Journal of Experimental Criminology (2005) 1: 397–415

Towards an epigenetic approach to experimental criminology: The 2004 Joan McCord Prize Lecture

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Abstract. One of the numerous important contributions of Joan McCord to criminology was her long term follow up of an exceptionally well designed experimental prevention study initiated in the 1930s. Her work influenced a large number of longitudinal and experimental studies which form the basis of developmental and experimental criminology. The aim of this paper is to highlight how developmental criminology, experimental criminology, and developmental genetics (epigenetics) are starting to blend together to explain the causes of antisocial behavior, and more importantly to help prevent chronic antisocial behavior. The paper uses physical aggression as an example of a developmental outcome of gene–environment interactions.

Key words: adolescent delinquency, criminal behaviour, developmental criminology, experimental criminology, gene–environment interactions, onset of physical aggression, physical aggression, prevention science, randomized clinical trials

Introduction

Joan McCord was not only a pioneer of experimental criminology, she was a pioneer of experimental prevention of criminal behavior, and a pioneer of developmental prevention of criminal behavior. In fact Joan McCord made many other important contributions (McCord 2002); however, this paper will concentrate on the links between developmental criminology, experimental criminology, prevention science, and gene–environment interactions. It will also focus on a specific category of criminal behavior: physical aggression.

Experimental prevention of crime in 1930: The Cambridge-Somerville youth study

Although Joan McCord received her basic training in criminology more than half a century ago, she was initiated to experimental criminology with one of the best designed experimental prevention trial in criminology. The Cambridge-Somerville experiment was planned in the early 1930s by Richard Clark Cabot, a Professor of Social Ethics and Clinical Medicine at Harvard University. His cousin, Judge Frederick Cabot, created the Judge Baker Guidance Center to assess and recommend the juvenile court regarding treatment of delinquents (McCord 1992). According to Gordon Allport (1951), Dr. Cabot had been strongly impressed by the results of Sheldon and Eleanor Glueck's (1934) study of 500

criminal careers. The words of Allport (1951, p. v), summarizing the Glueck's results published 75 years ago, read as if they had been written for the 2005 American Society of Criminology meeting: "Reformatories, they discovered, did not reform. Nor did any of the current methods for dealing with criminals seem to any degree to impede their antisocial course of conduct. Lines for delinquent careers, it turned out, are laid early in life, but the causative factors are so little understood that preventive and remedial social policies are largely ineffectual." That was also the story that had been told by the clinical work of Healy and Bronner (1926) who were invited by Judge Cabot to create the Judge Baker Guidance Center.

Having made important contributions to the etiology of heart disease and the diagnosis of numerous illnesses, Dr. Cabot realized that preventing criminal careers was an extremely complex enterprise that needed to be based on strong empirical facts (Allport 1951; McCord 1992). The Cambridge-Somerville experiment was meant to be a model for the challenge Dr. Cabot had thrown to the participants of the 1931 National Conference of Social Work. In his presidential address he exhorted social workers to "Measure, evaluate, estimate, appraise your results in some form, in any terms that rest on something beyond faith, assertion, and illustrative cases" (Allport 1951, p. vi). In fact, Cabot decided to go much further than simply "measure, evaluate, estimate, or appraise." He created a randomized clinical trial that remains a classic, although probably ignored by the large majority of social work and criminology professors. A total of 650 elementary school boys between 5 and 12 years of age were randomly allocated to a treatment and a control group after having been paired, based on teacher assessments of deviant behavior.

The treatment was meant to last 5 years to test Cabot's conviction that "Friendly understanding – implying an ingredient of love – is the basis of all therapy. This affiliative relationship, of course, should be established early in life before bad habits and an antisocial outlook become too firmly rooted." (Allport 1951, p. vi). Note that "early in life" here means between 5 and 12 years of age. From the perspective of "early" prevention not much changed over the following 60 years since the largest and most intensive randomized clinical trial to prevent antisocial behavior initiated in the 1990s targeted 6-year-old children (Conduct Problems Prevention Research Group 2002; Tremblay et al. 1999 in Quay). What has changed is the belief that "friendly understanding" is the corner stone for changing the behavior of disruptive elementary school children. However, we needed Joan McCord's and Richard Clark Cabot's beliefs in the best designed scientific experiments to learn this lesson. In the words of Allport (1951, p. vi) "He (Cabot) was a scientist as well as a physician and moralist: he wished to put his ethical propositions to a test. The hardheaded empiricism of the Gluecks' study had impressed him."

