

DANDRITE Lecture

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13:30 - 14:30

Building 1162, room 013



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A Neuroscientist Evaluates the Standard Biological Model of Depression

Neuroscientists widely hypothesize that "depression" arises from a brain disorder caused by some defect in a specific neural pathway. If so, we might identify and localize the defect, and then develop a rational therapy. However, recent evidence from multiple sources fails to support this hypothesis: (1) Neuroimaging does not identify brain abnormalities in depressed individuals; neuroimaging does not even distinguish between large populations of depressed vs healthy. (2) Genome-wide association studies identify hundreds of variants of small effect, but these do not identify a depressed individual, nor even a depressed population. (3) The "chemical imbalance" theory of depression has failed for want of evidence, thus depriving "antidepressant" drugs of a neuroscientific rationale. Perhaps unsurprisingly then, new analyses of clinical trials indicate rough parity for most participants between drug and placebo. (4) Depression, while weakly predicted by any "biomarker," is strongly predicted by childhood trauma and chronic social stress. Furthermore, depression is significantly reduced by physical repairs to the community (housing facades and vacant lots) and by psychological repairs through sharing experience of trauma and abuse. Thus, depression-given its lack of any reliable biomarker, its diverse and shape-shifting symptoms, its transience on the scale of human lifetime, and its positive response to renewed hope-would be most fairly characterized as a distressing psychological disturbance rather than as a brain disease or disorder.

Host: Sadegh Nabavi