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The existence of shared antigenic sites between the polysaccharides and the gangliosides may relate to the poor immune response against the *E. coli* K1 and *N. meningitidis* group B capsular antigens.⁶ This does not necessarily mean that antibodies against these polysaccharides are dangerous since naturally occurring autoantibodies against common glycolipids are present in human beings without causing obvious harmful consequences.⁷

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SODIUM HYDROXIDE DECONTAMINATION OF CREUTZFELDT-JAKOB DISEASE VIRUS

To the Editor: A useful method of chemical (sodium hydroxide) decontamination of the virus of Creutzfeldt-Jakob disease (CJD) has been successfully tested in our laboratory. In view of the mounting concern of medical and paramedical personnel about contact with patients with CJD or their tissues, we have attempted to develop practical decontamination procedures for laboratory and hospital use.

In an earlier report,¹ we documented the effectiveness of a one-hour exposure to 0.5 per cent sodium hypochlorite (1:10 dilution of household bleach) for inactivation of a guinea pig-adapted strain of CJD virus in tissue suspensions that contained about the same concentration of virus (5 to 6 log₁₀LD₅₀ per gram of tissue) as infected human brain tissue. In practice, however, the use of hypochlorite is limited by its variably corrosive effects on fabrics, metals, and skin.

Because 6 to 8 M urea has been reported to produce nearly complete or complete inactivation of partially purified scrapie virus,² and because urea is noncorrosive and innocuous to skin, it would appear to be an ideal candidate as a decontaminant; however, we have found 8 M urea to be almost without effect on crude tissue suspensions of either scrapie or CJD virus.

We have now found that exposure of a crude 10 per cent brain suspension or of brain-tissue fragments from guinea pigs infected with CJD virus to either 1 N or 0.1 N sodium hydroxide for one hour at room temperature inactivates all detectable infectivity: the untreated control brain contained 5.5 log₁₀LD₅₀ per gram of tissue, and there were no deaths at the lowest testable (10⁻¹) dilution of treated brain. We have also found that, unlike hypochlorite, 1 N sodium hydroxide sterilizes the much higher titers that are present in brain tissue of hamsters infected with the 263K strain of scrapie virus, and thus may be presumed to provide a greater margin of potency than hypochlorite for the inactivation of CJD virus. Sodium hydroxide at these concentrations is less corrosive to many fabrics, plastics, and metals than is hypochlorite ion. It may also be preferable for the decontamination of skin, since personal experience has

shown that a 10-minute exposure of finger or forearm skin can be tolerated with only minor irritation.

Previous experiments with the viruses of both CJD and scrapie^{1,3,4} have demonstrated that chemical inactivation by most disinfectants is largely completed within the first few minutes of exposure and that little if any additional inactivation occurs after longer periods of time. Experiments currently in progress will show whether this is also true for sodium hydroxide.

We still recommend autoclave sterilization for one hour at 121°C² or at higher temperatures⁵ when possible. When the autoclave is impractical we recommend a one-hour exposure to 1 N sodium hydroxide; we have found that sodium hydroxide is less corrosive than hypochlorite for all materials except aluminum and that its nonvolatile properties are especially desirable in the decontamination of work surfaces. We suggest that contaminated skin may be disinfected with little hazard by 5 to 10 minutes of exposure to 1 N sodium hydroxide, followed by extensive washing with water.

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HYPOPARATHYROIDISM IN WILSON'S DISEASE

To the Editor: We enjoyed Dr. Scheinberg's perceptive editorial (Oct. 13 issue) on the value of investigating rare diseases.¹ Elucidation of the mechanism of Wilson's disease and the subsequent successful treatment of this condition with penicillamine may also have shed considerable light on the etiologic mechanism of a rare birth defect, Ehlers-Danlos syndrome (dermatorrhexis cutis hyperelastica). This defect, which occurs spontaneously as a probable result of mendelian inheritance, also appears to be associated with maternal use of penicillamine in pregnancy.²

Penicillamine inhibits condensation of soluble tropocollagen because of affinity to the aldehyde. It chelates with unbound copper as well as with zinc. It also causes mesenchymal suppression and alters elastin cross-linking by lysine aldehyde condensation. It has been suggested that penicillamine is less teratogenic in pregnant women with Wilson's disease than in pregnant women taking it for other conditions.² One possible explanation for the reduction in teratogenicity is that in patients with Wilson's disease sufficient copper remains unchelated to meet fetal requirements. Support for this theory has been demonstrated by Hurley and her co-workers, who showed that the teratogenicity of penicillamine in rats could be reduced by dietary supplementation with copper.³

We offer this as another example of how the results of investigating a disease that practically "no one's got" may have some unusual and unexpected benefits.

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