## A01 - ACTIVE RESILIENCE MECHANISMS OF DOPAMINE MIDBRAIN NEURONS

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A01 aims at understanding the molecular mechanisms of maintaining the firing frequencies and patterns in dopaminergic (DA) VTA (ventral tegmental area) neurons, an established resilience mechanism after exposure to chronic social defeat (CSD). Therefore, A01 (i) identifies the involved DA VTA neuronal subtypes, (ii) identifies and characterizes voltage gated potassium channels (K channels) responsible for the maintenance of firing rates, (iii) tests a causal chain between mTOR-dependent changes in protein synthesis after CSD, K channel up-regulation, recovery of normal VTA dopamine neuron excitability and resilient outcome.

## A02 - UNRAVELING THE RELATION BETWEEN ADULT-BORN HIPPOCAMPAL NEURONS' CONNECTIVITY AND RESILIENCE

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Elevated levels of adult neurogenesis have been proposed as a resilience-conducive trait that protects against stress-related dysfunction by facilitating discrimination during and/or after stressor exposure, the latter being supposed to represent an active resilience mechanism. In A02, we aim at revealing the dependence of resilience to stress-related dysfunction on the degree of circuit integration of adult-born dentate granule cells (DGCs) as determined by rabies virus-based tracing of monosynaptic input connections. We will correlate circuit integration of DGCs and resilience to chronic social defeat stress and search for activity- and neurotrophin-induced manipulations that improve resilience outcome as a consequence of altered connectivity.

## A03 - NEURONAL ACTIN DYNAMICS SHAPING RESILIENCE: THE ROLE OF THE NOVEL ACTIN-INTERACTING PROTEIN 'DOWNREGULATED IN RENAL CANCER' (DRR1)

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A03 investigates the function of the protein DRR1, whose stressor-induced DRR1 up-regulation is a candidate active neural resilience mechanism. A01 proposes that DRR1 regulates the actin-dependent insertion of AMPA receptors at the synapse. Hippocampal AMPA receptor function in turn affects resilience, suggesting a link from stressor-induced changes in the trans-criptome via regulation of neural excitability in the hippocampus to resilience. The authors will further apply hypothesis-free approaches serving to discover further molecular candidates, including RNA sequencing of DRR1 overexpressing much after stress exposure.

## A04 - DEVELOPING A ZEBRAFISH MODEL TO IDENTIFY NOVEL MEDIATORS OF RESILIENCE MECHANISMS

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A04 will exploit the short generation times, space-saving housing and excellent genetic manipulability afforded by zebrafish. They develop the first zebrafish model of resilience to chronic stress, which will serve to discover new molecular candidates. Further, A04 applies BONCAT and FUNCAT techniques that allow for identifying newly synthesized (stressor-induced) synaptic proteins in a regionally specific manner. Also, they will use the model to validate discovered candidates by a battery of loss-of-function and gain-of-function approaches that are already well established in zebrafish.

## A05 - DECIPHERING THE EPIGENETIC BASIS OF RESILIENCE

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A05 will exploit natural inter-individual variability of resilience by comparing transcriptomes and genome-wide chromatin accessibility of hippocampus, nucleus accumbens and prefrontal cortex between resilient and non-resilient mice. Next, we will apply specific inhibitors of distinct epigenetic regulators to different groups of mice at the time of stress and compare their later behavioral outcomes with non-drug treated control groups subjected to identical stress. For mice treated with the inhibitor that showed the strongest resilience-promoting effects, we will then determine transcriptomes, genome-wide chromatin accessibility and associated epigenetic marks of the above mentioned brain regions.