), impulsivity, learning disabilities, antisocial behaviour and neurological deficits (Dore, Doris and Wright 1995).

The illicit nature of much substance use makes it difficult to establish the prevalence of substance use in pregnancy. However, Australian (Thomas, 1988) and international (Goode, 2000; McElhatton, 2000; Scully et al., 2004; Marcellus, 2002; Greenfield, Manwani, & Nargiso, 2003) research indicates that drug use in pregnancy is a serious and rapidly growing social problem (Love & Tsantefski, 2006). Consistent with international research, Victorian maternity units have recorded steadily increasing rates of substance use in pregnancy (Murphy, 2000). As these reports are based on self-disclosure of substance use, they are likely to underestimate prevalence (Love & Tsantefski, 2006).

Australian research (Swift, Copeland, & Hall, 1996) has also shown that of the women seeking treatment for substance dependence, approximately half are mothers, and most are using two or more drugs at, or near, the same time. These women also had a range of other co-occurring physical and psychological health concerns such as hepatitis, eating disorders and suicide attempts (Swift et al., 1996), and reported higher rates of negative childhood experiences, psychological distress, lower levels of perceived social support, homelessness and domestic violence (Harmer, Sanderson, & Mertin, 1999).

Illicit drugs and other psychoactive substances in infancy

It is well established that children who are raised in families with parental substance misuse often have poor developmental outcomes (Dawe, Harnett, & Frye, 2008). However, parental substance abuse often co-exists with other risk factors (such as domestic violence, low income, and transience) and it is the sum of these various influences that determines the child's lifelong outcomes (Dawe et al., 2008).

While it is difficult to obtain accurate data, it has been estimated that approximately 13 per cent of Australian children aged 12 years or less are exposed to an adult who is a regular binge drinker (Dawe et al., 2008). Additionally, just over 2.3 per cent of children aged 12 years or under are estimated to be living in a household containing at least one daily cannabis user and 0.8 per cent are estimated to be living with an adult who used methamphetamine at least monthly. However, these figures are widely acknowledged to be underestimated, given the self-reporting nature of data collection (Dawe et al., 2008).

Substance misuse by parents of very young children can compromise parental care in several ways, including intoxication, drowsiness and impaired attention, withdrawal symptoms, and engaging in illegal and dangerous activities.

When a parent is intoxicated, their ability to provide adequate care and protection to an infant is significantly compromised: intoxication will severely impair a parent's ability to provide a regular routine, clean environment (Dawe et al., 2008) and be responsive and sensitive to a child's emotional and developmental needs.

Substances that result in a state of extreme drowsiness and impaired concentration and attention, such as alcohol and heroin, clearly impact parental supervision, increasing risk of injury, neglect or harm by others. Regular use of substances such as amphetamines are also associated with a state of agitation, psychosis, restlessness and impaired judgement (Dawe et al., 2008). These states are clearly incompatible with sensitive and responsive parenting and may indeed increase the risk of neglect and abuse (Dawe et al., 2008).

A parent who is dependent on a substance will experience withdrawal symptoms when they are unable to use. While the experience of withdrawal varies across substance classes, such a physical state has the potential to impair the parent's ability to focus on the needs of their child over their own immediate physical and psychological distress (Dawe et al., 2008).

Illicit drugs such as opioids and amphetamine-type substances often require engagement in a range of illegal activities, such as theft or prostitution, in order to support the habit (Dawe et al., 2008). The use of these substances also comes with risks of exposure to injecting and other paraphernalia, association with other adults who use substances and, for some children, exposure to a physically dangerous environment when substances are being manufactured and/or not safely disposed. Child death reviews conducted by the Victorian Child Death Review Committee have consistently noted the high prevalence of parental substance misuse among deaths of infants known to the Victorian Child Protection Service (Victorian Child Death Review Committee, 2012).

Besides compromising parental care in various ways, alcohol or drug misuse can also increase the likelihood of child maltreatment by increasing the risk of violent tendencies (Flanzer 1993). Drugs such as crack, cocaine, heroin, LSD and marijuana have been proposed as direct causal factors that 'reduce inhibitions, unleash violent tendencies, and/or directly elicit violent behaviour' (Gelles 1993, p.183). Research shows that drugs (and alcohol) can lower the inhibitions that keep people from acting upon physically or sexually violent impulses (Araji & Finkelhor 1986). Furthermore, frustration tolerance may be lowered by alcohol or drugs, leaving a parent more likely to physically abuse a child when under their influence. Substance abuse may also diminish any shame or guilt a perpetrator feels after maltreating a child (Hayes & Emshoff 1993). The failure to experience negative emotions or inhibitors may perpetuate maltreatment as it minimises the negative consequences for the offender following an assault.

Co-morbidity

Over 50 per cent of heroin users, 20 per cent of amphetamine users, 16.5 per cent of cannabis users and 11 per cent of high-risk alcohol users reported diagnosis or treatment for mental illness in the past 12 months (Australian Institute of Health and Welfare, 2005, p. 99). When women are considered separately from men, the rates of co-morbid conditions are even higher, including trauma-related conditions (Conners et al., 2003; Najavitis, Weiss, & Shaw, 1997). Moreover, many families with parental substance abuse also experience low income/poverty, report high rates of unemployment, have unstable accommodation, and experience social isolation (Conners et al., 2003; Powis, Gossop, Bury, Payne, & Griffiths, 2000), compounding the effects of parental substance misuse.

Social isolation is a key feature of the lives of families with parental substance abuse. Typically, women with substance misuse problems feel unable to attend a range of community activities that are often the building blocks of community connectedness and support. Parents who have limited social support and live socially isolated lives are at greater risk for poor parenting practices. This is especially the case when these problems are further compounded by other risk factors, such as parental mental health problems and socioeconomic disadvantage.

6.2.3 Tobacco

There is conclusive evidence that smoking causes compromised fertility, and that parental smoking potentially has long-term and serious consequences for child health (Mitchell, Devlin, & Mannes, 2006). In 2010, 11.7% of pregnant women smoked before knowing they were pregnant, while 7.7% reported that they smoked after they knew they were pregnant. However, underreporting of smoking during pregnancy is a common practice, given the social stigma associated with smoking during pregnancy. High rates of underreporting have been reported in intervention trials (Mitchell et al., 2006). The likelihood of smoking during pregnancy was higher among teenagers, women in disadvantaged circumstances and Aboriginal women (Laws, Li, & Sullivan, 2010).

Smoking in pregnancy

Within minutes of inhalation of tobacco smoke, wherever the blood flows, many of the more than 4000 chemicals from tobacco smoke also rapidly flow (US Department of Health and Human Services, 2004). Nicotine induces the narrowing of the blood vessels, which affects the function of the placenta, restricting blood flow and reducing the supply of nutrients and oxygen to the foetus (US Department of Health and Human Services, 2004). There is growing evidence that smoking during pregnancy affects the normal development of the brain systems that regulate oxygen uptake and heart function, increasing the risk of stillbirth, neonatal death and SIDS (British Medical Association, 2004). Blood vessel constriction and high levels of carbon monoxide in the blood caused by smoking may induce hypoxia (oxygen deficiency), which has been implicated in placental abruption. Hypoxia can also result in the enlargement of the placenta, causing it to extend over the cervix, as seen in placenta praevia. Smoking during pregnancy has also been linked to the development of childhood obesity (Oken, Levitan & Gillman, 2008).

