These have also been looked at in a previous article on Opioid medication for neuropathic pain.

It has been found that elevation of the anti-opioid peptide cholecystokinin (CCK) is one of the likely causes of incomplete analgesia with opioid medication. This has been demonstrated in animal studies ([i]).

Nerve injury results in a rise in plasma CCK ([ii]) so that administration of an antagonist such as proglumide (originally developed as an anti-ulcer drug) may affect a return to the pain being opiate-responsive.

McCleane ([iii]) has found that proglumide can augment the analgesia from sustained-release morphine for neuropathic pain.

CCK is also raised by chronic opioid use and may contribute to development of tolerance. ([iv]) Watkins et al ([v]) have shown that proglumide can potentiate opiate analgesia and reverse morphine tolerance.

More studies need to be done to determine the best clinical use of these findings.

---

Chronic pain-related behaviours in spinally-injured rats; evidence for functional alterations of the endogenous cholecystokinin and opioid systems.

