Down-regulation of miR-206 is associated with Hirschsprung disease and suppresses cell migration and proliferation in cell models

Ankur Sharan^{b,c,#}, Hairong Zhu^{a,c,#}, Hua Xie^{a,c}, Hongxing Li^{a,c}, Junwei Tang^{a,c}, Weibing Tang^{a,c}, Hongwei Zhang^d, Yankai Xia^{a,b}

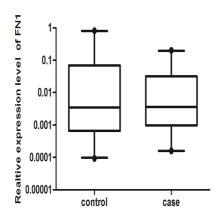
^aState Key Laboratory of Reproductive Medicine, Institute of Toxicology, School of Public Health, Nanjing Medical University, Nanjing 211166, China

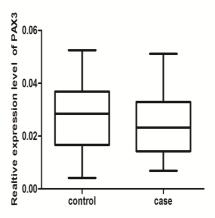
^bKey Laboratory of Modern Toxicology (Nanjing Medical University), Ministry of Education, China

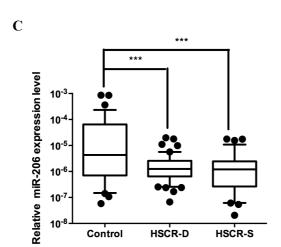
^cDepartment of Pediatric Surgery, Nanjing Children's Hospital Affiliated to Nanjing Medical University, Nanjing 210008, China.

^dDepartment of Pediatric Surgery, Xuzhou Children's Hospital Affiliated to Xuzhou Medical University, Xuzhou 221002, China.









Supplementary figure A-C. No significant differences between HSCR cases and matched controls in the expression level of FN1 and PAX3 mRNA. Extra expression level of miR-206 in controls, HSCR-stenosed segments (HSCR-S) and HSCR-dilated segments (HSCR-D).

A-B: The relative expression levels of FN1 and PAX3 in human HSCR tissues (n=80) and control tissues (n=80) were evaluated by qRT-PCR. Data were presented as box plot of the median and range of log-transformed relative expression levels. The top and bottom of the box represent the seventy-fifth and twenty-fifth percentile. The whiskers indicate the 10th and 90th points. * Significantly different compared with that of control (*P*<0.05). **C**: QT-PCR was conducted to investigate the expression level of miR-206 in 80 matched controls, HSCR-stenosed segments (HSCR-S) and 80 HSCR-dilated segments (HSCR-D). The results showed that the expression level of HSCR-D and HSCR-S were both much lower than controls.