Dose-response relationships exist between maternal smoking during pregnancy and stress or abstinence signs in the baby, including central nervous system visual stress and greater excitability (Law et al., 2003). These findings may indicate neonatal withdrawal from nicotine. Another study (Godding et al., 2004) found neurotoxic effects of maternal smoking in pregnancy, impacting a newborn's neurobehavioral outcomes, such as childhood learning disabilities.

Whether effective pharmacotherapies such as nicotine replacement therapy (NRT) are safe to use in pregnancy is still unknown. Nicotine itself has the potential to disturb the development of the embryo or foetus and there is no known safe level of nicotine that can be administered during pregnancy. Given that the ratio of potential benefit to harm is not conclusive, most recommendations are to consider pharmacotherapy only after psychosocial intervention has failed (Fiore and the Clinical Practice Guideline Treating Tobacco Use and Dependence 2008 Update Panel, 2008; Melvin et al, 2000).

Exposure to environmental tobacco smoke (ETS) or passive smoking during pregnancy is associated with a wide range of complications during gestation as well as in the perinatal and neonatal periods. Specifically, there is a high risk for preterm, low birth weight, small for gestational age infants, as well as for sudden infant death syndrome (Simón et al., 2017). Simón and colleagues (2017) studied the effect of the progressive introduction of smoke-free legislation in Spain, and found that even partial smoking bans resulted in reductions in preterm birth rates and low birth weight, with even greater reductions following the reduction of comprehensive bans (Collaco, Wilson & McGrath-Morrow, 2017).

Smoking in infancy

Exposure to environmental tobacco smoke (ETS) during infancy and childhood has also been associated with slower rates of growth in lung function and increased risk of asthma, middle ear disease and respiratory disease (British Medical Association, 2004). Some studies have found that compared to children of non-smokers, the children of smokers have a poorer performance at school, with lower scores in cognitive tests and greater likelihood of behavioural problems, including hyperactivity and shorter attention spans (British Medical Association, 2004). Exposure to environmental tobacco smoke (ETS) or passive smoking may also influence breastfeeding, with non-smoking women who are exposed to ETS stopping breastfeeding sooner than those who are not exposed (British Medical Association, 2004).

Nicotine distributes rapidly to and from breast milk. As maternal plasma nicotine concentration rises and falls, breast milk concentration also rises and falls. The mean elimination half-life of nicotine in breast milk is 95 minutes (Dempsey and Benowitz 2001). Mothers who smoke are less likely to start breastfeeding their babies than non-smoking mothers, and tend to breastfeed for a shorter time. Breast milk production is also lower in smokers than in non-smokers. In breastfeeding mothers who smoke, milk output is reduced by more than 250 mL/day compared with non-smoking mothers.

6.3 Stress

Researchers have consistently found that various types of chronic stress are linked to – and probably cause – shorter telomeres (Blackburn & Epel, 2012). Telomere shortness and stress have independently been associated with several common conditions, such as cardiovascular disease and diabetes. These associations are so widespread and consistent that even without a detailed understanding of the biochemical pathways involved, the message is clear. Failure to alleviate severe stress caused by prolonged threats such as violence, financial hardship, abuse and emotional neglect, particularly in children, will result in exponentially higher costs further down the line – personal, economic and otherwise.

6.3.1 Stress in pregnancy

As discussed, the prenatal period is a time of rapid change and extreme vulnerability for the foetus. It is during this period that the foetus is described as being in a state of ‘programming’: the process by which an event or insult during a sensitive developmental period has a long-lasting or permanent influence. The effects of programming depend on the timing of the exposure and on the developmental stage of organ systems. One primary risk factor for health outcomes resulting from foetal programming is prenatal exposure to maternal stress (Davis & Sandman, 2010).

During pregnancy, maternal stress impacts the foetal nervous system and reduces foetal growth and length of gestation. High levels of maternal anxiety are significantly associated with increased risk of intrauterine growth restriction (Ding et al., 2014; Grote et al., 2010). Poor growth in utero is a major risk factor for a number of subsequent health problems in the child’s later years, being linked to conditions such as heart disease, hypertension, and low birth weight, which increases the risk of developing conditions such as obesity and diabetes (Massin, Withofs, Maeyns, & Ravet, 2001; Shankaran, Das, & Bauer, 2006). Maternal stress levels are also associated with poorer birth outcomes, including preterm delivery, lower birth weight and gestational age, smaller head circumference, and poorer neurological scores at birth (Dole et al., 2003; Glover & O’Connor, 2006; Glynn, Dunkel Schetter, Hobel, & Sandman, 2008; Glynn, Wadhwa, Dunkel Schetter, & Sandman, 2001; Hobel & Culhane, 2003).

Similarly, a growing body of evidence has revealed that maternal gestation stress can negatively impact a range of health and developmental outcomes in infancy and early childhood (Monk, 2001; Ruiz & Avant, 2005; Talge, Neal, & Glover, 2007; Tegethoff et al., 2011, Greene, Olsen, Schaffner, & Meinlschmidt, 2011). These include cognitive development, language development, behavioural and emotional development, and physical and neuromuscular maturation.

Cognitive and language development. There is evidence that maternal elevated stress and anxiety during pregnancy is associated with delayed infant cognitive development (Brouwers, van Baar, & Pop, 2001; Buitelaar et al., 2003; Glover & O'Connor, 2006; Huizink et al., 2003; Sandman, Davis, Buss, & Glynn, 2012) and that this may persist well into adolescence (Mennes, Stiers, Lagae, & Van den Bergh, 2006). Maternal gestational stress, particularly in early pregnancy, has also been linked to lower language outcomes in infancy and early childhood (Henrichs et al., 2011; Laplante et al., 2004).

Behavioural and emotional development. A growing body of research indicates a correlation between maternal stress and child emotional and behavioural problems after birth (de Weerth, van Hees, & Buitelaar, 2003; Glover & O'Connor, 2006; O'Connor, Heron, Golding & Glover, 2003; Robinson et al., 2008; Sandman et al., 2012). For example, Robinson et al. (2008) found that the experience of multiple stress events in pregnancy was predictive of clinically significant levels of behavioural problems in the pre-school years after adjustment for multiple other risk factors. These effects were equally balanced across internalising and externalising symptoms. These effects can be very long-lasting. Epigenetic changes (facilitated in utero) relating specifically to the regulation of stress hormones have been found in adult children of parents with PTSD or other stress-related disorders (Yehuda et al., 2015).

Physical and neuromuscular maturation. Maternal stress in pregnancy also has an impact on children's physical and neuromuscular maturation (Buitelaar et al., 2003; Ellman et al., 2008; Grace, Bulsara, Robinson & Hands, 2016; Huizink et al., 2003; Sandman et al., 2012). Stress has an accumulative effect on the developing foetal motor system, especially in late pregnancy when the cerebellar cortex (the area of the brain responsible for regulating motor movements) is growing most rapidly. The negative effects of maternal gestational stress on offspring motor development becomes more evident as the child becomes older (Grace et al., 2016). The continued growth of the neurological systems throughout the first decade (Gramsbergen, 2003) may explain why the full impact on these systems is not evident until after puberty.

