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CX45 and CX36 gap junctions are differently regulated by pH_i and volatile anesthetics

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Gap junction (GJ) channels formed of connexin (Cx) proteins provide a direct pathway for electrical and metabolic cell-to-cell communication. GJ channels are highly sensitive to intracellular pH (pH_i), which can vary substantially under ischemia. It is generally assumed that GJ conductance (g_j) can be reduced by acidification of intracellular milieu and GJ uncoupling agents, such as isoflurane, a volatile anesthetic, or hexanol, while it increases in alkaline conditions. However, the extent of modulation may depend on the Cx type. In this study, we focused on two neuronal Cxs, Cx36 and Cx45, exogenously expressed in HeLa cells.

Double whole-cell patch-clamp and fluorescence microscopy were used to measure g_j and pH_i. We demonstrate here that in contrast to Cx45, g_j of Cx36 GJ channels is almost insensitive to acidification and is strongly reduced by alkalization. Moreover, in contrast to other Cxs, g_j of Cx36 GJs was strongly stimulated by isoflurane and hexanol. Also, the sensitivity of Cx45 and Cx36 GJs to these uncoupling agents was dependent and independent on pH_i, respectively.

Protonation level of histidines and cysteines may vary under physiological conditions (pK_a is ~6 and 8, respectively). Therefore we hypothesized that under acidic conditions, g_j of Cx45 GJs decreased due to protonation of histidines, and under alkaline conditions, g_j of Cx36 GJs decreased due to deprotonation of cysteines. We demonstrate by site directed mutagenesis that replacement of histidines in the intracellular loop of Cx45 reduced its sensitivity to acidification, and that C264S mutation of Cx36 reversed its response to uncoupling agents.

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Olfactory deficits in individuals with autism spectrum disorder

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Introduction: Olfactory abnormalities has been reported in subjects with Autism Spectrum Disorders (ASD), a neurodevelopmental disorder characterized by social deficits. However, previous studies considered mainly olfactory threshold or other dimensions of olfactory function, usually in isolation or at least in a combination with olfactory threshold. Aim of this study is to examine odor identification and discrimination in ASD compared to typically developing (TD) controls.

Materials and methods: Olfactory identification and discrimination were studied, with the 'Sniffin' Sticks' test, in 30 participants: 15 with ASD (2 female and 13 male, mean age 19.13 years) and 15 controls (3 female and 13 male, mean age 21.73). Participants with ASD showed IQ of 103.2 ± 18.5. TD controls were volunteer university students matched for chronological age with ASD participants.

Results: Receiver operating characteristic (ROC) analysis indicated a difference between participants with ASD and TD controls in odour identification [area under the curve (AUC) = 0.709, 95% confidence interval (CI): 0.515–0.859, $P = 0.028$] and in odour discrimination (AUC=0.782, 95% CI: 0.594–0.911, $P = 0.002$).

Discussion: According to previous studies, our results confirmed an impairment in odour identification and discrimination in subjects with ASD, which suggests deficits in orbitofrontal cortex, in medial temporal lobe and in cerebello-thalamo-cortical circuits. Our findings support the importance of olfactory evaluations as a non-invasive tool to identify cortical dysfunction in ASD.

