

'DEPRESSION': ONE DISORDER OR MANY?

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ABSTRACT

The article argues that depression is not a single disease but a cluster of syndromes. Consequently, these syndromes cannot and should not all be treated in the same way. To elaborate, four perspectives are briefly considered: psychopharmacology, psychodynamics, existentialism, and affect theory.

Depression, Seligman (1973) has famously said, is the common cold of psychiatry in that it has become so prevalent. But what if depression is like the common cold in a more fundamental way? This article makes the case that depression is not a single disease but a cluster of syndromes.

The common cold virus is defined by *Medilexicon* as: "any of the numerous strains of virus etiologically associated with the common cold, chiefly the rhinoviruses, but also strains of adenovirus, coxsackievirus, echovirus, and parainfluenza virus."

Any of the numerous strains of...associated with...chiefly...but also strains of:
this is the epitome of non-specificity.

Consider the DSM-IV criteria for major depression (American Psychiatric Association, 2000). The disorder is characterized by the presence of the majority of these symptoms nearly every day (at least five of the symptoms to be present, including at least one for the first two):

- depressed mood
- markedly diminished interest or pleasure in all, or almost all, activities
- significant weight loss when not dieting or weight gain, or decrease or increase in appetite
- insomnia or hypersomnia
- psychomotor agitation or retardation
- fatigue or loss of energy
- feelings of worthlessness or excessive or inappropriate guilt
- diminished ability to think or concentrate, or indecisiveness
- recurrent thoughts of death (not just fear of dying), suicidality

This is a grab-bag of mood, affects, physiological change, behaviours, feelings, and thoughts. What, if anything, is common? The biological scientist Sarpolsky: “If I had to define major depression in one sentence I would say it’s a biochemical disorder with a genetic component and early experience influences, where somebody can’t appreciate sunsets (2009)”.

My proposal is that, like the common cold, ‘depression’² is not a single syndrome, but rather a loose grouping or cluster of syndromes, some with some overlapping symptoms. And if that is the case, this must surely have consequences for treatment. As the topic is enormous I have chosen to consider it from four very different perspectives.

PSYCHOPHARMACOLOGY

The advent of anti-depressants has not solved the problem. Some depressive symptoms are relieved and not others (Tranter, O'Donovan, Chandarana, & Kennedy, 2002), thus requiring augmentation with other medications (Trivedi, Rush & Wisniewski, 2006). The same symptoms are relieved for one client but not for another (Ruhe, Huyser, Swinkels, & Schene, 2006). Symptoms for which the client had not sought treatment are relieved as well as symptoms not regarded as depressive at all—from anxiety, obsessive disorder (van Nieuwerburgh, Deforce, & Denys, 2008) to dysmenorrhea and snoring (Wulff, 2011). A famous recent review established that anti-depressants have a success rate only 2% better than placebo (Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008).

In his famous lecture on depression Sarpolsky (1994) proposes three features of depression, each to do with an absence of a different neurotransmitter. Anhedonia is to do with a lack of dopamine; psychomotor retardation is to do with a lack of norepinephrine; and grief/guilt is to do with a lack of serotonin.

² Having made my point about the depression not being a so-called entity, and in order to avoid being tiresome, I will avoid continuing to write the word within quotation marks.

For his part, Nathanson (1994) suggests that SSRIs (selective serotonin reuptake inhibitors) repair defects in shame biology (Dickerson, Gruenewald, & Kemeny, 2004); phenothiazines address fear-terror, tricyclics deal with the fusion of shame and fear known as guilt.

PSYCHODYNAMICS

The psychoanalyst McWilliams (2010) describes two types of depression: self-critical depression and the empty self. Each requires a different therapeutic approach. The person who feels empty does well when encouraged; the self-critical one doesn't. If the therapist says, "Its noteworthy how well you're doing considering how much you have had to deal with", the empty person will be bolstered, but the self-critical person will feel misunderstood. He or she needs to have their inner badness acknowledged: for instance, "It is true that you can sometimes be an X as you say, but perhaps not that much more of an X than many of us". Conversely, this approach will hurt and deflate the empty self.

McWilliams makes it clear that this is not a matter of there being two approaches which the therapist may choose from as he or she pleases. Using the wrong method will have negative therapeutic outcomes; each approach is designed to suit one type of depression and is contraindicated for the other.

EXISTENTIALISM

Existential angst (anxiety/depression) arises when a person is confronted with certain basic problems of existence. Yalom (1980) describes four such "ultimate concerns": death, freedom, isolation, and meaninglessness. Death is inevitable. Freedom means that we are free to act or not act, and to claim otherwise is bad faith. Isolation recognizes that each of us is alone in the universe. Meaninglessness stems from the first three. What

meaning does life have if we must die, if we construct our own world, and if each of us is ultimately alone?

Clearly the nature of the person's angst will depend on the existential issue at issue, and the focus of the therapy must be adapted accordingly. This is saying more than each therapy must be adapted for the individual; it is saying that there are several (but not a multitude) forms of suffering which we commonly think of as depression but—just like the 'bad back'—are more different than they are the same and require different treatment.

