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EDITORIAL

Cognitive and affective neuroscience and developmental psychopathology

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Can the connection between psychology and neuroscience provide a sufficient framework to support the study of the development of maladaptation and psychopathology? This Special Issue is devoted to papers that address this general issue within their specific domains. If we hope to provide a definitive answer to the question posed above, then it is important to know how cognitive and affective neuroscience arose, what are their distinctive findings to date, and, to the extent possible, predict what future developments can be expected. Before proceeding, we first examine the basic principles inherent to a developmental psychopathology perspective, as well as the multiple disciplines that played a critical role in its evolution as an interdisciplinary science.

Principles of Developmental Psychopathology

Historically, scientists in a variety of disciplines, including genetics, biology, neuroscience, embryology, psychology, and psychiatry, have stressed the importance of examining the interrelation between normal and abnormal patterns of development (see Cicchetti, 1990, for a historical review). Implicit in this perspective is an underlying commitment to understanding normal developmental processes so that we can begin to investigate the ways in which deviant development may eventuate. Furthermore, the examination of abnormal developmental processes and of the deviations from normal pathways of development may illuminate the range of individual variation inherent in the human organism with respect to neurobiological, cognitive, and affective functioning.

In part, as an outgrowth of these historical influences, over the course of the past several decades, developmental psychopathology has emerged as an integrative scientific discipline that strives to unify, within a lifespan framework, contributions from multiple fields of inquiry with the goal of understanding the relation between psychopathology and normative adaptation (Cicchetti, 1984; Cicchetti & Cohen, in press-a, in press-b, in press-c; Rutter & Garmezy, 1983; Sroufe & Rutter, 1984). Since its inception, work conducted within a developmental psychopathology perspective has incorporated theory and research from the fields of normal and abnormal development and advocated multidisciplinary approaches in its emphasis on examining the mutual interplay between normality and psychopathology with the ultimate goal of understanding individual patterns of adaptation and maladaptation (Cicchetti, 1984, 1990; Cicchetti & Toth, 1991; Sroufe & Rutter, 1984).

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In recent years, developmental psychopathologists have increasingly acknowledged that the investigation of developmental processes, both normal and atypical, is an inherently interdisciplinary enterprise. Scientists must utilize different levels and methods of analysis, depending on the questions being addressed in their research. Ideally, investigations must direct their energies toward an examination of multiple levels of analysis within the same individual. Although some problems are best addressed with the concepts and methods of a single discipline, other issues require interdisciplinary integration. In fact, history reveals that disciplines themselves often evolve from interdisciplinary efforts. For example, neuroscience developed as researchers working in a number of fields began to work in concert to solve some of the common scientific mysteries that existed about the nervous system (Albright, Jessell, Kandel, & Posner, 2000; Cowan, Harter, & Kandel, 2000). Importantly, the principles that neuroscientists have discovered have been utilized to inform research on the development of maladaptation and psychopathology (see, e.g., Cicchetti & Cannon, 1999; Cicchetti & Walker, 2001, 2003).

It is apparent from the questions addressed by developmental psychopathologists that progress toward a process-level understanding of maladaptive and psychopathological outcomes will require research designs and strategies that call for the simultaneous assessment of multiple domains of variables, both within and outside of the developing person (Cicchetti & Dawson, 2002). To comprehend psychopathology fully, all levels of analysis must be examined and integrated. Such research, almost by its very nature, must be interdisciplinary.

The National Advisory Mental Health Council (2000) recently concluded that interdisciplinary research should be accorded a higher priority in the basic science portfolio of the National Institute of Mental Health. Similarly, an Institute of Medicine report (Pellmar & Eisenberg, 2000) noted that it was critical to bridge research across disciplines to formulate a more comprehensive understanding of high-risk conditions and mental disorders. The power embodied by cross-disciplinary investigations that utilize multiple levels of analysis methodologies promises to significantly strengthen our capacity to decrease the burden of mental illness for society.

In this Special Issue, we examine the contribution that two prominent fields of neuroscience research can make to understanding the development of maladaptation and psychopathology. Specifically, we invited scientists working in the areas of cognitive and affective neuroscience to provide illustrations of how research in these two disciplines could elucidate the developmental processes eventuating in maladaptive or pathological outcomes.

