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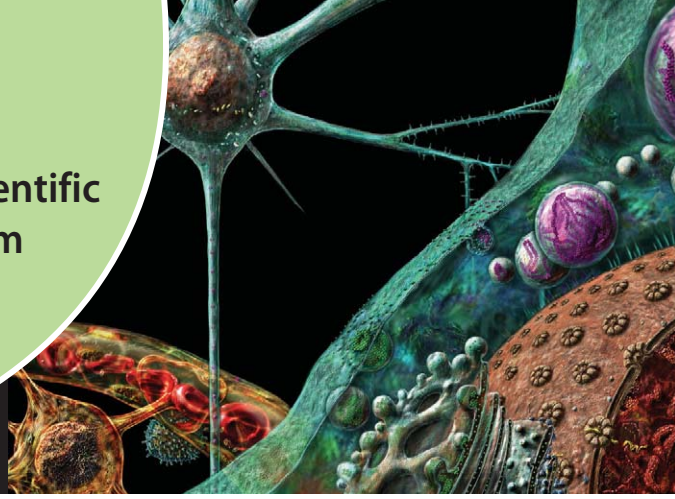
Prions: Introducing a Complex Scientific Controversy to a Biology Classroom



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It has been almost 50 years since Thomas Kuhn, in *The Structure of Scientific Revolutions*, posited that science does not progress by the steady accumulation of knowledge, but rather by a system of competition among paradigms. They vie for supremacy through greater parsimony, explanatory power, and popularity among the community of scientists (Kuhn, 1962). The current controversy concerning the identity of prions (PrPs) (proteins devoid of the nucleic acid) as the infectious agents of transmissible spongiform encephalopathies (TSE) elucidates all the issues involved in just such a debate.

While modern biology high school and university textbooks cover many scientific controversies that have been resolved decades and even centuries ago, they fail to cover current scientific disputes. This article is intended to address such an omission by introducing the prion controversy in biology classes in high schools and colleges.

In 1982, biochemist and neurologist Stanley Prusiner proposed a hypothesis concerning infectious proteins. He identified them as abnormal prions, proteinaceous infectious particles capable of converting normal prions (naturally present proteins in mammals) into an abnormal form causing a fatal disease of the central nervous system (CNS) in both animals and humans. Heretofore, it had been accepted that infections could be caused only by protozoans, fungi, bacteria, rickettsia, viruses, or viroids. Only nucleic acids, informational polymers, were known to be able to duplicate themselves, not proteins. For the discovery of prions which Prusiner posited can cause TSE, he received a Nobel Prize in 1997.

Were Prusiner's hypothesis correct, our understanding of the organic world would be changed forever. However, Laura Manuelidis (2007), one of most dedicated scientists in this field and the head of neuropathology at the Yale School of Medicine, contends that "prions thereby became canonized, although careful review of data revealed many discrepancies." Indeed, even Nobel Prize winners can err (Allchin, 2008), including Prusiner, and prions thus remain in the realm of a hypothesis (Manuelidis, 2007).

Despite overwhelming opposing data published in *The Lancet*, *Science*, *Virology*, *The Journal of Virology*, *Journal of Cellular Biochemistry*, *Viral Immunology*, *Journal of NeuroVirology*, *Proceedings of the National Academy of Sciences*, and many other scientific publications, most, if not all, biology textbooks in the U.S. present prions as the primary cause of TSE. While there are scientists convinced of the ability of abnormal prions to cause infections, there are other scientists who, based on their observations and experimental data, do not think that prions could become infectious. Manuelidis suggests that prions may simply be part of the late stage of a disease, not part of the cause (Mihailova, 2007), and PrP infectivity is questionable, and perhaps non-existent (Manuelidis, 2007).

TSE continues to spread throughout the world, killing people who eat the tissue of cattle infected with BSE, children treated with human growth hormone, patients in surgery ... herds of sheep, cattle, deer, elk, and mink.

Cannibalism & the Rise of TSE

Since students seem more engaged when instructors incorporate examples from popular culture into classroom discussions (Pryor, 2008), one might start a consideration of prions with mention of Kurt Vonnegut's science fiction novel, *Cat's Cradle* (1963), before introducing *Deadly Feasts* (1997), a shocking nonfiction case history of the discovery and epidemiology of the fatal disease TSE. Certainly, truth is stranger than fiction if one were to contrast Richard Rhodes' documented study and any of Vonnegut's science fiction novels. *Cat's Cradle*, concluding in an apocalyptic climax, concerns the ability of a nucleant that can turn water into ice, just as an abnormal prion can allegedly turn its host prions into abnormal forms, resulting in this fatal brain pathology. *Deadly Feasts* begins with a description of a burial ceremony that the women of the Fore tribe used to practice in New Guinea, "the last wild place on earth." Sixty or more native women with their babies and small children, the family of a deceased woman, would gather to bury her in their stomachs rather than abandon her to rot in the ground. "Why should we throw away good meat? It is not right," one woman told an anthropologist (Rhodes, 1997). The mourners would eat body parts, including "the bones, which they charred in the open fire to soften them," "even the feces would be eaten, mixed with edible ferns and cooked in banana leaves" (Rhode, 1997). Fore woman recalled that cannibalizing the corpses of their kindred "started within the lifetime of the oldest grandmother." "I eat you," was a Fore greeting (Rhode, 1997). The deceased who died of leprosy or diarrhea were not consumed.

By 1950, a disease called kuru (KOO-roo), which means shivering with cold or fear, had killed women in every Fore village. One of the most pronounced symptoms would be unprovoked laughter. Because of this, the disease became known as "laughing death." The victims would lose their ability to walk, shiver uncontrollably but not from cold or fear; their speech would become blurred. Finally, their ability to swallow would be so impaired that their relatives would have to chew food for the dying victims and force it down their throats. Such symptoms were considered to have been caused by bewitchment. Nevertheless, the flesh of women killed by sorcery was considered clean enough to be eaten by other