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THE TRANSMISSION OF AGGRESSIVENESS ACROSS GENERATIONS: BIOLOGICAL, CONTEXTUAL, AND SOCIAL LEARNING PROCESSES

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In this chapter, we present a theoretical perspective for examining the cross-generational transmission of aggressive or nonaggressive behavior. We begin by reviewing the evidence that there is substantial continuity of aggression within a generation across the life span. We note that this continuity is due as much to the continuity of unaggressiveness as to the continuity of aggressiveness. We then turn to examining the empirical evidence concerning the cross-generational continuity of aggression. We note that although a number of studies suggest such continuity, a number of methodological issues have not been resolved by most of the studies to date. Most notably, the number of prospective cross-generational life-span studies is still small. We then present a social–cognitive model that has evolved to explain aggressive behavior and its continuity in the life span, and we discuss how this model can be applied to understanding cross-generational continuity within a broader framework that explains such continuity as a product of four processes: genetic predispositions.

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interacting with environmental factors, continuities or discontinuities in environments, observational learning processes with a focus on parents as models, and conditioning processes with a focus on parents as the conditioners. Finally, we present some data from a 40-year, three-generational longitudinal study that shows that children’s aggressiveness after they grow up is predicted by their parents’ own childhood aggressiveness and their parents’ aggressiveness when the children were young or adults but not by their parents’ adolescent aggressiveness. It is shown that continuity of aggression within the life span plus transmission between generations during the critical childhood period of the second generation can adequately explain the cross-generational relations. These findings are consistent with intergenerational transmission through genetic and biological predispositions and learning processes during the childhood years.

WITHIN-PERSON CONTINUITY OF AGGRESSIVE BEHAVIOR ACROSS THE LIFE SPAN

One of the most consistently asked questions of prospective, longitudinal data is whether personality traits, behaviors, or other aspects of psychosocial functioning remain stable over the life course. The continuity of intellectual ability from childhood to adulthood is substantial, with correlations typically in the moderate range (about .50). Research, including our own, on the development of aggression suggests that adult aggression and antisocial behavior are also statistically predictable from childhood aggression and antisocial behavior (Farrington, 2003; Farrington, Ttofi, & Coid, 2009; Huesmann, Dubow, & Boxer, 2009; Huesmann, Eron, Lefkowitz, & Walder, 1984; Kokko, Pulkkinen, Huesmann, Dubow, & Boxer, 2009; Loeber & Dishion, 1983; Moffitt, 1993; Olweus, 1979; Pulkkinen, Lytta, & Kokko, 2009).

Our most recent estimates are based on analyses of data from the Columbia County Longitudinal Study, a study of a cohort of 856 eight-year-olds first evaluated in 1960 and then reevaluated at age 19 in 1970 to 1971, at age 30 in 1982, and at age 48 in 2000. Their parents were also interviewed in 1960, and 525 of their children were interviewed in 2000, providing data on three generations. We call the main sample the Generation 2 (G2) sample, and they have provided 40 years of data on continuity of aggression across the life span. When the continuity coefficients for 40 years are estimated with structural equation models that control for measurement error and method variance (see Huesmann et al., 2009), we obtain coefficients of .50 for males and .42 for females. These approach the continuity of intellectual ability. The consequences of such continuity can be serious. As Kokko et al. (2009) recently showed with data from Finland and the United States, early aggres-
siveness is more predictive of physical aggression and lack of self-control in later middle adulthood than it is of verbal aggression.

It is worth noting that the continuity correlations over time are due as much to low aggressive children staying low on aggression throughout life as they are to high aggressive children staying high on aggression throughout life (Huesmann et al., 2009). To demonstrate this fact, we grouped participants based on age-8 peer-nominated aggression into “high” and “low” categories using both median and one-third splits and examined how many stayed in the same category over 40 years. For the full sample, 37% (52 of 141) of individuals who were low in third grade stayed low through late adolescence (i.e., age 19), through young adulthood (i.e., age 30), and into middle adulthood (i.e., age 48) on the composite measure of aggression. Of individuals who were high in third grade, 35% (i.e., 31 of 89) stayed high through adolescence and into middle adulthood. In other words, continuity was as much due to low aggressives staying low as to high aggressives staying high. When we inspected these patterns by gender, interesting differences became evident. For males, 38% of those who were low in childhood stayed low through age 48. Similarly, 36% of females who were low in childhood stayed low into middle adulthood. However, the differences were striking with regard to those classified as high on aggression. Among males, 47% of those who were high in childhood stayed high into middle adulthood, whereas for females, only 18% who were high in childhood continued to be high into middle adulthood ($\chi^2 = 14.97, p < .001$).