Cognitive and Affective Neuroscience

The idea of connecting mental processes to brain activity goes back to the very beginning of psychology (Boring, 1950). The connection certainly received a considerable boost with the publication of Hebb's book, *Organization of Behavior* (1949), with its neural network approach to cognitive processes. Modern versions of cognitive neuroscience arose from the development of neuroimaging methods to view changes in the brain during thought processes such as occur during laboratory tasks (Albright, Kandel, & Posner, 2000; Gazzaniga, 2004; Posner & DiGirolamo, 2000). Imaging, first with positron emission tomography (Posner & Raichle, 1994), and later with functional magnetic resonance imaging (Rosen, Buckner, & Dale, 1998), opened up the human brain to detailed empirical examination of the nature and development of the neural networks Hebb proposed (for a recent review of this connection, see Posner & Rothbart, 2004). When combined with electrical or magnetic recording from outside the skull, it became possible to visualize in real time the circuits involved in competing aspects of an experimental task. The earliest imaging studies of language and attention fit quite naturally into the cognitive psychology and neuroscience frameworks and led to the development of journals and societies with the name cognitive neuroscience.

The study of the processing of positive and negative affect was also an important issue at

the start of the 20th century (Dagleish, 2004; James, 1890; Panksepp, 1998). Emotion was even more closely connected to physiology through animal research than was cognition. It was natural that neuroimaging would also take up issues of emotion (Davidson, 2000; Davidson, Jackson, & Kalin, 2000; Davidson, Scherer, & Goldsmith, 2003) and questions related to understanding the minds and emotions of others (Ochsner, 2004). Affective, social, and cognitive neuroscience have been built upon similar methods of neuroimaging applied to somewhat different problems. Moreover, the results of studies in all three areas have usually suggested specific networks of neural areas related to different cognitive, emotional, and social functions. These findings have the potential to draw the areas of social, affective, and cognitive studies closer together. For example, networks of brain areas related to regulation of cognitive and emotional functions have been shown to involve adjacent areas within the frontal midline (Bush, Luu, & Posner, 2000). Processing physical pain and the pain of social rejection appeared to involve overlapping areas of the anterior cingulate (Eisenberger, Lieberman, & Williams, 2003). Although anxiety and error can produce similar areas of activity in both cognitive and emotional tasks, many areas in the networks involved in emotion also can be quite distinct from those involved in unemotional cognitive tasks. The common network approach to diverse areas of psychology is beginning to lead to other commonalities in their study. For example, neural network models can be applied to cognitive, emotional (McClelland, 2001), or social psychological topics (Keysers & Perrett, 2004).

Although both cognitive and emotional tasks have revealed networks of brain areas common to most or all persons, there are also individual differences in details that influence the efficiency of the networks operation. These differences are likely to reflect both genes and experience. The rapid development of functional magnetic resonance imaging methods has begun to provide a basis for understanding differences among individual brains both anatomically and in terms of functional activations. Several studies have shown that individual differences in functional activation can be reliably assessed (Miller, Van Horn, Wolford, Handy, Valsangkar–Smyth, Inati, Grafton, & Gazzaniga, 2002; Ress, Backus, & Heeger, 2000).

These differences are to be expected because people are not identical in their thoughts, feelings, or behaviors. Studies also have examined the role of genetic differences in the strength of activation of networks involved in attention and memory (Goldberg & Weinberger, 2004). For example, studies in animals have identified a gene called brain-derived neurotrophic factor (BDNF) that plays a crucial role in long-term potentiation, thought to be a model of memory. One study examined the role of differences in two forms (alleles) of the *BDNF* gene (Egan et al., 2003). The behavioral part of the study compared performance of two groups with different forms of the gene performing a test of learning and memory. The two groups performed differently on the test, with the difference among alleles accounting for about 25% of individual differences on the test. When the test was run in the brain scanner on much smaller groups, significant differences between them were found in the hippocampus. Because the hippocampus is an important node in the network underlying explicit storage and retrieval in memory, these findings supported the importance of the *BDNF* gene in that function.

