Chapter 9

Puberty, Developmental Processes, and Health Interventions

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INTRODUCTION

Adolescence is increasingly recognized as a critical period in the life course, a time when rapid development of the brain, body, and behaviors opens a window of opportunity for interventions that may affect health throughout life.

Puberty results in very rapid somatic growth, brain development, sexual maturation, and attainment of reproductive capacity. It is accompanied by final maturation of multiple organ systems and major changes in the central nervous system and in psychosocial behavior (Patton and Viner 2007). The discovery of continued brain development through adolescence is one of the great advances of neuroscience in the past 20 years. A dramatic spurt in brain development begins during adolescence and continues until the mid-20s, with marked development of both cortical and subcortical structures (Goddings and others 2012).

This rapid development in the body and brain interacts with social changes, including increasing individuation and new peer groups, to facilitate transitions important for individuals to function as productive adults (World Bank 2006). A range of social determinants of health arise in adolescence, with peers, schools, and eventually the workplace becoming strong determinants of health and well-being as the influence of the family wanes (Viner and others 2012). These social changes are apparent even in traditional or more sociocentric cultures. More than half of the top 10 risk factors identified in the Global Burden of Disease study (GBD 2013 Risk Factors Collaborators 2015) are largely determined during adolescence.

Adolescence is also a time when young people may modify or alter the pathways to adult health or illness (Viner and others 2012). Early life experiences may reinforce both good and poor trajectories. Similarly, resilience during adolescence may improve outcomes for young people born into adversity. The transfer from primary to secondary school, sexual debut, and entry into the labor market may be critical points for preventing the accumulation of health risk (Viner and others 2012).

This chapter outlines the key dynamics of adolescent development and examines how they provide opportunities for intervention. Definitions of age groups and age-specific terminology used in this volume can be found in chapter 1 (Bundy and others 2017).

PUBERTY AND DEVELOPMENTAL PROCESSES

Puberty is a time of rapid growth in all body systems and changes in brain function and cognitive development (Patton and Viner 2007).

Stages of Puberty

The process of puberty begins earlier than most recognize, that is, between the ages of six and eight years with the early phase of adrenarche, the turning on of the

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adrenal glands. Adrenarche has few phenotypic signs in most children, but increasing evidence indicates that adrenal androgens may contribute to the structural and functional development of the brain and associated behaviors in adolescence (Whittle and others 2015). The timing of adrenarche affects the risks for mental health problems (Mundy and others 2015) and a range of cardiometabolic issues. Body mass index (BMI) is associated with adrenal androgens (Corvalan, Uauy, and Mericq 2013); children exhibiting premature adrenarche have been found to have higher levels of insulin and insulin resistance and a predisposition to higher BMI (Ibáñez and others 2008).

The second phase of puberty is gonadarche, the process of sexual maturation and achievement of reproductive capacity (Marshall and Tanner 1968). The production of gonadal steroids stimulates the growth and development of secondary sexual characteristics; it also kindles development across all organ systems, including the central nervous system. Other endocrine systems mature during puberty, including the growth hormone/insulin-like growth factor and thyroid axes.

Marshall and Tanner (1968) developed a system for identifying stages in the external signs of puberty. The earliest external changes—breast buds in girls and testicular enlargement in boys—typically appear about age 11 years, but vary among individuals. Despite a similar age of gonadarche in boys, these early changes are more visible in girls. Menarche, the onset of menstrual periods (menses), typically occurs in late puberty, approximately two years after breast budding. While menses appears to signal reproductive maturity for girls, it largely signals maturity of the uterus because early menstrual periods are irregular and girls are rarely fertile immediately after menarche (Hochberg and Belsky 2013).

Puberty is generally complete within two to four years following gonadarche, but other changes including fat and muscle patterning continue through adolescence. The timing of puberty is partly genetic (Day and others 2016), but intrauterine events, nutrition, family factors, stress, and socioeconomic conditions also play roles (Hochberg and Belsky 2013).

