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Early ciliary and prominin-1 dysfunctions precede neurogenesis impairment in a mouse model of type 2 diabetes

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Highlights

- Ependymal cilia are disorganized in type 2, but not in type 1, diabetic mice.
- Cilia disorganization is concomitant with a delocalization of prominin-1.
- Neurogenesis impairment followed ciliary dysfunction.
- Type 2 diabetes induced ectopic migration of neuroblasts.

Abstract

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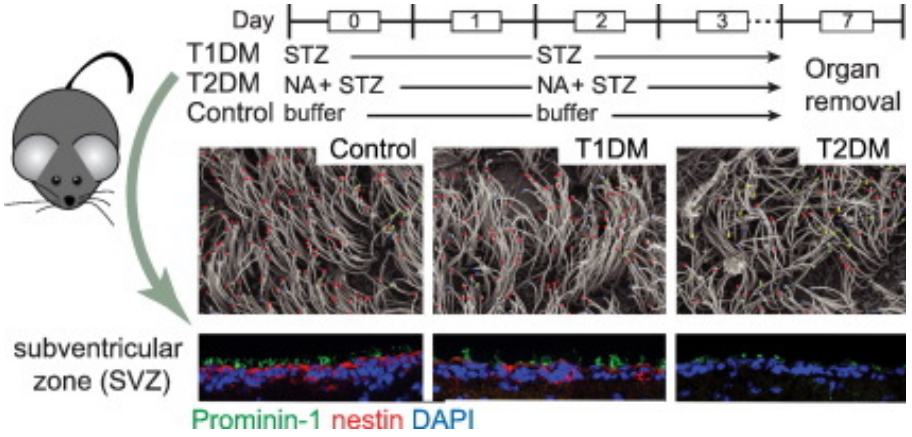
Surprisingly, only T2DM mice showed SVZ damage. The initial lesions were localized to ependymal cilia, which appeared disorientated and clumped together. In addition, they showed delocalization of the ciliary membrane protein prominin-1. Impairment of neuroprogenitor proliferation, neurogenic marker abnormalities and ectopic migration of neuroblasts were found at a later stage. To our knowledge, our data describe for the first time such an early impact of T2DM on the SVZ. This is consistent with clinical data indicating that brain damage in T2DM patients differs from that in T1DM patients.

Graphical abstract



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Abbreviations

AUC, area under the curve; CSD, circular standard deviation; CSF, cerebrospinal fluid; CLSM, confocal laser scanning microscope; DCX, doublecortin; DM, Diabetes mellitus; Glut, glucose transporter; GFAP, glial fibrillary acidic protein; IPGTT, intraperitoneal glucose tolerance test; NA, nicotinamide; RMS, rostral migratory stream; SEM, scanning electron microscopy; STZ, streptozotocin; SVZ, subventricular zone; T1DM, type 1 diabetes-like; T2DM, type 2 diabetes-like

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