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Compensatory psychiatric comorbidity: Freud (and others) remembered

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Jakovljevic and Crnčevic (2012) review the concept(s) of comorbidity in relation to mental disorders, which is timely. Yet they seem to ignore a longstanding and important notion of comorbidity, highlighted in psychiatry particularly by Sigmund Freud.

The ignored notion is that of compensatory (psychiatric and other) comorbidity. Compensatory comorbidity is a special case of compensatory phenomena in relation to disrupted health. An example may be auto-immunity, which causes morbidity due to a misdirected attempt of the immune system to fight a primary – infectious – disorder (in some cases, the infectious agent is long gone, in which case the auto-immune disorder becomes a primary disorder rather than a comorbidity). Another phenomenon that has for long been considered compensatory is the hyper-developed hearing of many blind people, albeit there is still some doubt regarding to what extent this phenomenon is compensatory (Pavani and Bottari, 2012). A standard – although apparently not yet fully confirmed (Ibid) – explanation for this phenomenon is that in some blind people parts of the brain cortex, such as the hearing-associated temporal cortex, take over from the vision-relevant part of the brain cortex. This is an explanation from brain neuroplasticity.

Although brain neuroplasticity is a relatively new discovery, researchers have used the notion of mental compensation for long. In particular, one of Sigmund Freud's most important general discoveries or innovations is that psychopathology can be a result of a defense mechanism gone awry (Fried and Agassi, 1983) rather than necessarily a result of the primary insult (such as the external trauma or internal drive which that defense mechanism addresses). That is, Freud viewed some if not most or all mental disorders as compensatory comorbidity (recognizing that the primary insult may not result by itself in overt disturbance, in which case the consequence is compensatory morbidity rather than compensatory comorbidity). Still, there was and still is no known neural correlate to these claims of Freud. How does this relate to neuroplasticity?

Spitzer demonstrated that the longer lasting a delusion, the larger the cortical brain area associated with it (Spitzer, 1995). This may be a prime example of neuroplastic – compensatory – mental (co)morbidity. But such compensation is maladaptive, as was that addressed by Freud, and therefore it is associated with (co)morbidity, in contrast to the adaptive neuroplasticity associated with some blind people's hyper-developed hearing. Is there adaptive mental and/or brain compensation in relation to mental disorders? I suspect there is, e.g., in relation to processes of recovery (Rudnick, 2008), although it is as yet uncharted research territory. If such adaptive compensation is found, knowledge of it may spur new interventions to facilitate recovery of people with mental disorders, e.g., perhaps enhancing such adaptive compensation using various – biological, psychological and/or social – interventions.

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