We are extremely lucky that in 1957, Joan McCord, a recent mother and a candidate student in philosophy at Harvard, "was offered a small sum of money to evaluate effects of the Cambridge-Somerville Youth Study on crime" (McCord 2002, p. 99), and that she had the foresight and the stamina to follow the subjects

up to the mid-1970s (McCord 1978; Dishion et al. 1999). Her work not only showed that the best intentions and a long term friendly relationship is not sufficient to help prevent criminal behavior with high risk elementary school boys; it also gave strong indications that such interventions could have serious negative long term effects. This demonstration is one of the best argument for the use of randomized clinical trials with long term follow-ups to test the effectiveness of preventive and corrective interventions.

The development of criminal behavior

The age–crime curve

With hindsight the importance of Sheldon and Eleonore Glueck's work on the development of criminal behavior is becoming more and more obvious. Not only did their work influence modern experimental criminology through Dr. Cabot and Joan McCord, but their subjects are still being used by the top ranking criminologists. Sampson and Laub (2003) recently published longitudinal analyses of official criminal data for 500 juvenile delinquents the Gluecks had originally assessed in the 1940s (Glueck and Glueck 1950, 1968). The longitudinal data from ages 7 to 70 (Sampson and Laub 2003) replicate the age–crime curve, and the age–violent crime curve results which have been observed in 20th century cross-sectional studies based on official crimes (Figure 1) (e.g., Blumstein et al. 1988). Violent offenses appear in preadolescence, increase sharply during adolescence, and then decrease slowly up to old age. If, as hypothesized by learning theories (Bandura 1973; Reiss and Roth 1993; US Human Capital Initiative Coordinating Committee 1997), young humans learn to physically aggress by imitation of aggressions observed in the home, in the neighborhood, and in the media, the increase in physical violence during adolescence makes sense. The older they are, the more they have observed acts of physical aggression, and the more likely they

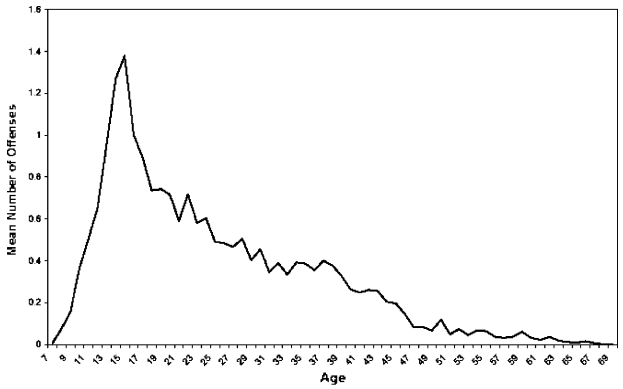

Figure 1. Actual mean number of offenses for total crime (Laub and Sampson 2003).

are to imitate what they have seen. The beauty of these data is not only that they confirm the prevalent psychological theory, but they replicate the data that Adolphe Quetelet, the Belgian astronomer-statistician, started collecting in the late 1820s (Quetelet 1833). They also replicate data on homicides in the 17th, 18th, and 19th centuries which were recently analyzed by Manuel Eisner (2003).

The age–crime curve is probably the most robust criminological observation. However, there are at least two major problems with that robust finding: first, as seen in Figure 1, it describes the mean trajectory of a sample of highly delinquent juvenile males, not the trajectory of the majority of humans; second, the age–violent crime curve studies limited their focus on adolescents and adults (Tremblay 2003a). The age–violent crime curve figures give the impression that physical aggressions appear with the legal age for criminal responsibility – as if lawmakers had chosen the age for criminal responsibility after detailed studies of child development. Those who decided to study elementary school children to understand the precursors of adolescent delinquency discovered that elementary school children used physical aggression before they had accumulated 12 or 13 years of social learning. In fact, Robert Cairns, a good friend of Joan McCord, and his colleagues (Cairns and Cairns 1994; Cairns et al. 1989) showed that the mean levels of physical aggression was decreasing from 10 to 18 years of age in samples of males and females in North Carolina.

