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Onset and Desistance in Criminal Careers: Neurobiology and the Age-Crime Relationship

RAYMOND E. COLLINS

Rutgers University

ABSTRACT Until recently, attempts to understand and explain criminal offending have been grounded in theories from sociological, legal, and psychological perspectives**.** In the preceding twenty years, or so, however, some research in the field has endeavored to look at offending from a psychobiological viewpoint. This research concerns the potential consequences of the effects of neurobiological influences on brain behavior and, consequently, human behavior. This paper discusses briefly some of the specific areas of neural research currently underway, including looking at the potential consequences for behavior, as it correlates with the age-crime curve, of the effects of neurotransmission. It also considers where the field of criminological research may be heading as a result of the insights into neurobiologically induced behavior. *[Article copies available for a fee from The Haworth Document Delivery Service: 1-800-HAWORTH. E-mail address: <docdelivery@haworthpress.com> Website: <http://www.HaworthPress.com> © 2004 by The Haworth Press, Inc. All rights reserved.]*

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Theories in criminology concentrate on the relationship of crime with many variables, including social, psychological, and to some extent biological factors apparently correlating with the commission of criminal acts to various de-

grees generally without specific reference to the age of the offender. "In contrast, developmental theories assume that different factors may have different effects on offenders of different ages" (Vold et al., 1998). Developmental theories discuss crime as a function of the life course: that is, in the progression through childhood, adolescence, adulthood to old age. A focal point in this progression for looking at desistance from criminal offending appears to be during the transition period from adolescence into young adulthood. This transition period has been seen as a critical stage in the "age-crime curve."

THE AGE-CRIME CURVE

Hirschi and Gottfredson (1983) have asserted that the age-crime relationship is generally strong and invariant. Of significant interest is the contention of Hirschi and Gottfredson (1986) that criminal offending declines with age. The decline in offending holds relatively constant for even persistent offenders and the relationship between age and criminal offending has been found to hold over time and throughout different cultures (Hirschi and Gottfredson, 1986).

As early as fifty years ago, Sheldon and Eleanor Glueck reported on the fundamental importance of the age-crime relationship (Sheldon and Eleanor Glueck, 1943). The Gluecks perceived the decline in criminal activity with the aging process as a kind of maturation result. Charles Goring also noted the correlation between age and crime as early as 1913 (Charles Goring, [1913] 1972). Even those critical of Hirschi and Gottfredson have admitted that age is a significant correlate of criminal offending (Blumstein, Cohen, and Farrington, 1988: 12-13).

In more recent psychological studies the observations of the Gluecks and Hirschi and Gottfredson have received substantial support. Frank Farley (1986) found that thrill seeking "is most often found among those in the 16-to-24 age range. From then it drops off gradually . . ." with its strongest expression in the late teens to early twenties preceding a general decline with age. Evidence presented by Farley is generally in accord with other psychological studies such as those of Colligan (1989) and Hare (1988). Colligan found that scores on the psychopathic deviation scale (Pd) of the Minnesota Multiphasic Personality Inventory were negatively correlated with age (i.e., evidence of psychopathic deviation declined as age increased) among both male and female subjects in a restandardization study (1989). He also observed the same correlation on the scores of the mania (Ma) scale.

Hare (1988) performed a 25-year study of Canadian offenders who had earlier been identified as either psychopaths or non-psychopaths. Hare presented evidence that "the criminal activities of the non-psychopath were relatively constant over the years, whereas those of psychopaths remained high until around age 40, after which they declined dramatically" (Hare et al., 1988). "If

decline in impulsivity and psychopathic deviation be accepted as reasonable operational approximations to 'burn out,' those clinical impressions seem to be psychometrically verified through the Mayo Clinic database" (Pallone, 1994: 181). Finally, in a study by Eysenck and Eysenck (1977), British prisoners scored significantly higher on psychoticism and neuroticism scales of the Eysenck Personality Inventory (EPI). This phenomenon occurred across all age groups, and score differences were not significant until age 30-39 when they were significantly lower. Moreover, up until age 30 prisoners scored higher on the social desirability scale than controls at which time scores began to reverse. "[T]hese findings are remarkably congruent with the general direction of research in *career criminality* [emphasis added] . . . that one cannot help but wonder whether a naturally-occurring psychological phenomenon (which might, as Pallone and Tirman [1978] suggested, be termed 'symptom abandonment' as a function of age) is not also reflected in decrease in overt criminal behavior" (Pallone, 1994: 181).

