Chapter 1

Puberty and adolescence: transitions in the life course

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Summary

- With increasing frequency, there are discordances between biology, social and physical environments (often termed 'mismatch') with repercussions across the life course.
- Social evolution has intensified in recent decades and has had a particularly strong impact on human health via substantial changes in family and social structure, lifestyle, nutrition and physical activity, and in human interactions via increasingly complex social environments.
- Transitions during the life course are particularly sensitive periods wherein this mismatch may be observed to have long term consequences. Adolescence itself as a transition period from childhood to adulthood has become prolonged relative to recent recorded history, and both expected and observed behaviours have subsequently changed as well.
- Strongly supported by experimental and observational physiological, neurological and sociological data, this prolongation of physical and social development has both positive and negative consequences for an individual.
- However, society is ill-equipped to mitigate the negative effects of these changes, resulting in increased observations of antisocial behaviour on a large scale.
- Recent studies suggest that events in the early life period most strongly influence later life health, behaviour and functionality, all of which influence social and physical health and economic productivity on both an individual and a population scale.
- Investments in early life that target both cognitive and non-cognitive skill formation have seen substantial economic rewards, but it is increasingly observed that non-cognitive skills have a broader impact on social adaptation and positive social outcomes.

- Later interventions as discussed elsewhere in this volume are also effective, but there are considerable data to suggest that prevention has broader effectiveness than remediation.

1. Introduction: a life course perspective

All mammals, including humans, have distinct phases to their lives. Some such as the transition from being a fetus to being a newborn are abrupt; others such as the transition from being a child to being an adult are gradual. The human life course comprises the fetal period, infancy, childhood, a pre-pubertal juvenile period, adolescence, adulthood and senescence, and generally extends in modern times over 7 to 8 decades. The transition from being pre-reproductive to being reproductively competent – that is, the process of puberty – starts some years after birth, and while males stay reproductively competent for the whole of their lives, women cease to be able to conceive at or prior to menopause. This slow life course with a long prepubertal period is distinct to the human and several other long-living species which have relatively few offspring (compared to other mammals) who receive a high parental investment over many years after birth [1]. The pre-pubertal juvenile period is essentially unique to humans [2], and refers to that period after about the age of 7 years when independent living is possible but does not occur; this phase is generally accepted as having evolved to allow humans to achieve greater cognitive skills before entering puberty, given the essential role of learning in our species.

Humans are, of course, more than their evolved biological componentry. We live within social networks and in social contexts, and this social environment – which has its evolved basis – creates, and merges with, the physical environment to interact with our biology and make us what we are. But arguably we are organisms that, more than other mammals, rely on learning and past experience to influence both our individual and social behaviour.

Recent research extends this paradigm considerably: past experience influences not only our behaviour through learning, but both our biology and our behaviour through chemical changes in the way our genes our controlled – a process termed epigenetics. This leads to long-lived (effectively permanent, although putatively reversible) changes in the way both our brain and other aspects of our biological systems respond [3, 4]. It is this information that leads to the crucial understanding that events in any one part of the life course can have important influences on how subsequent parts of our life course play out. We know, for example, that events before birth can have life-long consequences that manifest as a greater risk of heart disease or diabetes in adulthood [5]. And increasingly we recognise that events before birth, in infancy and in childhood have major influences on how the transition from child to adult – that is through puberty and adolescence – plays out [6]. Indeed, two long-running New Zealand studies based in Dunedin and Christchurch have been instrumental in establishing such influences (for example, [7, 8]). A major thrust of other chapters in this report is to highlight that, because of the critical importance of early life developmental factors, it is inevitable that if the transition through adolescence is to be improved, greater attention must be given to earlier stages in the life cycle.

It is self-evident that the human condition at any point in the life cycle is a result of genetic biology, developmental biology, learning, past and current environments and culture (in its broadest definition) interplaying with each other. Yet, because different academic disciplines have been focused on particular components which are well separated in their academic traditions, it is only recently that a greater integrated understanding of these interactions has emerged. These differences have been most pronounced in the artificial

but long standing 'nature' versus 'nurture' debates, where nature is generally used in a folk-biological sense to mean innate (which itself has multiple meanings [9]) or genetic and therefore not changeable, and nurture refers to learning and the context of the social and cultural environments. This has led to simplistic views of what is alterable and what is not, and to overzealous claims from different academic disciplines which have limited our understanding of human development. The emergent biologies of life history theory and epigenetics allow these concepts to be integrated – in effect, they cannot be separated [10]. Thus when a period of transition in the life course is being considered, such as puberty/adolescence, there are biological, developmental, social and cultural factors all at play. This chapter dissects these elements for explanatory purposes but they are of course intertwined. Evolutionary perspectives integrate these domains and are therefore a particularly useful heuristic.

2. Puberty

Puberty can be defined as the period in which a juvenile's previously inactive gonads (testes in the male; ovaries in the female) become activated, with the ensuing hormonal changes leading to physical and psychological changes allowing for reproductive competence. Puberty is not an instant in time; it takes 3 to 5 years to occur. It generally starts somewhat earlier in girls at between 7 and 12 years of age, and between 8 and 13 years of age in boys. In girls the first signs of puberty are the appearance of breast buds and then pubic hair. As these develop there is a growth spurt; late in the growth spurt ovarian action becomes cyclic, the girl starts having periods and she becomes fertile. In boys the first sign of puberty is the start of testicular enlargement followed by the development of pubic, armpit and facial hair, a change in the positioning of the larynx (leading to the voice breaking), the onset of sperm formation and a growth spurt. In both boys and girls this process leads to changes in body composition, with boys in particular becoming more muscular. The initiation of puberty is due to changed functions in the hormone control region of the brain called the hypothalamus, which starts to release a master control hormone; how this is switched on is poorly understood [11]. The physical features of puberty are reflected in an increase in sex hormones (primarily testosterone in boys; estrogen and progesterone in girls) released in the circulation by the activated gonads. These hormones also affect brain function, affecting the number of connections in the brain and perhaps the number of brain cells [12]. As a result the progression of puberty is associated with the development of certain aspects of brain function and, in particular, the development of psychosexual thoughts which are reflected in the interests and behaviour of the young person transitioning puberty. There is a presumed relationship between this psychosexual maturation and the changed way young people socialise and interact with their peers, and start engaging in attention seeking behaviours in relation to these peers.

