

# From the Guest Editors

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## Cognition and Psychopathology: Overview

*H. Carl Haywood*

Vanderbilt University, Nashville, Tennessee

*Stéphane Raffard*

University Montpellier 3, France

The bidirectional relation of cognition and psychopathology is discussed in historical context as an introduction to the special issue of *Journal of Cognitive Education and Psychology* devoted to contemporary research on this topic. Cognition refers to the processes by which information is transformed, elaborated, stored, recovered, and used. Psychopathology refers to disorders in behavior, cognition, and/or perception. Although rooted in studies of major psychiatric disorders and general intelligence, the field has moved to include developmental disabilities, neurological impairment, and less severe psychological disturbance on the one hand and finer grained cognitive processes on the other as well as a constant concern with language issues. Special methods of investigation reveal subtle effects but important facets of the cognition–psychopathology relation.

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Cognition refers to the processes by which sensory input is transformed, elaborated, stored, recovered, and used. Psychopathology is generally understood to refer to disorders in behavior, cognition, and/or perception. An extreme example of disordered cognition is delusional states, often observed in persons with schizophrenia. An extreme example of disordered perception is hallucinatory states, less often but sometimes observed in persons with schizophrenia. Disorders of behavior may be associated with either or both conditions, varying along a wide continuum of severity. Cognitive psychopathology is a recent

approach that uses the concepts and methods of cognitive psychology to study how cognitive dysfunctions (cognitive impairments, cognitive biases, and dysfunctional beliefs) contribute to the development, recurrence, and maintenance of psychopathological states. Adherents of this approach postulate that cognitive dysfunctions, which are closely related to emotional and relational processes, contribute to the development, maintenance, and recurrence of psychopathological disorders or symptoms. According to Bentall (1996), the main research goals of cognitive psychopathology are as follows:

- To provide better understanding of mental disorders
- To establish direct relationships between clinical symptoms and cognitive abnormalities
- To provide better understanding of the organization, functioning, and cerebral substrates of normal psychological processes, thereby emphasizing the interaction between cognition and emotion

From a clinical point of view, the principal goals of cognitive psychopathology are as follows:

- To develop new methods for assessing psychopathological symptoms
- To develop new treatments
- To contribute to new and improved classifications in psychiatry and abnormal psychology

We have assembled this group of papers as a sample, not necessarily representative, of contemporary psychological research on ways in which cognition and psychopathology are related and reflected in behavior, intellectual performance, social interaction, and nervous system phenomena. In doing so, we have defined psychopathology rather broadly to include psychotic, emotional, and behavior disorders as well as disorders primarily of development (e.g., intellectual disability, autism spectrum disorders, learning disabilities, and developmental language disorders). We have deliberately included language disorders because of the intimate relation of language and other cognitive processes.

Although cognition and psychopathology have traditionally been studied as relatively independent domains, there has been an observable trend over the last century to view and study them as interrelated in important and interesting ways (see, e.g., Beck, 1976; Beck & Bredemeier, 2016; Bleuler, 1911/1950; Dobson & Kendall, 1993; Haywood & Lidz, 2007; Hunt & Cofer, 1944; Kraepelin, 1919; Raffard et al., 2016; Roder & Müller, 2015; Shahar, 2013). Maher (1999) observed that “the mentally ill are generally defined in practice, if not in theory, by their manifest behavior. Abberations of their conscious experience, their sensations, feelings, thoughts and inner language are not open to direct observation” (p. 547). In spite of the “hidden” nature of these phenomena that are not directly observable, one can discern, from the papers in the present modest collection, unmistakable progress in quantifying and studying them.

Learning more about the interrelations of cognition and psychopathology is a scientifically important enterprise for both basic science and clinical applications. From a scientific standpoint, understanding how cognition and psychopathology are interrelated, and how each of these states and processes might possibly affect the other, will

advance the understanding of both. From a clinical perspective, the importance of understanding their interrelations will surely lead to advances in diagnosis and treatment, with potential treatment entry points in both domains of psychopathology and cognitive processes.

### MODERN (BUT NOT CONTEMPORARY) HISTORY OF INQUIRY INTO COGNITION AND PSYCHOPATHOLOGY

Both Bleuler (1911/1950) and Kraepelin (1919) were concerned with the relation of psychopathology, especially the schizophrenias, and intellectual functions, primarily as effects of severe psychiatric disorders on intelligence. To be sure, intelligence had not yet been analyzed into its subcomponents, so these pioneers in psychiatry tended to regard intelligence as a gross, undifferentiated phenomenon, in spite of the efforts of such investigators as Galton (1883) and Spearman (1927) to discover the components of intelligence. Both Bleuler and Kraepelin, as well as many of their contemporaries, observed that the acute onset of *dementia praecox* brought with it a significant decline in general intelligence, a decline that most psychiatrists of the time thought to be irreversible. Hunt and Cofer (1944) reported and challenged those observations. A series of studies done primarily in U. S. Veterans Administration psychiatric hospitals demonstrated a correlation of psychiatric symptoms and scores on intelligence tests, showing that IQ was at least partially recovered with diminution of psychiatric symptoms (see, e.g., Blaufarb, 1962; Hamlin, Haywood, & Folsom, 1965; Haywood & Moelis, 1963; Heinrich, 1991; Hunt & Cofer, 1944; Sclan, 1986; see also Haywood & Lidz, 2007, pp. 49–63). These studies revealed that special interactive techniques of assessment, now known as *dynamic assessment*, were instrumental in uncovering intellectual abilities that had been thought to be lost permanently.