The above cited evidence of measured decline in criminal activity occurs at two basic time periods in the age-crime curve. Offending among non-psychopathic individuals appears to peak at late adolescence and/or young adulthood and then decline, whereas offending by those described as psychopaths shows a later age of desistance (generally, between 30 and 40 years of age).

NEUROBIOLOGICAL EVIDENCE AND THE AGE-CRIME CURVE

The human neurobiological system consists of the brain and the spinal cord. The brain is highly organized and contains about 100 billion neurons. "The regulated transmission of chemical and electrical signals through circuits formed by chains of neurons is the *basis of all behavior* (emphasis added). Consequently, to appreciate current developments in psychiatry, it is necessary to have a basic understanding of the structural and molecular properties that make such intercellular communication possible" (Barondes, 1993: 65). Neurons are cells which consist of the same basic elements as other cells in higher organisms. Neurons have a nucleus which contains chromosomes and it is the area for cell transcription (transcription is the process by which DNA is replicated). The neuron cell also contains other organelles such as mitochondria (home for mitochondrial DNA which comes exclusively from the mother of the organism) and the Golgi apparatus which helps in the building of the cell plasma membrane. The plasma membrane is not only the cell's outer wall but functions as the site where neurobiological signals are sent and received.

Signaling occurs at sites called synapses which consist of signal sending areas called axons and signal receiving sites called dendrites. The space between the axon and dendrite is called the synaptic cleft. Neuronal interactions are

quite complex, consisting of about a dozen major neurotransmitters, each with distinctive properties. Some of these molecules are amino acids, including GABA (γ-aminobutyric acid), a major inhibitory neurotransmitter. Others are derivatives of amino acids (generally referred to as monoamines). These derivatives include dopamine, norepinephrine and epinephrine, which function as excitatory neurotransmitters (also called catecholamines since their composition includes a catechol ring) and serotonin (5-hydroxytryptamine or 5-HT). "These monoamines are of great importance in psychiatry, since they have been implicated in mood states, as well as in the experience of fear and pleasure" (Barondes, 1993: 80).

When neurotransmitters are released they perform their functions by binding to receptors on the postsynaptic membrane (dendrites). The effect of neurotransmission is halted when the neurotransmitter is removed from the receptors and eliminated from the synaptic cleft. This elimination occurs in two ways: enzymatic degradation and/or cellular reuptake. For example, serotonin and dopamine are degraded by the enzyme monoamine oxidase (MAO), hence the use of drugs such as Nardil (Phenelzine) and Parnate (Tranylcypromine) which function as both monoamine oxidase inhibitors and reuptake inhibitors. Also used are the SSRIs (serotonin-specific reuptake inhibitors) such as Prozac (fluoxetine), Paxil (paroxetine) and Zoloft (sertraline). Suffice it to mention there are two kinds of monoamine degrading enzymes (MAO-A and MAO-B), but a further discussion of the difference is beyond the scope of this review.

SEROTONIN AND AGE

The major central nervous system (CNS) tracts for serotonin function include the primary site of serotonergic cell bodies in the midbrain which radiates out into the basal ganglia, the *limbic system and the cerebral cortex* (emphasis added). The cerebral cortex includes the area of the frontal lobes which is the "region that determines how the brain acts on its knowledge [and is] the main feature that distinguishes the human brain from that of other primates and that lends it uniquely human properties" including judgment, comportment, executive function and motivation (Kaplan and Sadock, 1999: 94). The limbic system, "a circuit of phylogenetically ancient structures, is responsible for generating and modifying memories and for *assigning emotional weight to sensory and recalled experience*" (emphasis added) (Kaplan and Sadock, 1999: 89). One area in the limbic system, the amygdala, receives fibers from all sensory areas and is apparently the place where the brain assigns emotional significance to memories.

Studies on animals and humans suggest that serotonin is a crucial modulator of aggressive behavior. "Violent juvenile delinquents have been reported to have *decreased* [emphasis in original] platelet 5HT2 binding in one recent

study" (Fogel et al., 1999: 338, citing Blumensohn et al., 1995). Several studies have found lowered levels of serotonin markers (CSF 5-HIAA: cerebral spinal fluid 5-hydroxyindoleacidic acid) in interpersonally violent individuals, including generally aggressive behaviors, arson and impulsive manslaughter (Fogel et al., 1999: 338).

As revealing as this evidence is in implicating low levels of serotonin in impulsive and aggressive behavior, a much more interesting finding is that "most studies report increases of MAO activity with age. According to Rogers and Bloom (1985: 657), the most consistent change observed in 5-HT metabolism is an age-dependent increase in 5-HIAA." Serotonin, then, is found to increase with age and that increase may have a moderating effect on violent, impulsive and aggressive behavior during middle age. It is of some interest to note that as primates become aged (well into their senior years), serotonin begins a decline in the occipital cortex (Beal M., 1993: 707). This may be a contributing factor to the violence exhibited by some elderly individuals diagnosed with Alzheimer's disease (AD).