6.4 Summary

The most significant individual level factors in the first 1000 days which influence child health and development relate to nutrition, substance use and the experience of significant stress.

The presence or lack of good nutritional status of the mother and/or child is a critical factor in 'programming' the child for healthy development and positive long-term health and wellbeing outcomes. Women who are overweight or obese before pregnancy are at greater risk of complications during pregnancy, and giving birth to larger infants, who are at increased risk of developing obesity in later life. While excessive weight gain during pregnancy can also increase birth weight, inadequate gestational weight gain can increase the likelihood of poor foetal development.

After birth, factors such as excessive and rapid weight gain (and inadequate sleep) have been shown to contribute to childhood obesity. Exclusive breastfeeding has been shown to modestly protect against this, while the initiation and duration of breastfeeding can also influence this. Breast milk has also been found to decrease the likelihood of developing allergies in later life.

Complementary feeding (introduced at approximately 6 months of age) can commence when breast milk alone is no longer sufficient to meet the nutritional requirements of infants, and other foods and liquids are needed, along with breast milk. Because obesity, diabetes, cardiovascular morbidity, and neuropsychiatric diseases can all be considered paediatric diseases, disease prevention must start with improved nutrition.

Substance use in the first 1000 days is another noteworthy individual level factor which significantly impacts child health and wellbeing. Exposure to alcohol in the uterus is the leading cause of cognitive impairment and neurodevelopmental disorders, and the most common preventable cause of birth defects. The effects of alcohol on the embryo or foetus produce a spectrum of lifelong disorders that affect physical, learning and behavioural outcomes, the range of which is collectively termed 'foetal alcohol spectrum disorder' (FASD). Based on available research evidence, it cannot be stated that light drinking in pregnancy has been established to be safe.

The adverse effects of substance use can be considerable, and include (but are not limited to) premature birth, physical and/or mental retardation and birth defects. Pregnant women who are substance dependant are also more likely to present with co-occurring physical and psychological health concerns such as hepatitis, eating disorders and suicide attempts, increasing the risk of harm to their child before and after birth. Substance misuse after birth compromises parental care in several ways, including intoxication, drowsiness and impaired attention, withdrawal symptoms, and engaging in illegal and dangerous activities. These all impact a caregiver's capacity to respond to the child's immediate and long-term health, safety and wellbeing needs.

Exposure to tobacco in the first 1000 days is also detrimental to the health and wellbeing of the child. Within minutes of inhalation of tobacco smoke, wherever the blood flows, many of the more than 4000 chemicals from tobacco smoke also rapidly flow. Nicotine also restricts blood flow to the placenta and reduces the supply of nutrients and oxygen to the foetus. Following birth, exposure to environmental tobacco smoke in childhood is associated with slower rates of growth in lung function and increased risk of asthma (amongst various other adverse outcomes).

Toxic stress is the third major individual level factor that impacts health in the first 1000 days, and beyond. Toxic stress during pregnancy impacts the foetal nervous system and reduces foetal growth and length of gestation. Poor growth in utero is a major risk factor for a number of subsequent health problems in the child's later years, including: physical and neuromuscular maturation; behavioural and emotional development; and cognitive development. Toxic stress can also impact a parent's caregiving capacity and risk taking behaviour, once the child is born.

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So far, we have reviewed the evidence relating to the various factors and mechanisms through which child health and development can be influenced, for better or worse. The following section highlights the evidence surrounding how a child's experiences in the first 1000 days translates to outcomes in later life, and how long-lasting those effects are.

7. Beyond the first 1000 days

In considering how children's early life experiences are linked to later life outcomes, it is important to note that development is not simply interactional but is transactional. Rather than development being simply the product of an interaction between a child's genetic / epigenetic characteristics and the various caregiving and other environments they experience, it involves a dynamic transactional process whereby the child shapes the environment at the same time as the environment shapes the child (Bornstein, 2009; Mandy & Lai, 2016; Sameroff, 2009). Thus, infants and parent bring distinctive characteristics to their exchanges, but each also changes as a result of their interactions with one another, and both therefore enter the next interaction as changed individuals. This means that development is a function of the individual and the individual's environment and not of either alone (Bornstein, 2009).

With this consideration in mind, we will now explore several of the developmental pathways linking development during the first 1000 days and later outcomes.

7.1 Pathways to later outcomes

Four key ways in which early childhood experiences can have long-term effects have been identified (Boivin & Hertzman, 2012; Hertzman & Power, 2003; Keating & Hertzman, 1999; Shonkoff, Boyce & McEwen, 2009). They are:

- biological embedding
- accumulation effects
- developmental escalations of risk over time
- triple hit effects.

Although they are distinguishable from one another, these pathways are not mutually exclusive.

7.1.1 Biological embedding

The process of biological embedding has already been covered (see section 2.2.2 above). As we have seen, a great deal has been learned about the biological mechanisms underlying the developmental process of biological embedding – the role of epigenetics in general and telomeres in particular. Understanding these mechanistic causes of developmental outcomes is important because it enables us to understand the causal chains whereby these outcomes are achieved (Moore, 2015). However, epigenetic changes by themselves do not determine our subsequent development any more than our genes do. While they increase the risk of poor health outcomes in later life, whether or not these risks lead to actual poor outcomes depends upon the context (severity, length of time etc.) in which they are embedded and the complex interactions with other experiences and environments (Moore, 2015).

7.1.2 Accumulation effects

Development is also shaped by the cumulative effect of adverse environments and experiences (Boivin & Hertzman, 2012; Halfon, Larson, & Russ, 2010; Keating & Hertzman, 1999; Masten & Cicchetti, 2010; Nurius, Prince & Rocha, 2015). What Nurius and colleagues (2015) call stacked disadvantage, such as low parental education, low family income, single parent family structure, racial minority, and immigrant status, have additive effects on the life chances of children and young people. Adverse experiences during childhood also have cumulative effects, and the toxic stress they cause influence every aspect of health and wellbeing in childhood and beyond (Anda et al., 2009; Anda et al., 2006; Shonkoff et al., 2009; Shonkoff, Richter, van der Gaag, & Bhutta, 2012). These effects cascade across all areas of developmental functioning, thereby altering the course of development (Masten & Cicchetti, 2010). Over time, the cumulative wear and tear caused by exposure to chronic stress results in physiological changes to the body with long-term adverse consequences for health and wellbeing (Evans & Schamberg, 2009; Seeman et al., 2010).

This cumulative wear and tear on the body is known as allostatic load, and is caused by repeated mobilisations of multiple physiological systems over time in response to environmental stressors (Duncan, Kalil, & Ziol-Guest, 2013; Evans & Schamberg, 2009; Thompson, 2014). As we have seen, poverty is one such stressor, with longer periods of childhood poverty resulting in higher allostatic loads (Evans & Schamberg, 2009). This biological wear and tear eventually leads to maladaptive physiological responses that increase disease risk and undermine health. Childhood poverty may actually 'reset' the immune system in a manner that increases stress-related impairments in immune function, rates of infectious and chronic diseases, or blood pressure and cardiovascular disease incidence (Miller et al., 2009). On the other hand, the experience of relatively mild early-life adversity helps prepare for the future and promotes resilience to similar challenges in later-life (Daslakakis et al., 2013).

