Ask Stanford Med: Neuroscientist responds to questions on pain and love’s analgesic effects

Stanford neuroscientist Sean Mackey, MD, PhD, recently took questions about pain research and the analgesic effects of love as part of our ongoing Ask Stanford Med feature. Mackey began his responses with a message for everyone who submitted questions: “I would like to first complement the participants for their interest and their insightful questions. There is clearly a strong interest out there in pain – a condition that affects us all without discrimination.”

Below he responds to a selection of questions submitted using the hashtag #AskSUMed, an @reply to the @SUMedicine feed or the comments section on Scope.

**Nandini** asks: Does romantic love actually work as a painkiller? Or, does it just distract the person from feeling pain?

Romantic love actually does work as an analgesic. We were concerned when performing our study about the very issue you raise – that passionate love was simply acting as a distraction. To control for this, we added in an additional task that involved distraction. This was a well-adopted mental word generation task that involved, for example, having the subject “think about every sport that doesn’t involve a ball.” It turns out that this is a very distracting task and fairly well established.

What we discovered is that both romantic love and distraction work very well to reduce pain. In fact, they reduced both moderate and severe pain equally well. And, this fact that they work equally well played to our favor during our analysis. What was particularly exciting was that while distraction and love worked to about the same magnitude they both involved much different brain systems. Distraction engaged higher-level outer cortical brain systems that have been well established in previous research. However, love engaged very deep-seated brain and brainstem systems that are involved with our basic hungers, drives and cravings. These brain systems (nucleus accumbens and ventral tegmentum) are rich in dopamine neurotransmitters. So, in conclusion, love does affect like a painkilling drug.

**Frank** asks: What’s the difference between chronic pain and acute pain?

Acute pain can serve as a warning signal to protect us from harm and danger. It helps us learn from previous
experiences so that we do not engage in harmful activities again. Acute pain tends to be self-limited and respond to short-term analgesics. While we all might think it would be wonderful to live without any pain, there is a congenital condition that leads to complete insensitivity to pain. Unfortunately, these children often pass away at an early age because they do not have the protective signals that pain provides.

Chronic pain, on the other hand, appears to serve no physiologic basis. It is relentless and without mercy. Sometimes chronic pain is a symptom of another condition. When pain becomes truly persistent, however, it is now thought of as a disease in its own right – much like diabetes or asthma. As such, chronic pain may require long-term treatment. This treatment is often most effective when it combines components of pharmacologic therapy, physical and occupational therapy, interventional procedures, psychological and behavioral therapies and complementary medicine therapies. Unfortunately, we do not often have cures for chronic pain. Instead, we tend to focus on finding ways to better manage the disease condition so that people can have better quality of life and physical functioning.

@graciedoyle asks: What do we know about the way chronic pain changes the brain?

Since the advent of brain neuroimaging, we have been able to open windows into people’s brains and see what happens when “pain goes bad” – or, in other words, becomes chronic. We have learned that the central nervous system, including the brain, brainstem and spinal cord, can fundamentally rewire and change its function and structure as a consequence of chronic pain. Our studies, as well as the work of other investigators, have shown that chronic pain can lead to both shrinking and expansion of our brains’ gray and white matter. Pain can also lead to changes in localized function of the brain. And, finally, we have learned that pain can lead to changes in the networking of the brain – in other words, how the flow of information is transferred from one brain region to another.

What we have ultimately discovered is that pain can impact multiple brain systems, leading to disruption in non-pain related perceptions and behaviors. For instance, we now better understand how changes in the frontal regions of the brain can be associated with memory and cognitive deficits. Historically, we have attributed these deficits to the medications that patients were on. Now, we’re beginning to appreciate the pain itself can cause impairment in these brain systems.

The good news is that we are starting to see that, with appropriate treatment, some or all of these brain changes can be reversed. So there is hope that these changes are not permanent and are quite reversible.

@tracysherman asks: Your research clearly indicates that we can control pain. How does it work? On a similar note, Sruthi S asks: Your research has shown that chronic pain sufferers may be able to reduce pain levels by studying their own live brain images. How does this work?

We have known for thousands of years that we can voluntarily control pain and that some people are more adept at it than others (e.g., Buddhist monks). We have all developed cognitive strategies to learn how to reduce pain in our day-to-day life. These can include distraction, acceptance and reappraisal – or changing the overall meaning of the pain and whether we view it as threatening or not. Part of what we teach people at the Stanford Pain Management Center is how to better learn to control their own pain. This can be taught through pain psychologists who are trained to teach these skills to patients.

But I believe the question you might be asking here is related to our research on real-time fMRI neurofeedback. We developed technology that allows us to put people into a neuroimaging brain scanner, focus on any region or pattern of brain activity, process that information in real time and feed that information back to the subject so that they can control it. People have likened the process to biofeedback, but I would submit that it is somewhat different.

In traditional biofeedback, you are instructed to control downstream physiological processes that ultimately affect pain. For instance, you are asked to control muscle tension, heart rate, respiratory rate or the temperature of your arm. All of these maneuvers can reduce pain, but they often do so by reducing our sympathetic arousal, also known as the “fight or flight response.” In real-time fMRI neurofeedback, we’re
focusing directly on brain systems responsible for the perception and modulation of pain. We are, in essence, going right to the origin of pain. What we have found is that some people can effectively utilize this technique to reduce their pain. More research is needed, however, before we can use this as a clinical tool.

Over the next couple of months, colleagues and I will be launching a randomized clinical research study where participants with chronic lower-back pain will be assigned to undergo real-time fMRI neurofeedback, cognitive behavioral therapy, mindfulness-based stress reduction or acupuncture treatments. If you’re interested in learning more about the trial, please visit our lab website or the pain center website.

Previously: Ask Stanford Med: Neuroscientist taking questions on pain and love’s analgesic effects
Photo by 19melissa68

One Response to “Ask Stanford Med: Neuroscientist responds to questions on pain and love’s analgesic effects”

1. david Says:
   March 6th, 2012 at 2:11 pm

   Dr Mackey is overreaching when he says we can mentally control our pain-as if it were as simple as that. But his beliefs reflect the failure of modern medicine to find real solutions for people in pain-now he and others focus on peoples minds- as if to suggest peoples minds are defective in controlling pain. He fails to mention the idea of maldynia- an unproven concept as medicine fails to prove that pain can exist without some underlying pathophysiology. Dr Mackey may recall that not so long ago curare was used for anesthesia-and doctors routinely believed infants were incapable of feeling pain- and he knows today that often infants undergo painful procedures without any pain medication. Dr Mackey should take the issue of pain more seriously- its not all in our heads- as he suggests here.

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