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Spatiotemporal expression of NRAS and occurrence of giant congenital melanocytic nevi

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Statement of the Problem: The mechanism of giant congenital melanocytic nevus formation has not been fully understood. Mutant NRAS is the main driven gene in GCMN according to recent researches. Melanocytic precursor cells proliferate at the embryonic stage after acquiring NRAS mutation. However, the reason why GCMN undergoes intense proliferation in the embryonic stage but stops proliferating postnatal is still unknown. The current theory for this phenomenon is that the GCMN undergoes oncogene-induced senescence (OIS). However, insufficient evidence of senescence-induced growth arrest was found in GCMN. It is believed that excessively high mutated RAS signalling leads to cell senescence and excessively low mutated RAS signalling cannot drive cell proliferation. to understand the formation mechanism of GCMN, we still have to understand the expression level of the mutant gene in the physiological state of GCMN. Methodology & Theoretical Orientation: White et al. produced a high-resolution mRNA expression time course of embryonic development in zebra fish, and we found out the dynamic changes of the expression level of KRAS, NRAS and HRAS in the sequence data. The results showed that NRAS had a high expression level in the early embryonic stage and gradually decreased expression in the late embryonic stage, however the expression of KRAS and HRAS was relatively stable in the whole embryonic development stage. Findings: The NRAS gene is spatiotemporal differently expressed in the embryonic stage of zebra fish, which provides clues to the pathogenesis of giant congenital melanocytic nevi. Conclusion & Significance: In this study, we hypothesize that the expression level of the NRAS gene changes dynamically during the development and differentiation of neural crest cells into melanocytes, and it is the NRAS expression level that determines whether cell proliferation or quiescent.

Recent Publications

1. Charbel C, Fontaine RH, Malouf GG, Picard A, Kadlub N, El-Murr N, How-Kit A, Su X, Coulomb-L'Hermine A, Tost J, Mourah S, Aractingi S, Guégan S NRAS mutation is the sole recurrent somatic mutation in large congenital melanocytic nevi. J Invest Dermatol, 2014, 134:1067-1074.
2. Kinsler VA, Thomas AC, Ishida M, Bulstrode NW, Loughlin S, Hing S, Chalker J, McKenzie K, Abu-Amero S, Slater O, Chanudet E, Palmer R, Morrogh D, Stanier P, Healy E, Sebire NJ, Moore GE. Multiple congenital melanocytic nevi and neurocutaneous melanosis are caused by postzygotic mutations in codon 61 of NRAS. J Invest Dermatol, 2013, 133:2229-36.
3. Tran SL, Haferkamp S, Scurr LL, Gowrishankar K, Becker TM, Desilva C, Thompson JF, Scolyer RA, Kefford RF, Rizos H. Absence of distinguishing senescence traits in human melanocytic nevi. J Invest Dermatol, 2012, 132:2226-34.

Biography

Qingxiong Yu has his expertise in basic and translational medical research of giant congenital melanocytic nevus. He proposed that the spatotemporal expression changes of oncogenes may affect the occurrence and development of neoplasm. His research brings new perspectives to the pathogenesis of giant congenital melanocytic nevi.

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