5th International Conference on

Alzheimer's Disease & Dementia

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Ins2 JHQH H[SUHVVLRQ DQG IXQFWLRQ LQ WKH PRXVH EUDLQ

6XQGD\\$\$MD\L 'HUHN \$'LRQQH 'DULD) +XWFKLQVRQanootHDOPENFVD 0-R3KDQM-RC6DQMD 6RR 6KHUQD] University of British Columbia, Canada

nsulin de ciency and insulin resistance has both been reported in Alzheimer's disease. is study was designed to examine whether insulin protein or markers of insulin promoter activity can be observed in the mouse brain and to also determine the e ects of brain-speci c insulin genles(2) knockout on behaviour to ascertain the possible role of insulin produced locally in the brain. We have employed germline2knockout mice (hs2-/-), heterozygous mutant mice(h(2-/-)), and their wildtype littermate controls (hs2-/+), as well as cell type specine2knockout mice derived by crossing NesCre, SynCre, or CamkCre mice with mice harboring a oxetals2 allele (on the Ins1-/- background). Mice were genotyped using PCR. Insulin mRNA analysis using qPCR con rmed the deletion of the Ins2 gene in the germline knockout animals, but revealed a paradoxical increase infns2mRNA in many brain regions of thes1/-

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