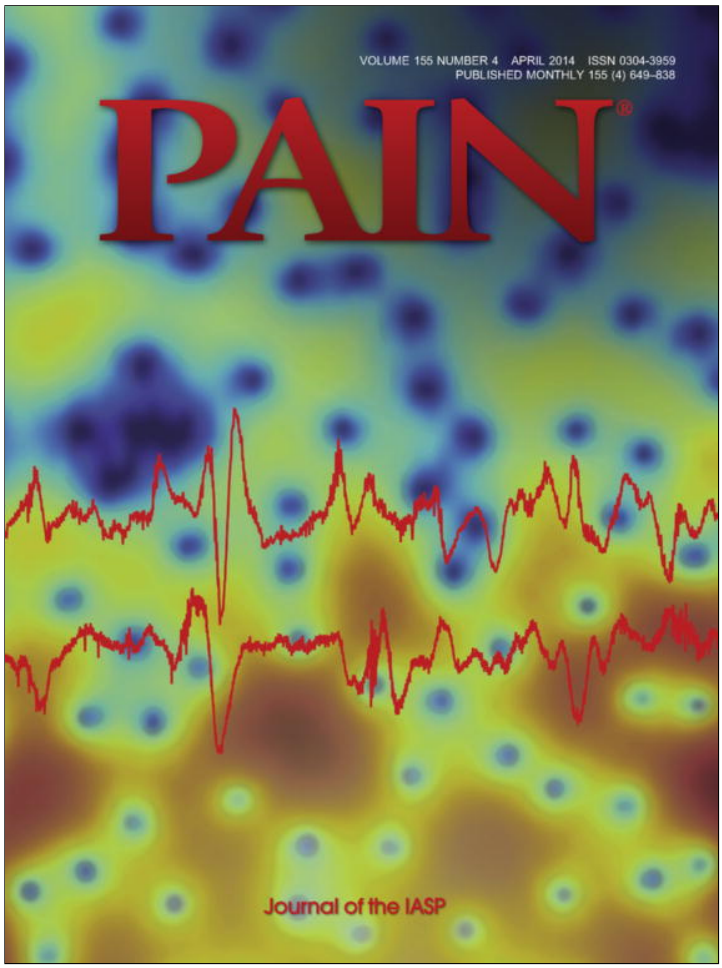

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Commentary

How does pain affect eating and food pleasure?

Sufferers of persistent pain have increased risk for many other health problems, including obesity. The mechanisms have not been well understood, however. In this issue, Geha et al. ask why chronic pain and obesity are so closely linked [8]. Drawing on the rich neuroscience literature which implicates the ventral striatum and medial prefrontal cortex (mPFC) in determining food pleasure [5], the authors point to evidence that chronic pain disrupts the normal structure and function of this circuitry. For instance, relief-related ventral striatal responses to offset of phasic pain were absent in patients with chronic back pain [1]. These patients also showed abnormal ventral striatal opioid signalling during pain [15], and functional connectivity between the ventral striatum and mPFC appears to play a role in the transition from acute to chronic pain [2].

Geha and colleagues tested 18 patients with chronic lower back pain (CLBP) and compared their responses to sweet and fatty foods with a matched control group. The average body mass index for each group indicated moderate overweight. Patients with CLBP showed a selective decrease in pleasantness ratings of fatty puddings. Importantly, they also displayed a disconnection between calories ingested during ad libitum food consumption and ratings of hunger and food pleasantness. That is, unlike for controls, there was no relationship between how much they liked the food and how much they ate. Strikingly, eating more also failed to predict a larger drop in hunger in this group.

Geha et al. propose that pain-related hedonic blunting (a reduced ability to experience pleasure) caused by disruptions in VS-mPFC brain circuitry may link chronic pain to obesity. This neuroscientific explanation is compelling, and future studies should address how pain affects the neural mechanisms of hedonic eating and relief of hunger.

The link between pain and pleasure is complex, however. One early study of patients with chronic facial or back pain suggested that anhedonia was more closely related to the patients' levels of depression rather than to the experience of pain itself [14]. Other conditions that can blunt pleasure in some domains, such as substance abuse (which also disrupts the ventral striatum-mPFC circuitry and opioid signalling), have been linked to increased liking of sweet tastes [9,10].

Pain has also been shown to enhance directly the enjoyment of sweet foods. After undergoing the cold-pressor test, healthy volunteers reported greater pleasure from eating chocolate [3]. In fact, pain offset appeared to increase sensitivity to a range of gustatory inputs [3]. This increased food enjoyment after pain offset may be related to a feeling of relief and is unlikely to occur in patients who suffer from persistent pain. In healthy humans, eating and drinking are often associated not just with pleasure but also with the emotion of relief (eg, the first coffee of the day, eating after a prolonged fast) [7]. The ventral striatum and medial prefrontal cortices which are so important for signalling food pleasure also appear to signal relief from physical pain [2] or threat of pain [13]. When persistent pain disrupts the function of this neurocircuitry, it may therefore also preclude the encoding of relief from hunger.

Pain can also affect food consumption in ways that are unrelated or only indirectly related to pleasure. Possible mechanisms range from a disrupted interpretation of physiological signals such as hunger, leading to food consumption that is determined largely by external signals or eating due to emotions other than hunger such as sadness (emotional eating) [6].