These results open the possibility that differential socialization of males and females places more pressure on females to reduce their aggression than it does on males. Early aggressiveness may also have more lasting serious consequences for males. For example, Huesmann, Eron, and Dubow (2002) found that early aggressiveness was the most important predictor of males being arrested by the time they were 30 years old, in comparison with a large variety of contextual and personal variables assessed in the Columbia County Longitudinal Study. The standardized odds ratio for age-8 aggression in predicting “ever arrested by age 30” was highly significant: 1.45, $p < .01$.

Of course, even continuity coefficients as large as .5 and odds ratios approaching 1.5 still mean that a substantial portion of adult aggressive and criminal behavior is not predictable from childhood aggressive behavior and is probably related to context (Broidy et al., 2003; Sameroff, Seifer, Baldwin, & Baldwin, 1993). In addition, it seems to be only aggressive and antisocial behavior that emerges early in life that has lasting negative consequences. As Moffitt (1993) suggested and a number of recent studies confirmed, some aggressive and antisocial behavior often emerges in adolescence and is relatively short lived. This kind of aggression seems to have few long-
term negative consequences, whereas life course persistent aggression that begins in early childhood has lasting detrimental consequences (Bergman & Andershed, 2009; Farrington et al., 2009; Huesmann et al., 2009; Pulkkinen et al., 2009).

Similar findings regarding continuity have been reported in studies of other indicators of adjustment over time. For example, Helson, Jones, and Kwan (2002) demonstrated quadratic changes in various indicators of personality functioning from early to late adulthood: Certain attributes such as dominance and independence peaked in middle adulthood. In her influential island of Kauai prospective study, Werner (2002) summarized long-term outcomes for children and adolescents with behavior disorders: By age 40, only one third of those males and one fifth of those females exhibited continuing difficulties (e.g., financial, marital, substance use). However, far less is known about the degree of continuity of positive psychosocial adjustment from childhood to adulthood, although this appears to be an emergent concern of longitudinal researchers. As an example, with data from the Jyväskylä Longitudinal Study, Pulkkinen and her colleagues demonstrated that prosocial behavior in childhood predicted greater self-esteem and subjective well-being and shorter lived periods of unemployment in adulthood (Kokko & Pulkkinen, 2000; Pulkkinen, Nygren, & Kokko, 2002). Flouris and Buchanan (2002) showed that good family relationships in childhood led to better marital adjustment in adulthood.

**RECENT STUDIES OF INTERGENERATIONAL CONTINUITY OF AGGRESSION**

The discovery of strong continuity of aggression across the life course has stimulated interest in the continuity of aggression and antisocial behavior across generations. Although many studies have investigated parent–child relations statically (i.e., cross-sectionally), longitudinal cross-generational investigations of continuity and discontinuity in personality, behavior, and adjustment are relatively few and are limited primarily to examining contemporaneous or retrospective links between parent and child behavior. Several discussions of the intergenerational transmission of aggression have been published in the child development literature over the past decade (e.g., Constantino, 1996; MacEwen, 1994). Those reviews indicated that most relevant studies have used self-report, retrospective questionnaire data obtained from two generations. However, response bias problems cloud the interpretation of such findings. Three studies in a 1998 special issue of Developmental Psychology did use prospective methodology and multiple methods of measuring aggression (Cairns, Cairns, Xie, Leung, & Hearne, 1998; Capaldi & Clark, 1998;
Serbin et al., 1998), and all reported modest to moderate cross-generational continuity. But those studies included only two generations.

