The term “neuroprotection” is often misused, overused, or misunderstood. A reasonable definition of neuroprotection refers to the preservation of “neuronal structure and/or function.” Although our knowledge about the cellular and molecular mechanisms of neurodegeneration has expanded, experimental systems and animal models that mimic the process or allow translation into clinical success remain limited. This editorial discusses reasons for this gap and strategies to close it. Experimental models can only mirror certain aspects of disease mechanisms in humans. Therefore, findings in these models need to be linked with patient data to improve real-life relevance. Successful neuroprotection depends on finding the right “window of opportunity” which varies from very short (stroke) to very long (Alzheimer’s disease), necessitating the need to focus on strategies for very early disease recognition. This need challenges the strategies to be chosen, trial approaches and methodologies, and the allocation of resources. Additionally, outcome measures are often not well suited to assess neuroprotection. To this end, surrogate measures, including biomarkers, are useful endpoints to demonstrate evidence of target directed therapeutic utility. Finally, studies have shown that
neuroprotection is not likely to succeed when targeting only one pathway. These obstacles have
reduced the level of enthusiasm for neuroprotection in certain disease areas (e.g., stroke). Academia,
industry, regulatory authorities, funding agencies and patient organizations have to cooperate to a
greater extent in order to overcome these impediments and to encourage nonclassical concepts.
These concepts will be interdisciplinary in order to achieve meaningful disease modification.

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