Summary of Adolescent Developmental and Neurodevelopmental Science in re Juvenile Life Without Parole

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Overview

In a series of US Supreme Court decisions, evidence from the developmental science of adolescence, including developmental neuroscience, has been cited in support of decisions eliminating capital punishment for juveniles and restricting the use of mandatory sentencing to life without parole for juveniles. This summary is intended to provide a brief descriptive overview of the developmental science used in those decisions, and of the continuing scientific progress in those relevant fields of research.[1] The most recent and ongoing research in these areas has added refinements to the overall picture, but the basics have been reinforced and extended to many other contexts.[2]

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[1] A summary of the developmental science used in *Thompson v. Oklahoma* (1988), *Roper v. Simmons* (2005), *Graham v. Florida* (2010), and *Miller v. Alabama* (2012) can be found in L. D. Steinberg, (2013): *The influence of neuroscience on US Supreme Court decisions about adolescents' criminal culpability, Nature/Neuroscience, 14,* pp. 513-518. This summary draws on that and its citations, along with other publications, including: Keating, D. P. (2012). Cognitive and brain development, *Enfance, 3,* 267-279; Keating, D. P. (2014); Adolescent thinking in action: Minds in the making. In J. Brooks-Gunn, R. M. Lerner, A. C. Petersen, & R. K. Silbereisen (Eds.), *The developmental science of adolescence: History through autobiography*. NY: Psychology Press. (Pp. 257-266); Keating, D. P., Demidenko, M., & Kelly, D. (2019). Cognitive and brain development in adolescence, *Reference Module in Neuroscience and Biobehavioral Psychology,* The Netherlands: Elsevier.

[2] Steinberg, L., Icenogle, G., Shulman, E. P., Breiner, K., Chein, J., Bacchini, D., & ... Takash, H. S. (2018). Around the world, adolescence is a time of heightened sensation seeking and immature self‐regulation. *Developmental Science, 21*(2), 1-13; Shulman, E. P., Smith, A. R., Silva, K., Icenogle, G., Duell, N., Chein, J., & Steinberg, L. (2016). The dual systems model:

The summary is organized into eight sections:

1. the relative immaturity of the prefrontal cortex, the executive functions, and the prefrontal goverance system (page 3);
2. the elevation of socio-emotional, incentive, and reward systems – the limbic system (page 5) ;
3. the developmental maturity mismatch (DMM) between those two brain systems (page 7);
4. the implications of current research for the prospects of rehabilitation among juvenile offenders (page 9);
5. the issue of age cutoffs (page 11);
6. the impact of adversity and trauma on the adolescent brain (page 13) ;
7. resilience and the capacity for further development (page 19); and,
8. notes on scientific methodology (page 23).

Review, reappraisal, and reaffirmation. *Developmental Cognitive Neuroscience, 17*103-117. doi:10.1016/j.dcn.2015.12.010

Sections

1. **Relative Immaturity of Prefrontal Cortex (PFC), the Executive Functions (EF), and the Prefrontal Governance System**

- *Executive Function, judgment, and decision-making*. The prefrontal cortex of the brain (the PFC) has long been understood to have the principal function of carrying out what are known as the "executive functions" (EF). These include basic functions such as working memory and planning, as well as where we direct our cognitive resources (known as "effortful control") essential for (1) impulse control (also known as the "inhibition of prepotent responses") and for (2) decision-making in complex situations. The PFC is known to begin developing in early childhood and to continue that development through the childhood, adolescent, and early adult years, showing full adult maturity in the early to mid-20s.[3] It is the functioning, and especially its gradual growth toward maturity, that is referenced in discussions of suboptimal adolescent judgment, especially in complex decision-making contexts that include competing demands.
- *Limited capacity*. Another key aspect of the PFC is that it has limited capacity. When fully engaged in one task involving effortful control, it has limited or no capacity to undertake additional tasks that require judgment. This has two implications: (1) having embarked on a plan to undertake a risky behavior, the execution of that plan may use up the available PFC resources, further

[3] This is found in research on the structure of neural circuitry, in neuroimaging in active performance situations, and in cognitive and behavioral evidence. The last section of this overview provides a brief description of the scientific methods used in the research described here and throughout the summary.

compromising the adolescent's ability to adjust behavior when circumstances warrant; (2) engagement with other activities that demand PFC resources, such as dealing with emotionally arousing situations or in the face of peer pressure, may make the limited PFC resource effectively unavailable.