'Matthew' effects¹⁸

Another way in which development is shaped by cumulative experience is through what are known as 'Matthew' effects. Learning and development are cumulative processes in that the skills acquired early form the basis for later skill development (Cunha, Heckman, Lochner & Masterov, 2006; Field, 2010; Rigney, 2010). Thus, the skills children possess when they get to school contribute to a chain of effects that either strengthens and improves their initial skills, or worsens initial difficulties and/or creates new ones (Alexander et al., 2001; Meisels, 1998; Rigney, 2010; Valeski & Stipek, 2001). 'Matthew' effects refers to this pattern of increasing advantage or disadvantage following early advantage or disadvantage (Morgan, Farkas & Hibel, 2008) where the gap between the advantaged and disadvantaged expands with time.

The economist James Heckman and colleagues (Cunha et al., 2006) have also analysed this phenomenon, using basic economic models to understand the life cycle of human skill formation. They view childhood as a multistage process where early investments feed into later investments: skill begets skill, and learning begets learning. They use the concepts of self-productivity and complementarity to explain the evidence on skill formation. Self-productivity describes the way that skill attainment at one stage of the life cycle raises skill attainment at later stages of the life cycle. Complementarity refers to the way that early investment facilitates the productivity of later investment. Together, these two processes explain why skill begets skill through a multiplier process. As a result, the returns to investing early in the life cycle are high, and the remediation of inadequate early investments is both difficult and costly.

18 This term, originally coined by Walberg and Tsai (1983), derives from a line in the Gospel of St. Matthew that says, 'For unto every one that hath shall be given, and he shall have abundance: but from him that hath not shall be taken away even that which he hath' (XXV: 29). Although this originally referred to the process whereby one's faith is strengthened, it is most commonly interpreted as 'The rich get richer, and the poor get poorer'.

Because children's learning is cumulative, early functioning is predictive of later functioning. However, behaviour and functioning at any point in time are actually more strongly influenced by the immediate social and physical environment than by past experience (Feinstein & Bynner, 2004; Lewis, 1997, 2005. Macmillan, McMorris & Kruttschnitt, 2004; van IJzendoorn & Juffer, 2006). The apparent contradiction between these two findings can be resolved by recognising that, for a variety of reasons, children's environments tend not to change (Sameroff et al., 1993). While this is not a problem if these environments are positive and stimulating, prolonged exposure to adverse environments can have adverse long-term effects upon children's development and learning (Anda et al., 2006).

7.1.3 Escalation of risks over time

Development is shaped by developmental escalations in risk over time (Boivin & Hertzman, 2012; Repetti, Taylor, & Seeman, 2002). An exposure or experience at one stage of the life course influences the probability of others later in the life course, as well as associated health and developmental outcomes (Hertzman & Boyce, 2010). This is akin to the notion of chains of risk (Kuh et al., 2003; Rutter, 1989), whereby a sequence of linked exposures raises disease risk because one bad experience or exposure tends to lead to another and then another. That is, risk begets risk, so that one bad adverse experience tends to lead to another. Such causal chains are common in living things and are known in biology as cascades:

Because our biological and psychological traits are caused by cascades of events, development can be understood as being the product of a history of cascading causes in which each subsequent change depends on prior changes

(Moore, 2015).

These sequential chains can, of course, work in the opposite direction, such that some individuals experience 'positive cascades of development' (Lewin-Bizan, Bowers, & Lerner, 2010).

7.1.4 Triple hit effects

The fourth pathway, the triple hit hypothesis, combines elements of the three previous pathways, and helps explain why some people who experience adversities respond with resilience while others develop stress-related physical and mental disorders. This pathway posits that three conditions or events are required to perturb development: an existing predisposition or vulnerability, a critical period of brain development, and exposure to environmental stressors. One version of this hypothesis comes from Casanova (2007, 2014) who argues that the great variation on the number and severity of symptoms observed in conditions such as autism spectrum disorders may be best explained in terms of a triple hit hypothesis. Thus, autism results when three factors combine: an underlying vulnerability (e.g. genetic predisposition), a critical period of brain development (e.g. early developmental plasticity), and environmental stressors (such as viral infections or toxins). Infants with genetic predispositions who are not exposed to environmental stressors are less likely to develop autism.

In another version of the triple hit concept, Daskalakis and colleagues (2013) seek to trace the early origins of adolescent mental health disorders. They propose that these are precipitated when three conditions are met: the adolescent has a genetic predisposition, has experienced a stressful early-life environment (when brains are most vulnerable), and was then exposed to further stressful experiences in adolescence (another period of high brain vulnerability). Similarly, Davis and colleagues (2016) propose that the development of schizophrenia may be driven by genetic vulnerability interacting with multiple vulnerability factors (including lowered prenatal vitamin D exposure, viral infections, nutrition and childhood trauma) during critical periods of neurodevelopmental vulnerability.

7.1.5 Measuring the cumulative effects of experiences and exposures

As depicted in this review, both early and later development are shaped by a wide range of environmental experiences and exposures, and by our biological responses to them. To help understand and measure the cumulative impact of these diverse experiences, exposures and responses, the concept of the exposome has been proposed (Miller, 2013; Wild, 2005, 2012). Conceived as a complement to the genome, the exposome is the cumulative measure of environmental influences and associated biological responses throughout the lifespan, including exposures from the environment, diet, behaviour, and endogenous processes (Miller & Jones, 2014). In contrast to the genome, the exposome is highly variable and dynamic and evolves throughout the lifetime of an individual (Coughlin, 2014).

The exposures that are taken into account by the exposome concept go far beyond those that are quantitatively evaluated in environmental epidemiology today (Coughlin, 2014). Various exposome initiatives are being undertaken, such as one that analyses baby teeth: because they form rings as they grow (as trees do, but daily instead of annually), baby teeth can reveal everything that an individual has been exposed to, including environmental toxins and stress hormones (Cernansky, 2016). These initiatives have led to proposals for a Human Exposome Project, an environmental analogue to the Human Genome Project (Niedzwiecki & Miller, 2017).

7.1.6 Summary

Beyond the first 1000 days, children's and young people's ongoing development and health are shaped by a combination of three processes: the way in which their biological development has been shaped by their earliest experiences and exposures, the extent to which their subsequent experiences and exposures are predominantly positive or negative, and the extent to which any negative experiences induce further negative experiences.

The underlying message here is that development is not a simple process where by an experience or exposure at one point in time will lead directly to a developmental outcome at a later point. As we have seen, development is always contextual (shaped by environmental experiences and exposures), transactional (the child both shapes and is shaped by the environment), and multi-determined (outcomes are the result of a combination of factors rather than any single factor.)

7.2 The long-term outcomes of early experiences and development

The evidence we have considered in this review highlights the considerable influence that experiences and exposures during the first 1000 days can have on health and development. Key questions to be addressed are: How much change is possible after the first 1000 days? How permanent are the effects of early experiences?

