A Proposed Mechanism for the Induction of Bone Loss as a Function of Chronic Envy

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ABSTRACT

The dorsal anterior cingulate cortex (dACC) has been shown to be uniquely activated when people engage in envy. Similarly, dACC is the major component of a proposed adversity processing circuit that predicts risks to prevent destructive behaviors and inhibit release of dopamine and serotonin, which play a significant role in skeletal health. A causal relationship may therefore exist between chronic envy and the induction of lowered bone mineral density (BMD) as a result of dACC activation.

A significant body of research shows that depression may induce bone loss, which has resulted in calls for it to be recognized as a contributing factor for lowered bone mineral density (BMD) and even the more advanced condition of osteoporosis. For example, rodents subjected to chronic mild stress with an experimentally established model of depression lead researchers to conclude that depression induces bone loss through stimulation of the sympathetic nervous system, associated with increases in skeletal epinephrine and serum corticosterone levels (1).

This raises the intriguing questions of whether other mood states may have a similar effect. Statistically reliable assessment instruments - Dispositional Envy Scale, Beck Depression Inventory, and Center for Epidemiological Studies Depression Scale – consistently showed a significant correlation between measures of envy and depression, $p < .001$ (2). Can envy play a role in the induction of bone loss as well? The dorsal anterior cingulated cortex (dACC) has been shown to be uniquely activated when humans exhibit envy (3). This "envy center" is also the warning generating component of a proposed adversity processing circuit that evaluates and predicts risks to stop us from making destructive choices. This circuit, along with dACC directly, may activate the lateral habenula (LHb) (4), where efferent projections to the midbrain and brainstem inhibit, rather than excite, release of dopamine and serotonin.

Deletion of the gene for the dopamine transporter protein (DAT), which is responsible for the transfer and rapid uptake of dopamine, resulted in skeletal deformities and reduced bone mass in mice (5). And serotonin increases, via brain-derived serotonin (BDS), bone mass accrual while limiting bone resorption through the sympathetic nervous system (6). This is significant because with age the resorption rate exceeds the rate of replacement by new bone growth which may lead to osteoporosis. It logically follows that BDS suppression, in sufficient quantities, can inhibit the growth of new bone tissue and skeletal health.

Persons exhibiting chronic, debilitating envy may well be tempted to engage in risky and destructive behaviors. The dACC would be activated by envy while stimulating the adversity processing center and LHb into action, resulting in lowered dopamine and serotonin release and a causal relationship between envy and reduced bone mass. Irrespective of the merits or shortcomings of the model proposed here, the role of envy and possibly other affective states in influencing skeletal health warrants further investigation.

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