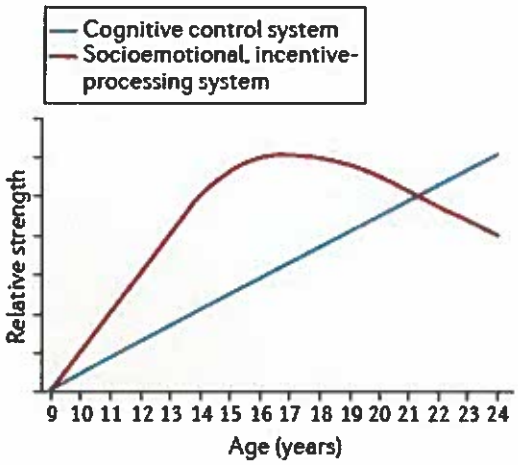
- *Governance of other brain systems.* In addition to the EF developments just described, the PFC shows development in a related function, the governance of other brain systems. This is also a gradual series of developments, as peripheral systems are brought more fully under the direction of the PFC. This is the basis of the colloquial designation of the PFC and its projections to other brain regions as the "top brain." It is not until the early to mid-20s that the ability to delegate tasks efficiently to other brain systems, relieving the PFC of its role to maintain effortful control and freeing up PFC space for other demands. Until that maturation has occurred, the ability of the rational, analytic, judgment, and governance functions of the PFC to override unanalyzed, poor decision-making is limited. This is a gradual process, so the maturational mismatch will on average be more marked the younger the individual.

2. Elevation of Socio-emotional, Arousal, Reward and Incentive Systems

- *Incentive systems*: Beginning in early to mid-adolescence, there is a sharp increase in what are termed "incentive systems" that entail complex neural circuitry, including emotional arousal (associated most strongly with the amygdala), sensation seeking (mediated by activity in the ventral striatum), and the heightened experience of rewards (mediated by a sharp increase in dopamine receptors) – a coordinated limbic system often referred to colloquially as the "bottom brain". These developments also coincide with (and may be partially explained by) significant changes in the hormonal balance associated with biologic pubertal shifts, principally as an activation of the HPG-axis (hypothalamic-pituitary-gonadal) whose endpoint is enhanced production of the steroids testosterone and estrogen, among others, differentially for males and females. These developments (neural hyperactivation and new hormonal set-points) are observed behaviorally and cognitively as a significant increase in exploratory and sensation-seeking behaviors during this same period of development when the governing capabilities of the PFC are limited (a mismatch described further below).
- *Benefits over risks*. There is substantial evidence that the factors above lead adolescents to focus more heavily on the benefits of risky behavior than on the possible negative consequences of their actions. This is not because adolescents are incapable of understanding or evaluating possible consequences of risky behavior, which under conditions of "cold cognition" (where little or nothing

that is upsetting, arousing or incentivizing is activated) is roughly the same as adults. Rather, they value the potential benefits of the behavior more highly than adults, altering the risk/benefit ratio in favor of undertaking unwise risks.

3. Developmental Maturity Mismatch (DMM) and Dual Process Models



- *Divergent developmental pathways*: The normative developmental pathways of the "top" and "bottom" brain diverge, with the limbic system advancing rapidly from early adolescence while the prefrontal system continues to grow, but at a slower pace, not reaching adult levels until the mid-20s. The term used to describe this is a "developmental maturity mismatch" (DMM), with significant consequences for the levels of all kinds of risk behaviors during the adolescent period. A schematic figure illustrates this[4].
- *Convergence of evidence*: The behavioral and cognitive evidence converges with the developmental neuroscience evidence here, with highly similar age-risk behavior profiles for a number of areas, including crime (the age-crime curve), accidental injuries, serious driving mishaps, and so on. All show peaks by mid- to late adolescence, with gradual drop-offs until they reach an asymptote in the mid-20s.
- *Dual process models*: The DMM is one version of a more general finding, known as dual process models. The research here shows that when performing a complex decision making task, there are two systems functioning. One is a

[4] This figure is from Steinberg (2013, see fn 1), although different versions of it have appeared in several publications.

rational, judgment based system that takes considerable cognitive effort. The second is a more automatic, "intuitive", non-analyzed system that is accessed more often (because it requires less time and energy). This occurs for automated tasks (especially in domains where expertise is high) but also and especially for "hot" cognition circumstances where there are competing demands – for example, from arousal and incentive systems. One line of research has proposed that the role of emotional response is sufficiently salient to consider a "triadic" model, in which the amygdala (key to emotion processing) is considered separately from the overall limbic system.[5] Although this is unresolved at the moment, it does not alter the basic picture of the DMM, in that the amygdala also shows a pattern of elevated development in the adolescent period.

[5] Ernst, M. (2014). The triadic model perspective for the study of adolescent motivated behavior. *Brain and Cognition, 89*, 104-111. See a summary of several current neuroscience models, and their similarities, in Demidenko, M., Huntley, E., Jahn, A., Thomason, M., Monk, C., & Keating, D. (2019). Cortical and subcortical response to the anticipation of reward in high and average risk-taking adolescents. *PsyArXiv*. https://psyarxiv.com/fnpvx/

4. Rehabilitative Prospects

- In addition to mitigation of sanctions owing to diminished culpability by reason of developmental immaturity, another implication of the developmental neuroscience evidence is that there are increased prospects for further developmental growth among juveniles. This is supported by the evidence already noted that major changes continue during this period. In addition, there is very substantial evidence for neural plasticity by way of a surge of new neural material ("synaptogenesis") followed by "synaptic pruning" based on ongoing developmental exposures and experiences that continue through this period of elongated adolescence until the mid-20s. Simply put, neural circuitry is shaped by the individual's experiences, such that the resulting mature circuitry is not settled until the mid-20s. Some plasticity continues throughout life, but never again as strongly as in adolescence. This potential for positive change was noted as a significant factor in recent Supreme Court decisions.
- *"Irreparably corrupt": Miller v Alabama* held that a sentence of life without parole is to be applied to juveniles only in rare cases of a determination of irreparable corruption. This would seem to require a determination that an individual can not be rehabilitated, which is assumed to be and is empirically found to be the case in a large majority of instances – desistance is substantially more probable than persistence. A successful methodology based on developmental science for determining irreparable corruption during the adolescent period has not been validated, and many of the measures have quite low predictability only a few years into the future, much less decades. Despite

efforts to construct accurate prediction well beyond adolescence, numerous limiting factors have made such efforts unsuccessful to date.[6]

[6] Fairfax-Columbo, J., Fishel, S., & DeMatteo, D. (2019). Distinguishing "incorrigibility" from "transient immaturity": Risk assessment in the context of sentencing/resentencing evaluations for juvenile homicide offenders. *Translational Issues in Psychological Science*, *5*(2), 132–142. https://doi-org.proxy.lib.umich.edu/10.1037/tps0000194

5. Age Cut-offs

- A perennial question in the developmental science of adolescence is what age should we use to identify the onset and offset of adolescent development. The first answer is that there are no hard and fast scientific markers for the onset and offset. Traditional language associates "adolescence" with the "teenage years," but there are important biological precursors (such as adrenarche, a shift in the functioning of the adrenal system) that begin to show changes as early as 8- or 9- years of age. The first markers of puberty per se are showing up on average at about 11-12 for girls and 12-13 for boys, with much individual variability.
- The offset of adolescence is also difficult to define, and it is this range that is most relevant in legal and criminal contexts. The cut-off of 18-years-old for consideration as a juvenile offender is a "bright line" legally, although the known biological markers support no such sharp delineation. A key measure of brain maturity is the ratio of white to gray matter. White matter is a marker for greater maturity through enhanced neural connectivity, because it shows up as white in MRI images because it has greater "myelination", the laying down of the fatty acid myelin that enhances neural connectivity. There is observable developmental change in this and in some other markers (volume, for example), especially in the PFC, but this change is gradual throughout the period from 12-13 through 23-25 on average.
- The elevated limbic system activation continues to decline through late adolescence/early adulthood as the prefrontal cortex continues to mature

throughout this same period. In that system, the evidence is more on levels of activation compared with structural shifts. A recent special issue in the journal *Nature* raised the question of how we regard the offset of the adolescent transition, and pointed to evidence that we should begin to think of an "elongated adolescence" that is not complete until the mid-20s.[7] Although this issue is not fully resolved scientifically or socially (with different ages as markers for driving, voting, entering enforceable contracts, juvenile offending, and so on), it is clear that markers are socially constructed rather than scientifically fixed, and have varied substantially across history. But the biological picture of brain maturity being attained only by the mid-20s is by now well established scientifically.