One explanation for this observation could be that, although the majority of youth reduce the frequency of physical aggressions as they grow older, a minority are increasing their frequency of physical violence and are being processed through the legal system which generates the statistics for the age–crime curve. To test this hypothesis, we needed to go beyond a description of the mean developmental trajectories of delinquents, and identify the different types of developmental trajectories that children are following. Nagin and Tremblay (1999) used a semi-parametric mixture model, with a longitudinal sample of more than 1,000 kindergarten boys in schools from low-socio-economic areas of Montreal, to describe these different trajectories. Why use males from low-socio-economic areas to study the development of physical aggression? The answer can be found in the report "Reducing Violence" from the Human Capital Coordinating Committee (1997, p. 11): "Certain environmental conditions not only trigger violence, they can also seem to teach aggressive or violent behavior patterns. For example, poverty is associated with both sudden violent outbursts and long-term, habitual aggression."

Results (Figure 2) from teacher ratings of boys from low socioeconomic areas in Montréal confirmed the North Carolina data. The large majority of boys from the poorest inner city areas of Canada were using physical aggression less frequently as they grew older. Only a very small group of boys (4%) did not show the declining trend; these were the boys who had the highest level of physical aggression in kindergarten and remained at the highest level until adolescence. When interviewed at ages 15 and 17, they were the boys who reported the highest frequency of physical violence, and they were the ones most frequently found guilty of infractions before 18 years of age. Thus, the increase in the age–crime

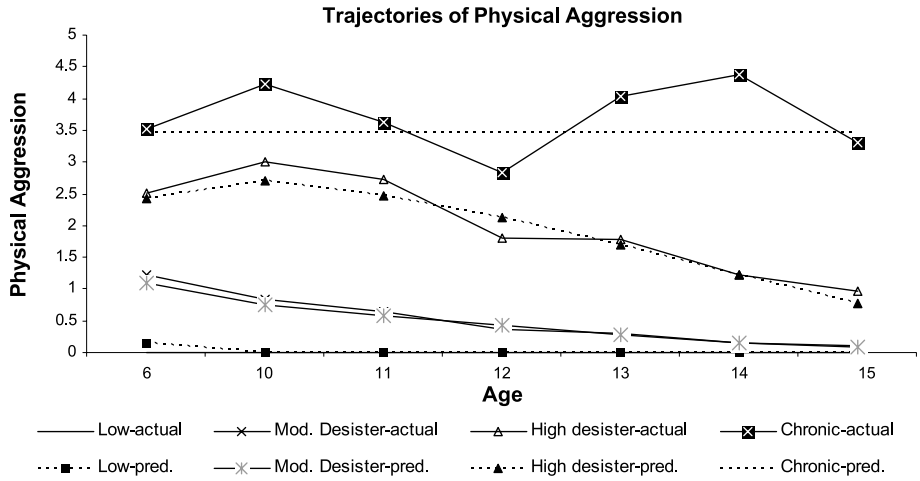

Figure 2. Physical aggression trajectories (Nagin and Tremblay 1999).

curve during adolescence for physical aggression would be produced by the fact that, during this period, the police and judicial system start arresting and convicting individuals who have been physically aggressing others at least since kindergarten. An international team of investigators replicated these finding using five other longitudinal studies in Canada, New Zealand, and the USA (Broidy et al. 2003).

Onset of physical aggression

Figure 2 clearly shows that all the boys tended to be at their peak level in frequency of physical aggression at 6 years of age, when they were at the end of their kindergarten year. If this is the case, when do they start to use physical aggression? There is a long history of case studies of physical aggression in young children. One of the first publication of a "developmental origins of aggression" study was published by Augustine of Thagaste (AD 397/1960, p. 49), later known as Saint Augustine. One millennium and a half later, in the "rage" section of the "The expression of the emotions in man and animals," Darwin (1872, p. 243) writes: "Every one who has had much to do with young children must have seen how naturally they take to biting, when in a passion. It seems as instinctive in them as in young crocodiles, who snap their little jaws as soon as they emerge from the egg."