It is important to note that most of the foregoing studies were focused on linguistic functions, such as vocabulary, verbal abstracting, and interpretation of proverbs, although some relied on general intellectual functioning (full-scale IQ). The series of studies eventually included individuals with intellectual and developmental disabilities (e.g., Gordon & Haywood, 1969) and traumatic brain injuries (Haywood & Miller, 2003), with strikingly similar results. Whereas, in cases of major psychiatric disorders, abilities that had once been developed appeared to be lost with the appearance of symptoms of the disorder, then to be at least partially recovered as psychiatric symptoms diminished, in cases of developmental disabilities, it has been possible, with the use of interactive/dynamic assessment strategies, to reveal intellectual and cognitive process abilities that had not been previously manifest as well as an unsuspected “teachability,” that is, ability to learn given teaching of fundamental cognitive processes, principles, and operations. Such findings lead us to conclude that special and more sensitive techniques of investigation and assessment are required to discover the masking of intellectual and cognitive processes that appears to accompany an astonishing variety of psychopathological conditions, reminding us of Sigmund Freud’s and Otto Fenichel’s references to “neurotic stupidity” (e.g., Fenichel, 1946).

Such special and sensitive methods of research certainly include brain imaging techniques that have become available in the last quarter century. Although recent analyses have questioned the validity of several fMRI studies (e.g., Eklund, Nichols, & Knutson, 2016), we

acknowledge that neurophysiological and neuroanatomical methods such as electroencephalography, magnetoencephalography, fMRI, and transcranial magnetic stimulation have provided an unprecedented opportunity to examine the neurocognitive components of mental disorders. For example, there is now evidence that auditory hallucinations in both clinical and nonclinical populations can be considered as erroneous perceptions or sensory deceptions without the presence of external stimuli and have been attributed to erroneous integration of sensory and cognitive processes that may influence conscious perception (Jardri, Pouchet, Pins, & Thomas, 2011).

From a neuroimaging perspective, structural brain imaging has allowed researchers to identify subtle but robust reductions in the gray matter volume in patients with auditory verbal hallucinations, particularly in areas involved in speech and language (Allen, Larøi, McGuire, Aleman, 2008; see also Caprihan et al., 2013), even if underlying mechanisms of auditory hallucinations extend beyond those processes (Waters & Jardri, 2014). Despite these important scientific advances, we must acknowledge that for the most part, brain imaging studies reproduce what has already been demonstrated via experimental and abnormal psychology, such as difficulty in identifying self-generated information, specific externalization bias, or failure to inhibit irrelevant memories (for a review, see Waters, 2012).

There is now clear evidence that cognitive psychopathology has allowed fundamental advances for our understanding of psychopathological disorders and brought significant advances for the treatment of individuals with mental disorders. Cognitive behavioral therapy and cognitive remediation have produced clear evidence of their efficacy (see, e.g., the review by Hofmann, Asnaani, Vonk, Sawyer, & Fang, 2012). It is important to note here that psychopathology cannot be explained solely by cognition. Biological, environmental, and societal variables, as well as emotional, motoric, and motivational processes, are important for our comprehension of mental states. As proposed by Kendler, Zachar, and Craver (2011), psychiatric disorders are defined not in terms of essences but in terms of complex, mutually reinforcing networks of causal mechanisms. Thus, we also would like to point out, following the work proposed by Belayashi and Van der Linden (in this issue), that the relationships between cognition and psychopathological states or psychiatric symptoms are complex, dynamic, and multidimensional and that cognitive interventions and cognitive theories must take account of this complexity.

## REFERENCES

- Allen, P., Larøi, F., McGuire, P. K., & Aleman, A. (2008). The hallucinating brain: A review of structural and functional neuroimaging studies of hallucinations. *Neuroscience and Biobehavioral Reviews*, *32*, 175–191.
- Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York, NY: International Universities Press.
- Beck, A. T., & Bredemeier, K. (2016). A unified model of depression: Integrating clinical, cognitive, biological, and evolutionary perspectives. *Clinical Psychological Science*, *4*(4), 596–619.
- Blaufarb, H. A. (1962). A demonstration of verbal abstracting ability in chronic schizophrenics under enriched stimulus and instructional conditions. *Journal of Consulting Psychology*, *26*, 471–475.
- Bentall, R. P. (1996). At the centre of a science of psychopathology? Characteristics and limitations of cognitive research. *Cognitive Neuropsychiatry*, *1*(4), 265–273.

