MOR1D MEDIATES OPIOID AND LIVER DISEASE-INDUCED ITCH VIA GRPR

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Background: Opioids, such as morphine, are used in pain management and have significant analgesic effects. However a common side effect of morphine is severe itching. Currently, morphine-induced itch is treated with antagonists against the mu opioid receptor. While these antagonists reduce itch, they also reduce the analgesic effects of morphine because the mu opioid receptor family mediates both the analgesic and itch-induced effects of morphine. New studies demonstrate a molecular separation between morphine-induced analgesia and morphine-induced itch.

Technology Description: Researchers at Washington University have shown that these two effects of morphine are mediated by different isoforms of the mu opioid receptor (MOR). As demonstrated through siRNA knockdown experiments in mice, MOR1 governs the analgesic response, whereas MOR1D specifically interacts with gastrin-releasing peptide receptor (GRPR) to mediate morphine-induced itch. The technology further describes a peptide that, in vitro, specifically disrupts the interaction between MOR1D and GRPR. In vivo, this peptide specifically blocked morphine-induced itch. Importantly, the analgesic effects of mice treated with morphine were unaffected, supporting the specificity of the peptide for MOR1D, with little off-target effects on other isoforms of the MOR proteins.

Key Advantages:

- Allows for the development of therapeutics to treat morphine-induced itch without affecting morphine's analgesic properties.
- Identifies a peptide that specifically targets the interaction between MOR1D and GRPR as a potential treatment for morphine-induced itch.