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Studies in the medicinal potential of *Tridax procumbens*

Orakwelu N O
Nnamdi Azikiwe University, Nigeria

The leaf of the plant *Tridax procumbens* was subjected to extraction by water and methanol Phytochemical screening on the extracts revealed the presence of saponins, tannins, flavonoids, anthracine glycosides, cyanogenic glycosides, protein, carbohydrate and ascorbic acid. The extract were found to be soluble in water, diethyl ether, petroleum ether with aqueous extract insoluble in ethanol and methanol extract soluble in ethanol. The effect of the extract were tested on *Escherichia coli, Staphylococcus aureus, Klebsiella pneumonia, Pseudomonas aeruginosa, Salmonella spp., Aspergillus flavus* and the yeast Candida albicans using agar disc and agar well diffusion methods. All clinical isolates except *Aspergillus flavus* and *Candida albicans* showed zones of inhibition ranging from 10-36 mm at different concentrations (800 mg/ml, 400 mg/ml and 200 mg/ml) using the well method 1.7 – 3.0 mm for both extracts while *S. aureus* and *E. coli* showed zones of inhibition ranging from 0.8 – 2.0 mm at different concentrations (800 – 200 mg/ml) using the disc method for both extracts.

ncheeme@yahoo.com

TAMing inflammation and enhancing myelination in mouse models of multiple sclerosis

Shafit-Zagardo B, Gruber RC, Ray AK, Johndrow CT and García de Frutos P
Albert Einstein College of Medicine, USA

Growth arrest-specific protein 6 (GAS6) is a soluble agonist of the TYRO3, AXL, MERTK, (TAM) family of receptor tyrosine kinases identified to have anti-inflammatory, neuroprotective and promyelinating properties. During experimental autoimmune encephalomyelitis (EAE), wildtype (WT) mice demonstrate a significant induction of *Gas6, Axl,* and *Mertk* but not *Pros1* or *Tyro3* mRNA. We tested the hypothesis that intracerebroventricular (ICV) delivery of GAS6 directly into the CNS of WT mice during myelin oligodendrocyte glycoprotein (MOG)-induced EAE would improve the clinical course of disease relative to artificial cerebrospinal fluid (ACSF)-treated mice. GAS6 did not delay disease onset, but significantly reduced the clinical scores during peak and chronic EAE. Mice receiving GAS6 for 28 days had preserved $SMI31^+$ neurofilament immunoreactivity, significantly fewer $SMI32^+$ axonal swellings and spheroids, and less demyelination relative to ACSF-treated mice. Alternate-day subcutaneous interferon-beta (IFNβ) injection did not enhance GAS6 treatment effectiveness. *Gas6*$^{-/-}$ mice sensitized with MOG_{35-55} peptide exhibit higher clinical scores during late peak to early chronic disease, with significantly increased SMI32+ axonal swellings, $Iba1^+$ microglia/macrophages, and enhanced expression of several proinflammatory mRNA molecules, and decreased expression of early oligodendrocyte maturation markers relative to WT mouse spinal cords with scores for eight consecutive days. During acute EAE, flow cytometry showed significantly more macrophages but not T cell infiltrates in *Gas6*$^{-/-}$ spinal cords than WT spinal cords. Our data is consistent with GAS6 being protective during EAE by dampening the inflammatory response, thereby preserving axonal integrity and myelination. Ongoing studies are examining targeted therapies to efficiently introduce Gas6 into the CNS.

bridget.shafit-zagardo@einstein.yu.edu