- Bleuler, M. (1950). *Dementia praecox or the group of schizophrenias* (H. Zenkin, Trans.). New York, NY: International Universities Press. (Original work published 1911)
- Caprihan, A., Jones, T., Chen, H., Lemke, N., Abbott, C., Qualls, C., . . . Bustillo, J. (2013). The paradoxical relationship between white matter, psychopathology and cognition in schizophrenia: A diffusion tensor and proton spectroscopic imaging study. *Neuropsychopharmacology*, *40*, 2248–2257.
- Dobson, K. S., & Kendall, P. C. (Eds.). (1993). *Psychopathology and cognition*. San Diego, CA: Academic Press.
- Eklund, A., Nichols, T. E., & Knutsson, H. (2016). Cluster failure: Why fMRI inferences for spatial extent have inflated false-positive rates. *Proceedings of the National Academy of Sciences, USA*, *113*, 7900–7905.
- Fenichel, O. (1946). *The psychoanalytic theory of neurosis*. London, United Kingdom: Kegan Paul.
- Galton, F. (1883). *Inquiries into human faculty and its development*. London, United Kingdom: Macmillan.
- Gordon, J. E., & Haywood, H. C. (1969). Input deficit in cultural-familial retardates: Effect of stimulus enrichment. *American Journal of Mental Deficiency*, *73*, 604–610.
- Hamlin, R. M., Haywood, H. C., & Folsom, A. T. (1965). Effects of enriched input on schizophrenic abstraction. *Journal of Abnormal Psychology*, *70*, 390–394.
- Haywood, H. C., & Lidz, C. S. (2007). Dynamic assessment in clinical settings. In *Dynamic assessment in practice: Clinical and educational applications*. New York, NY: Cambridge University Press.
- Haywood, H. C., & Miller, M. B. (2003). Dynamic assessment of adults with traumatic brain injuries. *Journal of Cognitive Education and Psychology*, *3*, 137–163.
- Haywood, H. C., & Moelis, I. (1963). Effect of symptom change on intellectual function in schizophrenia. *Journal of Abnormal and Social Psychology*, *67*, 76–78.
- Heinrich, J. J. (1991). *Responsiveness of adults with severe head injury to mediated learning* (Unpublished doctoral dissertation), Vanderbilt University, Nashville, TN.
- Hofmann, S. G., Asnaani, A., Vonk, I. J. J., Sawyer, A. T., & Fang, A. (2012). The efficacy of cognitive behavioral therapy: A review of meta-analyses. *Cognitive Therapy and Research*, *36*(5), 427–440.
- Hunt, J. M., & Cofer, C. N. (1944). Psychological deficit. In J. M. Hunt (Ed.), *Personality and the behavior disorders* (pp. 971–1032). New York, NY: Ronald Press.
- Jardri, R., Pouchet, A., Pins, D., & Thomas, P. (2011). Cortical activations during auditory verbal hallucinations in schizophrenia: A coordinate-based meta-analysis. *The American Journal of Psychiatry*, *168*, 73–81.
- Kendler, K. S., Zachar, P., & Craver, C. (2011). What kinds of things are psychiatric disorders? *Psychological Medicine*, *41*, 1143–1150.
- Kraepelin, E. (1971). *Dementia praecox* (E. Barclay & S. Barclay, Trans.). New York, NY: Churchill Livingstone. (Originally published 1919)
- Maher, B. A. (1999). Anomalous experience in everyday life: Its significance for psychopathology. *The Monist*, *82*(4), 547–570.
- Raffard, S., Bortolon, C., Burca, M., Novara, C., Gely-Nargeot, M. C., Capdevielle, D., & Van der Linden, M. (2016). Self-imagination can enhance memory in individuals with schizophrenia. *Cognitive Neuropsychiatry*, *21*(2), 168–181.
- Roder, V., & Müller, D. R. (Eds.) (2015). *INT-Integrated neurocognitive therapy for schizophrenia patients*. Cham, Switzerland: Springer Publishing.
- Sclan, S. G. (1986). *Dynamic assessment and thought disorder in paranoid and nonparanoid schizophrenic patients* (Unpublished doctoral dissertation), Vanderbilt University, Nashville, TN.
- Shahar, G. (2013). Self, cognition, and psychopathology: Introduction to the special section (Part 1). *International Journal of Cognitive Therapy*, *6*(3), 203–207.
- Spearman, C. (1927). *The abilities of man: Their nature and measurement*. London, United Kingdom: Macmillan.

- Waters, F. (2012). Multidisciplinary approaches to understanding auditory hallucinations in schizophrenia and nonschizophrenia populations: The International Consortium on Hallucination Research. *Schizophrenia Bulletin*, 38, 693–694.
- Waters, F., & Jardri, R. (2014). Auditory hallucinations: Debunking the myth of language supremacy. *Schizophrenia Bulletin*, 41, 533–534.

Correspondence regarding this article should be directed to Dr. H. Carl Haywood, 11 Burton Hills Blvd., Nashville, TN 37215. E-mail: [carl.haywood@vanderbilt.edu](mailto:carl.haywood@vanderbilt.edu)