Chapter 23
Reality Perception Spectrum Syndromes (Imagination, Dissociation, Conversion, Somatoform, Misattribution Somatization, Psychosis)

Contents

23.1 Gene × Meme Interaction and Evolutionary Adaptation

What is reality? In humans, reality is a representation, integration in the brain of complex perceptions. Once perception is encoded in memory as a percept, it is a meme, i.e., it is capable of reproduction and multiplication. Imagination is also a representation – a creation or reassembly of percepts within the brain. Imagination may be directed (e.g., imagine a smiling face) or nondirected. There is evidence that similar areas of the brain are activated in actual perception and in imagination (Iseki et al., 2008; Kim et al., 2007; Qiu et al., 2008).

Reality is a complex memeplex, i.e., it consists of sets of memes or percepts that have been accepted by the selfplex as "real," as opposed to other sets of percepts that the selfplex has either ignored or adjudged to be "unreal" or illusory.

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Existing perceptual memes replicate and gain force when attention is paid to them internally and when they are reassembled as imagination. When this process is intense as in fantasy, the perceptual memes being manipulated or processed by the brain may replicate to the extent that their intensity may equal those of "real" perception.

23.1.1 Imagination and Dreaming

Normal persons can differentiate between imagination and reality in spite of similar activation of brain areas, but in the dreaming state, normal persons actually experience hallucinations and delusions as reality. The REM state during which dreaming usually occurs is phylogenetically ancient and probably serves important physiologic function. Dreaming is significantly influenced by the activity of the frontal cortex (Solms, 2000) and may serve the function of rehearsing coping with threatening stimuli (Revonsuo, 2000).

Imagination and dreaming are necessary (see Chapter 2 for more about dreaming) for problem solving and creativity. Being unable to distinguish between imagination, dreaming, and reality and being unable to return to reality result in distress and thus mental illness. The inability to distinguish may arise from either reduction in the evaluative aspect of meme processing, attenuation of reality perception, or excessive attention to the internal processing of memes at the expense of the patency of the perceptual apparatus as in dreaming.

23.1.2 Dissociation

Dissociation, and the associated phenomena of depersonalization and derealization are not uncommonly experienced in normal persons when absorbed in a task or under stress. Dissociation may be adaptive and may be hardwired as an adaptive mechanism (Berrios and Sierra, 1997; Sierra and Berrios, 1998). In the face of acute stress situations where the individual lacks control over the environment and cannot localize the source of threat, the inhibition of nonfunctional emotional response may serve an adaptive function, i.e., not engage in useless fight/flight but heightening alertness for the environment. Such inhibition of emotional responses and suppression of autonomic arousal creates a feeling of unreality – depersonalization seen in dissociative states (Seligman and Kirmayer, 2008). In both trauma-related dissociation and hysterical conversion, there seems to be a sense of distancing or disconnection from self and the world that characterizes depersonalization and derealization, related to cortical inhibition of emotional processing. The experience of subjective loss of voluntary control over parts of the body appears tied to cortical inhibition of attention and awareness as well as disruption of the link between volition and execution. This is consistent with studies of hypnosis which show that, in highly hypnotizable individuals, specific suggestions can reduce conflict between ordinarily competing attentional processes (Raz et al., 2005). Functional brain imaging in this situation shows an effective disconnection between anterior cingulate

cortex, thought to be involved in monitoring cognitive conflicts, and other cortical regions involved in the cognitive or perceptual task.

Through these mechanisms, hypnotic suggestion can cause a functional dissociation that reduces conflict between otherwise incompatible cognitive processes (i.e., conflicting memes), allowing potentially contradictory streams of information processing to coexist.

Under circumstances that increase suggestibility, and in susceptible individuals, misattribution of distress to parts of the body rather than to a memetic stress may occur as in conversion and somatization syndromes, as well as misattribution of percepts from within the brain (imagination) to outside of the brain (hallucination). Hallucinations and delusions occur regularly in "normal" individuals in hypnotic trance or religious frenzy.