- One further issue is how close to some specific age cutoff one needs to be in order to be regarded as effectively "mature," and this often is brought up for individuals nearing their 18th birthday. But as noted above, 18 years of age is a legal, not a scientific cutoff, and 18-year-olds have considerable development, and thus neuroplasticity, remaining. Similarly, close age comparisons (within a couple of years) are beyond the scope of our current evidence.

[7] Ledford H. (2018). Who exactly counts as an adolescent? *Nature, 554*(7693):429-431; Worthman CM & Trang K. (2018). Dynamics of body time, social time and life history at adolescence. *Nature, 554*(7693):451-457

6. **The Impact of Developmental Adversity, Toxic Stress and Trauma on Adolescent Functioning**

- Many juveniles who have received or may be subject to receiving JLWOP sentences have experienced a history of trauma or significant adversity earlier in development, and this developmental history has been shown to substantially exacerbate the propensity for engaging in risky and/or potentially injurious behavior (to self or others) and to limit the capacity of juveniles with these developmental histories to inhibit or interrupt such behavior.[8] There is also substantial evidence, however, for the prospects of overcoming those obstacles through processes of resilience, prospects that are particularly salient during adolescent development.[9]
- *Elevation of risk behavior associated with early life trauma and adversity:* With the onset of new investigative techniques to look at the biological effects of earlier life trauma, including more widespread availability of neuroimaging and a growing understanding of epigenetic processes (in which developmental exposures and experiences change the ways that specific genes function, without altering the DNA[10] (also see Footnote 8 for an explanation aimed at a non-specialist audience), the behavioral association between early life trauma and developmental health problems later in life, increasingly studied in

[8] The general background for the impact of early life adversity on biology and behavior later in development is summarized in Keating, D. P. (2017), *Born Anxious: The Lifelong Impact of Early Life Adversity – and How to Break the Cycle.* New York: St. Martin's Press.

[9] Masten, A. (2001). Ordinary magic: Resilience processes in development. *American Psychologist, 56*(3):227-38.

[10] Keating, D. P., (2016). The transformative role of epigenetics in child development research, *Child Development, 87*(1), 135-142.

longitudinal cohorts over time, is now more clearly understood in terms of its underlying mechanisms.[11]

[11] References specific to early trauma and adolescent brain and behavior include:

Adams, Z. W., Moreland, A., Cohen, J. R., Lee, R. C., Hanson, R. F., Danielson, C. K., & ... Briggs, E. C. (2016). Polyvictimization: Latent profiles and mental health outcomes in a clinical sample of adolescents. *Psychology Of Violence, 6*(1), 145-155.

Alexander, N., Kirschbaum, C., Wankerl, M., Stauch, B. J., Stalder, T., Steudte-Schmiedgen, S., & ... Miller, R. (2018). Glucocorticoid receptor gene methylation moderates the association of childhood trauma and cortisol stress reactivity. *Psychoneuroendocrinology, 90*68-75.

Barzilay, R., Calkins, M. E., Moore, T. M., Wolf, D. H., Satterthwaite, T. D., Cobb Scott, J., & ... Gur, R. E. (2018). Association between traumatic stress load, psychopathology, and cognition in the Philadelphia neurodevelopmental cohort. *Psychological Medicine.*

Bernhard, A., Martinelli, A., Ackermann, K., Saure, D., & Freitag, C. M. (2018). Association of trauma, Posttraumatic Stress Disorder and Conduct Disorder: A systematic review and meta-analysis. *Neuroscience And Biobehavioral Reviews, 91*153-169.

Clark, D. B., Thatcher, D. L., & Martin, C. S. (2010). Child abuse and other traumatic experiences, alcohol use disorders, and health problems in adolescence and young adulthood. *Journal Of Pediatric Psychology, 35*(5), 499-510. doi:10.1093/jpepsy/jsp117

De Bellis, M. D., & Zisk, A. (2014). The biological effects of childhood trauma. *Child And Adolescent Psychiatric Clinics Of North America, 23*(2), 185-222.

Fox, B. H., Perez, N., Cass, E., Baglivio, M. T., & Epps, N. (2015). Trauma changes everything: Examining the relationship between adverse childhood experiences and serious, violent and chronic juvenile offenders. *Child Abuse & Neglect, 46*163-173.

Fragkaki, I., Cima, M., & Granic, I. (2018). The role of trauma in the hormonal interplay of cortisol, testosterone, and oxytocin in adolescent aggression. *Psychoneuroendocrinology, 88*24-37.