More recently, four studies on cross-generational consistencies in parenting and in aggressive and antisocial behavior were published in a special issue of the Journal of Abnormal Child Psychology (Capaldi, Pears, Patterson, & Owen, 2003; Conger, Nepple, Kim, & Scaramella, 2003; Hops, Davis, Leve, & Sheever, 2003; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003; see also Smith & Farrington, 2004). All four studies measured parenting by two generations (G1 and G2) and aggressive behavior in two generations (G2 and G3). Two of the four studies found significant inter-generational continuity of aggression. The two that did not report such continuity had the smallest sample sizes. All four studies also reported inter-generational continuity for some parenting factors relevant to aggression (e.g., negative affect in parent–child interactions, consistency of discipline). The results also suggested that child aggression mediates some of the continuity of parenting as well as parenting mediating some of the continuity of aggression. For example, Thornberry et al. (2003), using self-report questionnaire data, found a chain of relations for males from G1 parenting to G2 aggression to G2 parenting to G3 aggression. For females, the pattern was similar, although the link from G1 parenting to G2 aggression was not significant. Conger et al. (2003), using observational data, found both that G1 observed parenting had direct effects on G2 observed aggression and that G2 observed parenting had direct effects on G3 observed aggression. Hops et al. (2003), also using observations of parenting, obtained a fully mediated path from G1 parenting, to G2 aggression, to G2 parenting, and finally to G3 aggression. Capaldi et al. (2003) reported findings similar to those of the other studies, with an important methodological distinction: the use of multiple informants and sources of data, moving beyond the parent and child observational and questionnaire data used in the other studies to include teacher reports and archival records.

A plausible conclusion that can be drawn from these similar results in the four studies is that parenting behavior and aggressive behavior seem to have reciprocal influences on each other. Within generations, aggression in youth is often followed by aggression-promoting parenting. Aggression-promoting parenting, in turn, seems to contribute to aggression in offspring. However, this conclusion might be attenuated by important considerations related to the theory on which these cross-generational investigations are based. In spite of increasing empirical attention, the processes by which patterns of positive or negative adjustment are transmitted from parents to children are not yet well understood or firmly established, and more research is needed to explicate cross-generational links and the theory explaining such links (Dubow, Huesmann, & Boxer, 2003; Rutter, 1998; Shaw, 2003).
PSYCHOLOGICAL PROCESSES INVOLVED
IN AGGRESSIVE BEHAVIOR

To begin with, to talk theoretically about the cross-generational transmission of aggression, we need a model for the psychological processes through which predisposing personal factors and precipitating situational factors interact to determine whether a person behaves aggressively. The model needs to include a representation of the enduring psychological structures that control and influence these processes. Such a model has been provided by Huesmann (1998) in a unified information-processing model for social problem solving. According to this model that integrates previous theorizing of Huesmann (1988), Bandura (1977), Dodge (1982), and Anderson (Anderson & Huesmann, 2003), an individual’s emotional state and encoded schemas about the world interact with situational cues to lead the individual to make attributions about the situation. These attributions change the individual’s emotional state and prime the activation of scripts for behaving. The scripts are filtered through a set of normative beliefs about appropriateness until a script is accepted and followed.

Within this model, the long-term determinants of aggressiveness include (a) encoded cognitions represented by schemas about the world, (b) scripts for behavior, and (c) normative beliefs for filtering scripts, along with (d) the individual’s emotional predispositions. More specifically, hostile attributional biases (e.g., “People are mean”; Dodge, 1982; see also Chapter 9, this volume) occur when hostility is emphasized by the “world schema” that an individual has acquired. Attributions influence emotions and the type of script an individual will retrieve to deal with a social situation. Scripts (Huesmann, 1998) can be viewed as cognitive programs that have been acquired over time and are stored in a person’s memory and are used as guides for behavior and social problem solving. Not all scripts that occur to the child will be used. Before acting out the script, the child reevaluates the appropriateness of the script in light of existing internalized social norms—called normative beliefs (Huesmann, 1998; Huesmann & Guerra, 1997)—and examines the likely consequences of the script. These normative beliefs and expectations about outcome must also have been acquired over time.

Our focus on cognitive-information processes does not mean that emotions are unimportant. We view emotion regulation as an important influence on aggressive behavior that influences attributions, script selection, and evaluation of scripts. Emotion regulation has held a place of prominence in the study of behavioral development for some time, particularly with regard to developmental psychopathology (e.g., Cicchetti, Ackerman, & Izard, 1995; Frick & Morris, 2004).
Processes for the Intergenerational Transmission of Aggression

Given this model, how can a tendency to behave in a characteristic manner (e.g., aggressively or nonaggressively) be transmitted from one person (e.g., a parent) to another (e.g., the parent's child)? Our position is that there are four major processes by which this may occur that need to be considered: (a) through the transmission of genes that influence social behavior, (b) by changes parents make in the child's environment or through continuity in the parent's and child's environment, (c) through children observing parents' behaviors, and (d) through conditioning of the children's behavior in which the parent participates.