Memetic environment often determines the content of dissociative and misattribution experiences, e.g., "possession" by spirits, fibromyalgia.

23.1.3 Dissociative Identity Disorder (DID, Multiple Personality)

In one study, some 18% of women in the general population had some dissociative disorder, while about 1% had a diagnosis of dissociative identity disorder (Sar et al., 2007).

In Chapter 11, I argued that we are all multiple personalities, that our brain contains a number of different selfplexes that are hopefully coexisting in a democracy. In such a state, each selfplex is experienced as a part of *me*. So, I sometimes say, "A part of me agrees with your idea, but another part of me is in violent disagreement." This is akin to a country in which there are strongly opposing forces toward an issue, for example, a war. Problems arise when authoritarian governments take over in succession and forcibly suppress other points of view, often even suppressing historical memory. Such is the state in dissociative identity disorder when there is a disturbance in the formation of a coherent set of mutually recognized selfplexes. At times of stress or vulnerability, a suppressed, unrecognized selfplex may take over and suppress other selfplexes. The newly dominant selfplex may not be connected to the memory memes of previous selfplexes, thus may suffer from global amnesia. More often, there is a mostly dominant selfplex which is from time to time overwhelmed by one or more selfplexes that incorporate memes disavowed by the mostly dominant selfplex.

Why does tolerant democracy of selfplexes fail to develop in certain brains? Childhood physical and sexual trauma is usually considered to be associated with DID. With severe trauma, there may be a degeneration of the hippocampus which in turn reduces the meme-processing ability of the brain (Ehling et al., 2008; Kihlstrom, 2005). There may also be a developmental arrest of the orbitofrontal cortex involved with the sense of self (Forrest, 2001). The ventromedial cortex including the orbitofrontal cortex, the cingulate gyrus, and areas of temporoparietal cortex seem to be intimately involved in the sense of self (David et al., 2006; Devinsky et al., 1995; Lou et al., 2005). Thus, normal, integrated sense of self with

different personality aspects may represent a democracy of selfplexes that control the ventromedial cortex and associated brain areas.

In DID, competing and contradictory selfplexes may take over the brain areas, often incompletely. As selfplexes are clusters of meme-representing neurons, "taking control" means that the meme-containing neural clusters strengthen their connection to the sense of self areas of the brain.

In certain meme pools such as the Afro-Brazilians with the Candomblé religion, the religious memes provide a normalizing explanation for dissociative identity disorder and the stress that precipitates it. The Candomblé belief system involves the idea that a pantheon of deities, called the Orixas, control human destinies and human bodies. Orixas may choose to possess particular individuals. Hence, individuals experiencing acute psychosocial stress and dissociation may interpret these experiences as products of a spiritual disturbance caused by the Candomblé deities. By facilitating such interpretations, Candomblé encourages the belief that individuals may experience discontinuity among aspects of the self, including body, memory, responsibility, and personal identity. Such attributions allow individuals to understand their stressful experiences as non-self-implicating, thus alleviating the need to face the trauma directly. Belief in possession also allows them to embrace their dissociative experiences as spiritually productive, as such alterations in consciousness represent the replacement of their own self with that of a possessing deity. In fact, individuals learn how to induce such states of awareness in ritual contexts (Seligman and Kirmayer, 2008).

23.1.4 Misattribution Syndromes: Conversion, Somatization, Hypochondriasis, Chronic Pain (Somatoform Disorders)

Bodily symptoms without obvious physiological explanation are considered to be *somatoform* disorders. They presumably have psychological explanations.

Conversion disorder, or hysteria, is an age-old entity, first attributed to wandering uterus by Hippocrates. Conversion symptoms are such physical symptoms as anesthesia, paralysis, syncope, ataxia, globus hystericus, and seizures. Somatization disorder or Briquet's syndrome consists of physical symptoms in various parts of the body without demonstrable underlying pathophysiology. Chronic pain syndrome, or psychogenic pain, is pain without organic pathology or in excess of what is expected from organic pathology. Hypochondriasis is a preoccupation that one has the symptoms of a serious disease. There are other somatic symptoms such as chemical intolerance, fibromyalgia, chronic fatigue.

