



## Anat Elmann

Agricultural Research Organization  
Volcani Center, Israel

### Biography

Anat Elmann is a Research Scientist at the Department of Food Quality and Safety in the Volcani Center, Agricultural Research Organization. She got her PhD from the Weizmann Institute of Science, gained experience as a Researcher in a biotech company, and from the last 12 years she has been studying the effect of plant derived substances on glial and neuronal cells, which play important roles in neurodegenerative diseases and aging. She has presented her work at many different international conferences in Europe and USA, and she is the author of patents and papers on subjects including Neurobiology, Immunology, and the effects of phytochemicals and plant extracts on glial and neuronal cells.

[aemann@volcani.agri.gov.il](mailto:aemann@volcani.agri.gov.il)

## HARNESSING PHYTOCHEMICALS TO SUPPORT THE NERVOUS SYSTEM

Oxidative stress and amyloid beta toxicity are involved in the pathogenesis of Alzheimer's diseases. We have previously demonstrated that an extract prepared of the plant *Achillea fragrantissima* (*Af*) protected cultured brain astrocytes from oxidative stress-induced cell death and down regulated microglial activation. Using activity guided fractionation, we have purified from *Af* an active flavonoid named 3, 5, 4'-trihydroxy-6, 7, 3'-trimethoxyflavone (TTF). TTF protected cultured astrocytes from H<sub>2</sub>O<sub>2</sub>-induced cell death via interference with cell signaling (inhibition of SAPK/JNK, ERK 1/2, and MEK1 phosphorylation) and by reducing the levels of oxidative stress-induced intracellular reactive oxygen species (ROS). The mechanism of the protective effect of TTF against H<sub>2</sub>O<sub>2</sub>-cytotoxicity could not be attributed to a direct H<sub>2</sub>O<sub>2</sub> scavenging but rather to the scavenging of free radicals as was shown in cell free systems. In addition, TTF protected cultured neuronal cells from amyloid beta cytotoxicity via interference with cell signaling events and by reducing the amyloid beta-induced levels of intracellular ROS. Moreover, TTF exhibited anti-inflammatory activities and inhibited the LPS-elicited secretion of the proinflammatory cytokines interleukin 6 (IL-6) and IL-1beta from microglial cells. Our results suggest that TTF might be a therapeutic candidate for the treatment of Alzheimer's disease as well as other neurodegenerative diseases where oxidative stress, neuroinflammation and amyloid beta toxicity are part of the pathophysiology.



Note: