Title	The role of Sox10 in enteric neural crest cell migration
Author(s)	Li, Z; Leung, C; Zhang, M; Sit, HM; Sham, MH
Citation	The 2014 Hong Kong Inter-University Biochemistry Postgraduate Symposium, Hong Kong, 14 June 2014. In Program Booklet, 2014, p. 36, poster no. 46
Issued Date	2014
URL	http://hdl.handle.net/10722/203834
Rights	Creative Commons: Attribution 3.0 Hong Kong License

Poster Number

46

The role of Sox10 in enteric neural crest cell migration

Zhining Li, Carly Leung, Mei Zhang, Hon Man Sit, and Mai Har Sham
Department of Biochemistry, Li Ka Shing Faculty, The University of Hong Kong

Sox10 is a transcription factor essential for maintaining the multipotency of enteric neural crest cells (ENCCs). Sox10 mutant ENCCs fail to colonize the entire gut due to premature differentiation and migration defects. By time-lapse live-cell imaging analysis, we observed that *Sox10* $^{NGFP/+}$ ENCCs formed abnormal aggregates within the gut. In gut explant culture study, we found that mutant ENCCs displayed abnormal interactions on extracellular matrix with enhanced intercellular adhesion properties. Moreover, microtubules elongation seemed to be disrupted in mutant ENCCs when gut explants were stained with α-tubulin. We hypothesize that Sox10 may regulate ENCCs migration through modulating cell adhesion. To address the roles of Sox10 on cell-cell interactions, we examined the expression of candidate molecules including Cadherin and Eph/Ephrin family members by qPCR analysis. Cadherins engage in cell-cell junction formation. Disruption of Cadherin impaires cell-cell contact and adhesion properties. Ephrins/ Eph receptors are thought to mediate cell-cell repulsion to regulate directional migration of neural crest cells. We found that Cadherin-10/11 and EphA2/A4/B1/B2 expression were significantly changed. To access whether they are transcriptionally regulated by Sox10 involved in ENCC adhesion, we determined EphA2/A4 expression profile by immunostaining and found that they were expressed in both mesenchymal cells and ENCCs. Their ligand ephrinB2 was expressed in ENCCs, suggesting that ephrinB2 and EphA2/A4 interaction may be required for ENCC migration. Altered EphA2/A4 expression affects cell-cell interaction and may lead to reduced repulsion responses with neighboring cells. Our findings suggest a potential role of Sox10 in modulating ENCC adhesion by controlling EphA2/A4 expression.