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It is now widely accepted that astrogliosis represents a dominant response of the adult central nervous system to all types of injuries. This "reactive" state of the astrocyte can be induced by a diverse array of toxic substances as well as by neurological disease states and traumatic or ischemic injuries. Historically, astrogliosis was viewed as a permanent response involving cell division and scarring. We now are aware of the dynamic capacities of this neural cell type, including it's response to toxicant-induced injuries, i.e. time-dependent astrocytic hypertrophy, not hyperplasia. The reactive astrocyte, *in vivo*, can be monitored qualitatively and quantitatively by immunohistochemistry and immunoassay, respectively, of the astrocyte intermediate filament protein, GFAP. GFAP mRNA can now be reliably quantified, as well, using Taqman technology. The signaling mechanisms underlying the astroglial response to injury remain largely unknown but progress is being made through molecular analyses, e.g. by using inducible transgenic mice and the application of expression arrays to injury models. One area where the old dogma persists is in developmental neurotoxicology. The prevailing view remains that astrogliosis is diminished or absent in the developing nervous system. Despite this notion, we and others have used dose-, time- and region-dependent analysis of GFAP to show a robust astroglial response to diverse insults of the developing nervous system. These include early postnatal exposure to trimethyltin, triethyltin, cadmium, 6-OH dopamine, methamphetamine, NMDA, carbon monoxide, ethanol and bilirubin. Prenatal exposures to methylazoxymethanol, methylmercury and ischemia also result in a postnatal increase in GFAP. All of these responses were target appropriate and suggest that, as in the adult nervous system, astrogliosis is a characteristic response to neural damage, regardless of the nature or the site of damage. Assessments of reactive gliosis should be incorporated into future developmental neurotoxicity testing paradigms.