Toxic input from the meninges primes the brain for migraines

Latest Research: Dural stimulation in rats causes brain-derived neurotrophic factor-dependent priming to subthreshold stimuli including a migraine trigger.



Abstract - Migraine is one of the most common and most disabling disorders. Between attacks, migraine patients are otherwise normal but are sensitized to nonnoxious events known as triggers. The purpose of these studies was to investigate whether a headachelike event causes sensitization, or priming, to subsequent subthreshold events. Interleukin-6 (IL-6) was applied to the rat cranial dura mater which produced cutaneous facial and hind paw allodynia that lasted 24 hours. At 72 hours, IL-6-treated rats developed allodynia in response to dural stimulation with either a pH 6.8 or pH 7.0 solution and to a systemic nitric oxide (NO) donor, a well-known migraine trigger. Vehicle-treated rats did not respond to either pH stimulus or to the NO donor, demonstrating that IL-6 exposure primes rats to subthreshold stimuli. Inhibitors of brain-derived neurotrophic factor (BDNF) signaling given either systemically or intracisternally 24 hours after IL-6 eliminated responses to dural pH stimulation at 72 hours. Additionally, intracisternal administration of BDNF without previous dural stimulation produced allodynia and once resolved, animals were primed to dural pH 6.8/pH 7.0 and a systemic NO donor. Finally, hind paw IL-6 produced paw allodynia but not priming to paw injection of pH 7.0 at 72 hours demonstrating differences in priming depending on location.

Story at-a-glance

- Negative input from the dura primes the brain to be too sensitive to triggers that are normally not a problem.
- It appears that nociceptive input from the meninges causes sensitisation of the dural nociceptive system, and that this duranociceptive input and sensitisation causes neuroplasticity and may contribute to migraines.
- This brings relevance to chiropractic techniques that assess and affect the meninges, and their effect on the brain and its neuroplasticity.

These data indicate that afferent input from the meninges produces BDNF-dependent priming of the dural nociceptive system. This primed state mimics the interictal period of migraine where attacks can be triggered by normally nonnoxious events and suggests that BDNF-dependent plasticity may contribute to migraine.

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