This distinct life course pattern arose through the evolution of the human species. In evolutionary biological terms, the capacity to reproduce in an appropriate manner at the appropriate time in the life course is a major driver of what shaped our species. Thus it is to be expected that puberty, that is the shift to reproductive competence, is influenced by a number of environmental factors. For example, in modern hunter-gatherer societies the age of puberty is reduced in societies where the rate of mortality is high [13]. This is because the biological drive reflected in evolutionary processes is to ensure successful progeny, which in a risky environment is maximised by reproducing early. Recently Daniel Nettle's team has shown that similar drivers operate in Western societies. Extensive

studies of large UK cohorts showed that the life course is accelerated in those living in poverty ridden circumstances or those who experienced adverse early life familial events, where earlier age of puberty, earlier age of reproduction and shorter life span are observed [14-16]. Similarly, other threatening circumstances (in real or evolutionary terms) such as low parental supportiveness [17], being born small [18], childhood abuse [19] and dysfunctional paternal behaviour [20] can accelerate the onset of puberty. It is worth noting that the latter two studies looked at New Zealand children. Extreme circumstances such as severe childhood undernutrition, severe stress, and illness (e.g. anorexia nervosa) can also delay the onset of reproductive competence, and the processes underlying these occurrences demonstrate biological overrides on the timing of pubertal processes.

The age of onset and completion of puberty in Western populations has markedly declined over the past 200 years [21]. This is best documented for the female where the age at first period (menarche) provides an unequivocal time point in maturation. For European girls, the average age at menarche in the late 18th century was about 17 years; it has fallen progressively and quite rapidly over the next 150 years, then has continued to fall more slowly up to the present time, where it is now approximately 12 years of age. There is of course large variation in individual timing and a mean age of 12 years is associated with a normative range extending from 11 to 15 years. However, because menarche occurs relatively late in the progression through puberty, this means that possibly 50% of New Zealand girls have entered the pubertal process during primary school, and have had their menarche before they leave intermediate school [22]. Two generations ago, it was rare for a primary school girl to enter puberty but now an increasing number will have had menarche at primary school. This is a dramatic change in short time span and highlights the difficulties of imposing parental and grandparental concepts of maturation onto the current generation. There are some population differences in the age at menarche [21], but the extent to which they represent genetic differences or reflect populations in different stages of the nutritional and socioeconomic transition is unknown. In New Zealand, Māori and Pasifika girls tend to experience an earlier onset of menarche, with about 80% menstruating by age 13 compared to 62% of those of European origin; the disparity at age 11 is particularly concerning, with the number of Māori and Pasifika girls reporting menarche exceeding that of European girls by about 2.5 to 3.5 times [22].

While changes in the age of puberty are less well documented in the male they appear to have followed a similar trend of falling considerably over recent generations [23].

The evidence would suggest that this fall in the age of puberty onset – generally termed the secular trend – arose because of a marked improvement in maternal and infant health and nutrition since the Enlightenment in Europe at the beginning of the 19th century. Experimental [24] and epidemiological [25] evidence suggests that the improved state of maternal nutrition is important, alongside the generally accepted role of improved infant health and childhood nutrition [6]. Thus the fall in the age of puberty can be seen as a general reflection of an improvement in maternal and child health. It may be that excessive obesity in childhood further accelerates the fall in the age of puberty, and given the increasing rates of childhood obesity in developed societies to alarming proportions [26], this may be having some additional effect above that of the secular trend. There is little substantive evidence to suggest that the changing age of puberty in the normal population is linked to environmental contaminants such as toxins and contraceptive pills in the water supply, although there have been rare situations such as that of precocious puberty in Puerto Rican children in the early 1980s, suspected to be caused by inordinate hormonal use in farmed animals or by mycotoxin contamination of food [27, 28].

There can be a complex interplay between experiences before and after birth and the age of puberty. Within the normal population, the earliest ages at menarche are seen in those who were smallest at birth and who become relatively overweight in childhood [6, 29, 30]. This can be understood in life history terms [6]. Its most dramatic effects are in children who transition from highly deprived environments early in life to enriched environments in childhood. A classic example is that of girls from developing countries who are adopted in infancy to families in Europe [31]; some of these girls may undergo menarche at as early as 8 years or less and are therefore at considerable disadvantage, both psychosocially and physically, because menarche presages the end of the growth spurt.

While earlier (but not precocious) puberty is generally a sign of good child health in Western society, the problem is that because of the rapid fall in the age of puberty, there is an increasing mismatch which those aspects of maturation which are independent of sex hormone effects. The perception that an individual looks mature and is advanced in puberty can lead to inappropriate assumptions about other aspects of their maturity and behaviour. This presumption can occur both in how others perceive the child and how the child perceives himself or herself. These issues are addressed in more detail in the next section.

