documented in Mervis's article, however, the time for such a revival is growing short.

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Collaborating on Public Health Issues

MARCIA HAMBURG'S EDITORIAL "PUBLIC health preparedness" (22 Feb., p. 1425) reminds us that public health remains the essence of domestic security. We neglect it at our peril. But if the anthrax scare was a wake-up call, many states have hit the snooze-alarm, cutting basic public health services yet taking federal dollars earmarked for "bioterrorism." And while federal agencies give monies to states to "plan their planning efforts" and fund yet more "needs assessments," urgent public health needs go unmet.

Computer programmers in the Open Source movement (GNU/Linux) have shown how motivated talent can solve problems collaboratively regardless of institutional borders or commercial or official sanction. Academics and public health professionals in New England are organizing a mechanism to allow the many people with talent and experience in our region to contribute to solving the problems facing public health. We are also working with New England academic institutions to provide cross-institutional educational opportunities for public-sector workers who need additional training to meet the new challenges. New challenges sometimes require new solutions.

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Differing Views on Spinal Cord Repair

IN HIS VIEWPOINT "REPAIRING THE INJURED spinal cord" (Bodybuilding: The Bionic Human, 8 February, p. 1029), Martin E. Schwab summarizes four different repair strategies, namely, neutralizing growth inhibitors, grafting of peripheral bridges (both strategies for inducing regeneration), restoring the activity of remaining fibers, and increasing neuronal plasticity. His lack of emphasis on neuroprotection (rescue of spared axons from delayed posttraumatic degeneration) is puzzling because he points out that in patients with spinal injury, complete anatomical separation of the spinal cord is very rare. This would suggest that spared neurons should receive attention to ensure their continued viability and function. Such neuroprotection is a prerequisite for the therapeutic strategies he mentions.

Schwab's sole reference to neuroprotection concerns treatment with methylprednisolone, currently the only drug approved for use in patients with spinal cord injuries. He comments that whether inflammatory reaction causes further damage to the spared neurons is a matter of debate. We suggest that this statement is an oversimplification. Inflammation is not a single phenomenon of uniform manifestation, but rather a variety of processes that vary in nature, complexity, and outcome. Accordingly, and in light of recent findings in this connection, it would seem that the time has come to stop considering inflammation as "good" or "bad" for recovery, and instead to recognize that inflammation is the way through which the body heals itself and hence that therapeutic intervention should be aimed at controlling and boosting rather than suppressing it.

It is widely acknowledged that the immune system protects us from damage inflicted by external pathogens. A considerable body of evidence indicates that when the damage is caused by an insult that is the result not of foreign pathogens but of destructive self-compounds, protection can be achieved physiologically through an immune response directed against self-compounds. This autoimmune mechanism of spinal cord repair can be boosted by a variety of manipulations, such as transplantation of activated macrophages, passive immunization with autoimmune T cells, or active posttraumatic T cell–based vaccination with myelin peptides (*1*–*6*). These treatments do not merely "enhance myelin clearance." They serve the strategic purpose of boosting a well-controlled inflammation as a tool, directing immune cells to the lesion site (by vaccination with myelin antigens) and helping the body to apply its own repair mechanism for protection and regeneration.

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Response

SCHWARTZ AND HAUBEN MISS THE WELL-established point that neuroprotection and repair are two very different kinds of processes.

Neuroprotection has to occur acutely, in the case of the central nervous system within minutes, hours, and perhaps the first few days after the lesion. The processes involved in secondary injury are extremely complex, and the many ways that have been tried to effectively preserve central nervous system tissue after severe trauma or ischemia (stroke) have been largely unsuccessful. The recent data of Schwartz and collaborators showing protective roles of the immune system are very interesting but require confirmation, as findings from other labs emphasize a damaging rather than a protective role of the immune/inflammatory system. My own view is that there is probably a fine balance between positive and negative effects, both probably happen, and we certainly don't understand this system at present. Accordingly, the literature is vast and rather inconclusive. It is for these reasons that I restricted my Viewpoint to aspects of repair rather than neuroprotection.

Repair happens after the damage is done, and the mechanisms involved in spinal cord repair, especially as far as experimental manipulations are concerned, are very different from neuroprotective approaches. The separation of neuroprotection and repair is not only valid in the spinal cord field, but also in brain injury and particularly in stroke. I see no reason to confuse these issues, and I also do not think it is possible to make adequate and in-depth, balanced statements on neuroprotection in the context of a short note beyond what I have already done in the introductory paragraph of my Viewpoint.

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CORRECTIONS AND CLARIFICATIONS

REPORTS: "Field-effect modulation of the conductance of single molecules" by J. H. Schön *et al.* (7 Dec., p. 2138). An incorrect version of Fig. 4 appeared in print. The correct version appears here.