Greeson, J. P., Briggs, E. C., Layne, C. M., Belcher, H. E., Ostrowski, S. A., Kim, S., & ... Fairbank, J. A. (2014). Traumatic childhood experiences in the 21st century: Broadening and building on (Footnote 6 continued): the ACE studies with data from the National Child Traumatic Stress Network. *Journal Of Interpersonal Violence, 29*(3), 536-556.

Layne, C. M., Greeson, J. P., Ostrowski, S. A., Kim, S., Reading, S., Vivrette, R. L., & ... Pynoos, R. S. (2014). Cumulative trauma exposure and high risk behavior in adolescence: Findings from the National Child Traumatic Stress Network Core Data Set. *Psychological Trauma: Theory, Research, Practice, And Policy, 6*(Suppl 1), S40-S49.

Layne, C. M., Briggs, E. C., & Courtois, C. A. (2014). Introduction to the special section: Using the Trauma History Profile to unpack risk factor caravans and their consequences. *Psychological Trauma: Theory, Research, Practice, And Policy, 6*(Suppl 1), S1-S8.

- *Behavioral and cognitive patterns*: A history of early trauma and/or adversity has the capacity to become "biologically embedded", altering structural features of the brain and the functioning of key neurohormonal systems that are tightly linked to behavior and cognition. (The next section describes the underlying biology in summary format.) Among the most prominent of these outcomes are seen in the areas of:
  - Higher order cognition, especially executive functioning and impulse control. As noted above, in normative adolescent development, it is the behavioral/cognitive system that is crucial to inhibiting impulsive behavior, and effortful rather than unconsidered judgment about situations. This manifests in two crucial contexts: impulsively undertaking a high-risk behavior; and/or failing to think through options if the initial behavior leads into troublesome circumstances. This has been described metaphorically as the initial decision to board a train that is headed toward danger; and failing to get off that train if in fact the danger is becoming manifest. This insufficiency of executive and judgment resources (from the prefrontal system of the brain) is significantly worse for individuals who have experienced early life adversity or trauma, owing to structural brain changes described below.
  - The ability to regulate stress. As a result of epigenetic changes (more below), the stress response system in affected individuals lacks key

Lu, S., Gao, W., Wei, Z., Wang, D., Hu, S., Huang, M., & ... Li, L. (2017). Intrinsic brain abnormalities in young healthy adults with childhood trauma: A resting-state functional magnetic resonance imaging study of regional homogeneity and functional connectivity. *Australian And New Zealand Journal Of Psychiatry*, *51*(6), 614-623.

biological feedback controls that typically prevent the stress system from activating on a hair-trigger basis, and enable it to stand down when immediate danger has passed. This stress dysregulation can be expressed as heightened anger or aggression that is difficult to control (the fight response of "fight or flight"; and/or as internalizing tendencies or disorders, such as depression and anxiety (the "flight" option).

- There are lifelong health consequences from early adversity and trauma, exhibited in adulthood as increased morbidity in many aspects, and decreased longevity. These effects begin to show up as early as adolescence, however, especially in physical symptomatology, and in sleep disruptions, which is known to further aggravate stress dysregulation.

- *Brain changes*: The most replicated finding regarding changes to the brain as a result of early life trauma or adversity is in the prefrontal system. Differences in cortical thickness are seen as early as childhood and remain present into adolescence. There is also a normative development in adolescence of increased speed and accuracy of transmission along brain circuits, a function of "myelination" that insulates the circuits and is also known as white matter in the brain. This process proceeds more slowly for affected individuals, which leaves the prefrontal system with less management control over the rest of the brain. As a result of these processes, the effectiveness of the prefrontal system to act as a brake on risky behavior is compromised even more than for the typical adolescent.