Puberty is increasingly recognized as a time of distinct transitional physiology (Rosenfeld and Nicodemus 2003). The most dramatic change is the pubertal growth spurt, with boys typically growing 30 centimeters and girls growing 25–27 centimeters due to synergy between the sex steroids and growth hormone (Abbassi 1998). For girls, the growth spurt occurs early, with peak growth typically occurring about the time of the start of breast development. For boys, it occurs later. Girls typically stop growing by the end of puberty, adding only about 2.5 centimeters after the beginning of menses. Boys continue growing slowly after the end of puberty, achieving final height at about age 18 years.

The bone, renal, immune, and cardiovascular systems are also developing, and liver enzymes and blood lipids are maturing. Bone mineral accretion accelerates during puberty under the influence of gonadal steroids, with peak bone mass achieved by the early 20s (Loud and Gordon 2006). Cardiovascular and renal development means that blood pressure and heart rate make a transition to adult values, in parallel with growth in height and mass. The cardiovascular risk profile differs between the genders, with more adverse lipid patterns among boys than girls. Other blood markers, such as hemoglobin levels, similarly change to a sexually dimorphic pattern.

Timing of Puberty

The sequence of pubertal events is remarkably consistent across countries and ethnic groups, although timing varies by country. The timing of puberty is influenced partly by genetics, but largely by nutrition and economic development (Hochberg and Belsky 2013). The mean age at menarche is now 12–13 years in most high-income countries (HICs); it is usually later in low-income countries, even in affluent populations (Parent and others 2003).

The mean age of menarche stopped falling in most HICs after the 1960s, but it is still falling in low-income countries. However, data from the United States (Herman-Giddens and others 1997) and, more recently, Europe (Parent and others 2016) suggest that early pubertal events are occurring at younger ages but that late pubertal events are not. The reasons for this broadening of puberty are unclear, although exposure to endocrine disrupter chemicals and psychosocial stress are possible mechanisms (Parent and others 2016). Recent studies have suggested that earlier pubertal development is likely a response to changing environmental circumstances (Gluckman and Hanson 2006; Hochberg and Belsky 2013). By contrast, the timing of adrenarche appears to be relatively constant across populations (Hochberg 2009).

Effects on Health and Disease

In addition to puberty's direct influences on physiology and growth, the timing of puberty appears to program changes in lifelong health. In particular, strong evidence indicates that higher BMI accelerates the onset of puberty (Burt Solorzano and McCartney 2010; Frisch 1984; Hochberg and Belsky 2013), but early puberty also programs individuals for greater fat accumulation

over the life course (Power, Lake, and Cole 1997; Prentice and Viner 2013).

Early puberty is associated with cardiometabolic risk, increasing the risk of cardiovascular events and mortality, type 2 diabetes (Prentice and Viner 2013), and high blood pressure (Hardy and others 2006). These associations appear to be at least partly independent of childhood obesity (Prentice and Viner 2013). Mechanisms likely relate to stress reactivity, the growth hormone/insulin-like growth factor axis (Sandhu and others 2006), and glucose insulin homeostasis (Burt Solorzano and McCartney 2010).

Early puberty is linked to cancer in later life through several mechanisms. Longer exposure to gonadal steroids may increase the risk of steroid-dependent cancers such as breast and ovarian cancer in females (Ahlgren and others 2004; Jordan, Webb, and Green 2005) and possibly prostate cancer in males (Giles and others 2003). Mechanisms may include longer exposure to sex hormones, increased oxidative stress (Vincent and Taylor 2006), or hyperinsulinemia (Frezza, Wachtel, and Chiriva-Internati 2006) related to obesity in early developers or to behavioral risk factors such as substance use (Patton and Viner 2007).