3. Adolescence

For the purposes of this report, adolescence is defined as the period between the onset of puberty and the time when the individual is accepted as an adult by the society in which he or she lives. As such it involves a broader set of considerations including societal, cultural, behavioural as well as neurobiological aspects in its definition. There are clearly major biological substrates to progression through adolescence. Some aspects of brain function mature apparently independently, or at least partially independently, of puberty *per se*. In general processes of executive function including impulse control, judgement, evaluation of risk, reward behaviours and what might be called wisdom are not fully developed by the time puberty itself is complete, as discussed in the following section. Some aspects of psychosocial – as opposed to psychosexual – function also mature later although these probably reflect the executive functions detailed above. Society relies on this cluster of behaviours to assess when an individual can be accepted as an adult. Because this is highly variable among individuals, societies have tended to formalise this through legal ages to define adulthood or maturity. For example the age of legal responsibility, of legal sexual intercourse, of voting, of ability to leave school, of eligibility to join the military, of consenting to a medical procedure, of buying alcohol or hiring a car are all defined by law or regulation – these social milestones vary considerably both within a society and across rather similar democracies. There is therefore a high degree of ambiguity both for the individual and for the society in terms of how an adolescent should be evaluated in a particular situation.

In simple terms, adolescence can be envisaged as a period in which sexual maturation is occurring or has occurred, with neurobiological consequences promoting sexual thoughts, and risk and attention seeking behaviours. However depending on the society in which the individual lives, his or her behaviour, wisdom and judgement may not be seen as fully mature. The contextual variation of this is obvious across different populations: in simple societies such as those of traditional hunter-gatherers adolescence may be very short, particularly for females, whereas in modern Western society adolescence has become very prolonged.

Indeed it has been argued that prolonged adolescence is a relatively recent phenomenon in Western societies. As stated earlier, puberty had much later onset 100 years ago and for reasons discussed below, most individuals made a rather rapid transition from the pubertal stage to being an adult. When the age at menarche in a girl was between 16 and 17, there was generally a very short transition to being married or joining the workforce by the age of 18 to 20, and being accepted as an adult by that society. But now the age of puberty is much lower and yet, for reasons detailed in the next section, full psychological maturity is not exhibited in most until well into the third decade of life. This suggests that adolescence has extended from perhaps 1 to 3 years in length to between 7 and 15 years in length, with consequences which are at the heart of this report.

This discrepancy has been termed the mismatch of puberty [32], and there are data to suggest that this is a major driver of adolescent morbidity. For example, in girls where the timing of completion of puberty can be readily recorded from the age at menarche, earlier puberty – that is, a greater degree of pubertal mismatch – is associated with a greater risk of sexual activity, uptake of smoking and drinking, drug experimentation, aggression and eating disorders [33-35]. The curve of risk rises noticeably when the age at menarche is below 11 years. In studies of a large Swiss francophone cohort of teenagers subject to extensive psychological evaluation, earlier puberty was linked to a greater risk of acting out behaviours, substance abuse, sexual activity, and depression and anxiety in the young people [36]. In boys this was reflected in a significantly greater risk of attempting suicide. The study had a limitation in that puberty was not prospectively recorded and given the different social contexts in which populations operate, more research would be needed to evaluate whether generalisation of this observation is possible. Regardless, there is no doubt from the literature that earlier onset of puberty and the delayed age of completion of adolescence are associated with higher teenage morbidity [6, 37].

4. Brain maturation and adolescence

4.1 Biological maturation of the brain

In recent years, a wealth of neurobiological data have emerged from studies of Western adolescents suggesting that biological maturation of the brain, as reflected in brain imaging and functional studies, happens much later in life than was generally believed [38-41]. Many neuroimaging studies mapping changes in specific regions of the brain have shown that those regions associated with higher levels of executive function including task initiation and management, self-image, impulse control, judgement, strategising pathways and managing strong emotion, only fully mature well into the 20s [42-45]. Adolescents also have different responses within their brain pathways to reward-initiating stimuli compared to children and adults [46], and this is generally thought to relate to earlier maturation of striatal reward areas of the brain than fronto-cortical self-regulatory control regions [47-51]. An alternate and not mutually exclusive explanation is that the adolescent brain exhibits transient expression of reward-seeking pathways as an inherent part of the maturation of exploratory life skills [46]. Either way these data highlight the distinct features of the adolescent brain, which create challenges for society, families and individuals reflected in the acting out behaviours of adolescence. It explains why the adolescent is more vulnerable to poor decision making and risk taking behaviour [52] and is more sensitive to reward inducing stimuli such as peer pressure, drugs and alcohol [47].

The significance of these neuroimaging data showing late maturation of the brain regions important for executive functions is supported by behavioural studies of psychosocial maturity and other socially responsible behavioural traits in adolescents and adults. These show that 'maturity of judgement' measures such as responsibility, perspective (ability to assess short and long term consequences) and temperance are, on average, fully attained at about the age of 20 years – much later than was generally thought [53, 54]. Therefore, adolescents exhibit less psychosocial maturity than adults despite generally having similar cognitive abilities and being biologically mature.

These findings of asynchronous maturation raise three explanatory possibilities. Firstly, executive brain function has always taken a long time to mature but the late maturing components were not critical to normative behaviours in more simple societies. Secondly, we live in a more complex society than that in which we evolved to live, and as a result it takes longer to accumulate the full range of social skills to operate as a productive member of society. Thirdly, both modern society and the way children are bought up earlier in the life course have changed the manner in which the brain matures. In part this may also reflect the attitudes we bring to bear on young people – that is, our willingness, or lack thereof, to accept that they have reached the age of maturity. The interplay between how we respond to young people and the way their brains and behaviours mature is poorly understood. These are not mutually exclusive explanations and can be distilled to two factors: we live in immeasurably more complex societies than those in which we evolved [32], and we may be changing the way the brain matures as a result of educational and child rearing practices which have emerged in recent decades.

