



## The Placebo Effect

The Placebo Effect is defined by Oxford Dictionary as, “a beneficial effect produced by a placebo drug or treatment, which cannot be attributed to the properties of the placebo itself, and must therefore be due to the patient's belief in that treatment”. Usage of the placebo effect is rarely heard in pharmaceutical settings until recently. Many scientists have found that using the placebo effect can “mimic the action of active treatments” and “facilitate the activation of pain and nonpain control systems” in our bodies (Colloca, n.d.). Researcher Markus Rütgen theorizes the body can be its own painkiller through the general understanding of the placebo effect. In theory, the body will exhibit the same pain relief characteristics as a painkiller, without the actual use of a painkiller. Delving deeper into the power of placebo painkillers will further substantiate the hypothesis, “empathy for pain is partially grounded in first-hand pain by suggesting that this also applies to the underlying opioidergic neurochemical processes” (Rütgen, 2018).



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## The Study's Set-Up

Researchers in Rutgen's study split people into two equivalent groups: those who were given a placebo painkiller (labeled “placebo” group) and those who weren't (labeled “control” group). After the placebo group took the fake painkiller, researchers proceeded to zap the hands of both groups. The control and placebo groups were then surveyed on the inflicted pain.

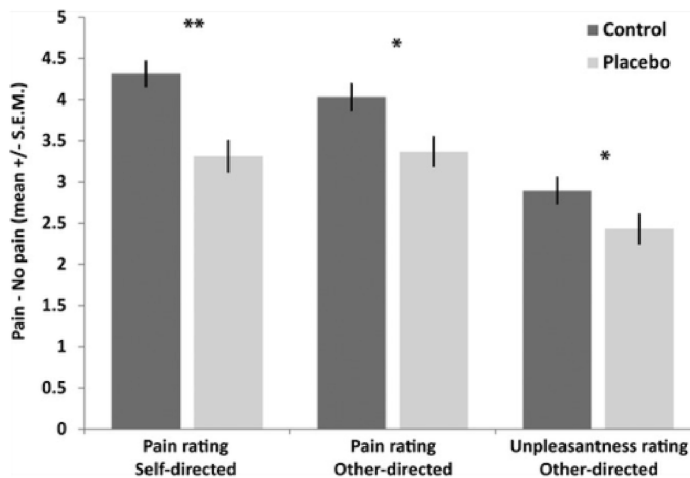


Figure 1. “Self-report results in the control (n = 53) and placebo group (n = 49), for ratings of self-directed pain (“how painful was this stimulus for you?”), other-directed pain (“how painful was this stimulus for the other person?”), and self-experienced negative affect (unpleasantness) when witnessing other-directed pain (“how unpleasant did it feel when the other person was stimulated?”).

Asterisks (\*P < 0.05, \*\*P < 0.01) mark significant planned comparisons (independent samples t tests) of the main hypothesis that placebo analgesia reduced both empathy for pain and its first-hand experience” (Rütgen, 2015).

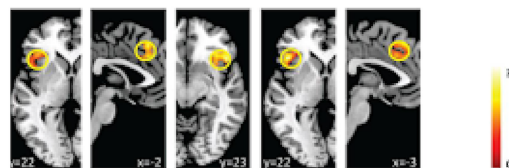


Figure 2. “Activation maps displaying the spatial distribution of brain activity within the ROIs (taken from a two-sample t test contrasting the two groups, for the contrast pain > no pain, separately for self- and other-directed conditions). The yellow circles mark the ROI sphere used to extract the mean activation. Note that these maps are shown for illustration purposes only (and for this reason are thresholded at P = 0.05 uncorrected) and that they are not independent of the ROI results” (Rütgen, 2015).

## Self-Reported Pain During Zapping

Generally, the placebo group reported feeling less pain after taking the placebo painkiller compared to the control group (Rütgen, 2015). Researchers proceed to run an MRI scan on the subject's heads. Within the MRI scan, scientists found reduced activity in the anterior insular cortex and the midcingulate cortex, which are responsible for emotional cognitive processes, control, and decision making (Sawa, 2017). What's more interesting is that the MRI scans show how there is more activity in the insular and midcingulate cortex from the placebo group than the control group (Rütgen, 2015). It can be inferred that the placebo group illustrates more brain activity is needed to create the pain relief effect onto the body than the control group. Without a catalyst, such as a painkiller, to be active in the body, the body needs to use more energy to create a similar effect. Therefore, if the mind can be tricked into thinking the body has taken painkillers, it will endure the same effects of pain relief.



So, what does this mean? The patients who took placebo were able to activate the opioid receptors of the brain by simply believing they took a strong painkiller. This reinforces the placebo effect and how the human brain can be tricked into reducing pain itself.

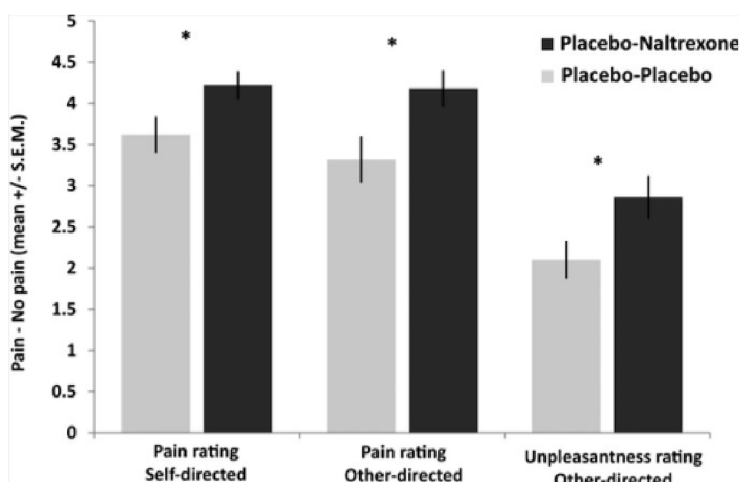


Figure 3. “Self-reported affect ratings of the psychopharmacological experiment in the placebo-placebo (n = 25) and the placebo-naltrexone group (n = 25), for the different types of ratings (self-directed pain, other-directed pain, and unpleasantness in response to other-directed pain). Asterisks (\*P < 0.05) mark significant planned comparisons (independent samples t tests) of the main hypothesis that naltrexone reduced the effects of placebo analgesia for both empathy for pain and its first-hand experience” (Rütgen, 2015).

## Future Research and Implications

So how will placebo painkillers contribute to future studies and, moreover, what does implicating placebo painkillers look like for ongoing clinical methods? While the use of placebo painkillers is still a work in progress, further progressions in the pharmaceutical industry can be made to strengthen drug development. Rütgen and his researchers even ran a final experiment a few years later where they gave the patients an actual painkiller. In this case, Naltrexone was used, “...to help narcotic dependents who have stopped taking narcotics to stay drug-free” (Mayo Clinic, 2023). Using this information, blah blah blah transition, “Self-report showed that blocking opioid receptors after the induction of placebo analgesia increased both first-hand pain and empathy for pain, replicating previous findings” (Rütgen, 2018). After taking naltrexone, researchers found that the opioid blocked the patients’ newly initiated opioid receptors, not only neutralizing the effects of the placebo painkiller but also verifying the effects of the placebo painkiller mimicking the effects of an actual painkiller. By continuing research on what is already known about placebo painkillers, a new realm of pharmaceuticals can be explored and utilized in medical practices today.

## References

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