neurotropic effects leading to encephalitis, meningitis, myelitis and myositis (2) indirect effects due to metabolic complications resulting in encephalopathy and cerebrovascular complications due to thrombocytopenia and platelet dysfunction and (3) postinfectious immune-mediated acute disseminated encephalomyelitis, Guillain Barré syndrome and optic neuritis.
Material and methods: This was a descriptive cross sectional study including seropositive patients diagnosed with Dengue fever (DF), Dengue with warning signs and Severe Dengue with neurological manifestations presenting to Medicine Department of LLR Hospital, Kanpur.
Results: 10 (2.6%) patients had neurological manifestations out of 383 seropositive patients. Out of ten, nine patients were male and only one patient was female. Among them 10% patients come under category of classical dengue fever, 10% patients suffered from dengue with warning signs and 80% with severe dengue .4 patients had encephalopathy,3 other patients had encephalitis, 2 patients presented with single episode of symptomatic generalized seizure and 1 patient presented as having an intra cranial hemorrhage.
Conclusions: Neurological manifestations of dengue are manifold and it is necessary to consider dengue as a cause for the above neurological presentations in endemic zones of the disease.

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CNS Infections 1
Safflower yellow inhibits the inflammatory response and regulates microglial polarization in LPS-stimulated bv2 cells

C. Ma[a], X.W. Yang[a], Y.H. Li[b], J.Z. Yu[b], C.Y. Liu[b], J.J. Huang[c], J.C. Liu[a], W.J. Jiang[a], Q.J. Feng[a], B.G. Xiao[d]. *[a]"2011" Collaborative Innovation Center/ Research Center of Neurobiology, Shanxi University of Traditional Chinese Medicine, Taiyuan, China; [b]Institute of Brain Science, Shanxi Datong University, Datong, China; [c]Department of Neurosurgery First Hospital, Datong Coalmine Group, Datong, China; [d]Institute of Neurology Huashan Hospital Institutes of Brain Science and State Key Laboratory of Medical Neurobiology, Fudan University, Shanghai, China*

Background: Safflor Yellow (SY), the main active constituent of the traditional Chinese medicine Safflower, is known as a neuroprotective agent that indirectly attenuates neuroinflammation. Macrophage/microglia have different phenotypic and functional states, M1 is associated with inflammatory responses, while M2 results in anti-inflammatory effects.
Objective: The purpose of this study is to discover the effect of SY on anti-inflammation and polarization of microglia stimulated with LPS as well as related molecular mechanism.
Material and methods: BV-2 microglial cell line was treated with LPS and/or SY. Molecular biological technique, flow cytometry, and immunohistochemistry were adopted.
Results: LPS-stimulated BV2 cells upregulated the expression of TLR4 ($p < 0.01$), Myd88 ($p < 0.01$), p-NF-κB ($p < 0.05$), p-P38 ($p < 0.01$) and p-JNK ($p < 0.001$), and the expression of inflammatory cytokines IL-1β ($p < 0.05$),IL-6 ($p < 0.05$),TNF-a ($p < 0.05$),NO ($p < 0.01$) and COX-2 ($p < 0.05$), but didn't influence the expression of p-ERK ($p > 0.05$). After SY stimulation, the expression of TLR4, Myd88, p-NF-κB and p-P38, and inflammatory cytokines declined ($p < 0.05$). Simultaneously, M1 markers iNOS ($p < 0.05$), CD16/32 ($p < 0.05$), IL-12 ($p < 0.05$) and M2 markers CD206 ($p < 0.05$), IL-10 ($p < 0.05$) were elevated after LPS stimulation, but M1 markers significantly declined after SY intervention ($p < 0.05$), while M2 marker CD206 ($p < 0.05$) and IL-10 ($p < 0.05$) were significantly elevated ($p < 0.001$). SY had no influence on M2 marker Arg-1, but the ratio of iNOS/Arg-1 declined compared with LPS-stimulated group ($p < 0.05$), indicating SY converted inflammatory M1 BV2 cells toward anti-inflammatory M2 microglia.
Conclusion: SY exhibited anti-inflammatory effect on BV2 microglia possibly through TLR-4/NF-κB/MAPK signaling pathways and the conversion of M1 to M2 microglia. (Grant: The 2011 Cultivation Project of Shanxi University of Traditional Chinese Medicine, 2011PY-1).

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CNS Infections 1
Rabies virus phosphoprotein induces mitochondrial dysfunction, oxidative stress, and neuronal process degeneration: Implications for future therapy of human rabies

A.C. Jackson[a], W. Kammouni[b], H. Wood[c], A. Saleh[d], C.M. Appolinario[e], P. Fernyhough[f]. *[a]Internal Medicine (Neurology), University of Manitoba, Winnipeg, Canada; [b]Internal Medicine, University of Manitoba, Winnipeg, Canada; [c]Zoonotic Diseases and Special Pathogens, Public Health Agency of Canada, Winnipeg, Canada; [d]Division of Neurodegenerative Disorders, St. Boniface Hospital Research Centre, Winnipeg, Canada; [e]Medical Microbiology, University of Manitoba, Winnipeg, Canada; [f]Pharmacology and Therapeutics, University of Manitoba, Winnipeg, Canada*

Background: Our previous studies in a mouse model of experimental rabies showed neuronal process (dendrites and axons) degeneration in association with severe clinical disease. Cultured adult rodent (mouse and rat) dorsal root ganglion neurons infected with the challenge virus standard-11 (CVS) strain of rabies virus (RABV) showed axonal swellings and reduced axonal growth with evidence of oxidative stress. We have shown that CVS infection alters a variety of mitochondrial parameters and increases mitochondrial Complex I activity and reactive oxygen species (ROS) production.
Objective: To understand basic mechanisms important in rabies pathogenesis.
Materials and methods: We have studied interactions of the RABV and Complex I using immunoblotting, immunoprecipitation, and immunofluorescence. We have expressed rabies virus proteins in cells after transfection of plasmids, including alanine mutagenesis of the RABV phosphoprotein (P), and evaluated Complex I activity and ROS generation.
Results: RABV P was detected by immunoblotting in RABV-infected purified mitochondrial extracts and in Complex I immunoprecipitates from the extracts. A plasmid expressing P in cells increased Complex I activity and increased ROS generation, whereas expression of other RABV proteins did not. Expression of a peptide from amino acid 139–172 of the P increased Complex I activity and ROS generation similar to expression of the entire P protein. Mutational analysis suggests particular importance of the 157 to 169 region of P.
Conclusion: Rabies virus infection is a mitochondrial disorder initiated by interaction of the RABV P and Complex I. This information will be important for the future development of novel therapies for rabies.

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