DOPAMINE

"Generally, cholinergic and catecholaminergic mechanisms seem to be involved in the induction and enhancement of predatory aggression, whereas serotonergic and γ-aminobutyric acid (GABA) seem to inhibit such behavior" (Kaplan and Sadock, 1999: 158). Dopamine induces aggressive behavior in rodents and humans. "Apomorphine, a potent dopamine agonist (an agonist increases production of the neurotransmitter it works on), can induce fighting in rats. Dopamine antagonists tend to reduce aggression but usually at doses that also slow motor and cognitive performance" (Fogel et al., 1999: 338).

"Some of the most consistent findings in the aging literature suggest that dopamine (DA) receptors are lost . . . during aging in a variety of species including humans" (Joseph J. and G. Roth, 1983: 246). Also found in the Joseph and Roth study (1983) is that "basal activity for dopamine sensitive adenylate cyclase shows consistent linear decrements with age." The decline in dopamine is found to begin between youth and middle age. Finally, in a 1995 study of the brain DA system, Joseph et al. obtained results which "suggest that signal transduction deficits may involve age-related structural alterations in membranes that interfere with receptor-G protein coupling and uncoupling" (Joseph et al., 1995: 185).

Since high dopaminergic activity is correlated with aggressive and manic behavior (the epitome of which is schizophrenia)–cocaine users are familiar with the uplift associated with cocaine as a dopamine reuptake blocker–there appears to be a correlation between the decreasing function with age of the DA system and the observed mellowing with age of criminal activity.

NOREPINEPHRINE

Catecholamine systems, which include norepinephrine (NE) and dopamine (DA), are associated with aggressive behavior. "Peripherally administered norepinephrine enhances shock-induced fighting in rats. α_2-Receptor agonists (α_2-Receptors and β-Receptors are linked to cyclic AMP [adenosine monophosphate] controlled axons which permit or inhibit passage of the catecholamines from the synaptic cleft to the cell) increase rat aggressive behavior, whereas clonidine (a catecholamine antagonist) decreases rodent aggressive behavior acutely" (Fogel et al., 1999: 338). Norepinephrine is found primarily as a regulating, excitatory neurotransmitter in the limbic system including the hypothalamus, the thalamus, the hippocampus, and the *amygdala* (emphasis added) as well as the entire neocortex.

NE shares with DA several enzymes of synthesis and catabolism and therefore shares many of the age related changes common to the DA system. Austin et al. (1978) and Ida et al. (1982) observed age-related decline of NE in the rat hypothalamus. More recently, NE decline was found in human postmortem studies (Spokes, 1979; Carlsson et al., 1980; Rogers and Bloom, 1985: 654-5). Of much interest is the finding of the loss of NE in the hypothalamus because the hypothalamus is part of the limbic system. "This system influences the perception and expression of intense emotion. It is the center for anger, terror, fear, happiness, pleasure, and sexual arousal. Nervous disorders affecting the limbic system can produce excesses or deficiencies in any of these emotions" (Christiansen et al., 1993: 227). Once again, age-related deficits of NE correlate with the decline of criminal activity between youth and middle age.

ACETYLCHOLINE

One of the major functions of acetylcholine appears to be for normal learning and memory function. Acetylcholine neurotransmitters "enervate all the cortical structures, including the neocortex, the hippocampus, and the amygdala" (Fogel et al., 1999: 89). Some of the earliest studies on neurotransmission and aggression centered on acetylcholine. Electrical stimulation causing release into the synaptic cleft of acetylcholine in the hypothalamus (part of the limbic system) of rats initiates predatory attack on mice previously tolerated by the rats in their cage. "Aggressive behavior after human exposure to cholinesterase (an acetylcholine degrading enzyme) inhibitors has been observed in several clinical case reports" (Fogel et al., 1999, citing Grossman, 1963).

Taylor (1993) reports an age-related decline in central cholinergic transmission as a direct function of the aging process. "In summary, we have reported a decline in cholinergic synaptic transmission in the hippocampus (part of the limbic system) of aged animals. This reduction was not the result of an overall decline in cholinergic function in the hippocampus but rather a specific

decline in functional synaptic transmission with age" (Taylor and Griffith, 1993: 514). What part might the decline in hippocampal cholinergic synaptic transmission play in the age-related desistance of criminal offending?