Recently, large Canadian longitudinal epidemiological studies using parent reports confirmed the case study observations. From 9 to 30 months of age the proportion of children who are reported to use physical aggressions increases substantially (Tremblay 2004); also, the frequency of use appears to increase up to the end of the third year after birth (Tremblay et al. 2004). This remarkable

increase is then followed by a continuous decline in frequency (Côté et al. in press; NICHD 2004; Tremblay et al. 1996) as seen in Figure 2.

The same general developmental picture is drawn whether we use data from different periods, data from different countries, data from different reporting sources (filmed interactions in day care, parents' detailed records, or parents' recall of behavior in the past months), or data from different methodologies (cross-sectional or longitudinal, calculated as an absolute frequency over a given period of time, a relative percentage of social behaviors, a percentage of individuals using the behavior, or a general estimate of the frequency of the behavior) (Bridges 1931, 1933; Côté et al. in press; NICHD 2004; Restoin et al. 1985; E.A. Sand 1966; Tremblay et al. 1996, 1999, 2004). Frequency of anger outbursts and physical aggression increase rapidly from the first year after birth to approximately the third, and then the frequency decreases. Unfortunately, none of the longitudinal studies tracing the developmental trajectories during early childhood are old enough to report on trajectories of physical aggression during adolescence and adulthood. However, we know from predictive studies that aggression during childhood is the best predictor of aggression during adolescence and adulthood (e.g., Broidy et al. 2003; Huesmann et al. 1984; Loeber et al. 2005). Thus, although physical aggressions by very young children appear qualitatively different from physical aggressions by adolescents and adults, the trajectory of the former appears to lead to the trajectory of the latter.

This overview of the available data on the development of physical aggression over the life-span is summarized in Figure 4: most humans have used physical aggression before they reach 36 months of age; humans use physical aggression most often between 18 and 42 months after birth; if humans are learning to physically aggress through imitation, this learning is happening in the first 2 years after birth, not by watching television and playing video games in middle childhood or adolescence; humans clearly learn not to use physical aggression, they learn to use alternative solutions, and this learning has started well before they enter school. For criminologists, it appears important to note that this phenomena is probably not restricted to the "crime" of physical aggression. There is growing evidence that it is true also for stealing (taking things from others), vandalism (destroying others' belongings), and fraudulent behaviour (e.g., lying) (INSERM, 2005; NICHD 2004; Tremblay 2004).

Genetics and physical aggression development

Onset of physical aggression and individual differences

The longitudinal data on physical aggression from infancy onwards, summarized in the previous section, indicates that physical aggression is not a behavior children learn like reading or writing, nor an illness children "catch" like poliomyelitis or smallpox. It is rather a behavior like crying, eating, grasping, throwing, and running, which young humans do when the physiological structure is in place.

The young human learns to regulate these "natural" behaviors with age, experience, and brain maturation. The learning to control process implies regulating your needs to adjust to those of others, and this process is generally labeled "socialization."

It is not hard to imagine why the evolutionary process would have given humans a genetic program coding for all the basic mechanisms in order to react to hunger and to threat. Young children's muscles are activated to run, push, kick, grab, hit, throw, and yell with extreme force when hungry, when angry, or when they are strongly attracted by something. However, stating that humans are genetically programmed to be able to physically aggress when needed, is different from stating that the frequency of the physical aggressions they use is genetically programmed. Since all 18-month-olds who have developed normally can, and possibly, do physically aggress, but not all do so at the same frequency and with the same vigor, to what extent are these individual differences due to the genetic program they have inherited or to the environment in which they have been growing?