4.2 Societal and cultural influences

There are academic arguments to support each of these explanations, but formal evaluation remains to be undertaken. Several possible societal and cultural changes need to be considered in this context. Firstly, 50 years ago primary school children had relative freedom outside school and undertook considerable independent and unsupervised play activity. Now parental attitudes of care and risk avoidance, and the shift of more women into the workforce, have meant most young children live more regimented lives. In contrast, adolescents previously had limited choices within the school system and their social activities were more regulated and limited. Now, they have a very broad range of choices and parents may tend to feel that they have relatively little control on the boundaries of behaviours that they exhibit. Access to money is very different for the modern adolescent; it equates to more freedom from the parent. This is not just a sociological issue – is it that the switch in rearing practices from a loose-to-tight progression to a tight-to-loose progression has changed the learning experiences that influence the maturation of executive function and its biological substrate in the brain?

Secondly, the human brain largely evolved in the Pleistocene period when humans lived in much simpler societies, generally in clan groups estimated to be between 50 and 150 people in size, with simple social and hierarchal structures [55]. Studies in primates of the part of the brain associated with higher function – the neocortex – show a tight correlation between normative social group size in that species and neocortical size. If humans are fitted on this relationship it suggests that we evolved to live in social networks of about 100–150 people, and Robin Dunbar has listed a number of social units which demonstrate that until recently this was indeed the way humans organised themselves [55]. But it is self-evident that in the modern connected world we live in expanded networks; we interact with far more than 150 people and even if much of that interaction

is telephonic or electronic it is still meaningful. This applies particularly to young people, who are fundamentally changing the shape of their social networks more rapidly than others through the use of the internet and social networks such as Twitter and Facebook. The concept of 'Facebook friends' means that young people are sharing the intimacy of their lives not just with the small family network of old but with literally hundreds of people. The nature of communication has also changed – communication is no longer associated with face-to-face contact, and non-verbal communication through purely electronic means is becoming increasingly pervasive. These sociological changes may also have neurobiological consequences. There is a growing and worrisome literature suggesting that brain structures may be altered by these changing patterns of interaction and communication [56]. Such changes could potentially have functional consequences. For example, there are suggestions that the nature of knowledge assimilation and learning as a result of technology usage has changed, and that this impacts on the ability of young people to learn and to pay attention to tasks [57]. This propensity towards distraction may have far reaching consequences – given the ubiquity of these new technologies, it is likely to have attendant influences on how young people learn and develop social skills [58].

4.3 Effects of the early environment

The third consideration beyond the accumulating evidence that these changed ways of learning and inter-personal exposure might affect the development of brain function and communication skills stems from another growing body of evidence, which suggests that the early environment of parental-infant attachment and of experiential (informal) learning can have permanent effects on brain function and maturation. Some of these effects are mediated through the actions of hormones such as oxytocin and vasopressin [59]. Some of these effects are likely mediated through epigenetic mechanisms which change the settings of gene switches. While most of this science is based on compelling data from animal studies [60], there are supportive data in humans showing that stresses and challenges early in life will change the settings of switches involved in pathways associated with social experiences and stress responses, with permanent effects [61]. Recent studies on suicide victims who had been abused during childhood showed epigenetic changes in the brains that were not found in non-abused victims [62]. Epigenetic changes have also been detected in women who experienced childhood abuse and later developed antisocial behaviour [63]. Attachment between mother and infant is more than simply a behavioural relationship; it involves the action of a number of hormones in the brain, and secure attachment is an important predictor of resilience in later life [64], including higher self-esteem, reduced anxiety and reduced hormonal responses to stress [61]. There is increasing evidence that this involves epigenetic processes and that these effects, which reflect the transmission of social determinants into a biological substrate, may have intergenerational consequences [65, 66]. In turn the same hormones involved in attachment appear to play a role in how individuals respond to each other and in creating cooperative and trusting relationships rather than aggressive tendencies later in life [67]. While this aspect of the science is still emerging, it is reasonable to postulate that these long lasting effects of poor attachment involve not only emotional aspects but also changes in the basic biology of the brain.

4.4 Transcultural differences

There are considerable transcultural differences in adolescent morbidity even among Western countries; New Zealand and other anglophone countries generally compare

poorly to non-anglophone European countries in, for example, teenage pregnancy and abortion rates [68, 69]. These transcultural differences should be informative but little research has been done to understand their basis. The age of puberty is similar across these populations, but what does differ is the balance of focus on early childhood education and support. It has been suggested that cultural differences in early childhood experience explain the disparateness in adolescent morbidity, and given New Zealand's dismal position it is a field of research meriting investment.

4.5 Cognitive versus non-cognitive skills

Such considerations are given greater weight by the work of James Heckman and others [70-72]. It is important to distinguish cognitive function as measured by formal IQ tests from other aspects of brain function, and in particular executive functions mediated through the prefrontal cortex. It is now clear that success in formal education and in many other areas is dependent on this latter class of brain function. Heckman and colleagues have pointed out that in early childhood, not only are formal cognitive skills starting to develop but this is also a critical window in which the broader range of non-cognitive but crucial life skills are established which promote school achievement, job performance and financial security [73]. Indeed, the evidence reviewed in Chapters 2, 3 and 4 highlights the growing data showing that it is the development of non-cognitive skills in early childhood that is critical to successful passage through later life. A large body of research has demonstrated that targeted investment in high risk populations through interventional programmes at this point in the life cycle pays social and economic dividends in terms of reduced incarceration, reduced arrest rates, higher employment and higher earning capacity later in life (described in the final section of this chapter). Once again this reinforces the notion that the biological, social and cultural factors conflate and cannot be separated.