Some of the disorders arise relatively early in life, often in families, and associated with stress. Freud considered hysteria to be a result of unconscious psychological conflicts: the symptom represents a compromise resolution of the unconscious conflict between an unacceptable wish (such as a gene-driven aggressive wish toward an authority figure) and a meme-driven inhibition (conscience), which compromise allows the conflict to be unconscious and thus avoid

experiencing the anxiety had it become conscious, but also drawing attention to the prohibited wish by the symptom itself, such as the paralysis of an arm that would have struck at the authority figure.

Conversion symptoms tend to occur more easily in patients with brain injury, and conversion seizures often coexist with electrical seizures. There may be specific cortical inhibition in certain conversion symptoms (Aybek et al., 2008).

In somatization, hypochondriasis, and chronic pain, there may be an amplification of mild discomfort into more severe one in the brain (Barsky and Borus, 1999; Barsky et al., 1988; Barsky and Wyshak, 1990).

Early-life stress and negative parental relationships have been associated with chemical intolerance syndrome in women (Bell et al., 1998). It is well known that fibromyalgia, chemical sensitivity, and hypoglycemia achieved epidemic proportions in recent time (Ross, 1999).

In general, the symptoms may arise through a process of brain's misattribution of distress to somatic (sensory and visceral) sources when they are central and often memetic in origin. This distress misattribution process may be facilitated in certain individuals through epigenetic factors, early memetic infection from another person who somatizes or is in the sick role, or both, and current stress that both causes the distress and reawakens the sick role memes.

23.1.5 Psychosis

Psychosis is an alternative mode of experiencing reality, often occasioned by an inability to distinguish fantasy and dreaming from reality. Drugs such as LSD as well as prolonged sensory deprivation can produce symptoms of psychosis. Susceptibility to psychosis may arise from genetic and epigenetic factors during the maturation of the brain. Auditory hallucinations may be a misattribution of the source of memes to the outside when it is actually a perception of memetic proliferation within the brain, particularly in the nondominant hemisphere. The "inner voice" may be actually heard as auditory hallucination in affected individuals. Crow proposed that such hallucination may be a result of failure of the left brain to exert complete dominance in language production and thus an inability to differentiate between self-generated thoughts and voices coming from outside (Crow, 2000).

The memetic content of psychosis is clearly dependent on the meme pool to which the individual has been exposed. Thus, delusions and hallucinations may be religious in religious societies, paranoid in secular societies, etc. (see Chapter 4).

23.1.6 Schizophrenia

Schizophrenia is a group of chronic psychotic syndromes characterized by a relatively early age of onset and a general decline in function without treatment. The

risk of developing schizophrenia in the general population is somewhat less than 1%, while the prevalence for parents of children who are known schizophrenics is 12%. The morbidity risk for schizophrenia for full siblings of schizophrenic patients is 13–14%. The risk for children with one schizophrenic parent is 8–18%. If both parents are schizophrenic, the morbidity risk for their children may be as high as 50%. In the case of twins, heterozygous twins have the same risk as other siblings, while homozygous (identical) twins have a concordance rate for schizophrenia of approximately 50%. (However, there is much variability in the concordance rate depending on the study, from practically 0 to 86%.)

In spite of the demise of the term *dementia praecox*, cognitive disturbance has recently become a cornerstone of understanding schizophrenia. Schizophrenia is conceptualized as a neurodevelopmental disorder resulting in a reduction in cortical volume and dysfunctions in glutamatergic, GABA (γ-aminobutyric acid)ergic, and dopaminergic transmission. There seems to be a hyperfunction of the mesolimbic and a hypofunction of the mesocortical dopaminergic transmission.

Mesocortical dopaminergic transmission is stimulated by glutamatergic transmission and reduced by GABAergic transmission, and it plays an important role in working memory often disturbed in schizophrenia (Romanides et al., 1999). There is evidence of dysfunction in schizophrenia of the GABAergic cortical chandelier cells that synchronize the firing of the glutamatergic pyramidal cells, which are necessary for proper functioning of the working memory (Lewis et al., 2004). A fundamental disturbance in schizophrenia seems to be an inefficiency of the prefrontal cortex, particularly the dorsolateral area, in processing information, and increased "noise" in the local microcircuit function (Meyer-Lindenberg et al., 2005; Weinberger, 2005).