Evolutionary Implications

The relationship of the timing of puberty to environment and nutrition has its origins in evolutionary biology. Severe environmental stress and malnutrition may result in delayed puberty, prioritizing the survival of the individual given that reproduction is not possible. Similarly, ideal environmental conditions may result in delayed puberty, maximizing the individual's later reproductive success. However, environmental stress that is not sufficient to threaten survival may accelerate pubertal development, increasing the likelihood of reproduction before death (Hochberg and Belsky 2013).

In this schema, adrenarche represents a point at which the environment can reprogram reproductive strategies (Del Giudice 2009). It may also allow children time to test their social status in a peer environment free from reproductive imperatives.

Accelerated puberty also increases health risk behaviors, such as early sexual activity and violence. Associations have been found between early pubertal development in girls and sexual abuse, severe psychosocial stress, and even absence of the father. For example, migrant children arriving in HICs frequently experience onset of puberty earlier than would be expected in either their home or host country. Their transition from a threatening to an ideal nutritional environment may accelerate their pubertal development (Hochberg and Belsky 2013).

In an evolutionary context, neurodevelopment during puberty is likely to optimize reproductive success by realigning emotional, social, and metabolic strategies to the external environment (Hochberg and Belsky 2013).

Brain Development

In adolescence, brain development involves two key processes: significant growth and change in regions of the prefrontal cortex (Paus, Keshavan, and Giedd 2008; Steinberg 2005) and improved connectivity between regions of the prefrontal cortex and regions of the limbic system (Casey 2015; Steinberg 2005). These changes are thought to underpin higher-order cognitive functions, such as reasoning, interpersonal interactions, the perception of both short- and long-term risk and reward, and the regulation of behavior and emotion (Paus, Keshavan, and Giedd 2008; Steinberg 2005).

Normative neurodevelopmental processes prepare the brain for responding to the demands of both adolescence and adult life, but may also make adolescents vulnerable to risk behavior and psychopathology (Paus, Keshavan, and Giedd 2008). Dual-system and imbalance models posit that adolescence is a particularly vulnerable period because of the imbalance between early maturation of the limbic motivational and emotional systems and slower, or later, development of the regulatory regions of the prefrontal cortex (Casey 2015). The dual-systems model emphasizes a developmentally normal mismatch between intense affective and behavioral reactions and motivations and limited capacity to regulate them (Steinberg 2005).

Recent studies, however, suggest a more complex picture (Mills and others 2014; Pfeifer and Allen 2012). For example, brain-imaging studies in adolescents do not provide consistent support for the association between immaturity in the frontal cortex and the emergence of risk behavior and psychopathology (Crone and Dahl 2012).

Recent attempts to quantify brain maturation have used measures of the whole brain, such as network-based measurements of resting-state brain function that are independent of specific tasks (Dosenbach and others 2010) or structural data from magnetic resonance imaging (Vértes and Bullmore 2015). Some of these measures may be related to both the emergence of more integrated self-regulatory abilities and plasticity in response to new learning experiences (Crone and Dahl 2012; Dosenbach and others 2010). However, the relationship between these neurodevelopmental patterns and cognitive, affective, and behavioral changes in adolescence is not fully understood.

Cognitive Development

Cognitive domains, including learning, reasoning, information processing, and memory, improve as adolescents develop. Executive functioning capabilities, which facilitate self-regulation of thoughts, actions, and emotions, continue to develop in parallel with changes in the prefrontal cortex (Kesek, Zelazo, and Lewis 2008). These increases in self-regulatory control are thought to support deductive reasoning; information processing; efficiency; and the capacity for abstract, planned, hypothetical, and multidimensional thinking (Steinberg 2005).

Cognitive and Affective Processing

Recent research has focused on both cognitive and affective processing, particularly regarding how these processes interact and influence each other in the context of decision making. First, cognitive skills allow improved self-regulation of affect—the capacity to initiate new or alter ongoing emotional responses—to achieve a goal (Ochsner and Gross 2005). Second, affective influences on cognitive processing, including decision making, risk taking, and judgment, change significantly during adolescence (Hartley and Somerville 2015; Steinberg 2005).

