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Neural correlates of migration background as a social environmental risk factor for schizophrenia

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Schizophrenia is a severe neurodevelopmental disorder with a complex etiology. The scientific literature on the pathogenesis of the disease suggests several environmental risk factors building on existing genetic predisposition. One of the most established environmental risk factors for schizophrenia is migration background. The risk for the disease is doubled in immigrant populations. The increased risk was observed not only in the first-generation but also in the second-generation migrants who did not experience the adverse effects of migration itself. These findings point out the importance of the post-migratory period where individuals might be exposed to chronic social stress and social defeat.

In line with the above, the current thesis investigates altered neural stress processing and its structural correlates using functional and structural magnetic resonance imaging. In the studies presented here, we have demonstrated that healthy second-generation migrants perceive higher chronic stress compared to native Germans. Additionally, migrants showed altered neural social stress processing in the perigenual anterior cingulate cortex (pACC). Further, the neural activation in the pACC, as well as in ventral striatum during social stress, was highly correlated with participants' perceived discrimination of their own ethnic group. Moreover, structural neuroimaging findings present that the healthy males, but not females, with migration background, demonstrate decreased gray matter volume in the pACC.

These studies are the first neurobiological investigations that are linking ethnic minority status to increased schizophrenia risk via altered neural stress processing. These findings further point out a potential neural target, the pACC, for future investigation of this severe neurodevelopmental disorder. The pACC has previously been shown to relate several other risk factors including urban upbringing and social status, and also demonstrated early structural and functional changes in psychosis patients. By identifying a convergent risk circuit involving the ACC, we propose chronic social stress as a mediator which may present itself as a target for future prevention and intervention programs. In the long term, a better understanding of these neural circuits will inform the causes for differential risk between certain groups in the development of schizophrenia. For this reason, experimental studies based on epidemiological literature, such as the two at hand, are essential to improve our understanding of risk mechanisms and to help translate biopsychosocial determinants of risk for schizophrenia into daily life.