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Developmental Origins of Chronic Physical Aggression: A Bio-Psycho-Social Model for the Next Generation of Preventive Interventions

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Abstract

This review describes a bio-psycho-social approach to understanding and preventing the development of chronic physical aggression. The debate on the developmental origins of aggression has historically opposed genetic and environmental mechanisms. Recent studies have shown that the frequency of physical aggression peaks in early childhood and then decreases until old age. Molecular genetic studies and twin studies have confirmed important genetic influences. However, recent epigenetic studies have highlighted the important role of environments in gene expression and brain development. These studies suggest that interrelated bio-psycho-social channels involved in the development of chronic physical aggression are generally the product of an intergenerational transmission process occurring through assortative mating, genetic inheritance, and the inheritance of physical and social

environmental conditions that handicap brain functioning and support the use of physical aggression to solve problems. Given these intergenerational mechanisms and physical aggression onset in infancy, it appears clear that preventive interventions should start early in pregnancy, at the latest.

Contents

1. INTRODUCTION

The study of physical aggression among humans includes a very large spectrum of topics, from the study of wars between countries to that of biting among toddlers in childcare centers. The causes and consequences of physical aggression among humans and animals are studied by a wide variety of specialists, such as anthropologists, biologists, criminologists, ethologists, historians, psychologists, neurologists, philosophers, psychiatrists, and surgeons.

2. THE LONG-STANDING NATURE–NURTURE DEBATE

The focus of this review is recent research into the developmental origins of chronic physical aggression by humans. However, to understand recent research questions and answers in a given research area, it is always useful to keep in mind its long-term history.

As illustrated by the story of Cain and Abel in the Bible, the topic of aggression among humans is probably as old as humanity, and questions concerning the developmental origins of aggression were investigated by many philosophers from ancient Greece to modern times. Not surprisingly, one central issue over the centuries has been the nature–nurture origin of aggression. For example, Aristotle, in his book *Politics*, concluded that humans grow from irrational to rational behavior

because "anger and will and desire are implanted in a child from their very birth, but reason and understanding develop as they grow older" (Aristotle 1943, p. 405). Some 800 years later, Augustin of Thagase and Hippo (Saint Augustine) played a major role in developing the idea of original sin based, in part, on his observations of young children's aggressive behavior [Augustine 1960 (397–401 AD)]. This line of reasoning was reiterated 1,200 years after Augustine by the British philosopher Thomas Hobbes. He noted, "Unless you give infants everything they want, they cry and get angry, they even beat their own parents," and concluded that aggressive adults were simply behaving like children [Hobbes 1998 (1647), p. 11]. However, a century later, Jean-Jacques Rousseau [1979 (1762), p. 5], one of the most influential philosophers of the Enlightenment and still a reference for many education specialists, strongly stated the nurture hypothesis in the first phrase of his famous book on the education of children: "God makes all things good; man meddles with them and they become evil." He also summarized the research agenda for many developmental psychologists, sociologists, and criminologists over the past century: "There is no original sin in the human heart, the how and why of the entrance of every vice can be traced" [Rousseau 1979 (1762), p. 56].

One of the best illustrations of the modern nature–nurture clash concerning the development of aggression is found in two books published approximately half a century ago. The first was *On Aggression* by the ethologist Konrad Lorenz (1966), originally written in German; the second was *Aggression: A Social Learning Perspective* by the psychologist Albert Bandura (1973). Lorenz's book was based on his observations of animal behavior and concluded that humans, like all other animals, inherited an aggressive instinct, which could lead to the destruction of humanity. Bandura's book was based on studies of children in a laboratory situation, where they were shown to spontaneously imitate an adult hitting a Bobo doll. Bandura, 200 years after Rousseau, reached a similar conclusion: "People are not born with preformed repertories of aggressive behaviors; they must learn them in one way or another" (Bandura 1973, p. 61). It is probably fair to say that most psychologists trained during the last 30 years of the twentieth century were convinced that humans learn to aggress from their environment, and most biologists trained during that period were convinced that humans, like other animals, instinctively use aggression.

To understand this long-standing philosophical and scientific debate, it is important to note that the term learn to aggress is used here to mean that, if a child never saw a human physically aggress another, this child would not be able to aggress even if he needed to do so to defend himself. The above quotations from Rousseau and Bandura appear to have that meaning. Those who argue that there is a genetic-instinctual basis to physical aggression generally do not deny that there are also learning components. It seems obvious that, for aggression, as for other physical skills that have a strong genetic-instinctual basis (eating, running, jumping, smelling, tasting, etc.), there is much that an individual must learn to use that skill effectively. This may be the reason why play fighting is common during the early development of cats, dogs, monkeys, and humans (Palagi et al. 2016, Parent & Meaney 2008).

At the end of the twentieth century, large-scale longitudinal studies were planned specifically to investigate the developmental mechanisms by which children learn to aggress from their environment. Many of these studies were driven partly by the hypothesis that children were becoming more aggressive because of violence on television (Eron et al. 1963, Huesmann et al. 1984). A few studies by psychologists during the 1930s had either observed young children's physical aggression (Murphy 1937) or questioned parents on their children's expression of anger (Goodenough 1931), but no studies had done long-term follow-ups from early childhood to adolescence to try to address the nature–nurture issue. In fact, to our knowledge, even biologists and psychologists who studied aggression in mammals, such as rats, mice, and primates, had not done developmental studies to unravel the developmental origins of aggression, probably because it appeared

obvious to them that aggression is an adaptive mechanism needed for a species to survive, as it appeared obvious to social psychologists and sociologists that humans learn to aggress from their environment.

3. DEVELOPMENTAL TRAJECTORIES OF PHYSICAL AGGRESSION FROM EARLY CHILDHOOD TO OLD AGE

The first prospective longitudinal studies with yearly assessments of physical aggression from childhood to adolescence reported surprising results from a social learning perspective, especially regarding the negative influence of television violence on children's aggression (Eron et al. 1963, Huesmann et al. 1984). If observing physical aggression on television made children more physically violent, we would expect that children's physical aggression would increase with age because the exposure to violence on television increases with age. However, results from longitudinal studies in North Carolina (Cairns et al. 1989) and Canada (Nagin & Tremblay 1999) showed, surprisingly, a substantial decrease in the frequency of instances of physical aggression from school entry to adolescence. To rule out the hypothesis that one particular group of children were increasing their frequency of aggression with age whereas most others decreased their frequency of aggression, developmental trajectory analyses of the data were made to identify differences in developmental trajectories. Results showed that no significant group of children went from a low frequency of physical aggression during childhood to a high frequency during adolescence. Those who frequently aggressed in adolescence were those who frequently aggressed during childhood. These results from a Canadian longitudinal study were replicated with longitudinal studies in Italy, New Zealand, and the United States (e.g., Broidy et al. 2003, Di Giunta et al. 2010).

The developmental trajectories of physical aggression during elementary school years and adolescence suggest that, if children learn to aggress from their environment, then this must happen before school entry. Thus, longitudinal studies of physical aggression were initiated with birth cohorts. Results from these studies showed (see **Figure 1**) that physical aggression is often initiated during the first year of life (e.g., Hay et al. 2014, Naerde et al. 2014, Tremblay et al. 1999), substantially increases in frequency with physical growth up to 3–4 years of age, and then starts decreasing (Campbell et al. 2006, Côté et al. 2006, Dearing et al. 2015, Naerde et al. 2014).

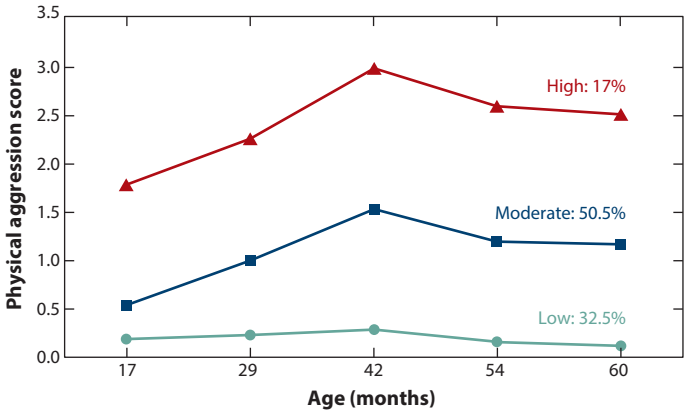

Figure 1

Developmental trajectories of physical aggression from 17 to 60 months old. Figure adapted with permission from Côté et al. (2007a).

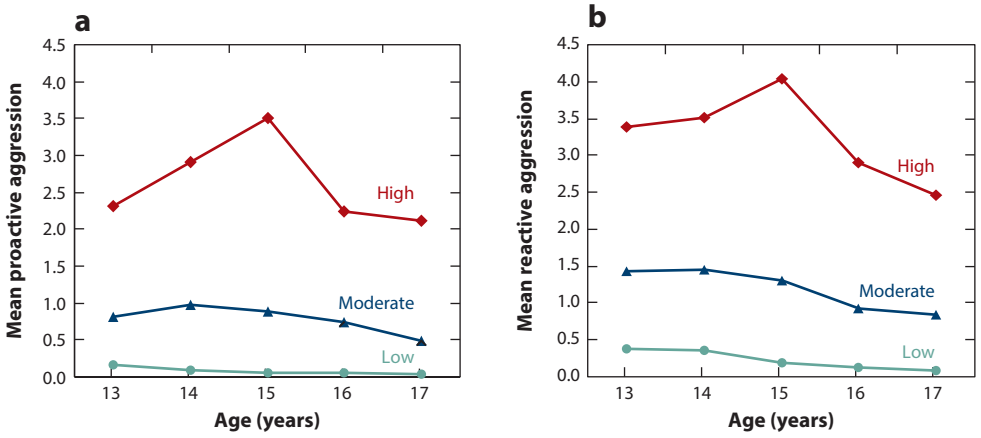

Figure 2

Trajectories of (*a*) proactive aggression and (*b*) reactive aggression from 13 to 17 years old.

These longitudinal studies of physical aggression during adolescence and early adulthood showed that the frequency of instances of physical aggression continues to decline with age for the majority of children. However, among the most physically aggressive individuals, there is often a slight increase during mid-adolescence, followed by a decline (Lacourse et al. 2002, van Lier et al. 2009; see **Figure 2**). Few studies have monitored the development of physical aggression from adolescence through adulthood with longitudinal studies. The best data comes from court records of a sample of male juvenile delinquents living in Boston in 1940 (Sampson & Laub 2003). The results showed a clear decline in arrests for violent crimes from early adulthood to old age. Similar results were obtained for physical aggression among couples from cross-sectional and short-term longitudinal studies (Bookwala et al. 2005, O'Leary & Woodin 2005, Suitor et al. 1990, Vickerman & Margolin 2008).

Thus, research on the development of physical aggression from infancy to old age indicates that (*a*) humans start to physically aggress before the end of their first year after birth, and (*b*) the frequency of physical aggression reaches a peak during the first 3–4 years after birth and then decreases until old age. A small group of individuals, mainly males, tend to use physical aggression more frequently than others throughout life, and they are more likely to increase the frequency of their physical aggression during mid-adolescence. Thus, from a learning perspective, one of the most important challenges for a young human is to learn not to physically aggress others.

4. BIO-PSYCHO-SOCIAL MECHANISMS THAT SUPPORT CHRONIC PHYSICAL AGGRESSION

To identify the mechanisms that support the developmental trajectories of physical aggression described above, we need studies that go beyond the mere description of these developmental trajectories. The fact that humans start to use physical aggression before they reach their first birthday suggests that they do not need to observe physical aggression by other humans to initiate physical aggression themselves. It also suggests that there is, indeed, a strong genetic-instinctual basis to human use of physical aggression. To understand the extent to which the different developmental trajectories of physical aggression are determined by genetic and environmental mechanisms, we need genetically informative research designs. However, to understand the bio-psycho-social

mechanisms that are triggered by genes and by environments at different periods during development, we also need designs that assess the different developmental pathways from conception to adulthood. These pathways may include neurophysiological as well as psychological and social processes. As we discuss below, this will require hard work by future generations of scholars.

4.1. Studies of Sex Differences

It has long been common knowledge that human males use physical aggression more often than human females. Sexual dimorphism is, indeed, one of the most robust findings from studies on aggression (Daly & Wilson 1990, Quetelet 1833). Men are found to use aggression more than females when studies focus on direct forms of aggression (e.g., physical or verbal aggression) and when the target of the aggression is an individual not known to the perpetrator. Conversely, females are found to use aggression more often than males when studies focus on indirect forms of aggression (e.g., psychological or social aggression) and when the target of the aggression is an individual known to the perpetrator (Archer 2000, Archer & Côté 2005).

However, the developmental origins of these sex differences in aggression have been studied only recently with large population samples of children. For example, with a large sample of Canadian children from 2 to 8 years of age (Côté et al. 2007b), significant differences were observed in the proportions of boys and girls who followed the highest and the lowest developmental trajectories of physical aggression, based on parent reports (see **Figure 3**). More boys than girls (53.6% versus 46.4%) were on the high physical aggression trajectory, whereas more girls than boys (57.2% versus 42.7%) were on the low physical aggression trajectory. The reverse was observed for indirect aggression (e.g., when the individual is mad at someone, they get others to dislike that person): More girls than boys (57.6% versus 42.3%) were on a high indirect aggression trajectory. With another large population sample and a different statistical approach, Baillargeon et al. (2007) reported that 5% of 17-month-old boys used physical aggression on a frequent basis compared to 1% of girls the same age, and the magnitude of the difference was the same one year later. Finally, in a comparison of six large longitudinal studies from Canada, New Zealand, and the United States, Broidy et al. (2003) reported similar sex differences based on teacher reports from school entry to adolescence.

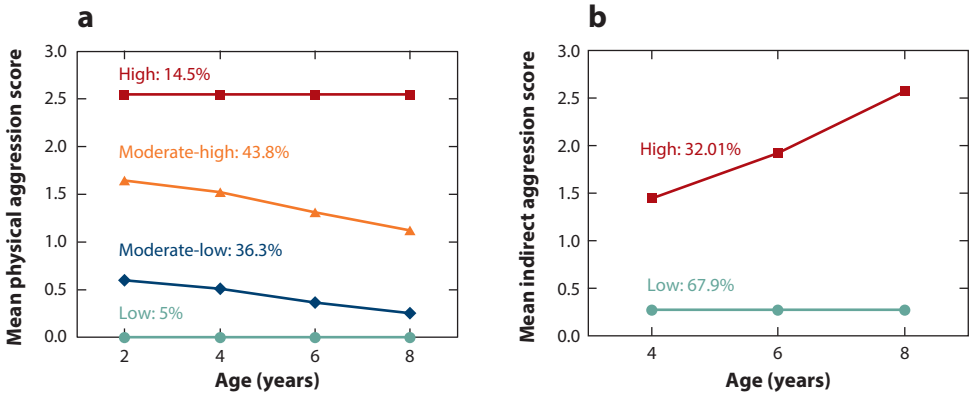

Figure 3

Developmental trajectories of (*a*) physical aggression between 2 and 8 years old and (*b*) indirect aggression between 4 and 8 years old. Figure adapted with permission from Côté et al. 2007b.

4.2. Twin Studies

One frequently used and genetically informative type of study involves comparing the frequency of aggressive behavior within and between pairs of monozygotic (MZ) and dizygotic (DZ) twins raised together. The basic logic behind these studies is that genetic mechanisms are likely to play an important role if MZ twins are more similar than DZ twins in their propensity to use physical aggression because MZ twins share the same genes, whereas DZ twins share only half of their genes, like non-twin brothers and sisters. Twin studies with elementary school children, adolescents, and adults have, indeed, shown that there is a relatively strong genetic component to the use of physical aggression (Brendgen et al. 2005, 2008; Burt 2009; Hicks et al. 2004; Rhee & Waldman 2002; Tuvblad et al. 2009). Not surprisingly, twin studies also indicate that genetic effects are probably mediated by brain development. For example, with a sample of twins in which continuity in proactive aggression (i.e., a type of predatory aggressive behavior) was shown to be influenced by genetic factors (Tuvblad et al. 2009), associations were found between the frequency of aggression, fear conditioning deficits (Gao et al. 2015), and a number of brain morphology characteristics related to the fronto-limbic-striatal circuit, such as cortical thickness in frontal regions (Yang et al. 2016).

To what extent are these genetic determinants of physical aggression active during early childhood? To address this question, a study of twins was initiated at birth and the frequency of physical aggressions was reported by parents at 20, 36, and 50 months (Dionne et al. 2003). At 20 months, the heritability of physical aggression was compared to the heritability of language development. Interestingly, the strength of the genetic influences on individual differences was higher for physical aggression (58%) than for expressive vocabulary (39%), whereas the strength of the shared environmental influence was high for expressive vocabulary (51%) but nil (0%) for physical aggression. Further analyses of the data on the development of physical aggression using the same twin sample indicated important genetic effects that changed over time (Lacourse et al. 2014). The contribution of genetic factors to the variance in frequency of physical aggression at 20 months was 60% and decreased to 50% at 50 months, whereas new genetic effects appeared at 36 and 50 months. Two separate sets of uncorrelated genetic factors accounted for the variation in initial level and growth rate. Results did not indicate any environmental effects (shared or nonshared) on the initial level of physical aggression (at 20 months) or on its stability and its growth rate from 20 to 50 months. Thus, during early childhood, when physical aggression is on the rise, genetic factors explain a substantial part of individual differences in the frequency of aggression (as seen in **Figure 1**). The fact that the genes involved appear to change on a yearly basis is not surprising considering that early childhood is a developmental period with accelerated growth of key instruments for the use and control of aggression, such as limbs and muscles, as well as neurological control over cognitive and emotional development.

Similar estimates of genetic effects were obtained from a larger longitudinal study with older twins (at 3, 7, and 10 years of age) using an assessment of aggressive behavior that was not limited to physical aggression (Hudziak et al. 2003). The developmental analyses from this study identified a similar dynamic process where genetic effects at 3 years old interacted with new genetic contributions at 7 years old. Interestingly, the important contribution of nonshared environmental factors was specific to a given age.

Although these two twin studies assessed different aspects of aggressive behavior (physical and nonphysical) by children from 20 months to 12 years old, they both support the hypothesis that the stability of aggressive behavior is influenced by both genetic and environmental factors from infancy to early adolescence. The larger study of children between 3 and 12 years of age had sufficient power to show that genetic effects were larger for boys and shared environmental effects

were larger for girls. It remains to be tested if this is true specifically for physical aggression, but developmental analyses of physical aggression during early childhood have shown that girls learn more quickly than boys to use alternatives to physical aggression and to replace physical aggression with indirect aggression (Baillargeon et al. 2007, Côté et al. 2006, Hay et al. 2011).

4.3. Molecular Genetic Studies

Molecular genetic studies of animals and humans have identified numerous genes involved in aggressive behavior. For example, a recent comparison of three genetic mouse models (Malki et al. 2014) identified a network of 14 genes associated with aggression-related behavior. Another mouse study (Yu et al. 2014) identified periods during development when the brain is particularly sensitive to the effects of dopamine and serotonin on aggressive behavior. Research in humans has similarly shown that various genes involved in serotonin and dopamine metabolism, for example, the *monoamine oxidase A* (*MAOA*), *dopamine receptor 2* (*DRD2*), and *serotonin transporter* (*5-HTT* or *SLC6A4*) genes (Belsky & Pluess 2013, Caspi et al. 2002, Pavlov et al. 2012), are associated with aggression in both humans and other animals. However, the genes that play a role in the development of aggressive behavior are not the same at different developmental periods, as the twin studies summarized above indicated. For example, Pingault et al. (2013a) modeled the age-dependent contribution of the *MAOA* gene to the development of physical aggression between 6 and 12 years of age and found that its influence appears to emerge over time. Using the same sample assessed during late adolescence and early adulthood, Ouellet-Morin et al. (2016) showed that the genetic moderation of adverse family environment by the *MAOA* gene, with reference to aggression and other forms of antisocial behavior, may be active only at certain levels of family adversity and varies according to the different antisocial outcomes that are being assessed.

Thus, molecular genetic studies with animals and humans have shown that numerous brain-related genes play an important role in the developmental trajectories of aggression. However, the genes involved vary with age and interact with the environment in which the individuals live.

4.4. Environmental Effects on Gene Expression (Epigenetics)

The study of environmental impacts on gene expression (epigenetics) and human aggressive behavior began only a decade ago. The basic point to remember concerning genetic influences and epigenetics is that genes can be turned on and off by environmental events through chemical signals, and genes can have effects only if they are turned on (Szyf 2009). Thousands of studies have shown that environments can play important roles in the development of behaviors and diseases by programming gene expression at different points during development. The study that led us to use epigenetic research to understand the development of chronic physical aggression was a mouse study where frequency of maternal licking at birth was shown to have a long-term impact on the offspring's ability to cope with stress through DNA methylation of the *glucocorticoid receptor* gene (Weaver et al. 2004).

It will take a few years before we have data on human behavior development and epigenetics as good as those provided by the mouse studies (Meaney & Szyf 2005), but numerous studies have shown that the epigenetic mechanisms observed in the mouse model probably apply to humans from the prenatal period onwards. For example, a recent study showed associations between placental methylation and the newborn reactive–poorly regulated profile, a well-known neurobehavioral profile (Paquette et al. 2015).

One of the key advantages of epigenetic research is that it provides a tool to study intergenerational transmission of behavior and health problems. The basic hypothesis is that parents' behavior

from conception onwards can impact offspring brain development, behavior, and health through its impact on the offspring's gene expression programming. This type of gene–environment interplay is well illustrated in the study of maternal mouse licking at birth, mentioned above. Parental behaviors in humans probably have epigenetic impacts on their offspring throughout development, and these epigenetic effects on brain development are likely to start early on during pregnancy (Glover 2011). One example of this mechanism is the putative impact of maternal mental health on offspring development. Numerous studies have shown associations between maternal mental health and children's behavior problems (e.g., Côté et al. 2007a, Herba et al. 2013). One of the underlying mechanisms of this link, besides genetic transmission, may be epigenetic impacts of maternal neuroendocrine functioning and behavior on offspring's brain development during pregnancy. For instance, Braithwaite et al. (2015) showed that maternal depression during pregnancy is associated with increased DNA methylation of 2-month-old male offspring's glucocorticoid receptor gene promotor region, which plays a critical role in the ability to respond to stress. Maternal depression during pregnancy was also associated with 2-month-old female and male brain-derived neurotrophic factor gene promotor, which plays an essential role in neurodevelopment.

The long-term epigenetic effects of maternal smoking during pregnancy are another good example of intergenerational effects of parental behavior on offspring's development. With a variety of research designs, researchers have associated smoking during pregnancy with many behavior problems in offspring, including chronic physical aggression during early childhood (Dolan et al. 2016, Gaysina et al. 2013, Huijbregts et al. 2008). In a UK longitudinal study of 800 newborn children (Richmond et al. 2015), epigenetic analyses of umbilical cord blood showed an association between maternal smoking during pregnancy and DNA methylation in seven gene regions. The duration and intensity of smoking during pregnancy also led to a dose-dependent response. The children were followed throughout development and blood samples were taken again at 7 and 17 years of age. The longitudinal analysis revealed that some methylation sites were persistently perturbed, whereas others showed reversibility. The investigators then focused on the methylation sites that were perturbed from birth to 17 years of age. Controlling for postnatal smoke exposure from mothers and fathers, they found that a critical window of exposure during pregnancy made the major contribution to the long-term perturbed DNA methylation sites.

The first epigenetic study on human chronic physical aggression (Provençal et al. 2013) compared DNA methylation in cytokines and their regulators in T cells and monocytes between Canadian boys who were on a chronic and a normal physical aggression trajectory from kindergarten to adolescence (Nagin & Tremblay 1999). Results provided evidence for an association between male physical aggression and differential DNA methylation in cytokines and their regulators in T cells and monocytes. A second study (Provençal et al. 2014), comparing the same two groups of boys (chronic aggression trajectory versus normal trajectory), used an epigenome-wide approach and identified 448 distinct gene promoters that were differentially methylated in the two groups. An identical study with females (Guillemin et al. 2014) showed that, for both males and females, the methylation of 31 gene promoters was associated with physical aggression.

Two further studies with the same sample of males focused on the association among chronic physical aggression, brain functioning, and DNA methylation. The first study (Booij et al. 2010) used positron emission tomography to compare brain serotonin synthesis in males on a high physical aggression trajectory with that of males on a low physical aggression trajectory. Results showed that males on the high-aggression trajectory had lower brain serotonin synthesis in the orbitofrontal cortex. The second study (Wang et al. 2012) hypothesized that this lower brain serotonin synthesis was associated with DNA methylation of critical genes in the serotonin pathway and detectable in peripheral white blood cells. Higher levels of methylation were, indeed, observed

in T cells and monocytes for the high-aggression group, who also showed lower brain serotonin synthesis.

Finally, Checknita et al. (2015) examined the association between the serotonergic system's *MAOA promoter* gene methylation and antisocial behavior during adulthood. The males from Wang et al.'s (2012) study who were on the normal trajectory from childhood to adolescence were compared to prisoners with an antisocial personality diagnosis. Results showed that *MAOA promoter* gene hypermethylation was associated with the antisocial personality disorder diagnosis, supporting the hypothesis that this hypermethylation is the mechanism that explains why impulsive aggression and antisocial behavior are associated with serotonergic system dysregulation.

4.5. Environmental Effects on the Brain and Aggressive Behavior

The epigenetic studies described above have shown that the environment can impact the development of aggressive behavior through its impact on gene expression, which, in turn, impacts brain development and eventually behavior. But the impact of the environment on behavior can be directly mediated by the brain. The clearest studies come from observations of animal behavior because they enable a more extensive control of the environment and more precise measurements of brain functioning and behavior. A useful recent example is a rat study that tested the hypothesis that chronic passive exposure to aggressive behavior can lead to aggressive behavior in the individual exposed. Suzuki & Lucas (2015) randomly exposed 72 rats to aggressive behavior for either 1 day or 23 days. Results showed that those who were exposed for 23 days had reduced dopamine density in the bilateral nucleus accumbens shell and increased amygdaloid receptor densities (serotonin). These effects on brain functioning interacted to lead to high levels of aggression. Thus, being forced to observe physical aggressions for long periods of time has an impact on brain functioning (dopamine–serotonin interactions) that can lead to high levels of aggression. However, this does not mean that a newborn rat needs to observe aggression to learn to aggress. Aggression, like mating, is a goal-directed innate social behavior associated with brain-regulated emotions and motivations and that can be measured in mice and flies as well as humans (Anderson 2016).

Numerous studies of school-age children have shown that aggression is associated with characteristics of the social environment (e.g., Anderson et al. 2010, Huesmann et al. 2017). In a large population sample study of the earliest social environment, the family, results showed that the best predictors of a high physical aggression trajectory between 17 and 42 months old were the following: young siblings, mothers with high levels of antisocial behavior before the end of high school, mothers who started having children at an early age, mothers who smoked during pregnancy, mothers with coercive parenting behavior, families with low income, and dysfunctional families (Tremblay et al. 2004).

However, few social environmental studies of aggression control for genetic effects on physical aggression or on social environmental variables that are correlated with physical aggression. Studies of genetically identical (MZ) twins are one of the best research designs to control for genetic effects while testing for environmental effects in human children. With a longitudinal study of 223 MZ twin pairs in Canada, Vitaro et al. (2011) examined whether having an aggressive friend increases one's aggression. This was done by testing if the difference in aggression between members of an MZ twin pair increased from kindergarten to first grade when one member of a pair had the more highly aggressive friend in kindergarten than the other. Results for both boys and girls showed that within-pair differences in friends' aggression among pairs of MZ twins in kindergarten significantly predicted increased within-pair differences in aggression the following year (first grade). These results clearly show that children's use of aggression can be influenced

through their close friendships even when we control for genetic effects. However, in a follow-up study that used the same sample of MZ twins and a similar control for genetic influences, Vitaro et al. (2016) found no effect of friends' aggression from middle childhood to early adolescence, possibly because older children, as compared to younger children, increasingly select their social environment in tune with their genetic dispositions (through a process known as a gene–environment correlation).

The same investigators also found that, although social experiences (such as friends' aggression) do not always predict changes in children's behavior, they may nevertheless operate to trigger the genetic liability for aggression. First, in two separate studies, they found that the genetic component associated with physical aggression was stronger when children had aggressive friends than when they had nonaggressive friends (Brendgen et al. 2008, van Lier et al. 2007). They also found that genetic influences were moderated by social experiences at the group level, such as classroom norms toward aggression. As expected, results confirmed that genetic factors did influence children's aggressive behavior, but that the genetic effects on aggression were attenuated or exacerbated depending on whether classroom norms were unfavorable or favorable to aggression, respectively, thus confirming that peer group norms impact genetic effects (Brendgen et al. 2013, Vitaro et al. 2015).

These peer group effects on the development of children's aggressive or antisocial behavior have been hotly debated as childcare has become a norm in Western societies (e.g., Belsky & Steinberg 1978). Data from a US longitudinal study specifically designed to investigate the effects of childcare did show that children who attended childcare for long periods of time were more likely to be disruptive during their elementary school years (Belsky et al. 2007). However, other longitudinal studies that also followed large random samples of children until the end of elementary school did not find these negative impacts (e.g., Zachrisson et al. 2013). Indeed, one population-based longitudinal study in Canada found that early initiation of childcare for children of mothers with low education helped these children learn not to physically aggress between 17 and 60 months (Côté et al. 2007a). Another study with the same sample compared the development of physical aggression, opposition, shyness, and social withdrawal between 6 and 12 years of age for children who went to childcare and those who did not (home care). **Figure 4** shows that, during their kindergarten year (age 6 years), the children who had not been in childcare were rated by teachers as less physically aggressive and less oppositional than those who had been in childcare. However, the difference between the two groups for both physical aggression and opposition disappeared by the second year in school (age 7 years). It is important to note that the reverse phenomenon was observed for teacher ratings of shyness and withdrawal. These results highlight the fact that, throughout development, humans adapt their social behavior to social group norms, but that this process takes time.

As with the twin studies discussed above, this does not mean that the social behavior of the children is totally explained by the group norms. The genetic and environmental effects can be observed when the research design is appropriate. An interesting example of this is a molecular genetic study (Belsky & Pluess 2013) using the same data as the US childcare study by Belsky et al. (2007). Results showed that the *dopamine receptor D4* (*DRD4*) gene moderated the effect of childcare quality on teacher-reported social skills (e.g., "makes friends easily," "controls temper when arguing with other children," "asks permission before using someone else's property"), but only in kindergarten and first grade. Interestingly, this time period is exactly the same school entry period in which significant differences in social behavior were observed between children with and without childcare experience in the Canadian study described in **Figure 3** (Pingault et al. 2015). These results highlight the fact that longitudinal studies need to differentiate transition periods from stable social environment periods.

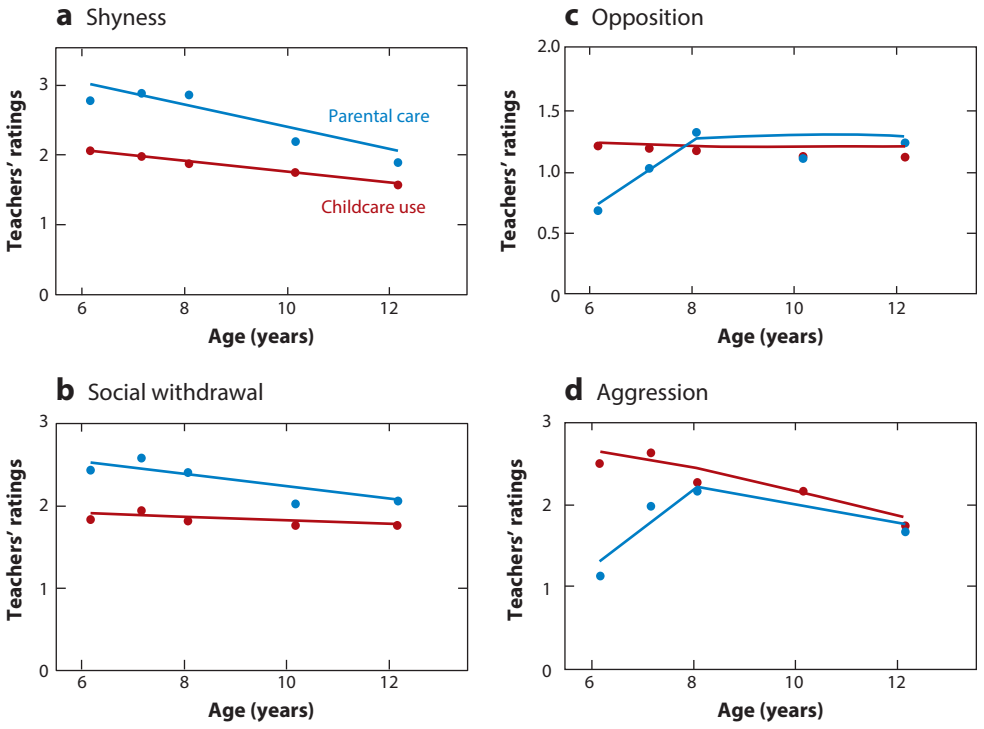

Figure 4

Developmental trajectories of behavior problems from 6 to 12 years old according to early childcare use. (*a*) Shyness. (*b*) Social withdrawal. (*c*) Opposition. (*d*) Aggression. Figure adapted with permission from Pingault et al. (2015).

Finally, it is important to briefly mention a completely different and very recent line of research on the interplay between the environment and the brain that has been labeled the gut-brain hypotheses. The human gut microbiome is a bacterial environment shown to have important impacts on the brain and on behavior. It has been linked to physical health problems but also to anxiety, depression, alcoholism, and aggression. Recent studies with animals have shown that the gut microbiome is important from the prenatal period to adulthood and that changing the bacterial environment of the gut can have significant and sex-specific impacts on social behavior, including aggression (e.g., Bailey & Coe 1999, Robertson et al. 2017, Sylvia et al. 2017).

4.6. Intergenerational Transmission: Numerous Interrelated Bio-Psycho-Social Channels

Intergenerational research on antisocial behavior, especially on the bio-psycho-social mechanisms involved, has been relatively limited. Longitudinal studies have shown that children of parents who were convicted of criminal behavior are more at risk of being themselves convicted of criminal behavior during adolescence and adulthood (see Farrington et al. 2017, Theobald et al. 2016). Adoption studies have also shown that children of parents with a criminal history are less at risk of criminal behavior when adopted by parents without a criminal record (Mednick et al. 1984). A Canadian longitudinal study on the early development of chronic physical aggression highlighted the fact that this association between the antisocial behavior of parents and that of children starts

very early in life (Tremblay et al. 2004). The study showed that maternal reports of antisocial behavior during their own adolescence predicted the chronic physical aggression of their child between 17 and 42 months old. This study further showed that the mothers of children who became chronically aggressive were, at the child's birth, more likely to be among the youngest and poorest, to be separated from the father, to have not completed high school, to have smoked during pregnancy, and to have had postpartum depression. This network of associations illustrates well the fact that the intergenerational transmission of behavior problems occurs through numerous interrelated bio-psycho-social channels. The child inherits a mix of their parent's genes, and their mother's smoking, stress, poverty, and depression during pregnancy impact the fetus' brain development through epigenetic mechanisms. From the postnatal period onwards, the physical and social environments created by a poor, young, depressed woman with low education, behavior problems, and coercive parenting in a dysfunctional family clearly fail to provide the care and education needed by the brain of a young child to learn to control their emotions and behavior. On the contrary, they may exacerbate further the expression of the child's genetic liability and contribute further epigenetic effects of their own.

However, the problems do not start with conception. We often forget that assortative mating (mating with someone who has similar characteristics) is one of the initial mechanisms that lead to the numerous interrelated bio-psycho-social channels that impact human development from pregnancy onwards. In a recent large (N = 707,263) population study in Sweden, Nordsletten et al. (2016) showed that there was more assortative mating for mental illnesses than for physical illnesses. The odds that a male with a psychiatric diagnosis had a mate with a psychiatric diagnosis was OR 2.24 (CI,2.21–2.27; $P < 0.001$), and the odds that a female with a psychiatric diagnosis had a mate with a diagnosis was OR 2.11(CI,2.08–2.14; $P < 0.001$). The correlation among mates in terms of diagnosis was 0.45 for attention deficit/hyperactivity disorder, which is strongly associated with chronic physical aggression from early childhood onwards (Carbonneau et al. 2016, Pingault et al. 2013b). It is important to emphasize that assortative mating brings together, for the reproduction of the next generation, mates that have similar histories, not only for physical and mental health problems but also for education, ability to self-control, and values on numerous crucial issues for children's development, such as nutrition, discipline, lifestyle, and respect for others (Domingue et al. 2014, Frisell et al. 2012, Grant et al. 2007, Kandler et al. 2012, Keller et al. 2013, Tognetti et al. 2014, Zietsch et al. 2011).

Figure 5 illustrates the complex network of interrelated genetic, epigenetic, neuropsychological, and social factors that are involved in the transmission of chronic physical aggression from one generation to another. This perspective is in line with the suggestion by the Nobel Prize winner and ethologist Nikolaas Tinbergen (1963) that behavior needs to be explained from four perspectives: the historical and survival value of the behavior in past generations as well as the development of the behavior within an individual's life and the proximal causal factors.

From the left to the right of **Figure 5**, we follow the intergenerational process with two lines of ancestors transmitting their genetic, economic, educational, cultural, health, and lifestyle characteristics to a male and a female who become parents of a child. The developmental changes in this child's DNA expression, brain, and behavior are shown to be determined by their intergenerational inheritance but also by the environments in which they will grow and interact. The uniqueness of this individual is largely determined by the genetic, economic, educational, cultural, health, and lifestyle characteristics they received from their ancestors through their parents but also by the unique environment in which they grow up.

To understand this complex process, it is useful to consider the developmental differences between MZ twins, DZ twins, and singleton siblings living with their parents or adopted by unrelated adults (e.g., Kendler et al. 2015, McAdams et al. 2015). Members of twin pairs, whether

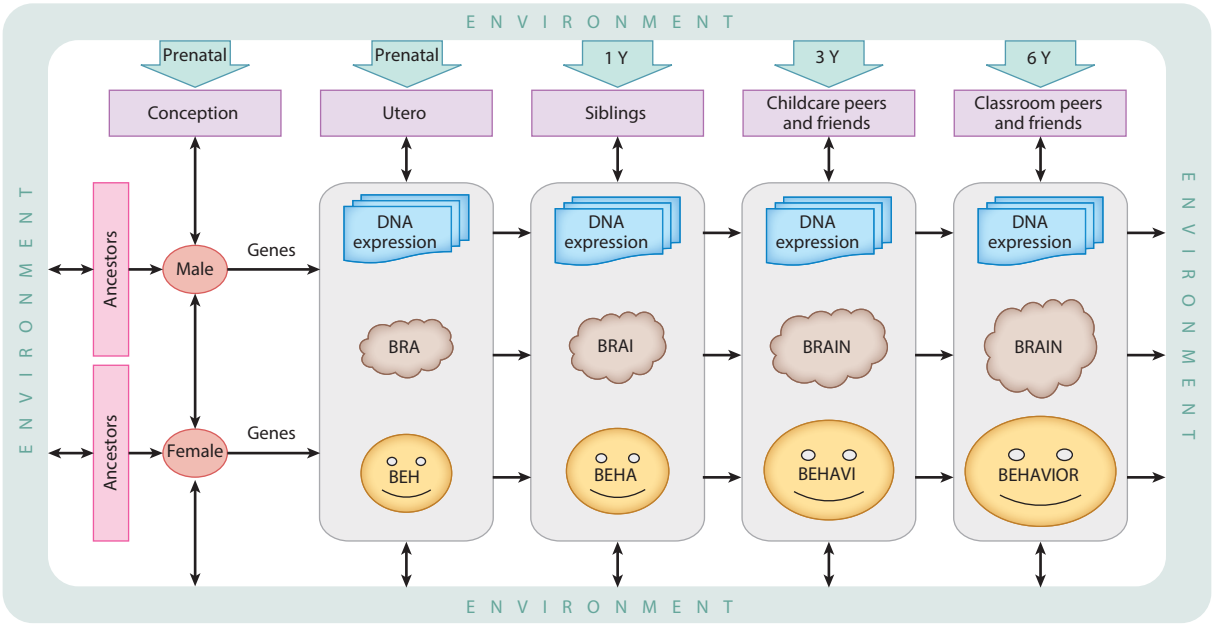

Figure 5

Network of interrelated intergenerational, genetic, epigenetic, neuropsychological, and social factors involved in the development of chronic physical aggression.

MZ or DZ, have the same ancestors and parents. When we consider DZ twins, we see that, with time, they become as different as ordinary siblings, but, because they share half of their genes, they become much more similar than adopted children living with the same parents. When we consider MZ twins, we see that, with time, they become much more similar than DZ twins and normal singleton siblings because they share the same genes. These comparisons highlight the power of genes. However, there is a limit to the power of genes, as can be seen from the fact that differences between MZ twins will increase with time because of the power of the environment, which impacts not only how they perceive the world and behave but also when their genes are expressed and, thus, how their brain functions and how they behave.

5. CAN CHRONIC PHYSICAL AGGRESSION BE PREVENTED?

Although understanding the development of physical aggression is, by itself, interesting, the aim of these studies is usually to provide information that will help prevent the development of chronic physical aggression problems and possibly reduce physical aggressions among humans. The numerous mechanisms involved in the development of chronic physical aggression described above suggest that we need to target many risk and protective factors. These include parents' behavior problems, family functioning, peer interactions, childcare, and school environments, as well as biological mechanisms related to brain functioning. However, one of the main findings of these studies is that humans start to use physical aggression during early childhood, and the children who are on a high physical aggression trajectory at that time are more likely to maintain higher frequencies of physical aggression into adulthood. This, of course, suggests that early childhood interventions are more likely to prevent chronic aggression problems than later interventions. This is clearly not a new idea. Erasmus [1529 (1985), p. 321] summarized it well in his essay on

education: "We should be especially careful with our children during their first years. For at this stage their behavior is guided by instinct more than by reason, so that they are inclined equally to good and evil—more to the latter perhaps—and it is always easier to forget good habits than to unlearn bad ones." However, the idea that physical aggression and other antisocial behaviors are learned from the environment mainly during adolescence led both the Surgeon General of the United States (Surg. Gen. US 2001) and the World Health Organization (WHO 2002, p. 31) to conclude that violent behavior starts during adolescence and that these violent adolescents neither were highly aggressive during childhood nor had behavior problems during early childhood. The result of these beliefs is that most experiments to prevent aggressive or antisocial behaviors target preadolescents and adolescents. In this section, we focus on the few studies that have targeted young children, starting with interventions during pregnancy.

5.1. Prevention Initiated During Pregnancy

The best known experimental intervention targeting pregnant women at high risk of having children with chronic antisocial behavior is the Elmira project in the United States (Eckenrode et al. 2010, Olds et al. 1986). The pregnant women were young, unmarried, and poor. Nurses visited the women once a month between the twenty-fourth week of pregnancy and the child's second birthday, giving support to the mothers in three areas of their life: (*a*) personal development, including education, workforce integration, and family planning; (*b*) health-related behavior, including smoking prevention and adequate nutrition for the mother and child; and (*c*) competent care of the child and maternal sensitivity. The nurses also helped link mother and child with community services. Results showed that the intervention reduced child abuse and neglect and maternal delinquency (Olds et al. 1986, 2007) and showed, at the 19-year follow-up, a significant reduction of arrests for the female offspring (Eckenrode et al. 2010). The latter finding is an especially important and encouraging result because female offspring will become the mothers of the next generation.

A more recent pregnancy experiment was carried out in a poor neighborhood of Dublin, Ireland (Doyle & PFL Eval. Team 2016). The study randomly allocated 230 pregnant women to a high- and a low-support program. The high-support program included home visits up to school entry at 4 or 5 years of age and a parenting support program. Results after 5 years showed significant differences between the two groups of children in numerous developmental outcomes, such as overall cognitive development, language development, attention, hyperactivity, motor skills, social competence, autonomy, and amount of hospital services used from birth to school entry. Surprisingly, however, there were no significant impacts on children's aggression, oppositional defiance, anxiety, and prosocial and respectful behavior, as assessed by teachers. One would expect that, if the wide-ranging positive impacts are maintained in the long run, they will reduce the likelihood of aggressive antisocial behavior by favoring social acceptance from normative peers and improved academic performance.

5.2. Prevention Initiated During Early Childhood

Children's cognitive abilities and parents' disciplinary skills have been the focus of most prevention programs during the preschool period. Head Start programs for socially disadvantaged children aged 3–5 years in the United States were shown to improve academic success in the short term and to prevent arrests and court referrals in the long term (Garces et al. 2002). The best known of these programs is the High-Scope Perry Preschool project, which targeted African American children age 3–4 years with parents with a low IQ. The 2-year program included a daily preschool program

aimed at increasing cognitive abilities and weekly home visits. Several long-term assessments up to age 40 showed that children who participated in the program were more likely to be employed, more likely to earn higher annual incomes, more likely to have graduated from high school, and less likely to have been arrested compared to the children who were part of the control group (Schweinhart et al. 1993). A study of the mechanisms that could explain these important long-term results concluded that the preschool program had impacts on the social skills of the children rather than on the targeted cognitive skills (Heckman et al. 2013).

Several parent training programs have been tested for preschool-aged children (e.g., Patterson 1982, Sanders et al. 2000). One of these programs for parents of preschoolers with behavior problems included components that also targeted the child and the teacher. A randomized control trial of its impact was made with a sample of 159 4- to 7-year-old children with oppositional defiant disorder (Webster-Stratton et al. 2001). The children were assigned to one of the following conditions: the parent program only; the child program only; the parent and teacher programs; the child and teacher programs; and the child, parent, and teacher programs. The five conditions were compared to a waiting-list control condition. At the end of a 6-month intervention period, children in the three conditions with the child program showed more prosocial skills with peers than the children in the control condition. No difference between treatment and control was observed for children in the two conditions without the child program. Similarly, parents in the three parent program conditions manifested less negative and more positive parenting and reported fewer child behavior problems than parents in the control condition and parents in the two conditions without parent training. Finally, children's behavior problems at school were reduced for those whose teacher participated in the teacher program relative to the control children. Overall, the conditions that included all three programs produced the best results after 6 months. At a 2-year follow-up, teachers reported an equal proportion of children with behavior problems across all five treatment conditions (i.e., around 50%). However, parents in the condition that included both the parent and teacher programs reported fewer cases of behaviorally disordered children relative to only one other condition, the parent-program-only condition, suggesting that more than one component may be necessary to achieve optimal results (see also Gardner et al. 2006). In an 8- to 12-year follow-up study of the children in the parent training condition, Webster-Stratton and her colleagues (2011) found that the majority maintained their postintervention progress, although one out of four reported major delinquent acts or had contact with the justice system.

5.3. Prevention Initiated During the Elementary School Period

Multitarget, multicomponent programs for preventing conduct problems in at-risk children have also been assessed during the elementary school years. For example, the Fast Track program (Conduct Probl. Prev. Res. Group 1992, 2004) had six components: (*a*) group parent training; (*b*) home visits; (*c*) group-based social skills training with the children; (*d*) peer-pairing, in which a target child and a no-risk peer participated in guided play sessions; (*e*) academic tutoring; and (*f*) teacher support. The program was implemented in four sites in the United States and targeted a large sample of children age 7 who scored above the ninetieth percentile on the aggressive–disruptive scale of the Child Behavior Checklist. The program was initially set to last until the end of elementary school but was eventually extended to tenth grade. After the first three years, the evaluation results were, at best, moderate, with effect sizes varying between 0.2 and 0.5 on teacher ratings of disruptive behavior. After five years (by age 11 or fifth grade), 37% of the randomly assigned Fast Track children had no conduct problem dysfunction, compared to 27% of control children (Conduct Probl. Prev. Res. Group 2002). Evaluations in late adolescence showed that the children in the prevention group were significantly better off with respect to

important outcomes, such as Diagnostic and Statistical Manual of Mental Disorders diagnoses for conduct disorder, criminal offenses, and interpersonal violence, compared to their counterparts in the control group, although the effect sizes remained moderate (Conduct Probl. Prev. Res. Group 2010). Finally, by age 25, 69% of the participants in the control group manifested at least one externalized, internalized, or substance-related problem, compared to 59% in the experimental group. Interestingly and importantly, mediation analyses showed that about one third of Fast Track's impact on later crime outcomes was accounted for by improvements in social and self-regulation skills during childhood (ages 6–11), such as prosocial behavior, emotion regulation, and problem solving (Sorensen et al. 2016).

A less intensive and shorter program was tested experimentally in Canada at approximately the same time and targeted kindergarten boys from schools in low socioeconomic areas who were among the most aggressive and hyperactive. The 2-year program (between 7 and 9 years of age) included a home-based parent training component and a school-based social-cognitive skills training component. Notably, the social-cognitive component was delivered at school in a small-group format that included one or two target boys and three or four prosocial peers. In comparison to the control group, boys who participated in the intervention were found to have less self-reported delinquency and substance use throughout adolescence (Castellanos-Ryan et al. 2013, Lacourse et al. 2002, Tremblay et al. 1995, Vitaro et al. 2001) and to have fewer criminal records by 24 years of age (Boisjoli et al. 2007). In addition, the intervention reduced property crime by age 28 but did not seem to impact violent crimes (Vitaro et al. 2013).

The experimental preventive interventions reviewed above show important short-, medium-, and long-term effects. But we must admit that we are far from having the impact we would like to have on the lives of these unfortunate children and parents that suffer from their behavior problems and create much suffering around them. How can we push forward to better understand the onset of these behavior problems and the means to change the life-course of these unfortunate people?

Long-term interventions that aim for long-term impacts will, of course, always appear to lag behind the advancement of knowledge when they are eventually published. For example, the preventive interventions described above did not include genetic, epigenetic, or brain imaging assessments because they were all initiated before the advancement of knowledge in these areas. We clearly need a new generation of preventive experiments that will make use of the recent knowledge on chronic aggression development to plan the interventions and to assess the long-term impacts. For example, to have significant long-term impacts, we will most likely need to tailor interventions with babies to their genetic, epigenetic, and neuroendocrine profiles, in the same way we tried to tailor the past interventions to the differences in behavior profiles and profiles of the family needs. We may also need to adopt a developmentally sensitive approach that includes the notion of stepwise continuous prevention for the extreme cases of individuals who do not revert to normative behavior during a given developmental period (Vitaro & Tremblay 2016). The next section gives examples of prevention experiments that would help achieve these aims.

5.4. Toward Integrated Bio-Psycho-Social Prevention Trials

We suggest that the intergenerational nature of physical and mental health problems highlighted above (**Figure 5**) is key in planning future prevention experiments and that this should lead to prevention experiments that start during early pregnancy at the latest. Parents who have had behavior problems carry with them pervasive high-risk environmental conditions (e.g., low education, low income, poor neighborhoods, and risky lifestyle choices such as use of tobacco, alcohol and drugs and unhealthy nutrition), which impact childhood and adulthood psychopathology through many interrelated channels, including impacts on the children's DNA methylation. Interventions that

do not start close to conception could not impact the many causal pathways that are already present during fetal life. Bovet (1951) already suggested using this intergenerational perspective in the first World Health Organization report, which happened to be on juvenile delinquency. Epigenetic research has now provided access to one of the important mechanisms by which the intergenerational problems are transmitted. If brain development is compromised through epigenetic effects during pregnancy, and if the child's parents also lack self-control, then it is easy to understand that the child will lack the cognitive and environmental support needed to develop the self-control that is essential to learning effective alternatives to physical aggression.

From this perspective, mothers are likely to have the greatest impact on early gene expression because their lifestyle during pregnancy has direct biological impacts on the child's development. Epigenetic effects during pregnancy suggest that we need to fundamentally revisit our thinking concerning early prevention of chronic physical aggression because, although male children are much more severely affected, pregnant women need to be our prime target to prevent a new generation of males and females with chronic physical aggression and similar intergenerational physical and mental health problems. This epigenetic perspective suggests that successful prevention of many physical and mental health problems may be easier to achieve by ameliorating the pre- and early postnatal environment, rather than by chasing bad genes (Bernet et al. 2007, Gluckman et al. 2008) or trying to change individuals' environments once they clearly have physical or mental health problems. Finally, we emphasize the fact that mothers, fathers, and children should not be blamed for the genes and the environment they inherit at conception and onwards. These families need to receive intensive and long-lasting support to break the intergenerational cycle of chronic behavior problems.

Intergenerational prevention trials should be initiated by enrolling in longitudinal and experimental studies large samples of young primiparous pregnant women with low levels of education. Intensive preventive interventions should be randomly allocated, especially to pregnant women with a history of antisocial behavior problems and their spouses, specifically targeting their lifestyle during pregnancy (i.e., smoking, drinking, drugs, spousal abuse) and their parental care and family life from birth onwards. Judging by the results of past experiments with these families (Boisjoli et al. 2007, Conduct Probl. Prev. Res. Group, 2005, Doyle & PFL Eval. Team 2016, Eckenrode et al. 2010), it is likely that supportive interventions maintained until the children are young adults will have the strongest impacts. Data collection would include genetic and repeated epigenetic information on parents and children and parenting behavior, as well as brain imaging and cognitive, health, and behavior development of the children. Longitudinal and experimental studies such as these will assess the short- and long-term impacts of the preventive interventions, but they will also advance knowledge on the causal mechanisms involved in the development of problem behaviors. The identification of these mechanisms will, in turn, help researchers design the following generation of interventions.

We believe that we will substantially advance our understanding of the mechanisms that lead to chronic physical aggression if, in these longitudinal and experimental studies, we regularly monitor the changes in epigenetic profiles, brain structure, brain functioning, cognition, and language, as well as changes in social-emotional behavior from birth onwards. This monitoring should, of course, include the changes in the children's environmental factors, such as parenting practices, family functioning, peer relationships, and social environments.

6. CONCLUSIONS

The aim of this review was to describe a bio-psycho-social approach to understanding and preventing the development of chronic physical aggression. We first highlighted the fact that, from a

long-term historical perspective, the debate on the developmental origins of aggression opposed those who supported a genetic mechanism (nature) to those who supported an environmental mechanism (nurture). We then presented recent longitudinal studies that show that human physical aggression starts at the end of the first year of life, reaches a peak in frequency before entry into kindergarten, and then decreases in frequency until old age. The appearance of physical aggression during the first year of life suggests that a human's very first acts of physical aggression are spontaneous reactions to anger (nature), rather than imitations of physical aggression in their environment (nurture). The very high frequency of physical aggression during early childhood and its decline throughout life also indicate that there are important brain maturation processes that are involved in the development of the ability to control physical aggression and that the environment plays an important role in learning alternatives to the use of physical aggression to solve problems. Molecular genetic studies with animals and humans, as well as twin studies, have confirmed important genetic influences on the use of physical aggression. However, more recently, epigenetic studies have highlighted the important role environments play, from conception onwards, in the expression of genes that impact brain development and behavior. From the present state of knowledge, we can conclude that the development of chronic physical aggression is generally influenced by genetic and environmental factors through numerous interrelated bio-psycho-social channels from conception onwards. The general mechanism that can be hypothesized from the recent bio-psycho-social studies is that the interrelated bio-psycho-social factors involved in the development of chronic physical aggression are generally the product of an intergenerational transmission process through assortative mating, genetic inheritance, and inheritance of physical and social environmental conditions that handicap brain development and support the tendency to use physical aggression as a means of solving problems.

Given these intergenerational mechanisms and the onset of physical aggression in infancy, it appears clear that preventive interventions should start early in pregnancy at the latest and continue throughout childhood and adolescence. In many cases, it may take more than one generation to break the intergenerational transmission. To our knowledge, no experimental preventive intervention with pregnant women has, to this day, specifically targeted risk for chronic physical aggression.

We suggest that the life-long beneficial impacts of preventive interventions with families of children at high risk of chronic physical aggression will be obtained only if intensive interventions are initiated early in pregnancy and maintained throughout the children's development. Based on results from previous studies (Eckenrode et al. 2010) and the key role of maternal health and lifestyle in brain development during fetal life and early childhood, we hypothesize that the effects of these very early preventive interventions will be greater for girls and that these effects will have a still greater impact on the next generation of boys and girls.

DISCLOSURE STATEMENT

The authors are not aware of any affiliations, memberships, funding, or financial holdings that might be perceived as affecting the objectivity of this review.

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