These are not easy questions to answer. As Prescott (2015) notes, it is difficult to answer exactly just how much of the non-communicable disease risk seen in ageing can be attributed to early life:

An 'unhealthy' start to life will reduce biological reserves, but this is then overlaid by maladaptive responses, and then by ongoing unhealthy behaviours. Some of this is impossible to regain. If we have fewer nephrons in our kidneys, if we have fewer islet cells in the pancreas, if our peak bone density is low, and if we have fewer neuronal synapses in our brain, our 'reserve' will be lower. We will cope less well with the age-related decline in all of these tissues. And we will be less resilient to challenges. Then, superimpose the added maladaptive metabolic responses that underlie conditions such as obesity and metabolic syndrome. Once these become established, they are very hard to change.

Nevertheless, there is evidence that many aspects of health and development continue to be shaped by later experiences and exposures. This review has identified the first 1000 days as the period of greatest developmental plasticity, and described the acute sensitivity of the foetus and infant to environmental experiences and exposures during this period. However, developmental plasticity does not end there, but continues to play a role in our ongoing development and functioning throughout our lives. The degree of plasticity is reduced, however: it takes stronger and more sustained environmental experiences to change us.

Epigenetic plasticity. The epigenetic changes that are passed on by parents or induced by adverse experiences in the womb or infancy are not fixed for life, but can be modified by subsequent experiences. As Moore (2015) points out, this is both good and bad news:

On the one hand, it means that even if a harrowing experience in childhood contributes to a person having a post-traumatic stress disorder today, the person will not necessarily remain symptomatic in the future; treatments for these symptoms exist, and continue to be refined. On the other hand, it means that there is no pre- or perinatal program of epigenetic manipulation that can ever ensure smart, happy, healthy offspring, because intelligence, contentment, and healthiness are characteristics that develop over many years of postnatal life

(Moore, 2015).

Telomere plasticity. While telomeres can be shortened by a variety of stressful experiences and unhealthy environments and exposures, they retain a significant degree of plasticity. Telomere shortening due to adverse experiences can be restored through exposure to positive environments, so the impact of early adverse experiences or inheritance can be counteracted through positive experiences, exposures and health practices. Telomere maintenance occurs through the activation of telomerase, an enzyme that counteracts shortening by adding DNA and building back the chromosome end each time a cell divides (Blackburn & Epel, 2017; Prescott, 2015).

Neurological plasticity. Beyond the first 1000 days, the brain continues to be capable of changing its structure and function in significant ways, and possesses a degree of neuroplasticity that is much greater than previously recognised (Doidge, 2007; Begley, 2009; Davidson & Begley, 2012). These changes come about in two ways – through experiences (external input) and through our own thoughts and intentions (internal input) (Davidson & Begley, 2012; Siegel, 2012). However, young children have not yet developed the capacity to modify their own behavior and brain structure through thoughts and intentions – that is one of the things they have to learn from adults – so are much more shaped by the external environmental experiences, especially their relationships with caregivers, than are older children and adults.

7.2.1 Summary

The experiences during the first 1000 days do not determine our subsequent development and health over the lifecourse. A measure of developmental plasticity is retained over the lifecourse – epigenetic changes can be modified, and brains rewired – it takes stronger and more sustained environmental experiences to change us.

The continuance of developmental plasticity means that we continue to be shaped by environments and experiences. This means that positive early experiences do not ‘inoculate’ us against subsequent adverse experiences – although they can convey greater resilience when confronted with later challenges. As Sameroff (2009) has observed,

Children are neither doomed nor protected by their own characteristics or the characteristics of their caregivers alone. The complexity of the transactional systems opens up the possibility for many avenues of intervention to facilitate the healthy development of infants and their families.

What this means is that we need to maintain support and provide appropriate intervention beyond the first 1000 days for those who have experienced a potentially compromised beginning to life. This involves maintaining positive conditions for families and children throughout childhood and adolescence. For children, these positive conditions include positive family relations, social support, safety (both physical and social), healthy environments, optimal nutrition, and exposure to natural environments. For families, positive conditions include supportive social networks, secure housing, secure employment / finances, healthy home environments, safe community environments, and ready access to family-friendly services and facilities.

8. Implications and key messages

8.1 Implications for action

The evidence we have reviewed tells us both how important our experiences and exposures during the first 1000 days are, as well as how pervasive they are. As summarised by Prescott (2015),

The importance of a healthy start applies to virtually all body systems. Adverse conditions in early life can have lasting effects on all aspects of growth and development. These very early effects on both structure and function shape our physiological (functional), immune, metabolic, and even psychological and behavioural response patterns to the environment, and can have lifelong effects. Most importantly, these effects can influence our susceptibility to diseases decades later.

Prescott (2015) goes on to argue that promoting 'optimal conditions' in early life is the best hope we have of hardwiring 'healthy' physiological, structural, immune, metabolic and behavioural-response patterns in order to prevent so many avoidable diseases.

There are three distinct developmental periods during the first 1000 days when actions to promote better outcomes can be taken: preconception, pregnancy and infancy.

- **Preconception.** The evidence has highlighted the impact that the health and wellbeing of parents prior to conception can have on the foetus from the moment of conception (Barker, 2015; Barouki et al., 2012; Chavatte-Palmer et al., 2016; Genuis & Genuis, 2016; Lane et al. 2014; Sun, Velazquez & Fleming, 2016). This has led to calls for specific strategies for both preconception and interconception care (Frayne et al., 2016; Genuis & Genuis, 2017; Ratcliffe, Rosener, & Frayne, 2017). Preconception care is defined as a set of interventions that aim to identify and modify biomedical, behavioral, and social risks to a woman's health or pregnancy outcomes through prevention and management. It is being advocated as a means to positively address the rising rates of adverse gestational and paediatric illnesses (Genuis & Genuis, 2017). Preconception care involves a two-pronged approach: (i) securing nutritional and physiological sufficiency—ensuring mothers have what they and the developing foetus require for optimal health, and (ii) avoiding or minimizing toxic exposures that predispose to adverse outcomes (Genuis & Genuis, 2017). Barker et al. (2016) see the preconception period as a 'teachable moment' for changes in diet and lifestyle. Interconception care is care provided to women beginning with childbirth until the birth of a subsequent child. It is a subset of preconception care that addresses the continuity of risk from one pregnancy to the next (Ratcliffe et al., 2017).
- **Pregnancy.** In this review of the evidence, the importance of development during the nine months in the womb has been highlighted again and again (Barouki et al., 2012; Barker et al., 2016; Robinson, 2013; Roesenfeld, 2015). The factors that impact on the health and development of the foetus during this period include nutrition, stress and exposure to environmental toxins. Given the importance of this phase of development, we need to rethink how best to support mothers and protect their babies during this period.
- **Infancy.** The importance of supporting parents and infants in the first two years after birth has been recognised for decades, and the factors that impact on health and development during this period are more widely understood than those that affect development in pregnancy (Barouki et al., 2012; Cozolino, 2014; Gerhardt, 2014; Gopnik, 2009; Siegel, 2012). However, this knowledge has yet to be translated into a comprehensive and integrated approach to supporting parents and infants during this crucial period.

This review has identified a number of specific aspects of health and development which have implications for action to improve outcomes. These include our cellular health, microbiome health, nutrition, and environmental toxins.