The social and emotional context for cognitive processing during adolescence may include factors such as the presence of peers or the value of performing a task, which are hypothesized to influence the motivational salience of specific contexts and the extent to which cognitive processing is recruited (Johnson, Grossmann, and Kadosh 2009). Moreover, some of these changes in cognitive and affective processing are linked to the onset of puberty (Crone and Dahl 2012), with flexibility of the frontal cortical network greater in adolescence than in adulthood (Jolles and others 2012).

Temporal Discounting

Temporal discounting refers to the inclination to discount the value of future rewards as compared with immediate ones (Christakou, Brammer, and Rubia 2011). This tendency declines sharply between ages 15 and 16 years (de Water, Cillessen, and Scheres 2014). Moreover, the neurobiological basis for temporal discounting is related to developmental changes in dopamine activity (Pine and others 2010). Specifically, increases are evident in both dopaminergic connectivity to the prefrontal cortex (Kalsbeek and others 1988; Verney and others 1982) and the density of dopamine transporters (Spear 2000). Dopamine receptors are overproduced in early adolescence, followed by pruning that is more evident in subcortical than in prefrontal regions (Spear 2000). The net effect is to shift the relative balance between subcortical and cortical dopaminergic systems, with increasing dominance across adolescence of the system responsible for valuing future rewards.

The impact of temporal discounting is consistent with the observed association between adolescence and risk-taking behaviors, despite adequate knowledge of risks (Steinberg 2005). It also suggests that affective valuation of immediate versus long-term outcomes (as opposed to conceptual understanding of them) is likely to be the main way in which adolescent decision making deviates from mature (adult-like) decision making.

Sensitivity to Peer Influence

Adolescents assign greater weight than adults to social outcomes such as peer acceptance. During the transition from childhood to adolescence, the amount of time spent with peers increases dramatically (Brown 2004) and peer and family values increasingly diverge (Gardner and Steinberg 2005; Steinberg 2008). These changes suggest that teenagers are less resistant to peer pressure than either children or adults, although susceptibility to peer influence per se declines over the course of adolescence (Steinberg and Monahan 2007).

Neuroscientific research has begun to explore this resistance to peer influence. Grosbras and others (2007) studied children age 10 years with high or low resistance to peer influence and found that, while viewing angry hand gestures and facial expressions, those with high resistance to peer influence showed more coordinated brain responses across parts of the brain associated with processing nonverbal behavior and with planning and executing movement. The better the brain is at coordinating its response to other people's nonverbal emotional expressions across the emotional and self-regulation networks of the brain, the better the person is at resisting peer influence. Consistent with this, Pfeifer and others (2011) found that adolescents who are better at resisting peer influence have greater activity in a region of the brain involved in reward, positive affect, and emotional regulation.

Executive Control

The literature on the structural and functional changes associated with brain maturation suggests a model in which some regions are tightly integrated into long-range networks, while other regions are segregated into short-range networks. Fjell and others (2012) demonstrated that developmental changes in cognitive control were associated with both the surface area of the anterior cingulate cortex and the properties of large fiber connections. Crone and Dahl (2012) proposed that because these patterns of long-range connectivity are still maturing, some aspects of executive control may

be less automatic and more flexible during adolescence, resulting in greater vulnerability when performing attentional and decision-making tasks under high demands (because the ability to integrate control is less automatic) and enabling adolescents to respond in novel and adaptive ways. Thus, specific learning or training experiences during adolescence may guide the final connectivity patterns in some of these long-range cognitive control networks.

ADOLESCENCE AS A TIME OF RISK

The developmental changes that occur in adolescence create greater vulnerability to emotional and behavioral dysregulation (Steinberg 2005).