The trajectories shown in Figures 2–4 clearly indicate that these individual differences exist at any given point, starting in early childhood; but the most interesting phenotype is the development over time. There is obviously intra-individual change over time. Most children learn to reduce the frequency of the use of a behavior which they apparently did not need to learn. However, relatively stable differences among individuals remain. What are the gene–environment mechanisms which explain the change and stability? They are possibly very similar to the mechanisms which explain the developmental trajectories of growth in height. Genes code for the growth mechanisms, but there are individual differences in these coding, as well as environmental differences (e.g., access to food) which lead to stable individual differences. Thus, the individual differences in the

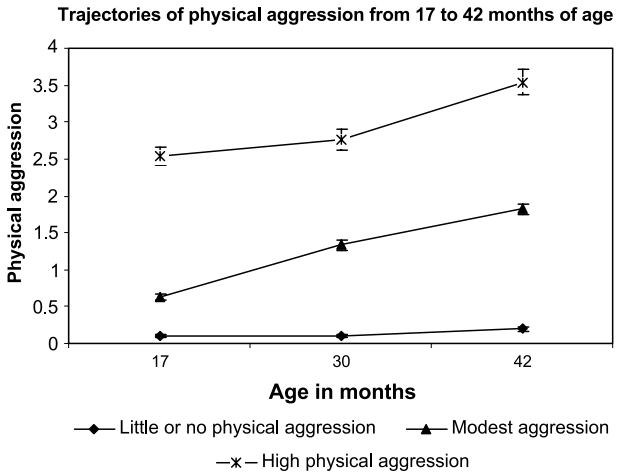

Figure 3. Trajectories of physical aggression from 17 to 42 months of age (Tremblay et al. 2004).

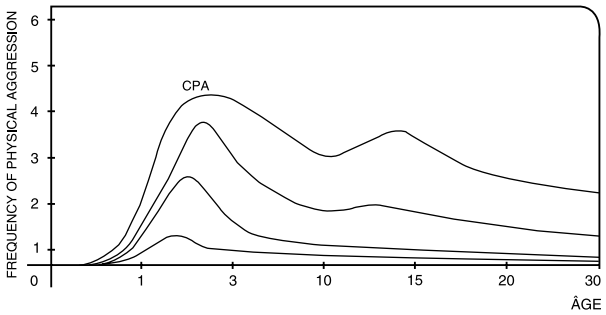

Figure 4. Age–physical aggression curves (Tremblay 2003).

frequency of physical aggression at one point in time, and over time, can be due to a large number of "causes," e.g., to individual differences in the genetic coding for serotonin (see Pihl and Benkelfat 2005), or testosterone (see van Goozen 2005), or language development (see Dionne 2005), or cognitive development (see Séguin and Zelazo 2005), or to environmental differences such as mother's tobacco use during pregnancy (see Wakschlag et al. 2002), birth complications (see Arseneault et al. 2002), parental care (see Gatti and Tremblay 2005; Raine et al. 1997; Zoccolillo et al. 2005), and peer characteristics (see Boivin et al. 2005). However, the individual differences that we observe are very likely to be due to interactions between many of these mechanisms, hence to epigenetic mechanisms (Francis et al. 2002, 2003; Weaver et al. 2004).

Twin studies

Knowledge on gene–environment interactions which could explain the development of chronic physical aggression is perilously close to zero. The first reason is that gene–environment interaction studies are recent. The second reason is that there are very few longitudinal studies that have included repeated assessments of physical aggression from early childhood to adulthood (see Rhee and Waldman 2002). However, the most important problem is that molecular genetic studies and twin studies have concentrated on global antisocial behavior phenotypes, generally assessed at one point in time. This is an old problem in the antisocial behavior literature (e.g., see Blumstein et al. 1988; Gottfredson and Hirschi 1990; Tremblay 2000, 2003a; Tremblay et al. 1991). Genetic studies have simply followed the main trend which tends to rely on measurement scales constructed by lumping items that are shown to correlate at one point in time.