Genetic Influences on Aggression

Genetic influences are well established empirically (see Chapter 8). Individual differences in emotional arousal, neurotransmitter levels, perceptual biases, and other characteristics relevant to aggression seem to be influenced by genes. The evidence for a heritable predisposition to aggression from twin and adoption studies is impressive (Cloninger & Gottesman, 1987; Deater-Deckard & Plomin, 1999; Mednick, Gabrielli, & Hutchings, 1984). Miles and Carey (1997) performed a meta-analysis of 24 genetically informative studies that included twin and adoption designs and found significant heritability estimates (in the .4 range) for self-report measures of aggression. In addition, a variety of adoption studies have revealed relations between children's aggressiveness or antisocial behavior and the aggressive or antisocial behavior of both their natural and adoptive parents.

However, these seem to be predisposing influences rather than deterministic influences. Estimates from behavior genetic analyses of low shared environmental variance depend on unlikely assumptions of genes being uncorrelated, noninteracting, and not influencing the environment of the individual. The weight of evidence suggests, rather, that biosocial interactions between genes and the environment are likely influences.

Recent studies are beginning to go beyond global estimates of the relative influences of genetic differences in accounting for phenotypic differences to look for the specific genes responsible (e.g., Chapter 8). Strategies based on genome scans have proved disappointing in the search for loci that influence behavioral phenotypes, but investigations of polymorphisms of candidate genes have been surprisingly productive. Although methods are still developing for statistical discrimination of reliable signals from noise when many candidate genes are studied, effects of monoamine oxidase (MAO) and serotonin promoters have been confirmed and others are under study (for a review, see Munafo et al., 2003). Studies by Caspi and colleagues (2002, 2003) have indicated that there are important childhood contextual–genetic
interactions in the expression of social behavior. For example, those authors found that a gene that causes slightly lower MAO increases risk of aggression only in a child who is exposed to high amounts of stress during childhood and adolescence. Similarly, the perinatal environment is known to affect the risk of aggressive behavior (Raine, Brennan, & Mednick, 1995). Whatever their source, a variety of individual differences in neurophysiology, neurotransmitters, hormones, and heart rate correlate with individual differences in early aggressiveness (Knoblich & King, 1992; Olweus, Mattsson, Schaling, & Loew, 1988; Raine & Jones, 1987).

**Intergenerational Environmental Continuity and Change and Their Influences on Continuity and Change in Aggression**

Many environmental influences on risk and resilience for aggression and violence have been identified. These include stress, poverty, abuse, parental rejection, peer behaviors, and religion, to name a few. To the extent that such environments are passed from parent to child, one can expect cross-generational continuity. To the extent that such environments change from parent to child, one can expect cross-generational discontinuity.

A major question in developmental research concerns how changes over time in the social contexts people inhabit affect development (Higgins & Parsons, 1983; Huesmann, Dubow, Eron, & Boxer, 2006; Sameroff, 1983). One needs to understand whether the degree of continuity in positive and negative adjustment over time and across generations is related to the degree of continuity in contextual factors. Is there continuity in parenting practices from what a current parent experienced in interactions with his or her parents to what the parent delivers to his or her child? Does continuity or change in socioeconomic status promote continuity or change in aggression? Many children have to cope with family changes: Their parents might divorce, get into trouble with the law, or lose their jobs. Evidence suggests that the stress engendered by such changes is a risk factor for children's socioemotional development (Caspi et al., 2002), thus increasing the likelihood of negative changes that might have long-term consequences. Our hypothesis is that similar discontinuities of a positive nature (e.g., a substantial improvement in the family's financial situation, a significant improvement in the child's academic performance) can turn trajectories of psychosocial adjustment upward toward greater success, achievement, and life satisfaction.

A second question in developmental research concerns the timing of exposure to contextual influences. What contexts at what points during childhood are most predictive of later outcomes? For example, Duncan (2002) found that poverty experienced during the early childhood years had the strongest effects on the number of years of education the child attained by early adulthood. This suggests that the environment in which the parent
lives and the child develops would be more important than the parent's own early environment.

A third issue of great relevance is the extent to which the larger historical context surrounding individual development influences the trajectories taken by cohorts embedded in those circumstances. As an example, what is the differential impact of being an adolescent in a society that is at war as compared with the same society experiencing relative peace, and how might the institution of selective or compulsory service magnify that impact?

