

that in mice and guinea pigs a minor metabolite of diethyl disulfide was ethyl methyl sulfone, the immediate precursor of which was presumably ethyl methyl sulfoxide (5). The fact that administration of dimethyl sulfide does not increase the urinary sulfate output in rats (6) suggests its oxidation to dimethyl sulfone (5). However, although there has been recent interest in dimethyl sulfoxide for many possible therapeutic uses (7), knowledge of its metabolism is lacking. DiStefano and Borgstedt (8) have shown the reduction of the compound to dimethyl sulfide in the cat, and our results in the rabbit demonstrate an additional metabolic route.

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Cradleboard Hoods, Not Corsets

Abstract. *Utmost caution should be exercised in interpreting archeological data out of context. Certain artifacts described in the literature on human paleopathology as therapeutic corsets are in fact hoods for cradleboards. Close cooperation among specialists in various disciplines is desirable in reconstructing aspects of prehistoric culture.*

Archeology is the subfield of anthropology which is concerned with prehistoric cultures, and modern archeology is more than mere collection of antiquities, in that archeologists do everything possible to both uncover and interpret the objects of prehistory in context. Context means both the

specific associations of an artifact at the time of discovery and the general pattern of the particular culture from which the artifact comes. Nonarcheologists who attempt to draw inferences from archeological data are frequently unaware of the necessity of such context, and sometimes draw functional inferences based solely on the observable attributes of the artifact itself in terms of their own culture. Such identifications are not necessarily incorrect, but, if made, should be used with extreme caution in drawing further inferences, and should be abandoned whenever positive proof of the real use becomes available. One such error in the literature on human paleopathology, written by medical men, is the erroneous identification of the bark hoods of prehistoric cradleboards (Fig. 1) from southwestern United States as therapeutic corsets. This identification implied that the ancient southwesterners had relatively advanced medical knowledge. Our purpose is to correct this misinterpretation and to point out the necessity for making inferences in context.

The objects in question are rectangularoids of heavy bark, about 75 cm long and 22 cm wide, bent into a U-shape. Two opposing corners are rounded, the others square. The margins of the objects are perforated.

The earliest tentative identification of one of these curved bark bands as an orthopedic corset appeared in a paper by Freeman in 1918 (1). It was based on examination of a specimen in the collection of the Colorado Historical Society in Denver. Freeman noted the artifact's close resemblance to modern orthopedic corsets used in the treatment of spinal lesions and stated further that the corsets "may have been used . . . for this purpose or for the treatment of rib fractures." The society's catalog gives no information on either the general provenience of this artifact or its specific associations, and questionably identifies it as a piece of bark armor.

Moodie in 1923 followed this earlier identification in both his popular book (2) and his scientific tome on paleopathology (3). In the latter he added that the band was "doubtlessly used for the treatment of spinal lesions and [suggests] . . . considerable knowledge of spinal disturbances." In 1924 Freeman (4) reaffirmed his earlier identification and called the object a "corset made of bark with eyelets and cord

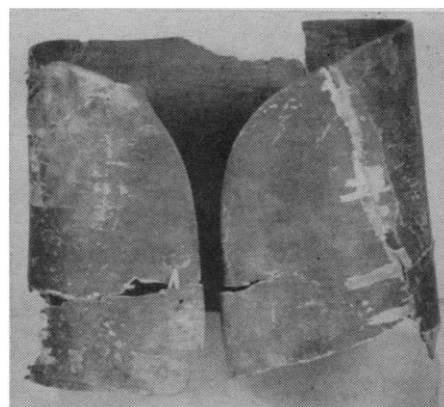


Fig. 1. Prehistoric cradleboard hood from an infant's grave at Tseahatso rock shelter. University of Colorado Museum specimen 2590.

for lacing it around the body. Possibly used for some orthopedic purpose." The most recent repetition of this identification occurred at a symposium on human paleopathology in January 1965 (5).

In terms of our own culture it is not an illogical speculation that these curved bands of bark served as orthopedic devices for bracing injured backs and that the eyelets were for lacing the edges together. However, specimens for which the archeological context is known demonstrate that these artifacts were hoods for cradleboards. As for the perforations, the holes along the lower margin were for binding the hood to the backrest, and, by analogy with the prehistoric Anasazi culture from which these cradleboards come, the perforations along the front edge were not eyelets for lacing cords, but mending holes. The latter was made by drilling two holes, one on each side of a crack. A cord through the two holes bound the crack and prevented further splitting. This method was widely used by the Anasazi for repairing baskets, skin bags, and, particularly, cracked pots.

Both actual cradleboards with this type of hood and clay effigies of them have been found in several localities in southwestern United States. Their associations indicate that they date to the period known as Pueblo III (A.D. 1050–1275) and belong to the local cultural tradition known as the Anasazi. The most complete specimen was illustrated by Guernsey in 1931 (6). It came from an infant's grave in a dry cave in Adugegi Canyon in northeastern Arizona. This specimen is in nearly perfect condition, with the hood still attached to the backrest.