A number of genes have been identified as candidate genes for the susceptibility to schizophrenia: catechol-*O*-methyltransferase *(COMT)* (chromosome 22q), *dysbindin-1* (chromosome 6p), *neuregulin-1* (chromosome 8p), metabotropic glutamate receptor 3 *(GRM-3)* (chromosome 7q), glutamate decarboxylase 1 (chromosome 2q), and disrupted-in-schizophrenia 1 *(DISC1)* (chromosome lq).

The *COMT* gene affects prefrontal cortical function by changing dopamine signaling in the prefrontal cortex and brainstem. *GRM-3* shows similar results on prefrontal function and has an effect on expression of various glutamate synaptic markers. *DISC1* affects hippocampal anatomy and function. *Dysbindin-1* seems to be a general cognitive capacity gene that is underexpressed in the cortex of schizophrenic patients.

As should be obvious from this discussion, schizophrenia is not a simple genetic disease; rather, it is a syndrome contributed to by susceptibility genes that have functions other than conveying susceptibility to schizophrenia. Such susceptibility may include susceptibility to psychosis in general including psychosis in bipolar disorder (Goes et al., 2008; Nickl-Jockschat et al., 2008; Prasad et al., 2008; Sullivan, 2008; van Haren et al., 2008).

Intrauterine infections may also play a role in the development of schizophrenia as well as early memetic environment and stress.

Schizophrenia has conferred a reproductive disadvantage on the afflicted. Why, then, is schizophrenia extant at more or less a constant rate across human populations? An obvious explanation is that the alleles that, in certain combinations, may predispose one to schizophrenia may be involved in other functions that are adaptive. Some of these may be involved in creativity and eccentricity.

In addition, the susceptibility genes may represent variations of ubiquitous genes subserving basic functions of the human brain. Crow proposed that schizophrenia may represent an extreme of normal genetic variation in the communication between the two hemispheres that is critical in language, a uniquely human acquisition. He postulates that Schneiderian first-rank symptoms such as thought insertion and withdrawal may represent a dysfunction of the coordinated hemispheric communication – a right hemispheric intrusion into left hemispheric linear thinking (Crow, 1997, 2007). Schizophrenia may represent an extreme of variations in the interconnectivity of various structures of the brain, particularly those involved in social cognition and the working memory.

In sum, certain individuals with problems in the normal development of the brain functions involved in meme processing may be susceptible to difficulty in distinguishing between internal and external sources of meme presentation, such that internal memes are perceived as external perception (hallucinations).

Psychotic symptoms, therefore, may represent an intrusion into consciousness of internal existing memes that are ordinarily unconscious but are available in adaptive processes such as fantasy and creativity. In some individuals, psychosis may also be a result of disorganization of the meme-processing functions of the brain due to severe internal or external stress. In such states, certain memes that are in conflict with the dominant selfplex may proliferate, having gained strength by recruiting the perceptual apparatus in the form of hallucination. It is one thing to hear your own conscience, quite another to hear God admonishing you.

Delusions may occur when the meme-processing ability is impaired such that irrational memes are reinforced and become convictions, beyond the control of the meme filter for unreasonable memes. Delusions are irrational memes that have either severed themselves from the rational meme-processing apparatus or overwhelmed it, and have taken up residence as a part of the dominant selfplex. No contradictory memes will then be able to enter the brain and take hold.

The meme pool of the environment may then determine the *presentation* of the psychosis, i.e., what is the "normal" way to become psychotic in that culture? Thus, in some cultures, presenting with acute agitation and uncontrolled behavior may be the accepted way of "being crazy," while in another culture, it may be becoming hypervigilant and paranoid. See Chapter 3 for further discussion of the interplay among culture, stress of migration, and psychiatric symptoms.

