



Letter to the Editor

Social navigation hypothesis of depression revisited

Dear Editor,

In our view, Daniel Nettle (JAD 81, 2004) tends to misinterpret the adaptationist social navigation hypothesis (SNH) for Major Depressive Disorder (MDD) proposed by Hagen (1999, 2002) and Watson and Andrews (JAD 72, 2002).

Others have hypothesized that depression is just an evolved signal of social need (e.g., Henderson, 1974). This is implausible because, as Nettle points out, depression elicits negative reactions in social partners (Segrin and Dillard, 1992), the opposite reaction expected of a simple signal of need. Emotions and behaviors, like sadness, grief, and crying, serve as adequate signals of need when there are few conflicts of interest between signal senders and receivers.

We argue that MDD functions to *credibly* signal need and, crucially, to *compel* support (1) when there is a high degree of mutual dependence in social groups, and (2) when there are conflicts of interest that hinder social support. In the small communities that characterized our evolutionary history, group members depended on one another for their mutual well-being. MDD symptoms, like loss of interest in virtually all activities and suicidality, put the well-being of all group members at risk. Individuals who were reluctant to help a fellow group member had little choice but to respond to a depressed and/or suicidal individual upon whom their own well being depended. There is evidence from small-scale societies that depression and particularly suicidality might serve such a social function (Hagen, 2003).

It is therefore a prediction of the SNH, not evidence against it, that (1) negative life events will not cause

MDD in all individuals. Such events will only cause MDD in those who need assistance *and* who have serious conflicts with important social partners. It is also a prediction of the SNH, not evidence against it, that MDD will often cause negative reactions in others because MDD serves as a strategy to manipulate them by withholding benefits.

There is also evidence that in the context of social conflict and low support, depressive symptoms elicit social benefits *despite* the negative reaction they cause in others. This occurs often enough that researchers worry these benefits reinforce depression (e.g., Sheeber et al., 2001).

Nettle is right when he states that significant heritability in the capacity for MDD would be strong evidence against any adaptationist hypothesis. However, once genetic control of exposure to environments that predispose to MDD is taken into account, the direct heritability of MDD is quite modest (e.g., Kendler et al., 2002). This is consistent with normal heritable variation in the triggering thresholds of a complex, multigene adaptation. There is little evidence that those who never suffer MDD are genetically incapable of it. Hagen (2003) addresses many of Nettle's other criticisms of the SNH.

Although far from proven, the SNH is quite consistent with the current evidence on MDD.

References

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