Work on attention has measured individual differences using the Attention Network Test (Fan, McCandliss, Sommer, Raz, & Posner, 2002). The test provides a measure of the efficiency of attentional networks related to maintaining the alert state, orienting to sensory information, and controlling conflict between competing responses. Studies of alert monkeys have shown that the orienting system is modulated by cholinergic input (Davidson & Marrocco, 2000) whereas dopamine is the principle modulator of the frontal areas important for monitoring conflict. Alleles of two cholinergic genes have been found to influence a visual search task related to the orienting network (Parasuraman, Greenwood, Kumar, & Fossella, in press); however, alleles of dopamine genes influence performance in the flanker task and, when compared, pro-

duced a significant difference in activation in the anterior cingulate (Diamond, Briand, Fossella, & Gehlbach, 2004; Fan, Fossella, Summer, & Posner, 2003).

These studies demonstrate that at least part of the variability in strength of activation is due to having different versions (alleles) of genes related to the network. However, genetic differences observed to date account for only a part of the variance found in behavior and imaging. Another potential contribution of the observed differences is that they serve as clues to the genes involved in the development of the common network. The genes related to individual differences can be examined in comparative animal studies to address questions such as how genes related to hippocampal development may have affected behavior in species even before there was a hippocampus and, in species for which the hippocampus plays a role in forms of memory, how these genes may be precursors of the explicit recollection found to be its role in humans. In the case of the *DRD4* gene, which in humans is related to attention deficit disorder (Swanson, Deutsch, Cantwell, Posner, Kennedy, Barr, Moyzis, Schuck, Flodman, & Spence, 2001), and a different allele to the normal monitoring of conflict (Fan et al., 2002), deletion of this gene in the mouse seems to be related to exploration of the environment (Grandy & Kruzich, 2004). These studies have the potential of improving our understanding of the role of genes in shaping the networks common to all humans. They also may eventually help us to understand how pathologies are related to genetic differences among people.

Now that the sequencing of the entire human genome has been completed (Venter, Adams, Myers, Li, Mural, Sutton, Smith, Yandell, & Evans, 2001), it is possible not only to examine the functional anatomy of brain networks but also to investigate how genetic differences might contribute to individual variation in the potential to use these neural networks in the acquisition and performance of skills. Nonetheless, the developmental pathway from genetic endowment to normal or abnormal performance will neither be simple nor independent from an understanding of the neural networks themselves.

The advent of contemporary cognitive and affective neuroscience and the effective integration and synthesis of the many previously independent disciplines that currently comprise the field (Albright et al., 2000; Albright, Kandel, et al., 2000; Cicchetti, 2002; Cicchetti & Cannon, 1999) also represent an unprecedented opportunity to augment current conceptual and methodological approaches to the study of resilience (Charney, 2004; Curtis & Cicchetti, 2003; Davidson, 2000). In addition, basic cognitive and affective neuroscience research on the development of maladaptation and psychopathology can be used to inform the diagnosis, prevention, and treatment of mental disorders. Such "translational research" (National Advisory Mental Health Council, 2000) is in direct accord with two of the major tenets of a developmental psychopathology perspective, namely, the reciprocal interplay between basic and applied research, and between normal and atypical development (Cicchetti, 1990; Cicchetti & Toth, 1998).

An outstanding example of the ability to use neural networks to approach issues of clinical treatment has emanated from studies of depression (Goldapple, Segal, Garson, Lau, Bieling, Kennedy, & Mayberg, 2004; Mayberg, 2003). These investigations have examined clinical interventions for depression based upon a neural network model. Treatments have involved drugs or cognitive–behavioral therapy. Both forms of therapy have been shown to be about equally effective based on percentages of persons showing improvement. However, neuroimaging data showed that the two therapies involved very different neural networks. The drugs remediated a largely subcortical network of brain areas that might be difficult to control voluntarily. The cognitive–behavioral therapy worked on cortical networks including areas involved in attention that would be more easily subject to voluntary control. These findings also suggest how genetic studies might allow better fits between the therapy used and the individual (Cicchetti & Blender, 2004).