The confusing hallucinations and/or delusions arising from faulty meme processing may force the patient to assume the "crazy meme" in the culture, which may in turn serve to reduce the distress, being labeled "crazy" and thus accepted in that role.

The clinical course of schizophrenia is clearly influenced by memetic stress. Memes in the form of expressed emotions, particularly negative ones, have been associated with exacerbations and hospitalizations both on a short-term and long-term basis (Kymalainen and Weisman de Mamani, 2008; Marom et al., 2005).

23.2 Treatment

23.2.1 Mild Dissociative Symptoms, Dissociation in Borderline Syndrome, PTSD

Generally, no treatment is necessary for mild dissociative symptoms including depersonalization and derealization as they tend to be time-limited and do not cause serious distress.

Severe, uncontrolled, and distressing dissociation may occur concomitant to strong emotional arousal with associated autonomic and endocrine arousal, often in the borderline syndrome and PTSD. Management and prevention of strong emotional arousal, especially anxiety, is through broad-spectrum anti-meme therapy and medications when indicated.

Memetic reinforcement of the selfplex may be helpful for the relatively transient dissociative symptoms, in the form of repetition of words or phrases asserting the identity or attributes of the selfplex, e.g., "I am Helen Stein, I am an internist, I am competent and respected." Such repetition will enhance replication of the desired selfplexes in the brain, and facilitate their regaining control over the sense of self brain areas. Memetic reinforcements in the environment, such as comforting photos and mementos may be also helpful.

Learning self-hypnosis may also provide a sense of mastery over the dissociation susceptibility.

23.2.2 Dissociative Identity Disorder (DID, Multiple Personality Syndrome)

Recognition that we are all multiple personalities at some level may provide the basis for acceptance of the multiple personality aspects within one's brain and pave the way toward a more cooperative selfplexes (see Chapter 11). The task is how to change a culture of successive oppressive authoritarian regimes to one of tolerance for differing and contradictory ideas and democratic power sharing and peaceful regime change in the brain.

Rational discussions concerning the existence of multiple selfplexes and the phenomenon of dissociation, and the need to accept their existence as one's own may lead to a strategy of treatment. Various psychotherapeutic techniques have been used successfully in treating DID – all the successful techniques eventually result in an

"integration" of the personalities, or an acceptance of different selfplexes to reside in the brain in a more harmonious way.

Hypnosis is a useful tool in inducing a dissociative state in which tolerance for coexistence of contradictory memes might be achieved as well as in investigating the presence of selfplexes that may be latent.

Some have argued that DID is an iatrogenic disorder that develops in the course of psychotherapy by therapists who are interested in the multiple personalities (Piper and Merskey, 2004; Reinders, 2008). As we are all "multiple personalities" as discussed previously in this chapter, it stands to reason that an emphasis on the differences of the selfplexes can lead to a diagnosis of DID in many "normal" people. Nevertheless, DID patients who seek help indeed suffer from the lack of communication among the different selfplexes, and treatment is often effective. DID may also be accompanied by a more serious syndrome such as PTSD and borderline syndrome – the treatment of the more serious syndrome may also reduce the symptoms of DID.

As proneness to dissociation is associated with childhood abuse and trauma, prevention should play an important role.

23.2.3 Misattribution Syndromes: Conversion, Somatization, Hypochondriasis, Chronic Pain, Fibromyalgia, etc.

A rational discussion concerning how stress, both external and internal memetic, can cause a dysequilibrium in the brain, which may in turn result in a misattribution of the source to a body part, can help the patient to recognize the presence of stress. This can also result in a discussion concerning the physiologic arousal accompanying stress which may further contribute to the distressing symptoms. The role of helplessness and depression should also be explored and discussed.

A careful history will usually reveal the original model for misattribution – e.g., a family member who always had a headache when stressed.

Through this process, the fear that the patient has a serious medical disease can be alleviated, at the same time recognizing that the patient has "real" physically experienced distress because of the brain's processing problems. Then methods can be developed to alleviate the distress through stress management, relaxation, and other broad-spectrum anti-meme therapies (see Chapter 17) as well as specific therapies such as cognitive–behavioral therapy as indicated. Pharmacotherapy may be indicated targeting anxiety and/or depression as well as pain.