Although adolescents are relatively physically healthy compared with other age groups, adolescence is a key phase of life for the establishment of risk factors for several highly burdensome diseases. The transition into early adolescence is marked by dramatic increases in morbidity and mortality, often associated with mental health disorders, substance use, and the consequences of risk taking and poor decision making (Blum and Nelson-Mmari 2004; Williams, Holmbeck, and Greenley 2002). The majority of mental health and substance use problems begin before age 21 years (Jones 2013), and poor health outcomes during adolescence may have ongoing and negative impacts on adult life (Sawyer and others 2012). For example, major noncommunicable diseases, such as heart disease and cancer, are acutely sensitive to lifestyle and behavioral risk factors that are often established during adolescence, such as nutrition, physical activity, sleep, obesity, stress, and substance use (Lowry and others 1996). The dramatic changes occurring in the brain during adolescence also make this a time of significant neuroplasticity, suggesting that behavioral patterns can become strongly encoded in the brain during this time (Crone and Dahl 2012).

Aggression and Violence

Aggression, including bullying and violence, increases dramatically and peaks in middle adolescence (Krug and others 2002; Patton and Viner 2007). Given the known effects of testosterone on aggression in animals and humans (Archer 1991), researchers focused on the relationship between puberty and aggression in males (Olweus and others 1988). More recently, large-scale studies have found good evidence that the risk of violence and aggression increases with pubertal stage in boys (Hemphill and others 2010).

Depression

Considerable evidence demonstrates that early puberty increases the risk of depression in girls (Hayward and others 1997; Kaltiala-Heino, Kosunen, and Rimpelä 2003; Kaltiala-Heino and others 2003; Mendle, Turkheimer, and Emery 2007). Angold, Costello, and Worthman (1998) found that pubertal stage predicts the risk of major depression in adolescents better than age. Rates of depression are higher in boys than in girls before puberty, but are higher in adult women than in adult men. Girls begin to surpass boys in depression at stage-3 puberty. Puberty may even reduce the prevalence of depression in males (Angold, Costello, and Worthman 1998).

Anxiety

Anxiety disorders increase markedly in both sexes during adolescence. However, the evidence for an association with pubertal timing is much less clear for anxiety than for depression. A recent review of more than 45 empirical studies found only moderate-quality evidence that both earlier timing and more advanced pubertal stage increase anxiety or symptoms in girls after adjusting for age. Findings for boys are even less robust (Reardon, Leen-Feldner, and Hayward 2009).

There is little evidence regarding anxiety disorders. Hayward and others (1992) found an association between panic attacks and pubertal stage, but Graber and others (1997) found no association between anxiety disorders and pubertal timing in boys or girls.

Deliberate Self-Harm

Deliberate self-harm, a major risk factor for suicide, rises sharply in early adolescence. In young women, it peaks about age 15–16 years and falls thereafter (Hawton and others 2002; Madge and others 2008). The literature on puberty and deliberate self-harm is much smaller than that on depression or anxiety. In a large population-based study, strong associations between deliberate self-harm and pubertal stage (adjusted for age) were attenuated when models were adjusted for depressive symptoms, showing that this association was largely or entirely mediated by depression (Patton and others 2007).

Eating Disorders

There is a strong association between puberty and eating disorders, at least in girls. A recent systematic review identified advanced pubertal status or early pubertal timing as a risk factor for eating disorders or disordered

eating in more than 40 studies in girls and more than 20 studies in boys. Early-maturing girls and boys have higher risk of a range of eating disorders, including anorexia nervosa and bulimia nervosa, as well as symptoms of eating disorders, including dissatisfaction with body, weight, or shape (Klump 2013). However, some studies found no association between eating disorders and puberty, particularly in boys, and others reported an association between early or advanced puberty and improved body image (McCabe, Ricciardelli, and Banfield 2001).