Another example of the use of neuroimaging is in the study of reading. Dyslexia can be defined as a low level of reading skill that cannot be accounted for by general intelli-

gence or poor educational opportunity (Shaywitz, 2003). Imaging studies have shown dyslexia to involve under activation in two brain areas important in normal reading, a posterior phonological area, and an area of the visual system called the visual word form area. The visual word form area is thought to be involved in chunking the letters into a unified whole. In normal readers, these two areas appear to work automatically to convert visual words to appropriate sounds, but in dyslexic children they show little or no activation until after training. It appears that phonological training may be an optimal way to induce activation of the phonological area, but we do not know what methods will prove optimal for training of visual word form. One study did show a change in the word form area after 1 year of phonological training (Shaywitz et al., 2004), but we do not know whether it was the phonology or extra reading that was most effective in the change. One set of studies suggests that word form activation in children occurs only for words that the child already knows, while in adults, activation is based upon orthography and shows similar activation for unfamiliar but pronounceable nonwords as it does for familiar words (Posner & McCandliss, 1999). This might suggest that word form development occurs as a part of practice in reading.

These findings show how important imaging the underlying neural network is likely to be in improving the effectiveness of therapies and in adapting them to prevention and to individual needs. Furthermore, the conduct of basic cognitive and affective neuroscience investigations of biological and psychological processes contributing to resilient functioning, in concert with the examination of the biological and psychological changes that occur as a function of resilience-promoting interventions, will greatly enhance our present knowledge base on the development of resilience and on the recovery of positive function.

In summary, the papers in this Special Issue serve to highlight the synergy that exists between cognitive and affective neuroscience and developmental psychopathology. To fully realize the potential that the cross-fertilization of these disciplines may yield, we must make an ongoing commitment to fostering interdisciplinary work addressing multiple levels of analysis. The potential for these endeavors to decrease the burden of mental illness is vast and therefore warrants the dedication of professionals, as well as the allocation of resources.

References

Albright, T. D., Jessell, T. M., Kandel, E. R., & Posner, M. I. (2000). Neural science: A century of progress and the mysteries that remain. *Cell*, *100*, and *Neuron*, *25*, S1–S55.

Albright, T. D., Kandel, E. R., & Posner, M. I. (2000). Cognitive neuroscience. *Current Opinion in Neurobiology*, *10*, 612–624.

Boring, E. G. (1950). *A history of experimental psychology* (2nd ed.). New York: Appleton–Century–Crofts.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences*, *4*, 215–222.

Charney, D. (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry*, *161*, 195–216.

Cicchetti, D. (1984). The emergence of developmental psychopathology. *Child Development*, *55*, 1–7.

Cicchetti, D. (1990). A historical perspective on the discipline of developmental psychopathology. In J. Rolf, A. Masten, D. Cicchetti, K. Nuechterlein, & S. Weintraub (Eds.), *Risk and protective factors in the development of psychopathology* (pp. 2–28). New York: Cambridge University Press.

Cicchetti, D. (2002). How a child builds a brain: Insights from normality and psychopathology. In W. W. Hartup & R. A. Weinberg (Eds.), *The Minnesota symposia on child psychology: Child psychology in retrospect and prospect* (Vol. 32, pp. 23–71). Mawah, NJ: Erlbaum.

Cicchetti, D., & Blender, J. A. (2004). A multiple-levels-of-analysis approach to the study of developmental processes in maltreated children. *Proceedings of the National Academy of Sciences*, *101*, 17325–17326.

Cicchetti, D., & Cannon, T. D. (Eds.). (1999). Neurodevelopment and psychopathology. *Development and Psychopathology*, *11*(3), 375–654.

Cicchetti, D., & Cohen, D. (Eds.). (in press-a). *Developmental psychopathology, second edition. Vol. 1: Theory and method*. New York: Wiley.

Cicchetti, D., & Cohen, D. (Eds.). (in press-b). *Developmental psychopathology, second edition. Vol. 2: Developmental neuroscience*. New York: Wiley.

Cicchetti, D., & Cohen, D. (Eds.). (in press-c). *Developmental psychopathology, second edition. Vol. 3: Risk, disorder, and adaptation*. New York: Wiley.

Cicchetti, D., & Dawson, G. (Eds.). (2002). Multiple levels of analysis. *Development and Psychopathology*, *14*, 417–666.

