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In the last 10 years, hundreds of studies have focused on the so-called "Hypo-Hippo" condition, associated with cancer development, where the downstream effector of the Hippo signalling pathway YAP (Yki in flies) is activated and upregulates its target genes, leading to unrestrained proliferation. More recently, a "Hyper-Hippo" condition has been associated with neurodegenerative phenotypes. In the few studies so far available, hyper-activation of upstream components of the pathway has been found to cause YAP/Yki inactivation and to block growth signals. This results in neuroinflammation and neuronal cell death both in mammals and in *Drosophila*. The Gaucher Disease (GD), described as the most common lysosomal disorder, arises from mutations in the *GBA1b* gene, which encodes the β-glucocerebrosidase acid enzyme. In more than 90 percent of patients, these mutations are responsible for a systemic disorder that can be treated with a substitutive enzymatic therapy. Few patients, often affected by the same mutation as the systemic Gaucher patients, develop a neuronopathic form, with neuroinflammation and neuronal cell death, and lack effective therapy. This observation opens to the possible involvement of other genes and pathways in this form of GD. Taking advantage of a *Drosophila* Gaucher-like model based on *dGBA1b* knock-out ($GBA1b^{KO}$), we analyzed the Hippo pathway activity in the mutant context. We found deregulation of some Yki target molecules, such as CycE, dIAP and MYC, which were all severely reduced both in terms of transcript and protein in the $GBA1b^{KO}$ context, suggesting a cell growth impairment. We also analyzed upstream components and found that Fat, an atypical cadherin upstream of the kinase complex, is up-regulated. In the light of this, we decided to examine some soluble factors known to be negatively regulated by Fat and involved in glial and synapse development, the *Drosophila* glypicans Dally and Dally-like. As expected, we found them downregulated in the $GBA1b^{KO}$ background, and we do believe this may be correlated with the neurological damage characteristic of the neuronopathic Gaucher. Preliminary functional data will be presented which support our current hypothesis.