response obtained for glucose with that obtained for L-serine and Methyl-aspartate.

827-Pos Board B596
Comparison of Resilience against Genetic Perturbations in Microbial Metabolism
Chintan Joshi, Ashok Prasad.
Colorado State University, Fort Collins, CO, USA.
In metabolic networks, microorganisms are able to survive genetic and environmental perturbations by redirecting the flux from one pathway to another. Hence, it is important to understand how design principles of metabolic networks confer this resilience to the organism. We used Flux Balance Analysis (FBA) and Minimization of Metabolic Adjustment (MOMA) in order to map the lethal gene deletions of two genome scale models of microbial metabolism - E. coli iAF1260 under aerobic growth and Synechocystis PCC6803 under heterotrophic and autotrophic growth - onto each other. Remarkably more than 50% of cyanobacterial lethal knockouts were non-lethal in bacteria. Comparisons of the pathways involved identifying some key subsystems of the bacterial metabolism and the electron flow machinery of cyanobacteria as being primarily responsible for conferring this resilience. We also used FBA to study epistasis in the cyanobacterial network, i.e. aggravating or buffering co-operativity in double gene deletions as compared to corresponding pair of single deletions. In accordance with previous results, heterotrophic and aerobic bacterial metabolism had both aggravating and buffering interactions between genes. Surprisingly however the autotrophic environment displayed a dominance of aggravating interactions with monochromatic interactions between subsystems. We also found that epistasis in an autotrophic organism was highly sensitive to light conditions, and under high light all interactions were aggravating. Our results suggest that the cyanobacterial metabolism is less resilient against genetic perturbations as compared to E. Coli, possibly due to greater specialization under autotrophic conditions.

Neuronal Systems & Modeling

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Optical Sensing of Axons in GaAs Ring Resonators
Aune Koitmäe, Cornelius Bausch, Daniel Diedrich, Eric Stava, Hoan Vu, Robert H. Blick.
University of Hamburg, Hamburg, Germany.
Microtubes herald a new approach to how semiconductors can be applied as actuators and sensors. This is mainly due to their three-dimensional nature, which enables to wrap the microtubes around an axon. This yields an increase in coupling, e.g. of action potentials, by orders of magnitude. While actuation can be established in a straightforward fashion, signal readout for sensing purposes is more intricate. In the current work, we present our approach of realizing microtubes as GaAs ring resonators for remote optical sensing. In practise the microtubes are first fabricated, while axons are grown at a later stage employing the tubes as growth guides. The microtubes are fabricated based on self-organized rolling-up of strained semiconductor layers. The diameter of the tubes is dialed in to be on the order of the diameter of an axon (2µm), while the length varies between 50 and 100 µm. To overcome the problem of toxicity of GaAs, the tubes are parylene-coated. Finally, transparent substrates are employed to measure optical modes in the bottle resonators without radiative losses [1].
We demonstrate our first results of optical sensing of axons in semiconductor microtubes on a transparent substrate.
[1] Strelow et al. Phys Rev. Lett. 2008, 101, 127403

829-Pos Board B598
Hippocampal Filopodia but not Growth Cones can Climb Over 600 nm Steps
Linh Thuy Lien[1], **Jelena Ban**[1], Massimo Tormen[2], Elisa Migliorini[2], Gianluca Grenci[2,3], Alessandro Pozzato[2], Vincent Torre[1].
[1]SISSA-International School for Advanced Studies, Trieste, Italy, [2]Istituto Officina dei Materiali (IOM-CNR), Trieste, Italy, [3]MIB-Mechanobiology Institute, Singapore, Singapore.
Guidance molecules, such as Sema3A or Netrin-1, can induce growth cone repulsion or attraction in the presence of a flat surface, but very little is known of the action of guidance molecules in the presence of obstacles. In order to address this issue, we analysed the action of Netrin-1 molecules in the presence of patterned polydimethylsiloxane (PDMS) surfaces with lines with a height varying from 100 to 600 nm. Filopodia can crawl over these lines easily, but not neurites. Indeed axons and thick dendrites are able to cross lines with a height of 100 nm, but progressively less when the line height is increased. When neurons are grown for 3 days over patterned surfaces it was possible to label selectively axons with axonal neurofilament marker SMI 312 and dendrites with microtubule associated protein 2 (MAP2): axons and dendrites do not differ in their ability to cross lines with a height varying from 100 to 600 nm. When axons and dendrites grow along lines, a clear staining for the adhesion marker paxillin was observed, but not when neurites cross the lines.