These issues lead to a fourth broad contextual concern: major life transitions or turning points in individual development (Rönkä, Oravala, & Pulkkinen, 2002; Rutter, 1996). *Turning points* are positive or negative events, over which the individual may or may not have control, which significantly alter the life trajectory. According to Rutter (1996), an event can be a turning point only if it leads to an enduring, long-term modification of the trajectory. Thus, turning points cannot be assessed contemporaneously for their impact. Longitudinal data are necessary because often individuals do not recognize turning points in their lives until some time has passed and the individual has understood the importance and meaningfulness of the event.

**Children Observing Parents' Behaviors**

The observation of parents' behaviors by children can be expected to influence social behavior in general and aggressive behavior in particular, and this can occur through two quite different processes: short-term stimulating processes and long-term learning processes. These processes operate more generally to make the child imitate anything he or she observes, including aggressive acts by others (Huesmann & Karrwil, 2007).

In the short run, when children see their parents behave aggressively, schemas, scripts, and normative beliefs associated with aggression are primed in the children's minds. In addition, emotions associated with the behavior are aroused in the child (e.g., anger may be stimulated). These processes alone can lead to short-term increases in the risk of aggressive behavior. In addition, however, because children generally identify with their parents, they are likely to mimic behaviors almost immediately. All of these short-term stimulating processes make it likely that a child will behave aggressively after observing his or her parents behaving aggressively.

However, the more important observational processes for the intergenerational transmission of aggression are probably long-term observational learning processes involving parent and child. As mentioned earlier, children generally identify strongly with their parents. Consequently, they tend to encode into their repertoire of scripts the scripts they see their parents using, they tend to adopt the world schemas they perceive their parents to be holding, and they tend to accept the normative beliefs of their parents about the
appropriateness of social behavior. For a long time, children's imitation of parents' behaviors was thought to be a relatively low-level childish form of behavior. “But recent work across a variety of sciences argues that imitation is a rare ability fundamentally linked to characteristically human forms of intelligence, in particular to language, culture, and the ability to understand other minds” (Hurley & Chater, 2005, p. 1). Imitation of parents’ behaviors appears to be innate and occurs automatically in young primate infants (Meltzoff & Moore, 1983). Specific mirror neurons seem to organize imitation in primate brains (Gallese, Fadiga, Fogassi, & Rizzolatti, 1996). However, recent work suggests that imitation goes far beyond the copying of specific sequences of behaviors to the encoding of social cognitions fundamental to the control of social behavior (Meltzoff, 2007).

These observational learning processes are also involved in the development of emotion regulation. Eisenberg, Cumberland, and Spinrad (1998) described three processes by which emotion socialization leads to social competence. First, through everyday family interactions, children display a wide range of both positive and negative emotions, thus providing parents with numerous opportunities to react in both positive (e.g., encouraging) and negative (e.g., punishing) ways. Second, when parents discuss various aspects of emotion (e.g., causes and consequences, emotional experience and regulation) with their children, they serve as models for understanding and coping with emotion. A child who receives a high degree of emotion knowledge from his or her parents should be better equipped to contend with emotionally charged events. Third, family emotional expressivity relates in important ways to children’s social competence. Through observation, children can learn positive (e.g., smiling) or negative (e.g., yelling) modes of expressing emotions, as well as heuristics for interpreting the emotions of others. This model of emotion socialization suggests individual and cross-generational continuity of emotional regulation and, in turn, social behaviors, as well as taking into account potential moderating influences of contextual or individual factors.

Conditioning of Children’s Behaviors to Be Similar to or Different from Parents

Of course, observational learning is not the only learning process crucial to intergenerational transmission of aggressive tendencies. As Patterson (1986) demonstrated, parents may unintentionally condition their children operantly to behave aggressively. Through coercive family interactions, they may unintentionally reinforce their children for behaving aggressively. They may also deliberately and directly reinforce their children for being aggressive if the parents believe aggression is appropriate.

Parents who harshly punish and abuse their children may also classically condition them to experience anger responses to stimuli that might seem
benign to others (e.g., persons of authority). They may classically condition alienation from society in this way and make appropriate socialization of the child by others in the child's environment difficult.