HYPERTROPHY, TESTOSTERONE AND THE LIMBIC AREA

Rance et al. (1993) found moderate hypertrophy (swelling) of infundibular neurons in older men that may be due to the reduced circulating testosterone levels associated with male aging. "Therefore, examination of infundibular neurons in aging men could be a useful paradigm to determine the sensitivity of human hypothalamic neurons to circulating levels of gonadal steroids" (Rance et al., 1993: 337). In the infundibular nuclei is located the highest concentration of GnRH (Gonadotropin-Releasing Hormone) which stimulates the release of LH (Lutenizing Hormone) and FSH (Follicle-Stimulating Hormone) which in turn affect the release of testosterone in males (Fogel et al., 1999: 136). High levels of testosterone have been correlated with greater incidence of violent offending among young adult prison inmates (Dabbs et al., 1987). Testosterone is the principle male sex hormone "and it appears related to a wide range of psychological and behavioral factors, including aggression, dominance, overall activity level, libido, sensation seeking, persistence, and sociability" (Rance, 1993: 174). The infundibular neurons are part of the hypothalamus which in turn is part of the limbic system.

γ-AMINOBUTYRIC ACID (GABA)

GABA appears to be the most abundant neurotransmitter in the human brain. GABA is synthesized from 1-glutamic acid and is catabolized by an enzymatic degrader, GABA-T (GABA-a-oxoglutarate transaminase). While most neurotransmitters found in the human brain are excitatory (i.e., promote signal transmission between neurons), GABA is the major inhibitory neurotransmitter (i.e., suppresses signal transmission between neurons) in humans. GABA regulates essentially every behavioral function of the brain, including the autonomic nervous system, sexual function, growth function, ingestive behaviors, motor functions, as well as the behavioral functions of anxiety, fear and aggression (Paredes and Agmo, 1991). Lowered levels of GABA have been found to be correlated with isolation-induced aggression, flight behavior and predatory killing behavior in rats (Paredes and Agmo, 1991). These studies have also reported that GABA has a direct effect on the brain's learning process. "From previously presented data, it appears that GABA antagonists enhance retention in different learning tasks" (Paredes and Agmo, 1991: 157).

Moreover, "[S]everal lines of evidence suggest that γ-aminobutyric acid inhibits aggression in animals and humans. GABA injected into the olfactory bulbs in rats

(part of the limbic area) inhibits mouse killing. Benzodiazepines and other agents that facilitate GABA can decrease isolation-induced fighting in mice and attenuate aggression caused by limbic lesions (organic damage)" (Fogel et al., 1999: 338). The dampening effect that GABA has on excitatory neurotransmission is even more important in light of the assertion by Rogers and Bloom (1985) that GABA is found in large amounts in vivo prenatally and decreases from birth until age 20 when the decline in GABA levels off through middle age (Rogers and Bloom, 1985: 659). The age-related loss in GABA postnatally in animals occurs in several areas of the brain including parts of the limbic system. What effect, then, might this decline in the major inhibitory neurotransmitter have on behavioral components linked to crimes of aggression, thrill seeking, impulsiveness and violence? Moreover, consider the implications of the onset of decline of the excitatory neurotransmitters after age 20 congruent with the leveling off of the decline of GABA during the same time period. Might this phenomenon be a neurobehavioral correlate to the age-related desistance in criminal careers?

FUNCTIONAL GENOMICS: A NEW BEGINNING IN BRAIN RESEARCH

Genome analysis of brain genetics is now under way. Using a variety of methods grounded in the analysis of genemicroarray databases, "a hint to the function played by a certain gene may be obtained if that gene is coexpressed with other molecules that have a well-defined function in a signaling pathway or a regulatory circuit" (Mariani, 2003). A different method of functional genomics considers the location of brain-related gene expression and analyzes the spatially defined patterns of coexpression with other known genes (Eichele, 2003). Eichele et al. are establishing a database of the spatial-temporal expression patterns of functional brain genes (20,000-30,000 genes) at the Max Planck Institute. Using this database, researchers will attempt to provide a molecular counterpart to the histologic mapping of the brain initiated by Cajal.

These new research methods for understanding brain function will, almost inevitably, contribute to criminologists knowledge of the correlates (if not the causation) of antisocial behavior. "As daunting as genome-wide analysis of brain-related genes is, however, one notion seems to be pursued more and more. Complex biological systems and complex phenotypic traits, such as behavioral traits or 'abnormal' behaviors, may not represent, as in the past, insurmountable obstacles" (Mariani, 2003).