The psychological meaning given to pain can also play a role. The experience of pain and suffering is closely tied to perceptions of justice. Perceived injustice (ie, Why should I suffer more than others?) is associated with problematic pain outcomes, such as prolonged disability and mental illness [16,17]. In experimental studies, pain perceived as being unfair significantly increased how many chocolates participants chose to eat [4]. In contrast, pain that seemed to be justified did not affect indulgence levels. Bastian et al. (2012) argued that unfair pain can induce feelings of entitlement and thereby increase indulgence in "guilty pleasures." A similar mechanism could explain the reduced drop in hunger after ad libitum in CLBP patients reported by Geha et al.: "I deserve more pudding because I am in pain." These patients may be responding to signals that are not directly related to hunger but instead to reestablishing a sense of psychological equilibrium and personal justice.

Finally, it is important to consider how evolved responses to pain may determine food-consumption behavior. Increased calorie consumption can be viewed as a rational response to pain, ie, amassing resources to deal with future pain and adversity [12]. However, the ready availability of rewarding and high-calorie foods in large parts of the world today renders this response maladaptive. Obesity increases the risk for numerous health problems, only one of which is chronic pain.

Pain and pleasure affect common brain circuits, including the ventral striatum and mPFC. Nevertheless, pain and pleasure are best understood within a framework that does not aim to conceptualize them as opposite experiences. As with other complex subjective experiences [eg, 11], it is possible to feel both pain and pleasure at the same time. Moreover, pain may both increase and reduce pleasure. Geha et al. make a convincing case for disrupted

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food hedonics as a mechanism linking pain with obesity, highlighting the study of pain, gustation, and eating as a fruitful area for future research.

Conflict of interest statement

The authors declare no conflict of interest.

References

[1] Baliki MN, Geha PY, Fields HL, Apkarian AV. Predicting value of pain and analgesia: nucleus accumbens response to noxious stimuli changes in the presence of chronic pain. Neuron 2010;66:149–60.

[2] Baliki MN, Petre B, Torbey S, Herrmann KM, Huang L, Schnitzer TJ, Fields HL, Apkarian AV. Corticostriatal functional connectivity predicts transition to chronic back pain. Nat Neurosci 2012;15:1117–9.

[3] Bastian B, Jetten J, Hornsey MJ. Gustatory pleasure and pain: the offset of acute physical pain enhances responsiveness to taste. Appetite 2014;72:150–5.

[4] Bastian B, Jetten J, Stewart E. Physical pain and guilty pleasures. Soc Psycholog Personal Sci 2012.

[5] Berridge KC, Kringelbach ML. Neuroscience of affect: brain mechanisms of pleasure and displeasure. Curr Opin Neurobiol 2013;23:294–303.

[6] Canetti L, Bachar E, Berry EM. Food and emotion. Behav Proc 2002;60:157–64.

[7] Desmet PMA, Schifferstein HNJ. Sources of positive and negative emotions in food experience. Appetite 2008;50:290–301.

[8] Geha P, deAraujo I, Green B, Small DM. Decreased food pleasure and disrupted satiety signals in chronic low back pain. PAIN® 2014;155:712–22.

[9] Green A, Kaul A, O'Shea J, Sharma E, Bennett L, Mullings EL, Munafo MR, Nutt DJ, Melichar JK, Donaldson LF. Opiate agonists and antagonists modulate taste perception in opiate-maintained and recently detoxified subjects. J Psychopharmacol 2013;27:265–75.

[10] Kampov-Polevoy AB, Garbutt JC, Davis CE, Janowsky DS. Preference for higher sugar concentrations and Tridimensional Personality Questionnaire scores in alcoholic and nonalcoholic men. Alcohol Clin Exper Res 1998;22:610–4.

[11] Larsen JT, Peter McGraw A, Mellers BA, Cacioppo JT. The agony of victory and thrill of defeat. Psychol Sci 2004;15:325–30.

[12] Leknes S, Bastian B. The benefits of pain. Rev Philos Psychol, in press. [SI: Pain and Pleasure]. http://dx.doi.org/10.1007/s13164-014-0178-3.

[13] Leknes S, Lee M, Berna C, Andersson J, Tracey I. Relief as a reward: hedonic and neural responses to safety from pain. PLoS ONE 2011;6:e17870.

[14] Marbach JJ, Richlin DM, Lipton JA. Illness behavior, depression and anhedonia in myofascial face and back pain patients. Psychother Psychosomat 1983;39:47–54.

[15] Martikainen IK, Peciña M, Love TM, Nuechterlein EB, Cummiford CM, Green CR, Harris RE, Stohler CS, Zubieta J-K. Alterations in endogenous opioid functional measures in chronic back pain. J Neurosci 2013;33:14729–37.

[16] McParland JL, Eccleston C. "It's not fair": social justice appraisals in the context of chronic pain. Curr Dir Psychol Sci 2013;22:484–9.

[17] Sullivan MJ, Scott W, Trost Z. Perceived injustice: a risk factor for problematic pain outcomes. Clin J Pain 2012;28:484–8.

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