23.2.4 Psychosis and Schizophrenia

Persistent psychosis and schizophrenia are major final common pathway syndromes that require both gene- and meme-oriented therapies. Pharmacotherapy is often critical in treating the symptoms of psychosis and standard textbooks of psychiatry

should be consulted for details. Broad-spectrum meme-oriented therapies include music, art, exercise, and dance therapies. Also useful are specific therapies geared to enhance positive selfplexes as well as specific problem-solving and coping skill enhancing therapies.

Acutely psychotic patients are hypervigilant and their meme-processing apparatus is flooded with threatening and confusing memes.

In acute psychosis or acute exacerbations of schizophrenia, temporary removal from the stressful memetic environment through hospitalization may be necessary.

Psychoeducation of the patient and family concerning the nature and symptoms of schizophrenia, need for reducing stress, adherence to medications, and follow-up visits is also important.

References

Aybek, S., Kanaan, R. A., David, A. S. (2008) The neuropsychiatry of conversion disorder. *Curr Opin Psychiatry*, **21**, 275–280.

Barsky, A. J., Borus, J. F. (1999) Functional somatic syndromes. *Ann Intern Med*, **130**, 910–921.

Barsky, A. J., Goodson, J. D., Lane, R. S., et al. (1988) The amplification of somatic symptoms. *Psychosom Med*, **50**, 510–519.

Barsky, A. J., Wyshak, G. (1990) Hypochondriasis and somatosensory amplification. *Br J Psychiatry*, **157**, 404–409.

Bell, I. R., Baldwin, C. M., Russek, L. G., et al. (1998) Early life stress, negative paternal relationships, and chemical intolerance in middle-aged women: support for a neural sensitization model. *J Womens Health*, **7**, 1135–1147.

Berrios, G. E., Sierra, M. (1997) Depersonalization: a conceptual history. *Hist Psychiatry*, **8**, 213–229.

Crow, T. J. (1997) Is schizophrenia the price that Homo sapiens pays for language? *Schizophr Res*, **28**, 127–141.

Crow, T. J. (2000) Schizophrenia as the price that homo sapiens pays for language: a resolution of the central paradox in the origin of the species. *Brain Res Brain Res Rev*, **31**, 118–129.

Crow, T. J. (2007) How and why genetic linkage has not solved the problem of psychosis: review and hypothesis. *Am J Psychiatry*, **164**, 13–21.

David, N., Bewernick, B. H., Cohen, M. X., et al. (2006) Neural representations of self versus other: visual-spatial perspective taking and agency in a virtual ball-tossing game. *J Cogn Neurosci*, **18**, 898–910.

Devinsky, O., Morrell, M. J., Vogt, B. A. (1995) Contributions of anterior cingulate cortex to behaviour. *Brain*, **118**(Pt 1), 279–306.

Ehling, T., Nijenhuis, E. R., Krikke, A. P. (2008) Volume of discrete brain structures in complex dissociative disorders: preliminary findings. *Prog Brain Res*, **167**, 307–310.

Forrest, K. A. (2001) Toward an etiology of dissociative identity disorder: a neurodevelopmental approach. *Conscious Cogn*, **10**, 259–293.

Goes, F. S., Sanders, L. L., Potash, J. B. (2008) The genetics of psychotic bipolar disorder. *Curr Psychiatry Rep*, **10**, 178–189.

Iseki, K., Hanakawa, T., Shinozaki, J., et al. (2008) Neural mechanisms involved in mental imagery and observation of gait. *Neuroimage*, **41**, 1021–1031.

Kihlstrom, J. F. (2005) Dissociative disorders. *Annu Rev Clin Psychol*, **1**, 227–253.

Kim, S. E., Kim, J. W., Kim, J. J., et al. (2007) The neural mechanism of imagining facial affective expression. *Brain Res*, **1145**, 128–137.