SOCIAL CONTROL THEORY AND DEVELOPMENTAL CRIMINOLOGY

The fundamental concept behind social control theory is that crime and deviance are more likely when individual bonds to society are tenuous or

breached. Laub (1996) argues for a differentiation by age over the life course and proposes that institutions of both formal and informal social control vary in their effect on individuals during the life span. Laub (1996) emphasizes the role of age graded "*informal* social control as reflected in the structure of interpersonal bonds linking members of society to one another and to wider social institutions (e.g., work, family, school). Unlike formal sanctions, which originate in purposeful efforts to control crime, informal social controls 'emerge as by-products of role relationships established for other purposes and are components of role reciprocities'" (Laub,1996: 246, citing Kornhauser, 1978: 24).

Grounded in the above concepts, Sampson and Laub (1993) have "developed a new theory of crime and delinquency over the life course" (Laub, 1996: 246) which consists of three components. The first component of the tripartite theory is the relationship between structural and process variables. Instead of considering structural variables as separate theoretical entities from process variables, what Sampson and Laub have done is integrate these two variables "along [with] individual characteristics like temperment and early conduct disorder into a single theoretical model" (Laub, 1996: 246). This model asserts that informal social controls derived from the family (discipline, awareness and emotional attachment) "mediate the effects of both *individual* [emphasis added] and structural backround variables" (Laub, 1996: 247). In this developmental model proximate factors (family discipline, e.g.) explain more variance than distal factors (formal institutional variables such as school and law enforcement, e.g.). Otherwise stated, process variables have more impact on individuals than structural variables.

The second component of this theoretical framework "incorporates the idea of continuity in childhood and adolescent antisocial behavior over the life course. In other words, antisocial behavior in childhood *predicts* a wide range of troublesome adult outcomes" (this is a strong and possibly warranted contention to be discussed later in this paper, emphasis added) (Laub, 1996: 247). Laub further contends:

> Our analysis found that independent of age, IQ, neighborhood socioeconomic status, and ethnicity, the original delinquents and nondelinquents in the Gluecks' study displayed behavioral consistency well into adulthood. Indeed, delinquency and other forms of antisocial conduct in childhood were strongly related to troublesome adult behavior across a variety of life's domains (e.g., crime, military offenses, economic dependence, marital discord). (Laub, 1996: 248)

The third component of the Sampson and Laub theory embraces the opposing concept of change in individual deviance and offending as one ages. "Having provided a role for continuity, we nonetheless believe that salient life events and social ties in adulthood can counteract, at least to some extent, the trajectories of early child development" (Laub, 1996: 248). Sampson and

Laub believe that social bonds in adulthood (e.g., labor force attachment and a cohesive marriage) can explain desistance in criminal behavior regardless of prior criminal propensity. "[W]e found that job stability and marital attachment in adulthood were significantly related to changes in adult crime–the stronger the ties to work and family, the less crime and deviance among both delinquents and control" (Laub, 1996: 248-9).

Apparently, then, the Sampson and Laub theory attempts a unification or fusion of "continuity and change within the context of a *sociological* understanding of crime during the life course [emphasis added]. Pathways and turning points are important concepts in the study of lives, and we have adapted this perspective to explore the lives of the disadvantaged sample of persistent adolescent delinquents" (Laub, 1996: 249). Laub continues: "Adaptation to life events is crucial, because the *same event or transition followed by different adaptations can lead to different trajectories*" (emphasis added) (Laub, 1996, citing Elder, 1985: 35). The following sections of this paper will be a critical look at some concepts leading toward a more comprehensive paradigm of developmental criminology, including a biobehavioral perspective, not included in the Sampson and Laub *sociological* paradigm.

SOME BIOBEHAVIORAL CONSIDERATIONS IN DEVELOPMENTAL CRIMINOLOGY

Wolfgang et al. (1972, 1987) in a study of juveniles in Philadelphia reported that 6% of juveniles accounted for 52% of all juvenile contacts with the police in that city and the same 6% were responsible for 70% of all felony offenses among juveniles in Philadelphia. Several other youth studies roughly confirm Wolfgang's findings that few juveniles in a cohort are responsible for an inordinate number of offenses (about 5 to 15 percent) (Shelden and Chesney-Lind, 1993; Facella, 1983; Thornberry et al., 1995 studies in Rochester, NY; 1995 Denver, CO; and 1995 Pittsburgh, PA, among others). There are neurobiological disorder correlates for these sociological findings.

