Mikhail Alexandrovich Filippov
Dr. sc. hum.

Tracing connexin26 expression using a genetic "knock-in" approach

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Diplom der Fachrichtung Biochemie, Moskauer Staatliche M.-W.-Lomonossow-Universität, Fakultät für Biologie

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Doktorvater: Prof. Dr. med. Hannah Monyer

A variety of connexins are expressed in the diverse cell types of the central nervous system (CNS) and are thought to regulate some of the functional properties exhibited by immature and mature cells. A proper understanding of the role of specific connexins in these processes requires an unambiguous characterization of their spatial and temporal pattern of expression. In order to define the cellular distribution of connexin26 (Cx26) in the mouse we have genetically altered the locus so that the *β-galactosidase* (*lacZ*) gene is expressed from the endogenous *Cx26* promoter. Although the modification resulted in homozygous lethality, the amount of lacZ protein produced appeared to be sufficient to serve as a marker for *Cx26* gene expression. Thus, *lacZ* gene expression was specifically visualized in tissues known to contain Cx26 such as, the meninges, liver, kidney, skin, cochlea, small intestine, placenta, and thyroid gland, and the distribution of lacZ activity in these tissues was in good agreement with that previously reported for Cx26. In the embryonic and mature CNS, however, lacZ was restricted to meningeal cells and could not be detected in either neurons or glia. The absence of *Cx26* expression in these cell types could also be confirmed by RT-PCR and *in situ* hybridization. Our experiments indicate that the $Cx26^{lacZ}$ mouse line can be used as a reporter of *Cx26* gene expression and suggest that Cx26, contrary to previous reports, is restricted to the meninges in both, embryonic and adult brain.