diffraction analysis of the mudflow clays (derived from Liassic shales) shows the presence of large amounts of montmorillonite clay together with illite and kaolinite (Fig. 1A). The mean ratio of montmorillonite plus illite to kaolinite is 9.5 : 1. The mudflow is seasonal in character and related to the hydration of clays in the shales by rainfall, which averages 105 cm per year.

On St. Lucia island, British West Indies, 13°44′N, 60°57′W, a series of coastal landslides are also associated with almost pure, swelling montmorillonite clay (Fig. 1B), derived from weathering volcanic bedrock. These landslides appear to move erratically and are related to water saturation of the clays during periods of heavy rainfall. Rainfall rates of up to 9.7 cm per day have been recorded in an area which locally averages 125 cm per year.

In the Scotland district of Barbados, 13°00′N, 59°30′W, Tertiary shales rich in montmorillonite, kaolinite, and illite (Fig. 1C) are exposed by erosion of a coral cap. These clay minerals contribute to widespread landslide activity, which is now being studied by the Soil Conservation Service of Barbados. This slope instability occurs within an area that receives 125 to 200 cm of rainfall annually.

The close association between landslide activity and the presence of montmorillonite-rich clays in Northern Ireland, St. Lucia, and Barbados is not coincidental. Bentonite clays are susceptible to changes in physical properties, such as plasticity, when subjected to wetting and drying. These changes are further enhanced in the presence of highly hydrated sodium and magnesium exchangeable ions (*5*). Sodium ions are associated with the clays in both St. Lucia (up to 0.59 percent) and Northern Ireland (up to 0.17 percent).

Climatic conditions which allow alternate hydration and dehydration of such clays can be found almost everywhere except in truly arid areas. Certainly, the periodicity of movement in bentonite landslides can be influenced by climatic factors. In arctic areas, hydration may be restricted to periods of thaw, thus imparting a seasonality to the landslide activity. Alternatively, in temperate and tropical areas effective precipitation can be reduced by evapotranspiration factors (*4*). However, there is a limit to the relevance of climatic criteria since groundwater supply of moisture may exceed the importance of direct precipitation. Thus, the primary factor in the location of many flow-type landslides is not a simple climatic one, but it is rather the presence of bentonite clay minerals in the slope materials. The distribution of bentonite landslides is thus largely geologically controlled.

The landslides described by Anderson *et al.* provide additional evidence of the relationship between slope instability and the mineralogy and the geochemistry of slope materials. But, it is readily apparent that there is no justification for associating these landslides with a particular climatic environment.

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It was not our intent to convey the impression that all bentonite-rich mud flows are unique to the Arctic. We recognize that mud and debris flows in clay soil occur on a worldwide basis. What we do believe may be unique about the bentonite flows near Umiat has to do with channel morphology, the relation between slope angle and the flow regime, the frequency of flow activity, and the density of the flow channel distribution on permafrost-underlain terrain. Climatic factors play a significant role in governing mud- and debris-flow regimes; however, we would not maintain that these factors are dominant over geologic aspects.

We do not maintain that bentonite debris flows are restricted to Arctic regions; we believe, however, that the frequency and morphology presented by the bentonite debris flows when they occur over permafrost are sufficiently distinctive that, on aerial photographs, bentonite debris flows may be differentiated from other types of flows with considerable reliability. The examples of mud or debris flows described by Prior and Ho do not appear to have a morphology like that of the Umiat bentonite flows. No confirmed examples of similar flows have been brought to our attention since our earlier communication, although one of us (Brown) has learned on a recent trip to Siberia that somewhat similar flows do occur there.

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α_1-Antitrypsin Deficiency

The unusually high incidence of heterozygous α_1-antitrypsin deficiency reported by Kueppers *et al.* (*1*) in both normal and emphysematous populations requires careful and critical examination of the method employed for detecting such heterozygosity. Kueppers *et al.* report that their antigen-antibody crossed-electrophoresis procedure distinguishes between normal α_1-antitrypsin and an electrophoretically distinct but antigenically identical species of α_1-antitrypsin in the heterozygote. This assertion is based upon the observation that all obligatory heterozygotes (offspring of known homozygotes) have a distinctive antitrypsin pattern on crossed electrophoresis. However, Kueppers *et al.* present no evidence to confirm the heterozygosity of those subjects with a heterozygous pattern in their healthy control population, as could be done through family studies. In addition, they say that a mixture of equal parts of serum from normal and deficient homozygotes yields the same pattern as that of a heterozygote. Kueppers and Bearn (*2*) earlier stated that serums from individuals homozygous for the deficient gene show a virtual absence of α_1-antitrypsin bands. Thus, one wonders why mere dilution of normal serum antitrypsin by another serum specimen lacking antitrypsin should result in a pattern resembling that seen in heterozygous deficiency. Kueppers *et al.* apparently did not determine the type of antitrypsin pattern that results when normal serum is mixed with another serum deprived of its α_1-globulin by

shaking with chloroform or acetone. If a "heterozygous" pattern results from this maneuver, then this pattern is due merely to low concentrations of antitrypsin globulin and is not the result of an "electrophoretically distinct molecule." In fact, we have observed that the electrophoretic mobility of the α_1-globulin on polyacrilamide gels varies with the dilution of serum protein.