Antisocial personality disorder (APD) "is an inability to conform to the social norms that ordinarily govern many aspects of people's adolescent and adult behavior" (Kaplan and Sadock, 1999: 784). The prevalence of antisocial personality disorder is about 3% to 7% in men and 1% in women (Kaplan and Sadock, 1999: 784; Hales and Yudofsky, 1987: 179-180). In prison populations, however, the prevalence of APD may be as high as 80% (Kaplan and Sadock, 1999: 794; Raine, 1999: 14, citing Hare, 1983; Correctional Services of Canada cited in Andrews and Bonta, 1998: 304). Recognizing the probable parameters of the APD syndrome in the offender population is important because "assessments of individual characteristics variously labeled 'antisocial personality,' 'psychopathic personality,' or 'weak self-control' are among the

strongest, most consistent correlates of criminality" (Andrews and Bonta, 1998: 80).

Looking at these data on the prevalence of APD in both the general population and the offender population, one is fascinated by the statistics. The largest amount of criminal offending appears to be committed by between 6% and 11% of the juvenile population. This percentage is even lower among the adult population as a direct result of desistance in criminal offending with age. In aggregate, then, criminal offending in the entire population (juvenile and adult) should average out to about 4.5% or less. This is almost exactly what would correlate to two standard deviations above the mean in a Gaussian distribution (extremely nonaggressive personalities would be found two standard deviations below the mean). Furthermore, assuming a neurobiologically induced dysfunction such as APD is a natural physical phenomenon, it would not be unreasonable to expect that it too would follow the same normal distribution in the population (see Kaplan and Sadock, 1999; Hales and Yudofsky, 1987).

NEUROBIOLOGY AND RISK-TAKING, SENSATION-SEEKING AND AGGRESSIVE BEHAVIOR IN HUMANS

It is clear from neurobiological studies that neurotransmitter dysfunction is correlated strongly with aggressive personality disorder and risk-taking behavior. Siever and Trestman (1993) found that in the noradrenergic system, clonidine-stimulated (α-adrenergic agonist) hormone responses were increased in patients with sensation-seeking and risk-taking disorders. Brown et al. (1982) reported a positive correlation between a history of aggressive behavior and deficits causing dysfunction in the β-adrenergic system (involving the neurotransmitter norepinephrine). This type of behavioral deficit is frequently associated with head injuries.

The most consistent data available involve the function of 5-HT (serotonin) in human aggression. Brown et al. (1982) reports that CSF 5-HIAA (cerebral spinal fluid 5-hydroxyindoleacetic acid), a catabolite of serotonin, was inversely correlated with clinician or self-reports of lifetime aggression. Other studies found negative correlations of CSF 5-HIAA with irritability, hostility, impulsive homicide, arson, maternal homicide, and with self-reported behavioral difficulties during childhood (Coccaro et al., 1993; Linnoila et al., 1983; and Virkkunen et al., 1989). In a more recent study, a correlation has been reported between a genotype (the gene type of an organism described by certain alleles [genes on a chromosome which may be dominant or recessive]) for a polymorphism (difference in DNA sequence among individuals) of an intron (non-coding section on a chromosome) in the gene for tryptophan hydroxylase (metabolite of 5-HT) and levels of CSF 5-HIAA in impulsive aggressive individuals (Nielsen et al., 1994). This finding suggests a connection between a

gene coding in the serotonergic system and impulsive aggression first postulated by Mednick et al. (1984).

There is also evidence that in the dopaminergic system genetic anomalies contribute to aggressive and dominant behavior. Several genes involved in dopamine function have been located and cloned including those for the DA receptors, the DA synthesizing enzymes tyrosine hydroxylase and β-hydroxylase, MAO_A and MAO_B, and several gene structures have been located which affect the metabolism and function of DA (Gejman et al., 1994; Itokawa et al., 1993; Rietschel et al., 1993). This is important because genetic defects in dopamine function have been found in impulsive behavior, antisocial behavior, drug abuse and aggression (Goldman et al., 1995; Comings et al., 1996).

NEUROBIOLOGICAL EVIDENCE AND ITS IMPLICATIONS FOR SOCIAL CONTROL THEORY IN DEVELOPMENTAL CRIMINOLOGY

Normal Neurology and the Age-Crime Curve

Sampson and Laub (1993) have developed a tripartite theory to explain onset and desistance in juvenile criminal offending. Based in social control theory which functions over the life course of an individual, their argument posits: (1) that structural variables should not be considered in absence of the effects of process variables; (2) that there is continuity in juvenile/adult antisocial behavior over the life course; and (3) that this continuity may be interrupted by structural and process variables. What their theory does take into account, then, is the environmental variables acting on juvenile/adult continuity in antisocial behavior. Social control theory sees the age-crime curve as a given phenomenon without asking why the phenomenon exists. But it is important to know that the age-crime curve may have a strong basis in normal neurochemistry.

