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Mad Cow Disease From Drinking Water?

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Bovine spongiform encephalopathy (BSE) is believed to be transmitted by the ingestion of proteinaceous agents called prions, which accumulate in the brain and spinal cord of infected bovines. Concern has been expressed about the risks of transmission of BSE to humans through BSE prions discharged to the aquatic environment from rendering plants, abattoirs, and landfills. The disease-related form of the prion protein is relatively resistant to degradation, and infectivity decays rather slowly in the environment. Levels of disinfection used for drinking-water treatment would have little effect.

Gale and co-investigators recently modeled the risks from a rendering plant disposing of cull cattle carcasses in the catchment of a chalk aquifer, which is used for a drinking-water abstraction. The risk-assessment approach focused on identifying the hydrogeological and physical barriers that would contribute to preventing BSE infectivity gaining entry to the aquifer. These barriers included inactivation of BSE agent by the rendering process, removal from the effluent by treatment at the plant, filtration and adsorption in the clay and chalk, and dilution in the ground water. The importance in environmental risk assessment of the cow-to-man species barrier is considered. Two key conclusions about the environmental behavior of the BSE agent are that

prion proteins are "sticky" and bind to particulates and that the millions of BSE prion molecules comprising a human oral ID₅₀ are subject to some degree of dispersion and hence dilution in the environment. Assuming the rendering plant processes 2,000 cull cattle carcasses per week, the risks to drinking-water consumers were estimated to be remote. Indeed, even using worst-case assumptions, an individual would have to consume 21 dL of tap water for 45 million years to have a 50% chance of infection through drinking water drawn from the aquifer.

FROM: Gale P, Young C, Stanfield G, Oakes D. Development of a risk assessment for BSE in the aquatic environment. *J Appl Microbiol* 1998;84:467-477.