A strong heritability of the aggression (read disruptive or obnoxious) score of the CBCL was shown with British, Swedish and USA preadolescent samples (Edelbrock et al. 1995; Eley et al. 1999). In fact the heritability for the aggressive behavior scale was stronger than for the delinquency scale. Eley et al. (1999) went

on to compare the preadolescent sample to an adolescent sample. They observed high heritability for both groups on the aggressive scale, and observed higher shared environmental effects for the delinquency scale, especially for the adolescent group. These results led them to conclude: a) that aggressive antisocial behavior (CBCL aggression scale) is highly heritable, and this confirms that life-course persistent antisocial behavior is highly heritable (Moffitt 1993); b) that non-aggressive antisocial behavior (CBCL delinquency measure, e.g., bad friends, truant, alcohol, drugs, run away, prefers older kids) is more influenced by the shared environment, confirming that adolescent-limited antisocial behavior is caused by environmental factors (Moffitt 1993). Note that these conclusions concerning a life-course developmental model were drawn from a cross-sectional study with an aggression scale that looks like an extraversion personality scale, and a delinquency scale which focuses on behavior that tends to start when children have become adolescents (drugs, alcohol, truant, run away). The phenotypes which are being assessed are not the developmental trajectories hypothesized by the theory. They are assessments at one point in time.

The study which started at the earliest point during development to assess gene–environment effects on physical aggression used a large sample of 18–19-month-old twins (Dionne et al. 2003). Results showed, with a large sample of female and male twins, that variance in mother reports of physical aggression was explained somewhat more by genetic factors (58%) than by environmental factors (42%) suggesting that there are strong genetic effects on physical aggression during early childhood. Based on the longitudinal studies described above, which show that physical aggression decreases with age, we would expect to observe increased environmental effects as they grow older, if the pressures on learning alternatives to physical aggression come from the environment. Unfortunately, studies with older samples of twins have used global scales of antisocial behavior (e.g., Arseneault et al. 2003; Edelbrock et al. 1995; Eley et al. 1999). If their results are a true representation of genetic effects on physical aggression, we would have to conclude that learning to regulate physical aggression is largely determined by genetic factors (see Arseneault et al. 2003). To settle this question, we need genetically and environmentally informative longitudinal studies, such as twin studies, that will focus on the development of physical aggression from early childhood to adulthood, to understand the role of genetic and environmental factors throughout development. The Dionne et al. (2003) study shows that both the genetic and environmental causes of a very specific socially disruptive behavior, physical aggressions, are in place by 19 months of age, long before the violent video games, the delinquent peers, and the effects of a Vietnam war.

Molecular genetic studies

Many molecular genetic studies have attempted to identify polymorphisms related to aggressive behavior, mainly with animal and human adult samples (see for example Pihl and Benkelfat 2005). Caspi et al. (2002) used a longitudinal study to

specifically address gene–environment interactions. They observed that the most maltreated males were at higher risk of being convicted of a violent crime before 27 years of age if they had the short version of the functional polymorphism in the gene coding for monoamine oxidase A (MAOA) activity. The MAOA enzyme metabolizes neurotransmitters linked in previous studies to behavior problems (e.g., dopamine, norepinephrine, and serotonin), and the short version of the allele leads to low activity. Effects were similar for conduct disorder assessed between 10 and 18 years of age, antisocial personality symptoms, and disposition to violence measured at 26 years of age. Individuals with a history of chronic physical aggression may be the driving force in these associations, since they are the most likely to be found in each of the assessed categories. This study is a good illustration of gene–environment issues related to prevention which need to be address. First, although the study was a longitudinal study from birth to 26 years of age, the analyses did not provide information on the developmental impact of the gene–environment interaction. Was the effect of the gene–environment interaction on physical aggression present in early childhood? Did it appear later during elementary school, adolescence, and even adulthood? These are important questions for preventive interventions. From the developmental data presented in the preceding section, one would expect that the effects were present early, and may have grown with time. A second question concerns the intervention strategies. Let's assume that the gene–environment interaction effects appear in early childhood and will increase with time if there are no early interventions. Which type of intervention should we use? For example, we could screen pregnant women soon after conception to identify those at risk of maltreating their child and offer a support program to help prevent the family from abusing the child (e.g., Olds et al. 1998). An alternative strategy would be to give the child a chemical treatment which would correct or compensate for the low MAOA activity (Caspi et al. 2002; Weaver et al. 2004). Both strategies could also be used with some cases. Thus, much research is needed to understand the gene–environment impact on the development of physical aggression, but research is also needed on the use of that knowledge to prevent the *development* of chronic physical aggression.