For example, Farley's (1986) finding that "thrill seeking" is most common among the 16- to 24-age group (and has neurobiological correlates) should be of interest to social scientists who are attempting to influence that behavior. The neurobiological components of juvenile behavior (both social and antisocial) include the findings that a major inhibitory neurotransmitter called GABA begins to decline after birth and the decline begins to slow in adulthood. As juveniles age, 5-HT begins to increase in the brain, and this neurochemical has been found to inhibit aggressive behavior. Moreover, while 5-HT begins to increase, DA activity begins to wane throughout the life course of an individual. Norepinephrine, too, begins to decline with age. NE is an excitatory neurotransmitter functioning in the limbic lobe (the seat of the brain's initial response impulses) as well as other areas of the brain (Christiansen, 1993).

Concurrent to the neurotransmitter changes in the brain there occurs moderate hypertrophy of the infundibular neurons linked to reduced circulating testosterone associated with male aging (Rance et al., 1993). Testosterone is related to such behavioral factors as aggression, dominance, and sensation seeking. Finally, Duffy et al. (1993) found an age-related decline in both male and female slow brain wave activity as measured by EEG (electroencephalogram).

Further research into the behavioral aspects of the neurobiology of the brain should shed more light on adolescent/adult transitional behavior. But it appears for now that neurobiological functions are distributed in the human population according to a normal curve with violent offenders at about two standard deviations above the mean. What does this assumption hold for the Sampson and Laub continuity of behavior theory?

Dysfunctional Neurology and Continuity of Behavior Through Life Course

There is probably much substance to the contention of the Sampson and Laub social control theory that institutions such as a strong marriage and attachment to solid employment contribute to the desistance of criminal offending into adulthood–for most juveniles. Those juveniles who do not make a successful transition to desistance in offending may be the ones, for the most part, who are found with a neurobiological system statistically two standard deviations above the mean toward aggressiveness. It may not be the case that these offenders do not make the transition to desistance because they do not avail themselves of society's normalizing institutions. It may be that these offenders do not make the transition to normal institutional attachment because of their rare neurobiology which may be a causal factor (variable) in failure to desist from antisocial behavior. As presented earlier in findings by Farley (1986), Colligan (1989), Hare (1988) and others, aggression as a function of neurobiology may run its course. But in a small number of individuals (Kaplan and Sadock, 1999; Raine 1999, citing Hare, 1983; Hales and Yudofsky, 1987; Andrews and Bonta, 1996) researchers have found links to disordered, or abnormal, neurobiological function in about 3% of the general population and (not surprisingly) much higher percentages of neurobiological dysfunction among convicted offenders. These may be among the individuals (though not exclusively) who fail to make the transition into desistance from criminal activity.

A Bio-Psycho-Social Paradigm for Developmental Criminology

It is becoming clearer to many of those in the criminal justice field that contributory causal variables to the onset of criminal offending should include what Pallone and Hennessy (1996) term "intrapersonal" variables (psychological

variables linked to the neurology of individuals) as well as "extrapersonal" variables (social control institutions). This kind of paradigm is somewhat illustrated by Felson (1986) in linking routine activities, informal control, and rational choice in onset and persistence in criminal offending (although neuropsychiatrists would take exception to calling all criminal decisions a function of "rational choice" even though those decisions may appear rational to individual offenders).

