

PC37

Immunohistochemical localisation of [Met]Enkephalin in the normal human colon and in Inflammatory Bowel Disease

G. Gibson¹, A.K. Foulis², D.L. Bovell¹ and A.D. Corbett¹¹Department of Life Sciences, Glasgow Caledonian University, Glasgow, UK and ²Glasgow Royal Infirmary, Glasgow, UK

Gastrointestinal activity is controlled primarily by the enteric nervous system through the release of different neurotransmitters, many of which are neuropeptides. The endogenous opioid peptides play an important role in regulating intestinal function and opioid peptides have been localised in enteric neurones of the human gastrointestinal tract (Kromer, 1988) and also in neuroendocrine (NE) cells within the mucosal crypts (Ahlman & Nilsson, 2001). Inflammatory bowel disease (IBD; Crohn's Disease and ulcerative colitis (UC)) is a condition of unknown aetiology that seriously affects gut function and it has been proposed that neuronal changes may contribute to the pathophysiology of IBD (Shanahan, 1998). In this investigation we have used immunohistochemistry to observe the distribution of the opioid peptide, [Met]enkephalin in the bowel wall of patients with IBD and of non-inflamed "normals". Archival paraffin embedded sections of diseased tissues and formalin fixed fresh specimens of "normal" tissue were obtained from Glasgow Royal Infirmary with informed patient consent and local ethical approval. Standard ABC techniques were employed and results visualised using light microscopy. Anti-[Met]enkephalin (Affiniti, UK) antibody was used at 1:800 dilution. [Met]enkephalin-like immunoreactivity was observed in the myenteric nerve plexus and in neurones throughout the muscularis externa in both inflamed and non-inflamed specimens, however, no significant difference in staining intensity was apparent. There was no evidence of immunoreactivity in the submucosa but [Met]enkephalin-like immunoreactivity was clearly present in the mucosal crypts. In all Crohn's disease specimens (n=5), intense staining was observed in numerous cells within the mucosa; these cells are most probably NE cells. In UC specimens, [Met]enkephalin-like immunoreactivity was also localised to these mucosal crypt cells in 6 out of 7 specimens, with varying degrees of intensity but in fewer cells than observed in Crohn's disease. In only 2 out of 5 non-inflamed specimens, weak immunoreactivity was seen in these NE-like cells. In conclusion, there appear to be alterations in the pattern of [Met]enkephalin-like immunoreactivity in the mucosal crypts of patients with IBD. These observations are in agreement with El-Salhy *et al.*, (1997), who have shown perturbations in the numbers of NE cells in IBD. Experiments are ongoing to determine if this mucosal immunoreactivity is indeed found exclusively in NE cells.

Ahlman H & Nilsson O (2001). *Ann Oncol.* 12(2): S63-S68.El-Salhy M *et al.*, (1997). *J Intern Med.* 242(5): 413-9.Kromer W (1988). *Pharm Rev.* 40(2): 121-162.Shanahan F (1998). *Neurogastroenterol. Mot.* 10: 185-187.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.

PC38

Changes in ankle and knee muscle activity during the running cycle in subjects with functional instability of the ankle.

B. Caulfield, T. Crammond, K. Monaghan, S. Reynolds and A. O'Sullivan

School of Physiotherapy, University College Dublin, Dublin 7, Ireland

Recent evidence suggests that changes in pre-programmed motor control may have a role to play in the development of chronic Functional Instability of the Ankle Joint (FAI), a disabling condition characterised by a tendency for the foot to repeatedly sprain or give way. A common mechanism of injury for subjects with FAI is the foot giving way at heel strike during the running cycle. Reber *et al* (1993) demonstrated activity of ankle musculature in the late swing phase of the running cycle in preparation for impending forces at heel strike. This pre-programmed muscle activity could be altered in FAI subjects. The purpose of this study was to compare activity of ankle and knee muscles pre and post heel strike in a group of FAI subjects and a control group. The study subjects included 7 subjects with unilateral FAI and 6 age and sex matched control subjects. Subjects ran on a treadmill at a velocity of 8kmph whilst electromyographic data was sampled at a frequency of 2000Hz from their quadriceps (VM), soleus (SOL), peroneus longus (PER) and tibialis anterior (TA) muscles using surface electrodes. Data from 5 consecutive running cycles for each subject was extracted for analysis, filtered (20-500Hz), full wave rectified, normalised with respect to peak amplitude, and averaged for each subject. Integral EMG activity during the 150ms linear envelopes relating to the periods immediately prior to and post landing were calculated for each subject and group means were obtained. T-tests were used to test for significant differences between groups. FAI subjects demonstrated significantly higher VM and SOL activity in the period prior to heel strike ($P < 0.05$) compared to the control group. There were no differences in pre heel strike activity of PER or TA or in post heel strike activity of any of the 4 muscles analysed. Table 1. IEMG activity (%max activity.150ms) pre and post heel strike. Values are Means \pm SD. * = significant difference from column to left ($P < 0.05$) These results demonstrate that pre-programmed motor control of muscle activity is altered during the running cycle in subjects with FAI. Changes were observed in activity of muscles at both the ankle and knee joints indicating that the change in motor control associated with FAI affects both local and adjacent joint function. The causes and consequences of observed changes are not apparent from these results and need further investigation.

Muscle	Pre Heel Strike		Post Heel Strike	
	FAI Group (n=7)	Control Group (n=6)	FAI Group (n=7)	Control Group (n=6)
VM	47.3 \pm 10.3	30.1 \pm 13.8*	128.6 \pm 30.6	136.6 \pm 13.3
SOL	60.3 \pm 34.4	24.7 \pm 16.2*	114.6 \pm 17.2	113.8 \pm 13.0
PER	38.0 \pm 20.3	55.1 \pm 45.0	92.8 \pm 35.7	117.7 \pm 22.1
TA	108.2 \pm 35.0	74.9 \pm 42.0	90.2 \pm 27.1	97.0 \pm 42.6

Reber, L., Perry, J. & Pink, M. (1993). *Am. J. Sports Med.* 21, 805-810.

Where applicable, the experiments described here conform with Physiological Society ethical requirements.