The slight variation in antitrypsin pattern by which Kueppers *et al.* distinguish heterozygous α_1-antitrypsin deficiency from normal very likely does include all heterozygotes. However, this pattern may also occur with serums from individuals without the inherited deficiency whose α_1-antitrypsin nonetheless is normally low.

Kueppers *et al.* point out that the concentration of antitrypsin can be raised to the normal range in heterozygotes by conditions such as infection and pregnancy. However, they imply that the diagnostic variations of antitrypsin patterns seen on antigen-antibody crossed electrophoresis remain unchanged. No evidence for this statement is presented.

The difference between heterozygous deficient and normal individuals may depend less on the subject's minimum unstimulated antitrypsin and more upon differences in ability to increase this level during an inflammatory process (*3*).

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On antigen-antibody crossed electrophoresis, the position of the different α_1-antitrypsin peaks characteristic for the different phenotypes remains the same over a wide range of concentrations. I have followed the α_1-antitrypsin pattern by antigen-antibody crossed electrophoresis of several heterozygotes for the deficiency gene, when their serum α_1-antitrypsin was low and when it was high (from 105 to 240 mg/100 ml of serum). The position of the peaks did not change; the height of the peaks varied with the concentration of α_1-antitrypsin. The precipitation pattern of a heterozygote can be explained by the presence of two groups of electrophoretically distinct but antigenically identical α_1-antitrypsin components (*1*). Although the α_1-antitrypsin bands in stained starch gels are usually weak or absent, homozygotes for the deficiency gene have a serum concentration of 25 ± 6 mg/100 ml (*2*).

As Lieberman suggests, heterozygotes respond differently to an inflammatory stimulus from normals. After an injection of typhoid vaccine normal individuals show an increase in serum of α_1-antitrypsin from approximately 200 mg to an average of 375 mg/100 ml, and heterozygotes show an increase from 112 mg to 196 mg/100 ml (*3*).

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Monosodium Glutamate: Specific Brain Lesion Questioned

Olney and Sharpe (*1*) administered, by subcutaneous injection, a single dose of monosodium glutamate (15 mmole per kilogram of body weight) to one newborn premature rhesus monkey and concluded that the lesion in the periventricular arcuate region of the hypothalamus was specifically induced by glutamate.

They stated "We have demonstrated susceptibility of a primate species to the mechanism of the glutamate effect" and "Presumably an elevated blood concentration of glutamic acid is an important requisite to the lesion formation." However, they did not supply data on concentrations of glutamic acid in the blood, nor did they supply control data for their experiment. A minimum requisite would have been the use of inorganic sodium salts administered in a way similar to that used for monosodium glutamate, the use of sodium salts of several other amino acids, and the administration of glutamic acid as the free acid. Perhaps most critical is the fact that they used only one animal.

An explanation as plausible as that presented is that the findings reflect a nonspecific solute effect. Finberg *et al.* (*2*) have shown that an intravenous or intraperitoneal dose of many univalent completely ionized salts (15 meq per kilogram of body weight) given to an infant cat reduces the cerebral spinal fluid pressure to zero in 180 minutes (the time interval used by Olney and Sharpe). Under these circumstances, there is always dilatation of capillaries and small vessels and occasionally, at this dosage, tearing of vessels in the Virchow-Robin space, with consequent hemorrhage. The specific lesion presumably varies with the conformation of the intracranial contents and skull of the animal model used.

The implication of a risk of brain damage as a result of human infants' consuming infant food products is unsupportable. Infant food products on the market, until recently, contained between 150 and 180 mg of monosodium glutamate per jar. A few contained as much as 600 mg per jar. A 5-kg infant would have had to consume, at one sitting, 20 jars of an infant food product containing 600 mg per jar to obtain the amount of monosodium glutamate used by Olney and Sharpe. The controversy surrounding monosodium glutamate in infant food products has aroused fear on the part of mothers that their infants would become mentally retarded as a result of their having been fed commonly available infant food products. There obviously was no such risk and no basis in fact that this could have occurred.

The issue of whether or not monosodium glutamate should be in infant food products is a different one. The amino acid serves no useful nutritional purpose either as a flavoring agent or as a supplement to an infant diet already high in glutamate as a result of milk and wheat content. Furthermore, the addition of glutamate probably increases the price of the product, and under these circumstances inclusion cannot be condoned. However, this issue is completely different from the proposition that a specific neurological lesion occurs in infants receiving these foods, and publishing information based on an experiment in a single animal with no controls is only confounding.

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