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Modena, 11-13 settembre 2023

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Retinoic gene signature in Merkel cell carcinoma

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The identification of the origin cell of Merkel cell carcinoma (MCC), a rare skin tumor, is prevented by the limited molecular knowledge of its two subsets, namely Merkel cell Polyomavirus-positive and -negative MCC (MCCP/MCCN). Given the expression of neuroendocrine, epithelial, fibroblast and B-cell markers in MCC, a multilinear origin has been assessed. However, the existence of two subsets may reflect oncogenic events occurring in two distinct origin cell types. Retinoic signaling activation has been reported to inhibit the epithelial-to-mesenchymal transition (EMT), while altered retinoic-associated genes have been identified in MCC. However, the role of the retinoic signaling in MCC is unclear. Retinoic gene signature was investigated herein in MCCP, MCCN and in epithelial/fibroblast control cells to elucidate the heterogeneous nature of MCC. Hierarchical clustering and Principal Component Analysis were conducted to categorize cells according to the retinoic gene signature. MCCP and MCCN cells were hieratically clusterizable from each other and from epithelial/fibroblast cells, according to their retinoic gene signature. MCCP vs MCCN differentially expressed genes (n=43) were identified. Among these, ten hub genes were identified *via* protein-protein interaction network. In particular, SOX2, ISL1, PAX6, FGF8, ASCL1, OLIG2, SHH and GLI1 were upregulated, whereas JAG1 and MYC were downregulated in MCCP compared to MCCN. Enrichment analyses indicated that differentially expressed genes are predominantly related to DNA-binding/transcription factors involved in development, regionalization, pattern specification and morphogenesis. MCCP-associated hub genes are transcription factors active in neurological development, stemness and EMT, thus suggesting that dysregulated retinoic gene expressions might confer a more pronounced invasive/transformation potential in MCCP than MCCN. Our findings

on retinoic signature in MCC may suggest the neuroendocrine origin of MCCP.

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