Physical Health

Less recognized are associations between puberty and physical illnesses. Puberty coincides with a rise in prevalence of many autoimmune conditions and a marked shift in gender ratio toward females (Beeson 1994). In both genders, type 1 diabetes begins in early puberty, although the peak age of onset occurs approximately two years earlier in girls than in boys, reflecting differences in pubertal timing (Pundziute-Lycka and others 2002). Early puberty is an independent risk factor for the persistence of asthma into adolescence and severity of asthma in adulthood (Varraso and others 2005). Seizures often become more frequent and new types of epilepsy emerge during adolescence (Klein, van Passel-Clark, and Pezzullo 2003). The pubertal growth spurt results in new musculoskeletal problems, and puberty is linked with various pain syndromes. The increase in back, facial, and stomach pains in early adolescence is associated with pubertal status in both sexes (LeResche and others 2005). Adult women have higher rates of migraine and tension headaches than adult men; this pattern is evident about age 11 years and is linked with puberty (Wedderkopp and others 2005).

ADOLESCENCE AS A TIME OF OPPORTUNITY

Adolescence is a key time for interventions to improve health. The benefits of intervention in early childhood are well described, and nations have made significant investments in maternal and child health and primary education (Commission on Social Determinants of Health 2008; Conti and Heckman 2012). Adolescence presents an opportunity to preserve investments made in childhood and to switch trajectories (Romeo 2010), while the emergence of new social determinants of health, such as peers, and connection with school, neighborhood, and workplace, offer new vehicles and venues for intervention.

Young people make five key transitions on the pathway to adulthood (World Bank 2006):

- Learning: Transition from primary to secondary schooling and from secondary to higher education
- Work: Transition from education into workforce
- Health: Transition to responsibility for own health
- Family: Transition from family living to autonomy, marriage, and parenthood
- Citizenship: Transition to responsible citizenship.

Transitions are accompanied by new behaviors, including the initiation of many health-related behaviors that track strongly into adult life. They are a time of great opportunity to tread new paths and embark on new trajectories toward health and well-being.

Secondary Education as a Health Intervention

Evidence is emerging that secondary education is efficacious against a range of health outcomes in adolescents and young adults, from sexually transmitted infections to adolescent fertility, mortality, and mental health (Patton and others 2016). Education is one of the strongest determinants of health and human capital (Commission on Social Determinants of Health 2008), and universal primary education is one of the key United Nations Millennium Development Goals. In both rich and poor countries, persons with more education live longer lives with less disability and ill health, and the relationship is likely to be causal (Baker and others 2011; Miyamoto and Chevalier 2010; Pradhan and others 2017, chapter 30 in this volume). The United Nations Sustainable Development Goals include a target for countries to provide every child with access to free primary and secondary education by 2030 (Barro 2013).[1]

Yet the health gains from secondary education have been studied less than those from primary education, despite a dramatic global expansion in the length of education in the past 30 years, with most gains in the late primary and early secondary years (IHME 2015). Among adults in HICs, upper-secondary education is most strongly associated with better health and mental health (Miyamoto and Chevalier 2010), although tertiary education confers additional benefits in U.S. studies (Case and Deaton 2015). Secondary education is known to promote better pregnancy and child health outcomes among adult women internationally (Grépin and Bharadwaj 2015; UNESCO 2010), and a small literature from Sub-Saharan African countries suggests that secondary schooling may have a stronger and more consistent effect on teenage fertility than primary education (Mahy and Gupta 2002).

Nutritional Interventions

Adolescence presents an opportunity to reverse earlier deficits from stunting or wasting in childhood (GBD 2013 Risk Factors Collaborators 2015). Nutritional sufficiency in adolescence is particularly important for pregnancy. Childbearing during adolescence places an additional nutritional burden on the mother and may explain some of the additional risk that pregnancy in adolescence poses to the 16 million teenagers who give birth annually and their offspring (Mundy and others 2015; Whittle and others 2015). Adolescent growth and development therefore provides an opportunity for preconception interventions to ensure adequate nutrition in adolescent girls. These issues are discussed in greater detail in chapter 11 in this volume (Lassi, Moin, and Bhutta 2017).

Psychosocial Interventions

Exposure to an enriching environment during adolescence may offset many of the negative neurobehavioral and physiological consequences of early life adversity (Romeo 2010). The onset of puberty marks the beginning of dramatic changes in the processing of rewards and emotional stimuli and social-cognitive reasoning (Crone and Dahl 2012). Efforts to sensitize young people to their social environment and push them to explore and engage provide opportunities to promote prosocial motivation and goals in early adolescence.