- *Neurohormonal changes*: Early life trauma, even as early as in fetal life arising from toxic stress for expectant mothers, makes significant alterations to the neurohormonal system. The most researched of these is to the stress response system, which operates along the hypothalamic-pituitary-adrenal (HPA) axis. A gene that is responsible for feedback to the HPA-axis to turn off is changed epigenetically, so that there is too much cortisol, a central "fight-or-flight" stress hormone. Increasing research strongly indicates related effects on other systems that can mitigate the stress response, including the serotonin system (a target of many anti-depressants) and the oxytocin system (the "trust" hormone). These are down-regulated, such that their ability to moderate some of the effects of an over-active stress system is compromised. The combined effect of these biologically embedded alterations due to early trauma is to increase the chances of a highly aroused and impulsive stress response, and a reduced capacity to move out of that highly stress reactive state, potentially allowing the prefrontal system (which is also, unfortunately, likely to be compromised) to exercise better judgment and inhibitory control. The typical adolescent's experience of the developmental maturity mismatch (DMM) is metaphorically described as "much acceleration, few brakes", whereas for these individuals it moves even closer toward "all acceleration, no brakes."
- *Risk factor "caravans" and polyvictimization*: As more longitudinal cohort studies (that is, following the same individuals across time) have become available, and have been aggregated to get a clearer picture of the patterns, several key findings have emerged. The most significant patterns capture a

picture that is intuitively reasonable, but ground that intuition in empirical observations. Known as "risk factor caravans" and "polyvictimization", the basic pattern is that individuals who have experienced one trauma often are in situations where additional traumas are likely to occur at the same time or as they go forward with their lives. The refinement from these aggregated longitudinal studies is that each added trauma or victimization increases the chances of negative life outcomes later in life, especially in the vulnerable period of adolescence. Thus the probability that an individual with a traumatic early life will experience greater propensity to engage in high-risk behavior is substantially increased.

7. Resilience and the Capacity for Further Development

- The increased research attention to the consequences of early life trauma have sparked a parallel and more hopeful line of research on resilience, which has two meanings: at a population level, the empirical finding that some proportion of individuals who have experienced early trauma and would be expected to suffer long term negative consequences are in fact successful on most measures; at an individual level, it is the set of characteristics and processes that promote positive outcomes in the face of adversity. Beyond the general case, there is also a literature specifically on the probability of desistance from criminal activity, which is always higher than the probability of persisting into a lifetime of criminal offending, and on the factors that promote such desistance. The factors promoting resilience and desistance are similar, and are likely to be related processes.
- *Patterns of resilience*: There are two major processes that have been found to promote resilience at any age: social connection; and mindfulness (and closely related processes of identity and life purpose). The evidence for social connection as promoting resilience is extensive and well replicated. This can happen at any age, although the effects are strongest at the developmentally sensitive periods of infancy/early childhood, and adolescence. The connections do not need to be within the immediate family (and family stressors may have been the initial trigger or cause of adversity or trauma), and connections with other caring adults in the extended family, or teachers, coaches, and mentors can work the "ordinary magic" of resilience. In adolescence, close friends or

romantic partners may have a similar impact. The operation of mindfulness can be developed (though not without effort) to allow the individual to promote the availability and effectiveness of the prefrontal system to bypass the automatic, dysregulated patterns that have become embedded. Whether these processes reverse or merely mitigate the problematic biology from early trauma, the end result behaviorally is very much the same.

- *Brain and neurohormone changes underlying resilience*: Although resilience operates clearly at the behavioral level, recent research has pointed to a better understanding of the underlying biological mechanisms. The normative brain changes of adolescence include a proliferation of new neural material (known as "synaptogenesis"), which ushers in a period of enhanced neural plasticity and the rewriting of some critical neural circuitry. The changes from enhanced social connections and mindfulness can be detected, although their precise form remains a topic of intense research. The evidence from neurohormonal changes is clearer. Social connections enhance resilience in part by elevating the activity of the neurohormones serotonin and oxytocin, both of which are associated with positive emotions. More critically, they are also biologial antagonists to cortisol, limiting and reducing dysregulated stress responses. At both the behavioral and biological level, resilience is possible, although it can be challenging to achieve.
- *In criminal behavior, desistance is the rule, persistence is the exception:* The "age-crime curve" is among the most replicated findings in the social sciences, dating back to Quetelet's work in the 19th Century. The general pattern is that criminal behavior, including serious offending, tends to peak in mid- to late-

adolescence, with a substantial decline into the 20s and an even sharper drop-off after that. With the recent work in understanding the adolescent brain, the connection between the DMM and criminal desistance has become of great interest, with parallel research into the conditions and circumstances associated with desistance versus persistence.[12] A key distinction has been made between "adolescence limited offending" (AL) and "life course persistent offending" (LCP). The prevailing type statistically is AL, with most individuals desisting in early adulthood – as evident in the age-crime curve. There has been a great interest in identifying the differences between AL and LCP offenders, in order to understand the differences, to use them if identified as predictors, and to develop interventions and programs to move potential LCP offenders toward the AL type. There are a number of important findings from this relatively

