Mechanism underlying distension-evoked peristalsis in guinea-pig distal colon: is there a role for enterochromaffin (EC) cells?

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Background: The mechanism underlying distension-induced peristalsis in the large intestine is unknown. Studies have suggested that the initiation of peristalsis is due to release of 5-hydroxytryptamine (5-HT) from enterochromaffin (EC) cells in the mucosa, which activates neighbouring sensory nerve endings. However, no direct evidence to support this hypothesis exists, since real time recordings of 5-HT release from EC cells have not been made during colonic peristalsis.

Methods: Real time amperometric recordings with video imaging of colonic movements were used to determine whether 5-HT release from EC cells was required for distension-evoked peristalsis in guinea-pig colon.

Results: Amperometric recordings revealed a basal and transient release of 5-HT from EC cells during peristalsis. This was evident when the carbon fibre electrode, lowered to within 100μm of the mucosal border, recorded an ambient release of 5-HT of 22μM (N=5). However, removal of the mucosa and submucosal plexus abolished all release of 5-HT (0.0μM; N=5), but did not prevent, nor inhibit the initiation of peristalsis, nor propulsive force generated during peristalsis. The propagation velocity of fecal pellets along the colon of dissected (mucosa-free) preparations (1.7 ± 0.2mm/s; N=14) was significantly reduced compared to control intact preparations (2.6 ± 0.2mm/s; N=25). This reduction in propagation velocity occurred even though there was no reduction in propulsive force generated during peristalsis in preparations devoid of mucosa and submucosal plexus. Maintained distension by fecal pellets generated cyclical peristaltic waves, which also persisted following removal of the mucosa and submucosal plexus, albeit with a reduced pacemaker frequency.

Conclusions: The initiation of colonic peristalsis evoked by physiological distension and the propagation of physiological colonic content does not require any release of 5-HT from EC cells, the submucosal plexus, nor activation of sensory nerve endings in the mucosa, as previously hypothesized. Our data lead to the inescapable conclusion that the mechanoreceptors activated by radial distension and sensory neurons which initiate peristalsis lie in the myenteric plexus and/or *muscularis externa*. Moreover, the pattern generator underlying cyclical generation of peristaltic waves in response to maintained distension also does not require release of 5-HT from the mucosa, or neural activity from within the submucosal plexus.