Pottery dating to Pueblo III, as well as other items, accompanied it. A second complete cradleboard with a bark hood was found in Moqui Canyon in southern Utah (7); it was poorly preserved but still held the remains of an infant. Pottery from the site, although not directly associated, suggested a Pueblo III time of placement. A third cradleboard with a bark hood (Fig. 1) was found in 1924, associated with an infant burial at Tseahatso, a large, dry, rock shelter in Canyon del Muerto in northeastern Arizona. This find has not previously been published. The hood had become detached from the backrest. Associated pottery indicated a Pueblo III date. Small clay effigies of infants in cradleboards with hoods of this shape have been found at Waterfall Ruin in northeastern Arizona (8) and near Flagstaff, Arizona (9); the latter effigies were parts of handles of ceramic dippers. The contexts clearly demonstrate the true function of these curved bands of bark. The earlier identification of these cradleboard hoods as corsets on the basis of a specimen lacking known context demonstrates the difficulties faced in attempting to interpret archeological materials out of context.

Calvin Wells wrote (10) that "it is most unwise for anthropologists who lack clinical training to venture into the infinitely subtle field of ancient disease." The specific corollary is obvious. To it we would like to add another: the cooperation of specialists in many professions is required to reconstruct the subtle aspects of prehistoric cultures.

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Antigen-Antibody Reaction: Nature of Complex Initiating Delayed Hypersensitivity

Abstract. *Two homologous lightly coupled dinitrophenyl conjugates of poly-L-lysine of differing average molecular sizes were compared with regard to their abilities to elicit in guinea pigs specific delayed hypersensitivity skin reactions, passive cutaneous anaphylaxis, and active Arthus reactions. Equal concentrations by weight (but not equimolar concentrations) of the two conjugates elicited equally intense delayed hypersensitivity reactions and Arthus reactions, whereas equimolar concentrations (but not equal weight-concentrations) elicited equally intense passive cutaneous anaphylaxis reactions. These results suggest that delayed hypersensitivity reactions are initiated by the reaction of antigen with antibody molecules in true solution, and not by the simple bridging by antigen of a small number of antibody molecules firmly fixed to cell membrane surfaces. Whether "sensitized cells" or circulating "delayed hypersensitivity antibodies" are the specific mediators of the delayed hypersensitivity reactions is discussed.*

Delayed hypersensitivity reactions (DHR) are characterized by their slow evolution, their histological appearance (mononuclear cell infiltrate), and their ability to be passively transferred by lymphoid cells, but not by serum, from hypersensitive donors. On the basis of these observations and other evidence, these reactions have been widely viewed as being mediated by "sensitized mononuclear cells" and not by freely circulating antibodies (1). The nature of "sensitized cells" has not been defined, but a classical possibility is that "sensitized cells" may be sensitized by having antibody molecules (2) firmly bound to their cell membranes. According to this model, DHR may be visualized as resulting from the following sequence: (i) Antigen reacts with and bridges a small number of antibody molecules firmly fixed on sensitized cell membranes. (ii) This simple bridging of the cell membrane in some way interferes with membrane function, and results in the release of toxic intracellular materials into extracellular environment causing tissue damage. A classical example of this kind of antigen-antibody reaction occurs in anaphylaxis (3, 4). Sensitized cells may

conceivably become so in other ways (5), and this consideration will be taken up below.

More recently, Karush and Eisen (6) have argued that the available experimental evidence does not provide an adequate logical basis for the "sensitized cell" hypothesis. Based on the known heterogeneity of the immune response and on other considerations, they have hypothesized that DHR may be mediated by freely circulating antibodies which have high antigen-binding affinities and which are present in serum in extremely low concentrations (6). According to this model, DHR may be visualized as resulting from the following sequence: (i) Antigen reacts with soluble (unrestricted in mobility) antibody molecules to form comparatively large complexes. (ii) These complexes (with or without substances bound from serum) chemotactically attract mononuclear cells. (iii) The interaction of preformed complexes and cells cause the release of toxic materials from the cells into the extracellular environment, causing tissue damage. A classical example of this kind of antigen-antibody reaction occurs in the Arthus reaction (4). Indirect experimental evidence supporting the notion that antibodies mediating DHR are of comparatively high binding affinities has recently been obtained (7).

I have attempted to choose between the two general kinds of antigen-antibody reactions already mentioned by considering them as different physical-chemical situations, that is, the interaction of antigen with a reactant which is restricted in mobility (tissue-fixed antibody) in comparison to its interaction with a reactant which is freely mobile (antibody in true solution). Experiments were set up comparing the abilities of two homologous and structurally well-defined antigens of different molecular sizes to elicit specific DHR.

In the first situation (restricted antibody), the two antigens should be precisely equally effective when their molar concentrations are the same, and in the second situation (freely mobile antibody), the two antigens should be equally effective when their concentrations by weight are equal (8), provided the following requirements are met: (i) Delayed hypersensitivity is specific for the same simple antigenic unit contained in the two antigens. (ii) The number of antigenic combining sites per unit weight conjugate is the same