[12] Barrett, D. E., & Katsiyannis, A. (2016). Juvenile offending and crime in early adulthood: A large sample analysis. *Journal Of Child And Family Studies, 25*(4), 1086-1097. doi:10.1007/s10826-015-0304-6

Brame, R., Mulvey, E. P., Schubert, C. A., & Piquero, A. R. (2018). Recidivism in a sample of serious adolescent offenders. *Journal Of Quantitative Criminology, 34*(1), 167-187. doi:10.1007/s10940-016-9329-2

Brame, R., & Piquero, A. R. (2003). Selective Attrition and the Age-Crime Relationship. *Journal Of Quantitative Criminology, 19*(2), 107-127. doi:10.1023/A:1023009919637

Jolliffe, D., Farrington, D. P., Piquero, A. R., Loeber, R., & Hill, K. G. (2017). Systematic review of early risk factors for life-course-persistent, adolescence-limited, and late-onset offenders in (Footnote 7 continued): prospective longitudinal studies. *Aggression And Violent Behavior, 3*315-23. doi:10.1016/j.avb.2017.01.009

Loughran, T. A., Nagin, D. S., & Nguyen, H. (2017). Crime and legal work: A Markovian model of the desistance process. *Social Problems, 64*(1), 30-52. doi:10.1093/socpro/spw027

Paternoster, R., Bachman, R., Kerrison, E., O'Connell, D., & Smith, L. (2016). Desistance from crime and identity: An empirical test with survival time. *Criminal Justice And Behavior, 43*(9), 1204-1224. doi:10.1177/0093854816651905

recent literature that has attempted to understand this from a developmental perspective. First, both AL and LCP offenders have a far higher frequency of early life trauma than non-offenders, much as we would predict from the patterns described earlier. The frequency of trauma between the two types is unclear, with some findings of greater trauma among LCP, and other research finding them to be roughly equivalent. Second, more serious offenders tend to show lengthier time to desistance, although after a decade or so, there is a desistance pattern more similar to those who have been imprisoned for less serious offenses. Desistance for serious offender seems to be delayed along the age-crime curve, but still occurs in the majority of cases. Third, the processes associated with desistance are, perhaps not surprisingly, those that are associated with resilience in general. Enhanced social connections, in the forms such as family formation or legal employment, are a significant predictor of desistance – along with "aging out". Developing an identity, sense of purpose, or self-awareness is also associated with a higher probability of desistance. In sum, early trauma is strongly linked to a higher risk of adolescent risk behavior and delinquency, but resilience (rehabilitation) remains possible for all.

8. Notes on Scientific Methodology

The sources of evidence used in this summary integrate several methodologies:

- *Structural neuroscience*: This refers to evidence on the changing structure of the "static" brain, that is, when it is not performing a task (in "resting state"). There are several methods for this, but the most prominent currently is diffusion tensor imaging (DTI), collected during a session of magnetic resonance imaging (MRI). This allows the characterization of the size of various parts of the brain, how they differ with age, and how they are connected with each other. It also is used for identifying structural anatomical features, and their development.
- *Functional neuroscience*: This assesses how the brain is working while it is engaged in a task, most prominently in functional MRI (fMRI) and various forms of electrical encephalography (EEG), such as evoked response potential (ERP). These use different physical methods (blood flow in fMRI, electrical signals in ERP), but they have the same goal, to elucidate the time and location of brain activity in different task conditions.
- *Cognitive and behavioral evidence*: In addition to the brain imaging evidence above, there are large amounts of behavioral and cognitive evidence that are relevant to the DMM, including self-reports of sensation seeking, impulsivity, and risk judgments, among others, as well as performance on cognitive tasks that assess EF, risk-reward trade-offs, and others.
- *Convergence of findings*: With respect to the confidence that is warranted with respect to the findings described above, one of the most important criteria (used in this summary) is to focus on findings where there is a ***convergence of findings across methods and content.*** Specifically, where the same

developmental pattern emerges from structural brain imaging, functional brain imaging, cognitive and behavioral evidence, and the epidemiology of risk behavior, we can have strong confidence in the major findings.

- ***Continued consistency of convergent evidence***. These overlapping areas of research have received ample attention before and subsequent to *Miller v Alabama*. This new work continues to provide strong support for the general conclusions drawn above on the basis of multiple lines of converging evidence.