Kymalainen, J. A., Weisman de Mamani, A. G. (2008) Expressed emotion, communication deviance, and culture in families of patients with schizophrenia: a review of the literature. *Cultur Divers Ethnic Minor Psychol*, **14**, 85–91.

Lewis, D. A., Volk, D. W., Hashimoto, T. (2004) Selective alterations in prefrontal cortical GABA neurotransmission in schizophrenia: a novel target for the treatment of working memory dysfunction. *Psychopharmacology (Berl)*, **174**, 143–150.

Lou, H. C., Nowak, M., Kjaer, T. W. (2005) The mental self. *Prog Brain Res*, **150**, 197–204.

Marom, S., Munitz, H., Jones, P. B., et al. (2005) Expressed emotion: relevance to rehospitalization in schizophrenia over 7 years. *Schizophr Bull*, **31**, 751–758.

Meyer-Lindenberg, A. S., Olsen, R. K., Kohn, P. D., et al. (2005) Regionally specific disturbance of dorsolateral prefrontal-hippocampal functional connectivity in schizophrenia. *Arch Gen Psychiatry*, **62**, 379–386.

Nickl-Jockschat, T., Rietschel, M., Kircher, T. (2008) [Correlations between risk gene variants for schizophrenia and brain structure anomalies.]. *Nervenarzt*, **80**(1), 40–42.

Piper, A., Merskey, H. (2004) The persistence of folly: critical examination of dissociative identity disorder. Part II. The defence and decline of multiple personality or dissociative identity disorder. *Can J Psychiatry*, **49**, 678–683.

Prasad, S. E., Howley, S., Murphy, K. C. (2008) Candidate genes and the behavioral phenotype in 22q11.2 deletion syndrome. *Dev Disabil Res Rev*, **14**, 26–34.

Qiu, J., Li, H., Liu, Q., et al. (2008) Brain mechanism of response execution and inhibition: an event-related potential study. *Neuroreport*, **19**, 121–125.

Raz, A., Fan, J., Posner, M. I. (2005) Hypnotic suggestion reduces conflict in the human brain. *Proc Natl Acad Sci USA*, **102**, 9978–9983.

Reinders, A. A. (2008) Cross-examining dissociative identity disorder: neuroimaging and etiology on trial. *Neurocase*, **14**, 44–53.

Revonsuo, A. (2000) The reinterpretation of dreams: an evolutionary hypothesis of the function of dreaming. *Behav Brain Sci*, **23**, 877–901, discussion 904–1121.

Romanides, A. J., Duffy, P., Kalivas, P. W. (1999) Glutamatergic and dopaminergic afferents to the prefrontal cortex regulate spatial working memory in rats. *Neuroscience*, **92**, 97–106.

Ross, S. E. (1999) "Memes" as infectious agents in psychosomatic illness. *Ann Intern Med*, **131**, 867–871.

Sar, V., Akyuz, G., Dogan, O. (2007) Prevalence of dissociative disorders among women in the general population. *Psychiatry Res*, **149**, 169–176.

Seligman, R., Kirmayer, L. J. (2008) Dissociative experience and cultural neuroscience: narrative, metaphor and mechanism. *Cult Med Psychiatry*, **32**, 31–64.

Sierra, M., Berrios, G. E. (1998) Depersonalization: neurobiological perspectives. *Biol Psychiatry*, **44**, 898–908.

Solms, M. (2000) Dreaming and REM sleep are controlled by different brain mechanisms. *Behav Brain Sci*, **23**, 843–850, discussion 904–1121.

Sullivan, P. F. (2008) Schizophrenia genetics: the search for a hard lead. *Curr Opin Psychiatry*, **21**, 157–160.

van Haren, N. E., Bakker, S. C., Kahn, R. S. (2008) Genes and structural brain imaging in schizophrenia. *Curr Opin Psychiatry*, **21**, 161–167.

Weinberger, D. R. (2005) Genetic mechanisms of psychosis: in vivo and postmortem genomics. *Clin Ther*, **27 Suppl A**, S8–S15.