AFFECT THEORY

Affect Theory is the systematic study affects and their physical responses into nine basic categories. The Affect Theorist Nathanson says that depression is "a collective noun, a wastebasket into which are thrown a wide range of dysphorias" (1994, p. 10). Instead, there are "six families of bad mood", any of which experienced steadily will be naively called 'depression':

- persistent distress-anguish (often called *sadness*)
- persistent fear-terror (*steady anxiety*)
- persistent anger-rage
 - when mild it is *irritability* or *annoyance*
 - when higher it is *bad temper*
- persistent shame-humiliation
 - *at withdrawal pole is loneliness, hurt feelings, and bad thoughts about the self*
 - admixed with fear terror it is called *guilt*

These must not be treated in the same way. Persistent shame, for example, is treated by considering impediments to interest-excitement and enjoyment-joy. Persistent distress, on the other hand, is treated by reducing the steady-state stimulus load. He

argues for a replacement terminology: for example, Persistent Dysphoria, fear-terror with secondary distress-anguish, somatic precipitant; or Persistent Dysphoria, inhibition of interest- excitement, cause unknown, possible biological shame syndrome (Nathanson, 1994, p. 10).

CONCLUSION

This article has approached depression from four different perspectives to build up a case that it is several disorders, not one. What people diagnosed as depressed people have in common is this—they are unhappy. However, following Tolstoy it might be useful to think that each unhappy person is unhappy in his or her own way³, or at least in many possible ways.

QUESTIONS RAISED BY THE TOPIC

Which treatment modality is best for depression? It depends on the variant of depression. Once one knows that, certain treatments suggest themselves (and others do not). Also, as Grawe (2007) has suggested, it may well be that the therapist decides to order one's interventions depending on what the client's brain is ready for at that time. Neurology has much to teach us about which areas of the brain function below par in which situations—it makes no sense to prematurely employ psychological treatments that depend on a relatively high level of functioning (Doidge, 2007).

PROGRESSING THE TOPIC

I am attempting to produce a model that accounts for the neurobiological, self, affective, and existential dimensions of what is called 'depression'. The task, of course, is to make it

³ The famous opening line of *Anna Karenina*: "All happy families are alike; each unhappy family is unhappy in its own way" (Tolstoy, 1878, p. 1).

complex enough to encompass the problem but simple enough for the practitioner to use.

REFERENCES

- American Psychiatric Association (2000) *Diagnostic and statistical manual of mental disorders: DSM-IV-TR*. Washington, DC: American Psychiatric Association.
- Dickerson, S. S, Gruenewald, T. L., & Kemeny, M. E. (2004). When the social self is threatened: shame, physiology, and health. *Journal of Personality*, 72(6), 1191-1216. doi:10.1111/j.1467-6494.2004.00295.x.
- Doidge, N. (2007). *The brain that changes itself*. Melbourne, Australia: Scribe.
- Grawe, K. (2007). *Neuropsychotherapy: How the neurosciences inform effective psychotherapy*. Mahwah, NJ: Lawrence Erlbaum Associates.
- McWilliams, N. (2010). *Individuality: Its dimensions and implications for therapy*. Paper presented at Auckland University of Technology, Auckland, New Zealand.
- Nathanson, D. (1994). The case against depression. *Bulletin of the Tomkins Institute*, 1, 9-11.
- Ruhe, H. G., Huyser, J., Swinkels, J. A., & Schene, A. H. (2006). Switching antidepressants after a first selective serotonin reuptakeinhibitor in major depressive disorder: a systematic review. *Journal of Clinical Psychiatry*, 67(12), 1836-1855. doi:10.4088/JCP.v67n1203.
- Sarpolsky, R. (2009). *Depression in U.S. Lecture Stanford University* [Video file]. Retrieved from <http://www.youtube.com/watch?v=NOAgplgTxfc>.
- Seligman, M. E. P. (1973). Fall into helplessness. *Psychology Today*, 7, 43-48. doi: 10.1111/j.1542-734X.1990.i302_109.x.
- Tranter, R., O'Donovan, C., Chandarana, P., & Kennedy, S. (2002). Prevalence and outcome of partial remission in depression. *Journal of Psychiatry & Neuroscience*, 27(4), 241-247.
- Tolstoy, L. (1878). *Anna Karenina*. Harmondsworth, England: Penguin Classics.
- Trivedi, M. H., Rush, A. J., & Wisniewski, S. R. (2006). Evaluation of outcomes with citalopram for depression using measurement-based care in STAR*D: implications for clinical practice. *The American Journal of Psychiatry*, 163(1), 28-40. doi:10.1176/appi.ajp.163.1.28.

- Turner, E. H., Matthews, A. M., Linardatos, E., Tell, R. A., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. *New England Journal of Medicine*, 358(3), 252-60. doi:10.1056/NEJMsao65779.
- van Nieuwerburgh, F., Deforce, D. & Denys, D. A. J. P. (2008). Serotonin related genes affect antidepressant treatment in obsessive-compulsive disorder. *Progress in Neurotherapeutics and Neuropsychopharmacology*, 3(1), 227-240, doi:10.1136/Si748232io7000146.
- Wulff, K. (2011). Circadian rhythms and their disorders. *Journal of Neurological Neurosurgery and Psychiatry*, 82, 2-3. doi:10.1136/jnnp-2011-300504.4.
- Yalom, I. D. (1980). *Existential psychotherapy*. New York: Basic.

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