- **Promoting cellular health.** The molecular biologist Elizabeth Blackburn and the health psychologist Elissa Epel (2017) believe that our cellular health is reflected in the wellbeing of our minds, bodies and even communities. They conclude their account of telomere effects with a Telomere Manifesto that outlines the key actions we can take individually and collectively to maintain our telomeres and contribute to a healthier world. At the individual level, they emphasise stress reduction, exercise, longer sleep, healthy eating, close relationships, and exposure to nature. At the collective level, they recommend improving prenatal care, protecting children from harm, reducing inequality, cleaning up environmental toxins, and improving food policies to ensure everyone has access to fresh, healthy and affordable food.
- **Promoting microbiome health.** While microbiome health is a relatively new and less well understood area of intervention, it is clearly of great importance for health and development (Blaser, 2014a; Dietert, 2016; Mayer, 2016; Prescott, 2015). We now understand that the microbiome acts as a 'personalised organ that can be modified by diet, lifestyle, prebiotics, probiotics, and antibiotics' (Indrio et al., 2013). This means we can overcome some of the major health challenges of the twenty-first century by learning how to ensure the early establishment of a healthy and diverse microbiome in infants (Dietert, 2016), understanding more about the interface between the microbiome and the immune system (Logan, Jacka & Prescott, 2016), and identifying mechanisms to re-establish a healthy complex microbiota after dysbiosis has occurred (Petersen & Round, 2014).

Three experts in this area offer a range of suggestions. The immunologist Rodney Dietert (2016) stresses the importance of building a healthy superorganism from birth, beginning with prompt 'seeding' of a baby's microbiome. He extols the virtues of breast milk as the ideal food both for the baby and the baby's microbiome (it contains certain sugars that our mammalian cells cannot digest but that are needed by our microbes). The microbiologist Martin Blaser (2014b) focuses on the specific issue of 'missing microbes', that is, the loss of diversity in our microbiomes. He recommends that we curb our use of antibiotics, stop feeding them to the animals we eat, and stop producing broad-spectrum antibiotics. (Strategies for reducing inappropriate antibiotic prescribing are now being developed, eg. Hersh & Kronman, 2017). We should also stop using so many sanitisers on ourselves and our children, reduce the use of caesarean section births, and continue to explore ways of restoring microbes to people with the damaged intestinal ecosystems. The gastroenterologist Emeran Mayer (2016) focuses on how and what to feed our gut microbes, recommending that we seek to maximise gut microbial diversity, cut down on fat, avoid processed foods, eat fermented foods and probiotics, eat smaller portions, fast periodically, avoid eating when stressed or angry or sad, and enjoy meals with others.

- **Promoting healthy nutrition.** The evidence is clear that early nutrition plays a major part in shaping children's lifelong health and wellbeing (Barouki et al., 2012; Prescott, 2015). This is one of the areas that is best understood, so there are guidelines available covering all periods of early development, including preconception (eg. Davies et al., 2016a, 2016b; Gluckman, Hanson, Seng & Bardsley, 2015). The paediatrician Susan Prescott (2015) sees nutrition as one of the most important and easily modifiable environmental factors in early life, and includes the recommendations on early life nutrition developed by the Australia and New Zealand Early Life Nutrition Working Party (Davies et al., 2016a, 2016b) as an appendix to her book. These recommendations cover the preconception period as well as nutrition during pregnancy and infancy. Prescott also recommends reforming the food industry, noting that while food producers are required to adhere to strict regulations to prevent acute poisoning, there are no regulations regarding the chronic food poisoning caused by their high-fat, high-salt and high-sugar products.

- **Promoting healthy environments.** The evidence clearly indicates that chemical exposures during pregnancy and early development are a major cause of neurodevelopmental disorders and can have a profound and life-long impact on human health (Heyer & Meredith, 2017; Schettler, 2010; Wang, Padula, Sirota & Woodruff, 2016). Given the complexity of the underlying mechanisms and their prenatal inception, treatment options are currently limited (Heyer & Meredith, 2017), so our efforts should be directed to trying to reduce exposures to environmental toxins in physical environments and food and consumer products by increasing public awareness and improving government and industry guidelines (Barouki et al., 2012; Gore et al., 2015; Heyer & Meredith, 2017; Landrigan & Goldman, 2011; Schettler, 2010; Wang et al., 2016). Ways of achieving this have been suggested (Afzal, Witherspoon & Trousdale, 2016; Children's Environmental Health Network, 2015; Wang et al., 2016).

The environmental health specialist Ted Schettler (2010) recommends introducing a screening program for endocrine disrupting chemicals in consumer products, air, food, and water; routinely testing the neurodevelopmental toxicity of pesticides; and phasing out any persistent, bioaccumulative toxicants in products to which people could be exposed. The World Health Organisation (2017) also focuses on the environmental health of children, recommending that we seek to reduce air pollution inside and outside households, improve safe water and sanitation, protect pregnant women from second-hand tobacco smoke, and build safer environments.

In considering how we might best address the wide range of possible interventions, the evolutionary biologist Daniel Lieberman (2013) suggests there are four possible courses of action:

- The first is to **let natural selection sort the problem out.** However, Lieberman notes that, although human evolution is not over, the chances of natural selection adapting our species in dramatic, major ways to common non-infectious mismatch diseases are remote unless conditions change dramatically.
- The second course of action is to **invest more in biomedical research and treatment.** While we should keep investing in fundamental biomedical research to promote further advances, Lieberman suggests we can expect little more than slow incremental progress at this stage. The developmental cognitive neuroscientist David Moore (2015) agrees, noting that, while research on epigenetics will ultimately yield revolutionary insights, this research has not yet produced many novel, trustworthy recommendations. For instance, although the evidence strongly suggests that our diets have significant effects on the functioning of our genes, we are still a long way from being able to prescribe diets that will protect babies from neurological conditions like autism. And no one currently knows how we might be able to use the results of epigenetics research to help us raise happy, healthy babies. Moore concludes thus:

The conclusions we can draw from behavioural epigenetics research are actually no different from the conclusion we can draw from a broader understanding of development. The research on epigenetics ... merely helps drive these points home. But the points are so valuable and so often overlooked that even if the takeaway lessons of behavioural epigenetics are the same as the lessons provided by 20th-century developmental science, they are worth rediscovering in the context of new data.

While this conclusion reflects our current state of knowledge, we can expect that further research will not only shed more light on the mechanisms underpinning development but also show us what we can do to prevent disturbances of development or ameliorate their effects. Such interventions are likely to take two forms. One will be at the biological level and involve highly sophisticated procedures at the genetic / epigenetic level – such as snipping mutant genes in embryos where there is a known carrier of a fatal disease, as recently reported in *Nature* (Ma et al., 2017). The other will be similar to personalised medicine but take the form of personalised environments: on the basis of knowledge about a child's particular genetic / epigenetic inheritance and their early exposures and experiences, we will seek to provide environments that are tailored to the child's individual needs. These environments will be designed to promote the biological, neurological and psychosocial characteristics that are poorly developed as a result of the child's inheritances and early history, while also protecting them from exposure to conditions that are known to be particular risks for the child in question.