4.6 Implications for research and intervention

While these data point to the vital importance of the early life as a period for intervention, action and targeted prevention, this does not mean that actions later in the life course are not justified or without benefit. However the complexities of the phenomena we are dealing with, and the strong evidence implying biological substrates that are not necessarily irreversible for the behaviours in question, mean that interventions must be well designed, research-based and carefully evaluated – unfortunately this is not always the case as will be discussed in Chapter 22. A framework that focuses on neuroscience within the wider context of adolescent research is needed [74], and there is a compelling case for including the neurobiologist with the more traditional social and behavioural scientist in promoting an interdisciplinary approach to the design of interventions [46].

5. A synthesis

Puberty is a biological process in which the individual's gonads are activated and he or she becomes reproductively competent. In evolutionary terms, reproductive competence would have evolved in parallel with the ability to cope as a reproductively active member of the society in which the individual lived. However society is now immeasurably more complex; the nature of social organisation and the skills required to function as an adult are very different from those operative 50,000 to 10,000 years ago. Adolescence marks the period in which sex hormone activity has been initiated, but due to an interplay of neurobiological, societal and cultural factors the individual is not accepted as fully mature.

What has definitely changed is the age of puberty, which has fallen quite dramatically in many populations in recent decades. This has a number of implications. The pubertal brain functions differently from the pre-pubertal brain and is particularly responsive to reward incentivising activities. Executive function placing control on these pathways is not fully mature. Further the big shift in age of sexual activation means it is increasingly likely to start during primary school, whereas the current structure of the school system, which separates primary, intermediate and secondary education, was designed on the presumption that pubertal behaviours did not get exhibited until secondary school. Current societal concepts of and attitudes to teenage sexual activity, for example, were largely developed in the 19th and early 20th centuries at a time when puberty was not completed generally until about the age then established in Western societies as the age of legal consent. There is increasing discourse on the implications of this emergent science for the justice system (e.g. [75]), and in particular of the potential for the immature brain to respond differently from the mature brain in response to punitive punishments, which may make recidivism more rather than less likely.

On the other hand it is now evident that aspects of executive function do not mature until much later, on average in the third decade of life. It is not clear whether this is a new phenomenon or whether its implications are only now emerging as important in a more complex society. The outcome however is a much prolonged period in which pubertal hormones are active but full maturity of executive function does not exist. The consequences are a period in which reward seeking behaviours are not adequately self-controlled, and as discussed in subsequent chapters, this is reflected in multiple behaviours and activities of concern.

Against this background of pubertal mismatch, the impact of widespread marketing aimed directly or indirectly at adolescents is worrisome. Marketing and media targeting young people contain a high content of promotion of risk-taking behaviours, alcohol consumption and sexuality. The celebrity culture, which is unabashedly marketed to the adolescent, creates role models and heroes out of behaviours which are particularly risky for young people with immature impulse control.

These issues will not disappear. The age of puberty has fallen largely because children are healthy. Technological developments mean that the complexity of society will not diminish. The internet greatly limits the control or regulation of communication among young people. The challenge therefore is to develop a better understanding of how to manage the life course so as to promote resilience in young people through nurturing of non-cognitive capabilities, which will in turn create greater resilience in adolescence. The life course approach would argue that one's focus should be on the early childhood period. It also suggests that greater attention to the understanding of neural maturation should be part of the research agenda. It may well be that significant changes in the structure of the educational system are needed to assist young peoples' maturation of executive function. Greater sensitivity is needed by society as a whole to the messages that are projected at young people during this vulnerable period.

The complexities of individual biology, developmental exposures and the contexts within which every young person lives mean that there is considerable individual variation in the timing and pattern of maturation, both biologically and behaviourally. Superficially, it might seem that resilience is related solely to the variation in timing and pattern of this maturation. However, as this chapter has tried to illustrate, it is now apparent that important aspects of brain maturation can be influenced by events earlier in the life course. This information provides preventative (as opposed to remedial) opportunities

to develop strategies enhancing the opportunities for more young people to transition to adulthood without harm to themselves or to the community.

6. An economic perspective on the life course

The life course perspective has economic as well as biological, behavioural and educational dimensions. Economists have used the life course approach to model the return on investment early in the life course on later outcomes. They have modelled the value of intervention in one phase of the life course in affecting progress through a later phase in the individual's life so as to estimate the costs and benefits of preventative and remedial interventions. The range of outcomes that can be considered include educational achievement, earnings, employment, health, relationships and interactions with the justice system (arrests/incarcerations). Human capital approaches have generally been used [76, 77].

Heckman and colleagues have considered the importance of cognitive and non-cognitive (executive) brain functions, and their development in early childhood with respect to outcomes in the high school and subsequent years. Importantly, they have studied high intensity targeted intervention programmes in early childhood such as the Perry Preschool and Abecederian programmes, both of which are US-based targeted high intensity intervention programmes for at-risk children, involving structured day care/kindergarten and home interventions with well-trained therapists. Their findings can be summarised as follows: targeted investments in early life increase high school completion rates [78], and intensive early childhood programs for at-risk African-American 3-4 year olds show long term positive effects in both genders; overall the greater effects are on non-cognitive traits and, in males, lead to crime reduction and improved self-control [79]. Throughout this body of work the development of non-cognitive executive skills is closely linked to performance on formal learning tasks. The importance of early childhood to the optimal manifestation of both cognitive and non-cognitive skills through adolescence is clear. While remediation of non-cognitive skills is possible, prevention is logical and more effective [80]. Heckman's group studied the economic return on graduates of the Perry Preschool programme through their third decade and showed a high level of economic return, with the greatest impact on crime reduction [77, 81]. Similar data were obtained from follow-up of the Abecederian study [82]. The relationship between crime and low educational achievement is well documented [83].

