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Epigenetics of autism

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First described by Kanner, 1943, autism is a neurodevelopmental disorder characterized by repetitive, stereotypical behaviours and impaired expressive communication, which has since been folded into the broader classification of autism spectrum disorders (ASD). There are no single known causes of ASD and there are ongoing challenges for diagnosis and treatment. One of the most critical consequences of ASD is the deficit on social behaviour. We are trying to search for epigenetics (methylation) patterns that could significantly differentiate between ASD and non-ASD (healthy) individuals and are associated to genes that could be related with this impaired social communication, often attributed to misreading of emotional cues. Our research started with the olfactory function. Olfactory function and memory are strongly related with the social communication in mammals and is still not clear how the olfactory system is affected with ASD. Also, is still unclear why individuals with ASD misread emotions. Recent results implicate social chemosignaling as a sensory substrate of social impairment in ASD, implicating a difference in the way of doing an interpretation of social odors for ASD and TD (typically developed) individuals. Research emerging within the past two decades suggests that immune dysfunction (also in mothers during pregnancy) is a viable risk factor contributing to the neurodevelopmental deficits observed in ASD. Specific haplotypes of immune genes within the major histocompatibility complex (MHC) of human chromosome 6 – human leukocyte antigen (HLA) and complement – have been implicated in ASD. Using cluster analysis to separate methylation levels and an ANOVA analysis with random subject effects, we are obtaining a quite interesting group of significant CpGs and related to genes potentially associated to ASD, as the HLA family. Further data processing and statistical analyses are needed in order to arrive to final conclusions. Together, these data suggest that immune dysregulation resulting from environmental exposures may underlie the pathogenesis of ASD.