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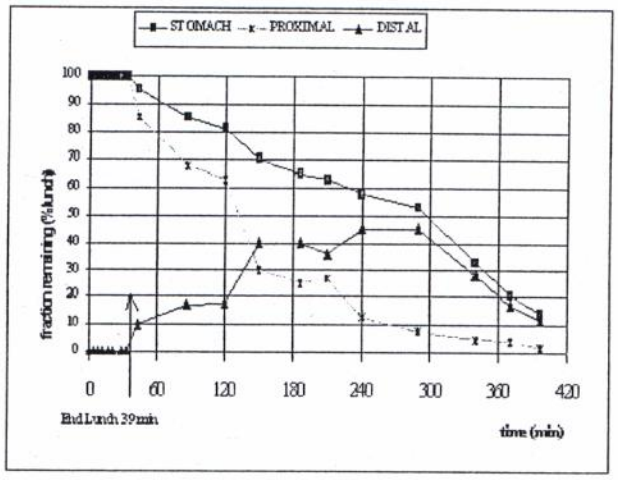
Program & Abstract



PS094 **Poster Presentation**

Effects of Somatostatin and Prostanoids (PGE2) on Jejunal Contractile Activity in Schistosomiasis Infected Mice

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Background/Aims: Somatostatin (SOM) has proven useful in the treatment of visceral pain and motor dysfunction. Prostaglandins E2 (PGE2) is known to modify intestinal motility, but the receptor mediating its actions have not been fully characterized. The aim in this study was to investigate the effect of somatostatin on schistosomiasis induce hyperactivity of intestinal motility and to determine the role of PGE2 in regulating intestinal motility through inflammation.

Methods: Experiments were performed in Swiss mice 4- and 8-weeks following infection with S. mansoni compared to untreated controls. Jejunal motility was assessed using a Trendelenburg preparation to study aboral directed motor complexes (MC) as described previously (Abdu et al., AJP. Am J Physiol Gastrointest Liver Physiol 2002; 282(4): G624-633). Histological evaluation was used to determine the physiological characteristics of inflammation. Data were expressed as mean±SEM (n=6-16) and were analysed by paired or un-paired t tests as appropriate.

Results: Wall thickness of 4-wk infected jejunum was similar to control. However, 8-wk post infection, the muscularis and the mucosa were thickened compared to control and 4-wk infected animals.

Infection had no significant effect on MC amplitude and intervals at 4-wk compared to control (Table 1 and 2), whereas, in the 8-wk post infection there was a significant increase in MC and decrease of intervals compared to control.

PGE2 (1 μM) caused a significant decrease in amplitude and intervals of control, 4-wk and 8-wk post-infection (table 1). SOM (300 nM) in control animals significantly inhibited MC intervals but had no significant effect on MC amplitude ($P>0.1$), table 2. The effect of SOM on MC intervals was attenuated at 8-wk post infection ($P < 0.03$) compared to control.

Conclusion: PGE2 and SOM play a potential role in modulating motor dysfunction during inflammation.

Key Words: somatostatin, prostaglandins, intestinal motility, inflammation

Table1:Effect of PGE2 On contractile activity in schistosomiasis infected mice.
* = P<0.05 vs control; * = P<0.05 vs baseline

	Amplitude			Intervals	
	Baseline	PGE2		Baseline	PGE2
Control	5.97±3	3.03±0.9*	Control	43.88±0.05	19.81 ±1*
4 weeks	5.92±1	3.13±0.3*	4 weeks	49.64±0.9	21.47±3*
8 weeks	20.68±2*	10.14±2*	8 weeks	38.37±3*	23.40±1*

Table 2: Effect of SOM on contractile activity in schistosomiasis infected Mice

	Amplitude			Intervals	
	Baseline	SOM		Baseline	SOM
Control	5.53±2	4.56±1	Control	44.14±2.1	279.29±50*
4 weeks	5.41±2	4.82±2	4 weeks	43.05±1	144.69±41*
8 weeks	21.57±4	16.58±3	8 weeks	31.23±1*	81.58±20*

PS095 **Poster Presentation**

Histopathology of Colonic Mucosa in Postinfectious Irritable Bowel Syndrome

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Background/Aims: Postinfectious irritable bowel syndrome (PI-IBS) develops in 3-30% of individuals with bacterial gastroenteritis. The low grade inflammation has been focused on as one of the pathophysiology of IBS and recent studies also demonstrated increases in inflammatory components in gut mucosa mostly rectum. So we aimed to clarify the histological changes in PI-IBS in colon and rectum.

Methods: The subjects were recruited from our previous study, in which we investigated the incidence and risk factors of PI-IBS in patients with shigellosis 3 years after its outbreak. We compared four groups, PI-IBS (n=4), non PI-IBS (n=7), D-IBS (n=7, diarrhea predominant type) and controls (n=8). All of them underwent colonoscopic biopsy at three areas of descending colon (DC), sigmoid colon (SC) and rectum. which were assessed for serotonin-containing enterochromaffin (EC) cell, T lymphocyte, mast cell and resident macrophage.

Results: All subjects had no structural or gross abnormalities at colonoscopy. Hematoxylin and eosin stain showed no significant differences among four groups. PI-IBS showed increased EC cells at DC and rectum compared with other groups ($p<0.05$). Intraepithelial T lymphocytes (IEL) were increased in D-IBS at SC compared with controls ($p<0.05$). T lymphocytes in the lamina propria were increased in PI-IBS, non PI-IBS and D-IBS compared with control significantly. Mast cell counts made no significant differences among four groups. PI-IBS showed increased macrophages at rectum compared with other groups as well as at DC compared with non PI-IBS ($p<0.05$).

Conclusion: The immunocytes were sporadically increased in PI-IBS, non PI-IBS and D-IBS compared with control. Although three years had passed since acute infection, T lymphocytes, macrophages and EC cells were increased in PI-IBS which may contribute to development of PI-IBS.

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Key Words: PI-IBS, inflammation, colon