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ABSTRACTS

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Conclusions: Our results indicate that EA could be comparable and even superior to Celecoxib for the treatment of neuropathic pain. They further suggest that EA could involve other analgesic mechanisms, more than mere inhibition of COX-2 expression in the spinal cord.

PS3-28

ABNORMAL ENTERIC NERVOUS SYSTEM DEVELOPMENT IN A *SOX10^{EGFP}* MUTANT MOUSE

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SOX10 mutations have been identified in human Waardenburg-Hirschsprung patients who displayed a varied degree of intestinal aganglionosis. The spontaneous *Sox10* mutant *Dom* and the null mutant *Sox10^{lacZ}* are characterized by the absence of enteric ganglia in the myenteric and submucosal plexuses in the distal hindgut. It was suggested that in the mouse mutants, the enteric neural crest-derived progenitors failed to maintain their multipotency, resulted in a reduced progenitor cell pool and aganglionosis.

We have generated a novel mouse mutant *Sox10^{EGFP}* in which the HMG DNA-binding domain and the transactivation domain of *Sox10* have been replaced by the EGFP marker. In the gut of heterozygous *Sox10$^{EGFP/+}$* mutants, the migration of the enteric neural crest cells was delayed at 12.5dpc and the neural crest cells failed to populate the full length of the gut by 14.5dpc. This abnormal phenotype was also observed by immunohistochemical analysis using antibodies against neuronal markers such as TUJ1 and NADPH-diaphorase biochemical assays. In homozygous *Sox10$^{EGFP/EGFP}$* mutants, enteric neural crest cells could only detected in esophagus. We have isolated the mutant enteric neural crest cells and cultured them at clonal density in neurosphere cultures. We are currently studying the differentiation potential of the mutant neural crest cells, in order to correlate the *Sox10^{EGFP}* mutation with the phenotype and to further investigate the functions of *Sox10*.

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CHARACTERISATION OF NEURAL CREST STEM CELLS DERIVED FROM THE MOUSE EMBRYONIC GUT

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Enteric neuropathies comprise a vast and disparate array of congenital and acquired disorders of enteric nervous system (ENS). The use of ENS stem cells to replenish the damaged ENS can be a potential therapeutic measure other than surgical resection. The present study focused on the isolation, culture and charactertzation of ENS stem cells *ex vivo* from the gastrointestinal tract of mouse embryos at embryonic day 14.5 (E14.5). Expression of various cell markers for neural crest (p75, Sox10), neural stem cells (nestin, Sox2), proliferation (PH3), differentiated neuronal/glial cells (Tuj1, GFAP) and myofrbroblasts (□-SMA) was examined with specific immunohistochemical labelling, and the proliferation rate and neurosphere-forming frequency