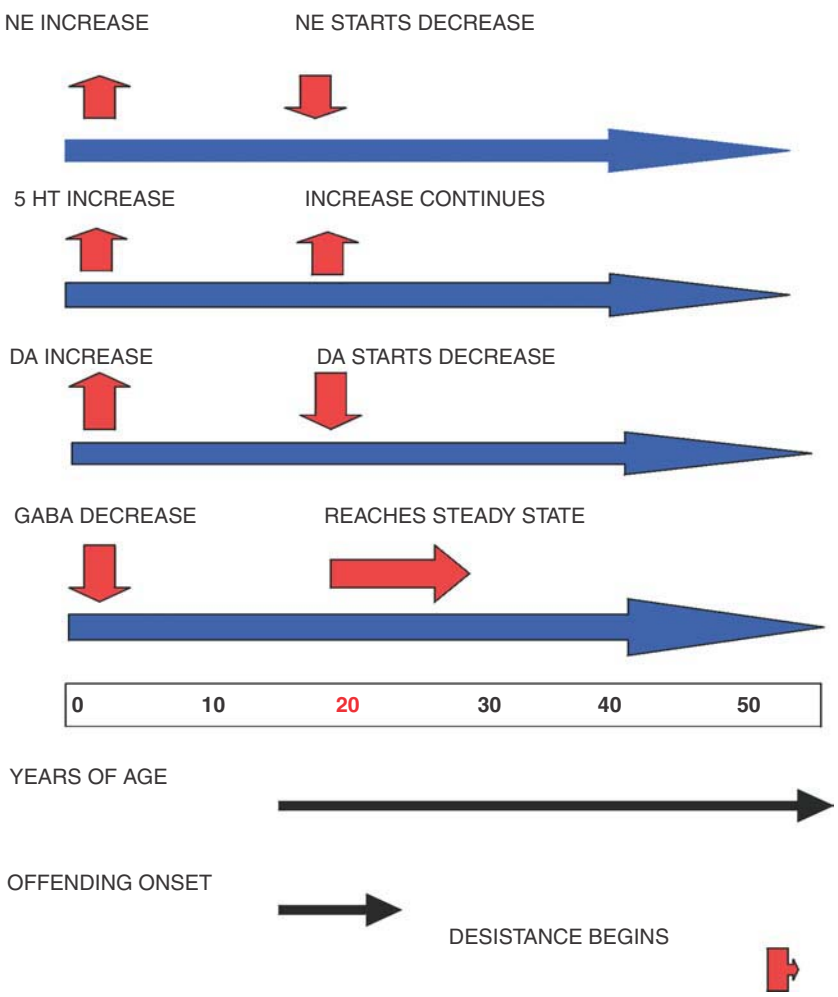
Figure 1 is a graphic representation for the beginnings of a model representing the interactive process that links neurology, demography and patterns of socialization and social influence as interactive precursors to each other and to criminal offending. This paradigm supposes that neurological influences are necessary but not sufficient variables for the onset/desistance of criminal offending. This chart represents basic normal neurology correlating to key focal points in the onset and desistance of antisocial behavior and/or criminal offending. A notable variant in this paradigm is the apparent desistance period which begins for psychopaths (in the late thirties or early forties) somewhat later than for offenders in general. Some (but certainly not all) of those who persist in criminal offending beyond the normal period of desistance may be found to reflect the psychopathic profile, but whatever the case it seems that most youthful offenders follow the normal neurological profile for desistance beginning in early adulthood.

THE BEGINNINGS OF A NEW PARADIGM FOR CRIMINAL OFFENDING

Until recently, criminal justice has been dominated by theories from the fields of law and sociology. During the last two decades, however, there have been attempts to develop an interdisciplinary criminology grounded not only in law and sociology but in biology (especially the neurosciences) as it inflects on psychology, sociology and the law. "These new sciences of behavior must be integrated into an interdisciplinary system with the environmental sciences of geography, urban-planning ecology, and sociology, and with the policy sciences of law, ethics, and political science" (Jeffrey, 1996).

Criminal justice practitioners might well heed the observations of Pallone and Hennessy (2000: 22-1) that "it is an unexceptionable proposition that paradigm shifts within any single discipline radiate outward to (as well as inward from) adjacent disciplines rather slowly, at least as those disciplines are represented . . . in the trappings of academic structure [and insulated as specific disciplines]. Hence, we have come to expect a palpable 'paradigm lag' between analytic models of behavior anchored in contemporary scientific psychology [with intellectual exchange from the neurosciences] and those anchored in other social science disciplines; what remains less clear is the typical duration of such a paradigm lag and its implications for interdisciplinary communica-

Figure 1: A Graphic Representation of Hypothesized Relationships Between Neurochemical Variables and Crime Onset and Desistance



tion and conceptual cross-fertilization." According to Coyle (1988), "the nearly logarithmic growth in neuroscience research" since the 1960s has yielded a major paradigm shift, producing in the process new methods for understanding the neurological functions of the brain. In resounding the view of Pallone (2000: 22-11), it becomes evident that identifying neurologic variables associated with criminal behavior can only provide risk markers at the psychological level: "What needs to happen next to apply such knowledge to social and personal betterment will be largely the work of the applied social sciences" (2000: 22-11). There is much to consider in the argument that any effort by the applied social sciences to alter human criminal conduct "in the absence of appropriate attention to . . . neurologic variables associated with [criminal behavior] will necessarily remain fragmentary until the communication gap between the neurosciences and the social sciences is securely bridged" (2000: 22-12).

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AUTHOR'S NOTES

Raymond E. Collins is a candidate for the PhD at the Graduate School of Criminal Justice at the Newark campus of Rutgers–The State University of New Jersey. His core area of research is in the effects of neuropsychological inflections on offending behavior.

Address correspondence to Raymond E. Collins, 765 Myrtle Street, Elizabeth, NJ 07202 (E-mail: raymondcollins@msn.com).