- The third course of action identified by Lieberman is to **educate and empower**, that is, to provide people with useful, credible information about how their bodies work and what they need to do to optimise health and wellbeing. However, Lieberman argues, knowledge is not enough on its own - we also require motivation and reinforcement to overcome basic urges in order to make healthy choices in environments replete with plentiful food and labour-saving devices.
- Hence, the need for the fourth course of action, which is to **change the environment**. Lieberman's argument here is that, since all diseases result from gene-environment interactions, and we cannot reengineer our genes, the most effective way to prevent mismatch diseases is to re-engineer our environments. This is also the argument made by the epidemiologist Michael Marmot (2015, 2016) who emphasises the importance of addressing the social determinants of health and disease, the 'causes of the causes'. One of the principle causes is the growing economic inequalities that are evident in many developed nations, including Australia. Marmot also argues that the best way to reduce social inequities in health at older ages is undoubtedly to start at the beginning of life.

The argument that we should seek to change the environment and address the conditions under which families are raising young children is supported by a number of others (Blackburn & Epel, 2017; Moore & McDonald, 2013; Prescott, 2015). For example, Prescott (2015) argues that trying to address only the superficial 'causes' of non-communicable diseases (such as bad nutrition, smoking and inactivity) without addressing the wider 'causes of the causes' (the social, cultural and economic determinants of health) will be certain to fail. And Blackburn and Epel (2017) call for policy of societal stress reduction in the form of broad social policies that have the goal of buffering the ubiquitous socioenvironmental and economic chronic stressors faced by so many.

A number of general strategies to guide future actions have been identified in this and other evidence reviews.

- **Holistic approach.** Humans are complex systems and no single factor affecting health and development should be seen in isolation (Kappagoda, 2013). Most chronic disease processes are characterised by multi-causality and complexity, and understanding such processes requires a more holistic approach that focuses on systems (International Scientific Committee of the International Conference on Fetal Programming and Developmental Toxicity, 2007; Kappagoda, 2013).

- **Multilevel approaches.** Conditions such as obesity are multifactorial, and prevention strategies need to address the individual, the family, the physical environment, the social environment, and social policy (Campbell, 2016). To improve long-term outcomes for children experiencing significant levels of disadvantage, a multilevel, ecological approach is required involving actions at three levels: program level interventions delivered directly to children and families; community and service system level interventions that seek to build more supportive communities and better co-ordinated and effective service systems; and structural and societal level interventions that address the structural (e.g. government policy) and wider social factors (e.g. attitudes and values) that influence child and family outcomes (Moore & McDonald, 2013; Moore et al., 2016a). Key elements of this approach include service systems based on principles of progressive or proportionate universalism (Barlow et al., 2010; Boivin & Hertzman, 2012; Feinstein, Budge, Vorhaus & Duckworth, 2008; Human Early Learning Partnership, 2011; Marmot Review, 2010; Statham & Smith, 2010) and place-based or collective impact approaches (CCCH, 2011; Moore, 2014b; Moore et al., 2014b; Moore & Fry, 2011; Moore, McDonald, McHugh-Dillon & West, 2016b).
- **Flexible and responsive service systems.** In addressing complex and ‘wicked’ problems, service systems need to become more agile and responsive than they have been traditionally (Daniel, Taylor & Scott, 2010; Fox et al., 2015; Moore et al., 2016a). This means engaging families and communities as partners and co-producers in determining what local conditions need to be addressed, the goals being sought and what strategies are to be used to achieve them (Dunston et al., 2009; Moore et al., 2016b).
- **Prevention.** The value of prevention as a general strategy in human services has been recognised for decades (eg. Chehimi & Cohen, 2013; Cohen, Chehimi & Chavez, 2010; Cohen & Iton, 2014; Coie et al., 1993), and has been highlighted several times in this review as being critically important during the first 1000 days (eg. Balbus et al., 2013; Barouki et al., 2012; Hanson & Gluckman, 2015; Prescott, 2015). A true prevention approach addresses the underlying causes of problems (O’Connell, Boat & Warner, 2009; Maziak, Ward & Stockton, 2008; Stagner & Lansing, 2009) and seeks to improve the conditions under which families are raising young children. As Prescott (2015) states, ‘promoting optimal conditions in early life is the best hope we have of hardwiring ‘healthy’ physiological, structural, immune, metabolic and behavioural-response patterns in order to prevent so many avoidable diseases.’

8.2.1 Conclusions

A huge amount of research has been summarised in this review, yet clearly there is plenty of scope for more. Despite the argument that we should not expect quick answers, basic research on the neurological and biological processes underpinning development should continue to be supported. Given the wide range of disciplines involved, interdisciplinary research should be encouraged, and ways of integrating research findings explored.

In terms of taking immediate action to promote a healthy start to life, the third and fourth courses of action identified by Lieberman (2013) – educate and empower, and change the environment – are the most viable.

The *educate and empower* strategy is essentially a public health approach. Barker (2015) argues that empowerment should form the basis of a new approach to public health, pointing out that an empowered public demand for better access to better food could go a long way towards improving maternal, infant and family nutrition, and therefore the health of generations to come. Developing better ways of educating and empowering the general public about the evidence identified in this paper should be a priority. From a policy perspective, a population-wide public health strategy is clearly indicated.

The *changing the environment* strategy involves efforts to improve the conditions under which families are raising young children, as well as strategies to address the social determinants of health and wellbeing, and reducing the social inequities that create social gradients in health and development (Carey & Crammond, 2015; The Marmot Review, 2010; Tarazi et al., 2016). Central to shifting the current social gradient in health outcomes, and the social and economic burden of chronic diseases is health equality starting in the first 1000 days. This can only be done through a coordinated policy approach which addresses the needs of children from conception, thus laying the foundation for their future health and wellbeing (The Marmot Review, 2010). As such, governments must adopt a social determinants framework across the policy and programmatic functions of all government departments and strengthen its leadership role in supporting a social determinants approach that addresses the gradient across the whole of government (Commission on the Social Determinants of Health, 2008).

There is a fifth course of action that is not mentioned by Lieberman and that has been beyond the scope of this review – provide service-based interventions to promote effective parenting or to address specific problems. This has been the default approach adopted by governments and service providers, and will continue to play an important role in ensuring the health and wellbeing of children and families. However, relying solely on targeted health and other services has not been sufficient to make a significant difference to the complex health problems that are prevalent today, and by ignoring the holistic nature of childhood development this approach risks jeopardising children’s optimal development. This course of action needs to be complemented by the two strategies above, educating and empowering communities, and changing the environments in which families are raising young children during the first 1000 days. A reduction in chronic diseases and developmental problems is more likely to result from the combined effect of a range of social, health and economic policies that are based on equity and the even distribution of resources.

