

Cellular resistance to methylmercury.

[Neurotoxicology](#). 1996 Spring;17(1):27-36.

[Sarafian TA](#), [Bredesen DE](#), [Verity MA](#).

Department of Pathology, UCLA Center for Health Sciences 90095, USA.

The destructive pathologic and biochemical consequences of methyl mercury have been extensively described. Relatively little is known, however, about the defensive mechanisms neurons employ to protect themselves against mercurial injury. Studies using a variety of cell types have disclosed several inducible biochemical responses to heavy metal and pro-oxidant insult. These responses include modulation of cellular levels of glutathione, metallothionein, hemoxygenase, and other stress proteins. With few exceptions, however, neurons appear to be markedly deficient in these responses. This suggests the possibility that the phenomenon of selective vulnerability of cells in the nervous system to mercury and other heavy metals may arise from a critical absence of inherent protective mechanisms. Transfection of neural cells with the anti-apoptotic gene, bcl-2, confers partial resistance to a variety of neurotoxins, including methyl mercury. This approach to providing neuroprotection may represent an efficacious strategy for combating tenacious neurotoxins such as methyl mercury.