Gene–environment interactions, epigenetics and prevention experiments

To address causal issues and identify effective preventive interventions, we need to be able to manipulate putative causal variables. Twin studies and molecular genetic studies can address the gene–environment interaction issue. However, concerning causal mechanisms leading to chronic physical aggression, they are still limited to a correlational analysis. Cross-fostering experiments like those done with rats, mice and monkeys (e.g., Francis et al. 2003; Suomi 2005; Weaver et al. 2004) would give better insights into causal mechanisms than studies which are limited to correlational analyses. Obviously, it is hard to conduct such studies with humans, except with adoption studies which will be discussed later in this section. However,

prevention experiments aimed at helping high risk families and children can be used to understand the mechanisms that prevent children from chronic physical aggression.

The Cambridge-Somerville study was a prevention experiment which Joan McCord showed to have long term negative impacts even with the best of intentions (McCord 1978, 1992). It also proved that interventions after early childhood could have long term significant impacts. More recent prevention experiments have shown that interventions with elementary school children can also have relatively long term positive impacts (e.g., Hawkins et al. 2005; Kellam et al. 1994; Lacourse et al. 2003; Vitaro et al. 1999). However, from our present understanding of the development of physical aggression described above, we would expect that intensive preschool interventions would have more positive (or negative) long term impact than intensive elementary school interventions.

For example, the Perry Preschool experiment with 3- and 4-year-olds showed impressive long term reduction of criminal behavior among males; unfortunately, there is apparently no information on the development of physical aggression in this study. Olds et al. (1998) experimented with an earlier intervention. They randomly allocated a nurse home visitation program to young underprivileged pregnant women at high risk of child abuse and neglect. These children were obviously also at high risk of chronic physical aggression. The long term follow-up of the children from the intervention group showed that they were less frequently abused and neglected, and were also less likely to exhibit delinquent behaviors during adolescence. One would expect that the intervention group learned more rapidly to regulate physical aggression and was less frequently involved in physical aggression during childhood and adolescence. Unfortunately, based on the published material, the development of physical aggression, or any other antisocial behaviour, appears not to have been included in the follow-up assessments. Regular assessments of physical aggression during childhood and adolescence in the two experiments would have helped us understand to what extent an intervention starting 3 years after birth and targeting cognitive development has as much of an impact on developmental trajectories of physical aggression, as an intervention which started during pregnancy and which could affect early brain development. These are important questions for assessing the cost and benefits of interventions during the developmental cycle, but they are also important questions for understanding the mechanisms which lead to or prevent chronic physical aggression. To what extent are the control mechanisms plastic, and over what period of time? Preventive studies with disruptive children after school entry indicated that there is still some plasticity, but somewhat limited and costly to achieve (Ialongo et al. 1999; Lacourse et al. 2002; Tremblay 2003a).

Drug companies are developing a new research field which has been labelled "pharmacogenomics." Its aim is to create the knowledge which will enable the creation of pharmaceutical products specifically meant to match the genetic makeup of an individual. It is easy to imagine that we can do the same with psychosocial interventions: match the intervention to the genetic profile of the client. Weaver et al. (2004) recently showed how an environmental event during

early childhood (rat pups licked by their mothers) activated gene expression which influenced the development of the HPA axis and increased life expectancy. This phenomenon is a good illustration of the complex epigenetic mechanisms which are involved in gene–environment interactions. Genes need an environment to be turned on and off. Psychosocial interventions can offer programs which will help activate the right genes at the right time. This is more probable during pregnancy and early childhood than at later stages of development. Psychosocial interventions can also compensate for defective genes, as is often done with nutrition (e.g., Scriver and Kaufman 2001).