8.2 Key messages

- During the first 1000 days, the developing foetus and infant are at their most vulnerable to external exposures and experiences, good or otherwise. At the same time, developmental plasticity is at its greatest, giving us the biological capacity to adapt to the particular physical, social, and nutritional worlds we are born into. This is a powerful capacity that contributes greatly to humans’ success as a species, but it is a double edged sword – adapting to adverse experiences may help in the short term but have negative biological and developmental implications in the long-term.
- What makes this new knowledge about developmental plasticity and programming so important is the evidence that the changes made during the first 1000 days can have lifelong effects. Environmental exposures such as stress or undernutrition during critical periods of development can have long-term effects on chronic disease risk by ‘programming’ organs, tissues, or body system structures or functions. Adult conditions such as coronary heart disease, stroke, diabetes, and cancer that once were regarded solely as products of adult behaviour and lifestyles are now seen as being linked to processes and experiences occurring in pregnancy or infancy.
- Instead of being passive during pregnancy, the foetus actively responds to changes in the intrauterine environment, using the cues provided by the mother’s bodily and mental states to predict the kind of world they will be born into and altering their own bodily structures accordingly. This works well when the antenatal and postnatal environments are optimal and stable, but when these environments are less than optimal and when the prenatal and postnatal environments do not match, the adaptations made by the foetus can compromise later health and development.
- These processes are important because they affect every aspect of our development and our functioning. This is because all bodily systems function as an integrated system – the mind and brain and body function as a unit, and what happens in one bodily system affects all others to a greater or lesser extent.

- This integrated system also includes the microbiome, all the bacteria and other microbes that live on us and in us and with whom we have co-evolved. So closely linked is our development and health with that of our microbiome that humans can be considered to be superorganisms rather than a stand-alone species.
- The range of factors that are known to impact upon biological and developmental functioning during the first 1000 days is considerable. Some predate conception – parental health and lifestyle, and even grandparental nutrition and experiences of trauma can result in transgenerational transmission (genetic, epigenetic, telomeres). Thus, what children inherit from their parents (and grandparents) is not just their genes, but also non-genomic changes that can place them at greater risk of disease and other developmental problems. Others factors that affect biological and developmental functioning occur during pregnancy (e.g. maternal physical and mental health, maternal nutrition, maternal stress, maternal consumption of teratogens, exposure to environmental toxins) or at birth (caesarean section births, premature births). Still others occur in infancy (e.g. parental neglect and abuse, poor nutrition, exposure to environmental toxins).
- When we consider the factors that underlie these factors – the ‘causes of the causes’ – we can see that the conditions under which families are conceiving and raising young children have a profound and direct impact on their capacity to care for and raise the children as they (and we) would wish. Our development and functioning are ecologically shaped – the immediate physical and environments in which we are raised are what we respond and adapt to. Parental and family functioning are also ecologically shaped.
- What makes this so significant is that we have dramatically altered the physical and social ecology over the past 60 or so years, a period of unprecedented change with unpredictable effects. The combined effect of these changes is to dramatically alter the conditions under which families are conceiving and raising young children. While much of the economic, technological and health developments have been beneficial for most people, they have also had unintended consequences that we are now beginning to recognise. These range from the growth of social inequities (with accompanying social gradients effects on development and functioning) to alterations to the composition of our microbiomes, which have evolved with us and play an important role in maintaining our health and wellbeing.
- Rapid social, economic, technological and ecological changes have also altered the nature of the problems facing society and governments: these are now more likely to be complex or ‘wicked’ problems that cannot be resolved using traditional governance and leadership models or by service-driven approaches. Examples of these entrenched wicked problems include child protection, family violence, Aboriginal disadvantage, social exclusion, health inequalities, entrenched poverty, and obesity. Some wicked problems (e.g. poverty, child abuse) are not new, but have become more of a concern because of an increasing awareness regarding the adverse consequences of these problems upon child development, and the complex nature of their underlying causes.
- Another result of rapid social and other changes is that modern living conditions are very different from those that our bodies were evolutionarily adapted for, and fail to provide the kinds of normal stressors that our bodies need to develop in a healthy fashion. As a result, we are subject to a wide range of ‘mismatch’ physical and mental health conditions, many with origins in the first 1000 days. These are chronic conditions, and are known as non-communicable diseases to distinguish them from the acute communicable diseases that were the focus of health services in much of the 20th century. These non-communicable diseases or conditions are now so prevalent as to constitute 21st century epidemics.

- Rapid advances in scientific technologies have prompted the emergence of new scientific sub-disciplines and an explosion of new knowledge about the underlying mechanisms whereby experience shapes biological and neurological development. These mechanisms involve changes at a cellular level (telomeres), biological level (epigenetic processes), neurological level (synaptic growth and pruning), and the microbiome level. As yet, we do not know how to intervene directly with these mechanisms, although continued growth in our understanding of how they operate may eventually make such interventions possible. However, we do understand a lot about what causes these various mechanisms to change in the way that they do, and therefore what we need to do to ensure that such changes have positive developmental and biological outcomes.
- A key question concerns how permanent or otherwise these changes are. Some changes are partly or largely reversible through changes in the environments – telomere shortening can be reversed, brains rewired, and epigenetic effects modified. These changes can be for better or worse; the effects of early adverse experiences can be ameliorated through exposure to safer, more responsive and more stimulating environments, but a positive start to life may be compromised if later social and physical environments are markedly less positive. However, other changes may not be so reversible. The prenatal biological sequence and timetable for some organ development is completed at birth and cannot be altered, so that any perturbations resulting from adverse exposures, infections or random mutations during pregnancy can leave lasting effects. Some of these changes have ‘sleeper’ effects, being detectable many decades after the precipitating experience while having little obvious impact on health and wellbeing during the intervening period.
- Whether the effects of experiences and exposures in the first 1000 days have long-term effects depends in some cases on what happens later in childhood and later life; sometimes a double or triple ‘hit’ later in childhood or adolescence is needed to trigger severe disturbances (as in the case of schizophrenia). If children and young people can be protected from these later adverse experiences, then the early adverse experiences or inheritance will not lead to adverse outcomes.
- However, reversing early adverse adaptations or inheritances gets progressively harder after the first 1000 days as developmental plasticity is reduced. Moreover, children exposed to adverse environments and experiences early are likely to continue to be exposed to such experiences and therefore accumulate adverse experiences. In this way, a poor start to life in the first 1000 days may be the start of a cascade of events that reinforce the earlier neurological and biological adaptations. While it is never too late to make changes in our lives, the first and best opportunity we have for change is during the first 1000 days.
- A huge amount of research has been summarised in this review, yet clearly there is plenty of scope for more. Although we should not expect quick answers, basic research on the neurological and biological processes underpinning development should continue to be supported. Given the wide range of disciplines involved, interdisciplinary research should be encouraged, and ways of integrating research findings explored.

9. Final comment

The rapid social, economic and technological changes that have occurred over the past half century or so have been enormously beneficial in many ways. There have been dramatic improvements in longevity, standards of living, and health care, along with reductions in violence. But these gains have come at a cost that we are only just beginning to recognise - in the form of the epidemics of non-communicable diseases that threaten to reverse the longevity gains, the growth in inequalities and the corrosive effects these are having on social trust and capital, the emergence of wicked / complex problems, and even climate change. This is the 'social climate change' story – how the dramatic changes that have occurred in our societies over the last half a century have affected the conditions under which families are raising young children, and therefore children's developmental and health outcomes. While some of these changes are for the better, some appear not to be. Our task is to understand the mechanisms that underpin development, learn how these can be disrupted by adverse experiences and exposures, and identify the environmental changes that are having these adverse effects so we can address them. This paper is a progress report on where we have got to with that task.

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