To quote Heckman and Masterov [81]:

> *"An accumulating body of knowledge shows that early childhood interventions for disadvantaged young children are more effective than interventions that come later in life. Because of the dynamic nature of the skill formation process, remediating the effects of early disadvantages at later ages is often prohibitively costly (...) Skill begets skill; learning begets learning. Early disadvantage, if left untreated, leads to academic and social difficulties in later years. Advantages accumulate; so do disadvantages. A large body of evidence shows that postschool remediation programs like public job training and general educational development (GED) certification cannot compensate for a childhood of neglect for most people."*

Much of this disadvantage will manifest in adolescence through dropout rates, asocial and acting out behaviours associated with poor impulse control, crime and reinforcement of disadvantage for the next generation. It is also clear that much of the advantage of early intervention is mediated through non-cognitive skill development and that this differential

can be anticipated to be particularly manifest in adolescence [84], when immaturity of executive function in relation to biological maturation is most likely to confer individual and community disadvantage.

7. References

1. Gluckman PD, Beedle AS, Hanson MA. Principles of Evolutionary Medicine. Oxford: Oxford University Press; 2009.
2. Bogin B. Patterns of Human Growth. 2nd ed Cambridge: Cambridge University Press; 1999.
3. Zhang TY, Meaney MJ. Epigenetics and the environmental regulation of the genome and its function. Annual Review of Psychology. 2010; 61: 439-466.
4. Gluckman PD, Hanson MA, Buklijas T, Low FM, Beedle AS. Epigenetic mechanisms that underpin metabolic and cardiovascular diseases. Nature Reviews Endocrinology. 2009; 5: 401-408.
5. Godfrey K. The 'developmental origins' hypothesis: epidemiology. In: Gluckman PD, Hanson MA, eds. Developmental Origins of Health and Disease. Cambridge: Cambridge University Press; 2006: 6-32.
6. Gluckman PD, Hanson MA. Evolution, development and timing of puberty. Trends in Endocrinology and Metabolism. 2006; 17: 7-12.
7. Melchior M, Moffitt TE, Milne BJ, Poulton R, Caspi A. Why do children from socioeconomically disadvantaged families suffer from poor health when they reach adulthood? A life-course study. American Journal of Epidemiology. 2007; 166: 966-974.
8. Boden JM, Fergusson DM, Horwood LJ. Risk factors for conduct disorder and oppositional/defiant disorder: evidence from a New Zealand birth cohort. Journal of the American Academy of Child and Adolescent Psychiatry. 2010; 49: 1125-1133.
9. Mameli M, Bateson P. Innateness and the sciences. Biology & Philosophy. 2006; 21: 155-188.
10. Bateson P, Gluckman P. Plasticity, Robustness, Development and Evolution. Cambridge: Cambridge University Press; In press.
11. Grumbach MM. The neuroendocrinology of human puberty revisited. Hormone Research in Paediatrics. 2002; 57: 2-14.
12. Krause DN, Duckles SP, Pelligrino DA. Influence of sex steroid hormones on cerebrovascular function. Journal of Applied Physiology. 2006; 101: 1252-1261.
13. Walker R, Gurven M, Hill K, Migliano A, Chagnon N, De Souza R, et al. Growth rates and life histories in twenty-two small-scale societies. American Journal of Human Biology. 2006; 18: 295-311.
14. Nettle D, Coall DA, Dickins TE. Early-life conditions and age at first pregnancy in British women. Proceedings of the Royal Society B: Biological Sciences. 2010; doi: 10.1098/rspb.2010.1726.
15. Nettle D. Dying young and living fast: variation in life history across English neighbourhoods. Behavioral Ecology. 2010; 21: 387-395.
16. Nettle D. Flexibility in reproductive timing in human females: integrating ultimate and proximate explanations. Philosophical Transactions of the Royal Society B: Biological Sciences. 2011; 366: 357-365.
17. Ellis BJ, Essex MJ. Family environments, adrenarche, and sexual maturation: a longitudinal test of a life history model. Child Development. 2007; 78: 1799-1817.
18. Adair LS. Size at birth predicts age at menarche. Pediatrics. 2001; 107: E59.
19. Romans SE, Martin JM, Gendall KA, Herbison GP. Age of menarche: the role of some psychosocial factors. Psychological Medicine. 2003; 33: 933-939.
20. Tither JM, Ellis BJ. Impact of fathers on daughters' age at menarche: a genetically and environmentally controlled sibling study. Developmental Psychology. 2008; 44: 1409-1420.