830-Pos Board B599
Quiescent Response of Cerebral Cortical Astrocytes to Nanoscale Scaffold Properties
Volkan Mujdat Tiryaki[1], Virginia M. Ayres[1], Kan Xie[1], Ijaz Ahmed[2], David I. Shreiber[2].
[1]Michigan State University, East Lansing, MI, USA, [2]Rutgers, The State University of New Jersey, Piscataway, NJ, USA.
Astrocytes cultured on electrospun polyamide nanofibers whose nanophysical properties may reproduce key aspects of native extracellular matrices have demonstrated promising results in both in-vitro and in-vivo situations. In vitro, astrocytes cultured on nanofibrillar scaffolds assumed morphologies that appeared to recapitulate those observed in native tissues and also demonstrated increased neurite outgrowth by co-cultured neurons. In vivo, the same scaffolds introduced into spinal cord wound sites promoted accelerated hindlimb recovery measured by standardized observational scoring with aligned and fasciculated axon development and revascularization throughout wound sites. The in-vitro and in-vivo results suggest that nanofibrillar scaffolds could induce preferential astrocyte differentiation leading to minimized glial scar formation, which has positive implications for improved treatment options for central nervous system injury repairs.
The present investigation [1] examined the hypothesis that external physical cues of the nanofibrillar scaffolds can trigger specific signaling cascades with morphological consequences. The nanophysical cues of macro- and nano- scale surface roughness and surface polarity were investigated in this study. The morphological response of cerebral cortical astrocytes to nanophysical properties of the nanofibrillar scaffolds was investigated at high-resolution using AFM. The three-dimensional capability of AFM was also used to characterize cell spreading. An initial study of the corresponding activation of GTPase upstream regulators was performed using immunocytochemistry, focusing on investigation of the main GTPase regulators for the observed morphological responses: filopodia, lamellipodia, stress fiber formation, and stellation. The results support the hypothesis that the nanophysical extracellular environment can trigger preferential activation of members of the Rho GTPase family with demonstrable morphological consequences for cerebral cortical astrocytes.
[1] Tiryaki VM, Ayres VM, Khan AA, Ahmed I, Shreiber DI, and Meiners S ‘‘Nanofibrillar scaffolds induce preferential activation of Rho GTPases in cerebral cortical astrocytes’’, Int. J. Nanomedicine, 07:3891-3905 (2012).

831-Pos Board B600
Obesity Influences the Regulation of the Intrinsic Excitability of AgRP/NPY Neurons by Leptin
Kristen M.S. O’Connell, Shannon Guyot, Scott B. Baver.
University of Tennessee Health Science Center, Memphis, TN, USA.
The adipostat leptin binds the long-isoform of the leptin receptor (LepRb) signaling nutritional status in mammals. The hypothalamic arcuate nucleus (ARH) is a major brain region involved in the regulation of food intake; LepRb are highly expressed in the ARH in both orexigenic NPY/AgRP neurons and anorexigenic POMC neurons. Leptin exerts opposing effects on the activity of these two neuronal populations, inducing net activation of the ARH appetite-suppressing circuit. In diet-induced obesity (DIO), the leptin-sensitivity of these neurons is diminished, producing persistent activation of the orexigenic circuits in the ARH. However, the underlying changes in the ion channels that mediate excitability remain to be fully elucidated. In this study, we show that DIO generates a persistent activation of NPY neurons, even in well-fed animals. NPY neurons were found to have a delayed rectifier K^+ current whose activation is significantly shifted to more hyperpolarized potentials by leptin. However, in acute brain slices from DIO mice, the leptin-sensitivity of this current is significantly reduced. The biophysical properties of this current combined with immunohistochemistry suggest that Kv2.1 underlies the leptin-sensitive IK. In HEK cells coexpressing Kv2.1 and LepRb, leptin induces a significant, large hyperpolarizing shift in the voltage-dependence of activation of the Kv2.1-mediated K^+ current that is qualitatively similar to that observed in NPY neurons. This effect was specific to Kv2.1, as leptin had no effect on the function of the closely related channel Kv2.2. We propose that dynamic modulation of somatic Kv2.1 channels by peripheral signals of satiety and hunger regulates the intrinsic excitability of AgRP/NPY neurons to modulate both the spontaneous activity of these neurons and the integration of synaptic input onto the neurons that comprise the energy balance circuits in the ARH.