Furthermore, neurodevelopment likely affects a young person's ability to engage with or benefit from interventions, particularly those that target decision making and risk behaviors in peer and affective contexts. In particular, immaturity in cognitive processes, such as temporal discounting, may necessitate a different approach to intervention in early adolescents than in middle to late adolescents. Furthermore, adolescents are uniquely vulnerable to peer influences, both antisocial and prosocial, and this vulnerability can be used to enhance health outcomes. Indeed, resisting negative peer influences is important for self-regulation. Finally, specific experiences may affect neurodevelopment—psychosocial interventions may enhance self-regulation and can have benefits not only during adolescence but also later in life.

Research to date has yielded some efficacious early intervention and prevention approaches to mental disorders during adolescence. For example, both psychosocial and pharmacological treatments with established efficacy are available for treating depression (Kazdin 2003). In particular, research on interpersonal and cognitive behavioral therapy as well as on the use of fluoxetine found that 60 percent to 75 percent of adolescents will recover by the time of posttreatment assessment (Asarnow, Jaycox, and Tompson 2001; March and others 2004). Despite these favorable results, the long-term outcomes of current treatment approaches are unclear. One meta-analysis of psychotherapy for depression in youth found no lasting effects one year following treatment (Weisz, McCarty, and Valeri 2006).

Mental disorders, once established, are difficult to ameliorate fully, highlighting the importance of evidence-based strategies to prevent or slow the onset of disorders in vulnerable individuals. To achieve this goal, it is necessary to identify developmentally significant, modifiable risk factors and to target change in them.

In recent decades, numerous controlled studies have evaluated the effect of programs to prevent mental illness (Durlak and Wells 1997, 1998) and substance use (Tobler and others 2000); problems at school and depression (Gillham, Shatté, and Freres 2000); and aggression and behavior problems, especially in children (Tremblay, LeMarquand, and Vitaro 1999); along with many other conditions. These studies have shown that some programs may strengthen protective factors, such as social and problem-solving skills, stress management skills, prosocial behavior, and social support, and reduce the consequences of risk factors, symptoms, and substance use.

However, few studies have examined how to prevent the onset of case-level mental and substance use disorders, mainly because of the challenges associated with designing and funding studies with enough statistical power to detect such effects (Cuijpers 2003). Such programs have had modest effects (Horowitz and Garber 2006), and there is a need to ascertain which individuals are most likely to benefit from specific interventions. In sum, while prevention and intervention approaches delivered early in life are promising, there is a clear need to understand how to match interventions with individuals to increase their impact and cost-effectiveness.

CONCLUSIONS

Adolescence is a time of great developmental plasticity and risk for the onset of a range of disorders that can carry a high burden of disease throughout the lifespan. It offers a critical developmental window of opportunity for intervention and prevention. Puberty and brain development during adolescence are responsible for dramatic shifts in burden of disease, away from childhood conditions toward injuries and emerging noncommunicable diseases. Knowledge of the unique developmental processes that characterize adolescence and the role they play in both risk and opportunity during this phase of life is expanding rapidly. What remains is the task of

translating this knowledge into intervention and prevention methods that target modifiable, developmentally sensitive mechanisms to maximize the effectiveness of intervention approaches during this phase of life.

NOTES

World Bank Income Classifications as of July 2014 are as follows, based on estimates of gross national income (GNI) per capita for 2013:

- Low-income countries (LICs) = US$1,045 or less
- Middle-income countries (MICs) are subdivided:
  a) lower-middle-income = US$1,046 to US$4,125
  b) upper-middle-income (UMICs) = US$4,126 to US$12,745
- High-income countries (HICs) = US$12,746 or more.

1. See the Sustainable Development Knowledge Platform, http://sustainabledevelopment.un.org/.

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