21. Tanner JM. Fetus Into Man: Physical Growth From Conception to Maturity. Cambridge: Harvard University Press; 1978.
22. Ministry of Health. NZ Food NZ Children: Key results of the 2002 National Children's Nutrition Survey. 2003. Wellington: Ministry of Health.
23. Herman-Giddens ME. Recent data on pubertal milestones in United States children: the secular trend toward earlier development. International Journal of Andrology. 2006; 29: 241-246.
24. Sloboda DM, Howie GJ, Pleasants A, Gluckman PD, Vickers MH. Pre- and postnatal nutritional histories influence reproductive maturation and ovarian function in the rat. PLoS One. 2009; 4: e6744.
25. Espetvedt Finstad S, Emaus A, Potischman N, Barrett E, Furberg A-S, Ellison P, et al. Influence of birth weight and adult body composition on 17β-estradiol levels in young women. Cancer Causes and Control. 2009; 20: 233-242.
26. Wang Y, Lobstein T. Worldwide trends in childhood overweight and obesity. International Journal of Pediatric Obesity. 2006; 1: 11-25.
27. Sáenz de Rodriguez CA, Bongiovanni AM, Borrego LCd. An epidemic of precocious development in Puerto Rican children. The Journal of Pediatrics. 1985; 107: 393-396.
28. Massart F, Saggese G. Oestrogenic mycotoxin exposures and precocious pubertal development. International Journal of Andrology. 2010; 33: 369-376.
29. Cooper C, Kuh D, Egger P, Wadsworth M, Barker D. Childhood growth and age at menarche. BJOG: An International Journal of Obstetrics & Gynaecology. 1996; 103: 814-817.
30. Sloboda DM, Hart R, Doherty DA, Pennell CE, Hickey M. Age at menarche: influences of prenatal and postnatal growth. Journal of Clinical Endocrinology and Metabolism. 2007; 92 46-50.
31. Parent AS, Teilmann G, Juul A, Skakkebaer NE, Toppari J, Bourguignon JP. The timing of normal puberty and the age limits of sexual precocity: variations around the world, secular trends, and changes after migration. Endocrine Reviews. 2003; 244: 668-693.
32. Gluckman P, Hanson M. Mismatch: Why Our World No Longer Fits Our Bodies. Oxford: Oxford University Press; 2006.
33. Kaltiala-Heino R, Marttunen M, Rantanen P, Rimpelä M. Early puberty is associated with mental health problems in middle adolescence. Social Science & Medicine. 2003; 57: 1055-1064.
34. Bratberg GH, Nilsen TIL, Holmen TL, Vatten LJ. Sexual maturation in early adolescence and alcohol drinking and cigarette smoking in late adolescence: a prospective study of 2,129 Norwegian girls and boys. European Journal of Pediatrics. 2005; 164: 621-625.
35. Najman JM, Hayatbakhsh MR, McGee TR, Bor W, O'Callaghan MJ, Williams GM. The Impact of Puberty on Aggression/Delinquency: Adolescence to Young Adulthood. Australian & New Zealand Journal of Criminology. 2009; 42: 369-386.
36. Michaud PA, Suris JC, Deppen A. Gender-related psychological and behavioural correlates of pubertal timing in a national sample of Swiss adolescents. Molecular & Cellular Endocrinology. 2006; 254-255: 172-178.
37. Golub MS, Collman GW, Foster PMD, Kimmel CA, Meyts ERD, Reiter EO, et al. Public health implications of altered puberty timing. Pediatrics. 2008; 121: S218-S230.
38. Sowell ER, Thompson PM, Holmes CJ, Jernigan TL, Toga AW. In vivo evidence for post-adolescent brain maturation in frontal and striatal regions. Nature Neuroscience. 1999; 2: 859-861.
39. Steinberg L. Risk taking in adolescence. Current Directions in Psychological Science. 2007; 16: 55-59.
40. Yurgelun-Todd D. Emotional and cognitive changes during adolescence. Current Opinion in Neurobiology. 2007; 17: 251-257.

41. Fareri DS, Martin LN, Delgado MR. Reward-related processing in the human brain: Developmental considerations. Development and Psychopathology. 2008; 20: 1191-1211.
42. Gogtay N, Giedd JN, Lusk L, Hayashi KM, Greenstein D, Vaituzis AC, et al. Dynamic mapping of human cortical development during childhood through early adulthood. Proceedings of the National Academy of Sciences of the United States of America. 2004; 101: 8174-8179.
43. Lebel C, Walker L, Leemans A, Phillips L, Beaulieu C. Microstructural maturation of the human brain from childhood to adulthood. Neuroimage. 2008; 40: 1044-55.
44. Tamnes CK, Østby Y, Fjell AM, Westlye LT, Due-Tønnessen P, Walhovd KB. Brain maturation in adolescence and young adulthood: regional age-related changes in cortical thickness and white matter volume and microstructure. Cerebral Cortex. 2009.
45. Dumontheil I, Hassan B, Gilbert SJ, Blakemore S-J. Development of the selection and manipulation of self-generated thoughts in adolescence. Journal of Neuroscience. 2010; 30: 7664-7671.
46. Somerville LH, Casey BJ. Developmental neurobiology of cognitive control and motivational systems. Current Opinion in Neurobiology. 2010; 20: 236-241.
47. Spear LP. The adolescent brain and age-related behavioral manifestations. Neuroscience & Biobehavioral Reviews. 2000; 24: 417-463.
48. Somerville LH, Jones RM, Casey BJ. A time of change: behavioral and neural correlates of adolescent sensitivity to appetitive and aversive environmental cues. Brain and Cognition. 2010; 72: 124-133.
49. Galvan A, Hare TA, Parra CE, Penn J, Voss H, Glover G, et al. Earlier development of the accumbens relative to orbitofrontal cortex might underlie risk-taking behavior in adolescents. Journal of Neuroscience. 2006; 26: 6885-6892.
50. Steinberg L. Risk taking in adolescence: what changes, and why? Annals of the New York Academy of Sciences. 2004; 1021: 51-58.
51. Van Leijenhorst L, Zanolie K, Van Meel CS, Westenberg PM, Rombouts SARB, Crone EA. What motivates the adolescent? Brain regions mediating reward sensitivity across adolescence. Cerebral Cortex. 2010; 20: 61-69.
52. Geier CF, Terwilliger R, Teslovich T, Velanova K, Luna B. Immaturities in reward processing and its influence on inhibitory control in adolescence. Cerebral Cortex. 2010; 20: 1613-1629.
53. Cauffman E, Steinberg L. (Im)maturity of judgment in adolescence: why adolescents may be less culpable than adults. Behavioral Sciences and the Law. 2000; 18: 741-760.
54. Luna B, Sweeney JA. The emergence of collaborative brain function: fMRI studies of the development of response inhibition. Annals of the New York Academy of Sciences. 2004; 1021: 296-309.
55. Dunbar RIM. The social brain: mind, language, and society in evolutionary perspective. Annual Review of Anthropology. 2003; 32: 163-181.
56. Bickart KC, Wright CI, Dautoff RJ, Dickerson BC, Barrett LF. Amygdala volume and social network size in humans. Nature Neuroscience. doi:10.1038/nn.2724.
57. Ophir E, Nass C, Wagner AD. Cognitive control in media multitaskers. Proceedings of the National Academy of Sciences of the United States of America. 2009; 106: 15583-15587.
58. Lin L. Breadth-biased versus focused cognitive control in media multitasking behaviors. Proceedings of the National Academy of Sciences of the United States of America. 2009; 106: 15521-15522.
59. Carter CS. The chemistry of child neglect: do oxytocin and vasopressin mediate the effects of early experience? Proceedings of the National Academy of Sciences of the United States of America. 2005; 102: 18247-18248.
60. Weaver ICG, Cervoni N, Champagne FA, D'Alessio AC, Sharma S, Seckl JR, et al. Epigenetic programming by maternal behavior. Nature Neuroscience. 2004; 7: 847-854.
61. Champagne FA. Epigenetic Influence of social experiences across the lifespan. Developmental Psychobiology. 2010; 52: 299-311.

62. McGowan PO, Sasaki A, D'Alessio AC, Dymov S, Labonté B, Szyf M, et al. Epigenetic regulation of the glucocorticoid receptor in human brain associates with childhood abuse. Nature Neuroscience. 2009; 12: 342–348.
63. Beach SRH, Brody GH, Todorov AA, Gunter TD, Philibert RA. Methylation at 5HTT mediates the impact of child sex abuse on women's antisocial behavior: an examination of the Iowa adoptee sample. Psychosomatic Medicine. 2011; 73: 83-87.
64. Sroufe LA. Attachment and development: a prospective, longitudinal study from birth to adulthood. Attachment & Human Development. 2005; 7: 349-367.
65. Champagne FA. Epigenetic mechanisms and the transgenerational effects of maternal care. Frontiers in Neuroendocrinology. 2008; 29: 386-397.
66. Gluckman PD, Hanson MA, Beedle AS. Non-genomic transgenerational inheritance of disease risk. Bioessays. 2007; 29: 145-154.
67. De Dreu CKW, Greer LL, Handgraaf MJJ, Shalvi S, Van Kleef GA, Baas M, et al. The neuropeptide oxytocin regulates parochial altruism in intergroup conflict among humans. Science. 2010; 328: 1408-1411.
68. Unicef. A league table of teenage births in rich nations. 2001. Florence, Italy: Unicef Innocenti Research Centre.
69. Ministry of Health. Sexual and reproductive health strategy: phase one. 2001. Wellington: Ministry of Health.
70. Heckman JJ. Schools, skills, and synapses. Economic Inquiry. 2008; 46: 289-324.
71. Doyle O, Harmon CP, Heckman JJ, Tremblay RE. Investing in early human development: timing and economic efficiency. Economics & Human Biology. 2009; 7: 1-6.
72. Cunha F, Heckman JJ, Schennach SM. Estimating the technology of cognitive and noncognitive skill formation. Econometrica. 2010; 78: 883-931.
73. Knudsen EI, Heckman JJ, Cameron JL, Shonkoff JP. Economic, neurobiological, and behavioral perspectives on building America's future workforce. Proceedings of the National Academy of Sciences of the United States of America. 2006; 103: 10155-10162.
74. Johnson SB, Blum RW, Giedd JN. Adolescent maturity and the brain: the promise and pitfalls of neuroscience research in adolescent health policy. Journal of Adolescent Health. 2009; 45: 216-221.
75. Steinberg L. Adolescent development and juvenile justice. Annual Review of Clinical Psychology. 2009; 5: 459-485.
76. Almond D, Currie J. Human capital development before age five. In: Ashenfelter O, Card D, eds. Handbook of Labor Economics. Vol. 4. Amsterdam: Elsevier; 2010.
77. Heckman JJ. Skill formation and the economics of investing in disadvantaged children. Science. 2006; 312: 1900-1902.
78. Cunha F, Heckman JJ. The economics and psychology of inequality and human development. Journal of the European Economic Association. 2009; 7: 320-364.
79. Heckman JJ, Moon SH, Pinto R, Savelyev PA, Yavitz A. The rate of return to the HighScope Perry Preschool Program. Journal of Public Economics. 2010; 94: 114-128.
80. Heckman JJ. Policies to foster human capital. Research in Economics. 2000; 54: 3-56.
81. Heckman JJ, Masterov DV. The productivity argument for investing in young children. Applied Economic Perspectives and Policy. 2007; 29: 446-493.
82. Campbell FA, Ramey CT, Pungello E, Sparling J, Miller-Johnson S. Early childhood education: young adult outcomes from the Abecedarian project. Applied Developmental Science. 2002; 6: 42-57.
83. Lochner L, Moretti E. The effect of education on crime: evidence from prison inmates, arrests, and self-reports. American Economic Review. 2004; 94: 155-189.
84. Heckman JJ, Stixrud J, Urzua S. The effects of cognitive and noncognitive abilities on labor market outcomes and social behavior. Journal of Labour Economics. 2006; 24: 411-482.