Ruprecht-Karls-Universität Heidelberg
Medizinische Fakultät Mannheim
Dissertations-Kurzfassung

Placebo Analgesic Responses and the Role of Treatment History in Patients with Chronic Pain

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Placebo analgesia is the reduction in pain following administration of an inert treatment. In healthy humans, it is induced by expectation of pain relief and modulated by learning and prior treatment experiences. Research into placebo analgesia in chronic pain patients is sparse, although its underlying psychological and neurobiological mechanisms may differ from healthy humans. This dissertation project investigated how treatment expectation, learning and treatment history modulate placebo analgesia in chronic pain patients.

In the first study, a pharmacological placebo intervention was administered in a sample of chronic musculoskeletal pain patients (n=49) in a clinical treatment setting. Analyses were based on placebo responders. We found that the verbal information of a potent pain relieving effect of the intervention resulted in substantial placebo responses to both acute experimental and clinical pain. An additional conditioning procedure did not enhance placebo analgesia. Treatment history modulated the magnitude of the placebo response to clinical pain: patients with a more negative treatment history reported significantly larger pain relief in chronic pain after our placebo intervention. The placebo responses to acute and to chronic pain were not significantly related.

In the second study, the pharmacological placebo intervention was applied in a sample of 31 chronic back pain patients (CBP) on two subsequent occasions one week apart. A healthy control group (HC) was added (n=31). The neurobiological underpinnings were examined by functional magnetic resonance imaging. We observed that the verbal information of potent pain relief induced strong placebo analgesia in CBP and HC. Responses were not enhanced by conditioning. In CBP, placebo responses to acute and to chronic pain were again not related. A positive treatment history and a higher expectancy of pain relief predicted the placebo response to acute experimental pain in CBP on the first placebo application. Responses at the repeated intervention and to chronic pain were predicted by low anxiety in CBP. The placebo response to acute experimental pain entailed enhanced neural modulation within the perigenual anterior cingulate, amygdala and hippocampus in CBP. Treatment history modulated neural activation in CBP.

We could show that expectancy-induced placebo analgesic responses to acute and to clinical pain are strong in chronic pain patients. Different mechanisms may underlie the placebo responses to acute and to chronic pain. Treatment history modulates placebo analgesia in chronic pain patients. Our findings highlight that placebo responses and treatment history are highly relevant in the treatment of chronic pain. Further research into the psychological and neurobiological underpinnings of placebo analgesia in chronic pain patients is necessary as findings from research in healthy humans may not easily be transferable to chronic pain populations.