Cicchetti, D., & Toth, S. L. (1991). The making of a developmental psychopathologist. In J. Cantor, C. Spiker & L. Lipsitt (Eds.), *Child behavior and development: Training for diversity* (pp. 34–72). Norwood, NJ: Ablex.

Cicchetti, D., & Toth, S. L. (1998). Perspectives on research and practice in developmental psychopathology. In W. Damon (Ed.), *Handbook of child psychology* (5th ed., Vol. 4, pp. 479–583). New York: Wiley.

Cicchetti, D., & Walker, E. F. (Eds.). (2001). Stress and development: Biological and psychological consequences. *Development and Psychopathology*, *13*, 413–753.

Cicchetti, D., & Walker, E. F. (Eds.). (2003). *Neurodevelopmental mechanisms in psychopathology*. New York: Cambridge University Press.

Cowan, W. M., Harter, D. H., & Kandel, E. R. (2000). The emergence of modern neuroscience: Some implications for neurology and psychiatry. *Annual Review of Neuroscience*, *23*, 323–391.

Curtis, W. J., & Cicchetti, D. (2003). Moving research on resilience into the 21st century: Theoretical and methodological considerations in examining the biological contributors to resilience. *Development and Psychopathology*, *15*, 773–810.

Dagleish, T. (2004). The emotional brain. *Nature Reviews in Neuroscience*, *5*, 582–589.

Davidson, M. C., & Marrocco, R. T. (2000). Local infusion of scopalamine into intraparietal cortex slows cover orienting in rhesus monkeys. *Journal of Neurophysiology*, *83*, 1536–1549.

Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *American Psychologist*, *55*, 1196–1214.

Davidson, R. J., Jackson, D. C., & Kalin, N. H. (2000). Emotion, plasticity, context, and regulation: Perspectives from affective neuroscience. *Psychological Bulletin*, *126*, 890–909.

Davidson, R. J., Scherer, K. R., & Goldsmith, H. H. (2003). *Handbook of affective sciences*. New York: Oxford University Press.

Diamond, A., Briand, L., Fossella, J., & Gehlbach, L. (2004). Genetic and neurochemical modulation of prefrontal cognitive functions in children. *American Journal of Psychiatry*, *161*, 125–132.

Egan, M. F., Kojima, M., Callicott, J. H., Goldberg, T. E., Kolachana, B. S., Bertolino, A., Zaitsev, E., Gold, B., Goldman, D., Dean, M., Lu, B., & Weinberger, D. R. (2003). The BDNF val66met polymorphism affects activity-dependent secretion of BDNF and human memory and hippocampal function. *Cell*, *112*, 257–269.

Eisenberger, N. I., Lieberman, M. D., & Williams, K. D. (2003). Does rejection hurt? An fMRI study of social exclusion. *Science*, *302*(5643), 290–292.

Fan, J., Fossella, J. A., Summer, T., & Posner, M. I. (2003). Mapping the genetic variation of executive attention onto brain activity. *Proceedings of the National Academy of Sciences of the United States of America (Washington, DC)*, *100*, 7406–7411.

Fan, J., McCandliss, B. D., Sommer, T., Raz, M., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, *14*, 340–347.

Gazzaniga, M. S. (Ed.). (2004). *The cognitive neurosciences: III* (3rd ed.). Cambridge, MA: MIT Press.

Goldapple, K., Segal, Z., Garson, C., Lau, M., Bieling, P., Kennedy, S., & Mayberg, H. (2004). Modulation of cortical–limbic pathways in major depression. *Archives of General Psychiatry*, *61*, 34–41.

Goldberg, T. E., & Weinberger, D. R. (2004). Genes and the parsing of cognitive processes. *Trends in Cognitive Science*, *8*, 325–335.

Grandy, D. K., & Kruzich, P. J. (2004). Molecular effects of dopamine receptors on attention. In M. I. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 260–268). New York: Guilford Press.

Hebb, D. O. (1949). *Organization of behavior*. New York: Wiley.

James, W. (1890). *Principles of psychology* (Vol. 1). New York: Holt.

