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Gene expression alterations in a mouse model of cerebral malaria

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Despite extensive research, malaria remains an important cause of morbidity in the developing world. Previously we demonstrated that *Plasmodium berghei* ANKA (PbA)-infected C57BL/6 mice develop cerebral malaria (CM) associated with demonstrable cognitive deficits that are associated with inflammation in the brain and alterations in neuronal axons and cerebral blood flow. To further investigate the downstream effects of CM on the brain parenchyma, we infected six week-old female mice with PbA. At day 6 of infection the brain lysates of infected and control mice were analyzed by cDNA microarray. Analysis of pathways of significantly altered gene expression decreases in several pathways indicative of neuronal dysfunction including a decrease in synaptic pathways (z score 3.516 permuted p = 0.008), and a decrease in energy synthesis and utilization when looking at parameters such as alcohol metabolism (z score = 2.931 permuted p = 0.02) and hydrogen transport (z score = 4.048 permuted p = 0.001). This was in contrast with an increase in G-protein-coupled receptor activity (z score = 3.7 permuted p = 0.001). We also observed an increase in inflammatory pathways and growth factor activity (z score = 2.331 permuted p = 0.026). These data indicate that acute infection with PbA results in substantial changes in expression of neural genes, consistent with neurological sequelae, including cognitive deficits, associated with CM.