The ultimate prevention experiment to test early interventions and gene–environment interactions are adoption studies. Instead of trying to change an individual's environment by attempting to modify the behavior of his or her parents, adoption simply changes the parents. For example, Duyme et al. (1999) showed that adoption into a family with a high social class compared to lower social class had substantially increased children's IQ by adolescence. Adoption is often used to replace clearly inadequate parents. However, what I am suggesting here is not an intervention strategy, but a research strategy using the adoptions that are being made to test hypotheses concerning gene–environment interactions and epigenetic effects on the development of antisocial behavior. Furthermore, because one would expect that the environment provided by good adoptive parents would be much better than the environment created by at-risk parents who are being supported by a nurse or a social worker, adoption studies should give a good indication of the maximum effect one could obtain with the best psychosocial interventions targeting biological parents. An old study by Van Dusen et al. (1983) is a good example. Using Danish records over many years, they collected the criminal convictions of adopted females (N = 6,374) and males (N = 5,649) with reference to the biological and adoptive parents' socioeconomic status (SES). Their results showed, as expected, that the level of criminal convictions for males was close to six times that of females. However, males with high SES biological parents were less likely to have a criminal conviction if they were adopted into a high SES family (9.3%) than if they were adopted into a low SES family (13.8%). Similarly, males with low SES biological parents were less likely to have a criminal record if they were adopted into a high SES family (12.98%) than if they were adopted in a family with the low SES of his biological parents (18.04%). Note that the former (low SES biological–high SES adoptive) were as likely to have a criminal record as the high SES biological–low SES adoptive (12.98% vs. 13.8%). The largest differences between groups of males was between the high SES biological–high SES adoptive (9.3%) and the low SES biological–low SES adoptive (18.04%). The latter are close to two times more likely to have a criminal conviction. If we were successful in changing the behaviour of high risk low SES parents, it would be surprising that we would achieve better results than placing a high SES biological parent male into a high SES adoptive family. The data also indicated that the interventions with females may have proportionally more impact (0.64% vs. 3.02%), although the number, and probably the severity, of convictions are substantially lower for females.

Experiments using an adoption design could randomly allocate children available for adoption to parents who are offering to adopt children. These experiments can control for types of children (e.g., sex, age, families of origin, genetic characteristics) and types of adoptive families (e.g., parents' age, education, income, parenting skills, place of residence). By starting such adoption studies during pregnancy, and by including regular assessments over time of environment, behaviour, physiology, and genetic expression we will be able to address genotype–environment interactions, including timing of the change in environment, from the perspective of epigenetic mechanisms with males and females.

The question is to what extent the environment during birth and after birth can modify the developmental trajectories that are expected from the genotype, and reduce the probability of criminal behavior. An important sub question is why such differences between females and males. To give a good answer to these questions we need well designed experiments starting as close as possible to conception.

Conclusion

Joan McCord made exceptionally good use of a very well designed experimental prevention study initiated in the 1930s. Her work influenced a large number of longitudinal and experimental studies which form the basis of developmental and experimental criminology. Most of the studies which were initiated within her lifetime focused on school age children and young adults. However, she promoted the extension of these studies to early childhood, and was extremely excited to see the first results (e.g., McCord and Tremblay 1992; McCord et al. 2001).

Joan McCord lived to see the advent of modern genetic studies which are showing important genetic effects in the development of antisocial behavior. A philosopher and sociologist by training, she was at first skeptical. But she was first and foremost a scientist motivated by the advancement of knowledge (McCord 2002). Having stressed the importance of parenting and the importance of maternal behavior (McCord 1990, 1991), she would have been extremely excited by the results published a few months after her death showing that the licking behavior of rat mothers with their pups regulates the expression of genes influencing the development of their brain and their long term behavior (Weaver et al. 2004). These studies are clearly showing the strong impact the environment has on the expression of our genes, especially at the beginning of life. Long term behavior and long term health is apparently determined by gene–environment interactions which start *in utero* and continue throughout our lives (e.g., Caspi et al. 2002; Kajantie et al. 2004; Weaver et al. 2004). To understand the most effective means of preventing criminal behavior, the next generation of Richard Clark Cabot and Joan McCord will need to work in multidisciplinary teams, using experiments to manipulate putative environmental causal factors, while controlling for genetic characteristics, and assessing the effects of the manipulation on the behavior of the

subjects as well as on the expression of the genes during development (epigenesis). Since humans appear to learn to control their emotions and their social behavior largely during early childhood, one would expect that the most effective preventive interventions to promote long term prosocial behavior will have targeted pregnant women and infants.

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