Keysers, C., & Perrett, D. I. (2004). Demystifying social cognition: A Hebbian perspective. *Trends in Cognitive Science*, *8*, 501–507.

Mayberg, H. S. (2003). Modulating dysfunctional limbic-cortical circuits in depression: Towards development of brain-based algorithms for diagnosis and optimized treatment. *British Medical Bulletin*, *65*, 193–207.

McClelland, J. L. (2001). Failures to learn and their remediation: A Hebbian Account. In J. L. McClelland & R. S. Siegler (Eds.), *Mechanisms of cognitive development* (pp. 97–122). Mahwah, NJ: Erlbaum.

Miller, M. B., Van Horn, J. D., Wolford, G. L., Handy, T. C., Valsangkar–Smyth, M., Inati, S., Grafton, S., & Gazzaniga, M. S. (2002). Extensive individual differences in brain activations associated with episodic retrieval are reliable over time. *Journal of Cognitive Neuroscience*, *14*, 1200–1214.

National Advisory Mental Health Council. (2000). *Translating behavioral science into action: Report of the National Advisory Mental Health Counsel's behavioral science workgroup* (No. 00-4699). Bethesda, MD: National Institutes of Mental Health.

Ochsner, K. N. (2004). Current directions in social cognitive neuroscience. *Current Opinion in Neurobiology*, *14*, 254–258.

Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. New York: Oxford University Press.

Parasuraman, R., Greenwood, P. M., & Kumar, R. F., J. (in press). Beyond heritability: Neurotransmitter genes differentially modulate visuospatial attention and working memory. *Psychological Science*.

Pellmar, T. C., & Eisenberg, L. (Eds.). (2000). *Bridging disciplines in the brain, behavioral, and clinical sciences*. Washington, DC: National Academy Press.

Posner, M. I., & DiGirolamo, G. I. (2000). Cognitive neuroscience: Origins and promise. *Psychological Bulletin*, *126*, 873–889.

Posner, M. I., & McCandliss, B. D. (1999). Brain circuitry during reading. In R. Klein & P. McMullen (Eds.), *Converging methods for understanding reading and dyslexia* (pp. 305–337). Cambridge, MA: MIT Press.

Posner, M. I., & Raichle, M. E. (1994). *Images of mind*. New York: W.H. Freeman.

Posner, M. I., & Rothbart, M. K. (2004). Hebb's neural networks support the integration of psychological science. *Canadian Psychologist*, *45*, 265–278.

Ress, D., Backus, B. T., & Heeger, D. J. (2000). Activity in primary visual cortex predicts performance in a visual detection task. *Nature Neuroscience*, *3*, 940–945.

Rosen, B. R., Buckner, R. L., & Dale, A. M. (1998). Event relation functional MRI: Past, present and future. *Proceedings of the National Academy of Sciences of the United States of America*, *95*, 773–780.

Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In E. M. Hetherington (Ed.), *Handbook of child psychology* (4th ed., Vol. 4, pp. 774–911). New York: Wiley.

Shaywitz, S. E. (2003). *Overcoming dyslexia*. New York: Knopf.

Shaywitz, B. A., Shaywitz, S. E., Blachman, B. A., Pugh, K. R., Fulbright, R. K., Skudlarski, P., Mencl, W. E., Constable, R. T., Holahan, J. M., Marchione, K. E., Fletcher, J. M., Lyon, G. R., & Gore, J. C. (2004). Development of left occipitotemporal systems for skilled reading in children after a phonologically-based intervention. *Biological Psychiatry*, *55*, 926–933.

Sroufe, L. A., & Rutter, M. (1984). The domain of developmental psychopathology. *Child Development*, *55*, 17–29.

Swanson, J., Deutsch, C., Cantwell, D., Posner, M. I., Kennedy, J., Barr, C., Moyzis, R., Schuck, S., Flodman, P., Spence, A. (2001). Genes and attention-deficit hyperactivity disorder. *Clinical Neuroscience Research*, *1*, 207–216.

Venter, J. C., Adams, M. D., Myers, E. W., Li, P. W., Mural, R. J., Sutton, G. G., Smith, H.O., Yandell, M., & Evans, C.A. (2001). The sequence of the human genome. *Science*, *291*(5507), 1304–1351.