Analysis of Intergenerational Continuity in the Columbia County Longitudinal Study

The first analyses of the four waves of data spanning three generations from the Columbia County Longitudinal Study suggest that genetic and dispositional processes, learning processes operating during the child-rearing years, and environmental continuities may all contribute to cross-generational continuity of aggressiveness. The sample we used in these analyses consists of 349 of the 551 G3 offspring who were interviewed when their G3 parent was 48 years old. The 349 represent one child of each G2 subject interviewed—the youngest child for subjects with more than one child (in 202 families two children were interviewed). The ages of the G3 sample at the time of the interview ranged from 6 to 33, with a median age of 18.

In these analyses, we assessed aggression in the G2 generation with the same composite measure we developed to assess continuity of aggression across the life span in the G2 generation. The indicators we used in that analysis of four waves of G2 data were peer nominations at age 8 and 19; severe physical aggression at ages 19, 30, and 48; and aggressive personality at ages 19, 30, and 48. The "severe physical aggression" measure assessed how often the person punched, choked, beat up, or used a knife or a gun against another person in the preceding year. The "aggressive personality" measure was the sum of the F, 4, and 9 scales of the Minnesota Multiphasic Personality Inventory (MMPI; Hathaway & McKinley, 1940). For the G3 data, we used the same two indicators of adult aggression as with G2: aggressive personality and behaving severely physically aggressively. However, we analyzed these indicators separately because the sample sizes are quite different for the two variables, given that only those G3 offspring who were over 13 received the aggressive personality assessment.

In Table 7.1, the intergenerational correlations are shown from G2 to G3 for these variables. One can see substantial cross-generational correlations between the G2 parent's aggression at age 8, 19, 30, and 48 and the G3 offspring's aggressive personality and severe physical aggression at the time the G2 parent was 48. For a subsample of 125 of the G3 offspring we had data on the aggressiveness of the other parent as well. Their aggressiveness correlated about .20 (p < .05) with the G2 spouses' aggressiveness at both age 30 and 48 and with the G3 offsprings' aggressiveness. However, the total prediction of G3s' aggression was not enhanced by adding in this other parent's aggression to a prediction equation.
TABLE 7.1
Correlations Between Generation G2’s (Parents’) Aggression at Four Ages and Generation G3’s Aggression When G2 Parent Was Age 48

<table>
<thead>
<tr>
<th>G2 parent’s aggression at different ages</th>
<th>Child aggression when parent was age 48</th>
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<tbody>
<tr>
<td></td>
<td>G3 child’s aggressive personality (N=212)</td>
</tr>
<tr>
<td>Age 8</td>
<td>0.24***</td>
</tr>
<tr>
<td>G2’s composite aggression</td>
<td></td>
</tr>
<tr>
<td>Age 19</td>
<td>0.26***</td>
</tr>
<tr>
<td>G2’s composite aggression</td>
<td></td>
</tr>
<tr>
<td>Age 30</td>
<td>0.46***</td>
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<tr>
<td>G2’s composite aggression</td>
<td></td>
</tr>
<tr>
<td>Age 48</td>
<td>0.19***</td>
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<tr>
<td>G2’s composite aggression</td>
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* \(p < .10\), ** \(p < .05\), *** \(p < .01\).

The correlations with G2s’ age-8 aggression are consistent with genetic and biological predisposition models of intergenerational transfer, whereas the correlations between G2s’ later aggression and the G3 offsprings’ aggression are consistent with learning models that emphasize transmission of aggressive behavior during the child-rearing years. Although the differences are not large, an inspection of the correlations suggests that the strongest relations with the offspring’s aggressive personality are from the parent’s aggression years earlier when the parent was a child, adolescent, or young adult. However, the strongest correlations with the G3 offsprings’ actual physically aggressive behavior are with the G2 parents’ aggressiveness later in life at age 19, 30, and 48 (i.e., during the early child-rearing years). These results are consistent with the concept that characteristically aggressive personalities are influenced by predisposing genetic and biological factors modified by learning experiences during the early child-rearing years, whereas the emergence of adult aggressive behaviors is influenced more by the child-rearing environment in which the offspring develops. The highest correlations with G3s’ aggressive personality around age 18 is with the parents’ age 30 aggressiveness for both genders 18 years earlier. However, the highest correlations with G3s’ physically aggressive behavior around age 18 is with the parents’ concurrent age 48 aggressiveness.

