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Stable isotope assisted metabolic profiling reveals chronicinflammation and oxidative stress as a consequence of a functional lossof the Parkinson's disease related gene DJ-1

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Profiling cellular metabolism provides an insight into the actual state of a livingsystem which represents the integrated result of all regulatory events for a certainenvironment. Routinely applied metabolomics techniques monitor static metaboliteconcentrations. On the other hand, the application of stable-isotope labeled tracers canextend metabolomics techniques and provide quantitative information on intracellularreaction rates or metabolic fluxes. Mutations of the gene *DJ-1* (*PARK7*) have been relatedto a familial autosomal recessive form of Parkinson's disease with an early onset ofdisease progression. Although it has been shown that DJ-1 function is required to protectneurons against oxidative stress, its exact role in cellular homeostasis is still matter ofdiscussion.Here the author will report recent studies of metabolic and cellular effects causedby a DJ-1 deficiency in cells of the central nervous system. Recently, our group showedthat mammalian immune cells produce the antimicrobial metabolite itaconic acid underinflammatory conditions. Intriguingly, we could detect increased synthesis of thismetabolite as a consequence of DJ-1silencing in mammalian microglia cells. Based onfurther analysis of metabolic fluxes and intracellular metabolite profiles, we couldobserve a weak but constitutive pro-inflammatory state of the cells. We could furthervalidate these observations by the analysis of cytokine expression profiles and cytokinerelease. Furthermore, by comparing the cellular state of different cell types based onmetabolic fluxes we could show that a lack of DJ-1 protein results in increased levels ofoxidative stress but that the specific inflammatory metabolite signature is specific formicroglia cells. Finally, using stable isotopic tracers we could show that metabolic fluxesfrom serine to glycine and to the tetrahydrofolate (one-carbon) pool are especiallyaffected by oxidative stress.

Biography

Johannes Meiser finished his Ph.D. in 2011 at the Institute of molecular plant physiologyof the Saarland University, Germany. In August 2012 he started an appointment as apostdoctoral fellow in the Metabolomics Group of the LCSB in Luxembourg. Since thattime, he put his research interest on neuronal metabolism and biochemistry.

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