Fibromyalgia patients show an abnormal dopamine response to pain.


Abstract

Fibromyalgia is characterized by chronic widespread pain and bodily tenderness and is often accompanied by affective disturbances. Accumulating evidence indicates that fibromyalgia may involve a dysfunction of modulatory systems in the brain. While brain dopamine is best known for its role in pleasure, motivation and motor control, recent evidence suggests that it is also involved in pain modulation. Because dopamine is implicated in both pain modulation and affective processing, we hypothesized that fibromyalgia may involve a disturbance of dopaminergic neurotransmission. Fibromyalgia patients and matched healthy control subjects were subjected to deep muscle pain produced by injection of hypertonic saline into the anterior tibialis muscle. In order to determine the endogenous release of dopamine in response to painful stimulation, we used positron emission tomography to examine binding of [(11)C]-raclopride (D2/D3 ligand) in the brain during injection of painful hypertonic saline and nonpainful normal saline. Fibromyalgia patients experienced the hypertonic saline as more painful than healthy control subjects. Control subjects released dopamine in the basal ganglia during the painful stimulation, whereas fibromyalgia patients did not. In control subjects, the amount of dopamine release correlated with the amount of perceived pain but in fibromyalgia patients no such correlation was observed. These findings provide the first direct evidence that fibromyalgia patients have an abnormal dopamine response to pain. The disrupted dopaminergic reactivity in fibromyalgia patients could be a critical factor underlying the widespread pain and discomfort in fibromyalgia and suggests that the therapeutic effects of dopaminergic treatments for this intractable disorder should be explored.

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