These specific relations are illustrated in more detail in Figure 7.1, which displays the mean aggression scores for the offspring of G2 parents who scored low (i.e., < 25th percentile), medium, or high (i.e., > 75th percentile) on aggression concurrently at age 48 or 18 years earlier at age 30. The effects
Figure 7.1. In the upper panel, the relation between a parent's age-48 aggression and their offspring's concurrent severe physical aggression when the offspring averaged 18 years of age, $F(2, 312) = 11.7, p < .001$. In the lower panel, the relation between a parent's age-30 aggression and their offspring's aggressive personality 18 years later (i.e., when the offspring averaged 18 years of age), $F(2, 113) = 6.24, p < .01$. G3 aggression is low if G2 is below the 25th percentile, medium if G2 is between the 25th to 75th percentile, and high if G2 is above the 75th percentile.
are significant and illustrate that it is the offspring of parents who were above the 75th percentile on aggression at age 30 who are most at risk of developing an aggressive personality, and it is the offspring of parents who are above the 75th percentile on aggression at age 48 who are most at risk of being seriously physically aggressive around age 18.

Given these relations, we decided to test a model of cross-generational transmission that combines continuity of aggression within the G2 generation with transmission across generations only during the age 30 to age 48 period when the G3 child is being reared. The model incorporated both the measure of aggressive personality and the measure of severe physical aggression. This two-generational structural model for continuity of aggression is shown in the left panel of Figure 7.2. The model assumes that the only path from generation G2 to generation G3 is the path from the aggression of the G2 parent at age 30 to the aggression of the child 18 years later. The model fits the data well with a nonsignificant chi-square statistic and other reasonable goodness-of-fit statistics. The path from G2 aggression at age 30 to G3 aggression 18 years later is a highly significant and large effect.

We next expanded the model to incorporate all three generations we had studied. The results are shown in the right panel of Figure 7.2. Unfortunately, our only good measure of G1's aggression is the individual's tendency to hit G2 when G2 was age 8. This measure is related to G2's aggression, but it probably represents G2's tendency to behave badly as well; thus, the path relating it to G2's aggression at the same time is bidirectional. Still, the model fits the data well, and the best estimates of the path coefficients for the model suggest a total cross-generational effect from G1 to G3 of .15. Adding a path from G2's aggression at age 48 directly to G3's concurrent aggression did not significantly improve the fit of the model, indicating that the strong relation between G2's age 48 aggression and G3's concurrent severe physical aggression (see Table 7.1 and Figure 7.1) is a consequence of the stability of G2's aggression through the child-rearing years. Thus, the estimated path coefficients are consistent with the concept that aggression is transmitted across generations primarily during the child-rearing years, with genetic and biological influences exerting long-term effects through within-generation continuity.

SUMMARY

In this chapter, we argued that cross-generational continuity in aggressiveness is undoubtedly the product of four factors: biological predispositions that are inherited by offspring, continuity in environment across generations, and conditioning and observational learning experienced by the child. We elaborated a model that takes into consideration all of these factors, and
Continuity of Aggression Over 40 Years Across 2 Generations

Chi-Sq(43) = 47.9, p > .28, RMSEA = .018, CFI = .989

Continuity of Aggression Over 40 Years Across 3 Generations

Chi-Sq(52) = 56.1, p > .32, RMSEA = .015, CFI = .991

Figure 7.2. In the upper panel, the continuity of aggression over 40 years across 2 generations (G2 and G3). $X^2 (43) = 47.9, p > .28, \text{RMSEA} = .018, \text{Cumulative Fit Index} = .99$. In the lower panel, the continuity of aggression over 40 years across 3 generations (G1, G2, and G3). $X^2 (52) = 56.1, p > .32, \text{RMSEA} = .015, \text{Goodness of Fit Index} = .991$. # indicates that the measurement parameter was fixed at the value estimated in the within-generation model. Correlated errors were specified for the same measures across ages. PrAgg = peer nominated aggression; Sev Phys Agg = severe physical aggression; MMPI F49 = the sum of scales F, 4, and 9 on the Minnesota Multiphasic Personality Inventory; G1 Par Hit G2 = the amount of hitting of G2 child at age 8 by the G1 parents; G2 Agg 'n' = G2's composite aggression score at age 'n'.

*p<.05 **p<.01 ***p<.001.

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we applied it to data from a three-generation longitudinal study. The data showed significant cross-generational continuity including significant relations between an offspring’s aggressive behavior as an adult and their parents’ aggressive behaviors in both childhood and during the time the child was being raised. A model fits the data well in which the cross-generational effects across two and three generations are mostly mediated by the parent’s aggression during the child-rearing period.

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