Recasting plaster in Late Stone Age

The Neolithic or Late Stone Age was a time of tremendous social and technological change in the Near East. Until recently, many saw it as a revolutionary period leading to the formation of the first complex societies. For instance, researchers have maintained that the domestication of crops and animals swept the Near East around 8,000 B.C., causing nomadic hunters and gatherers to settle down in villages and intensify agricultural production. Another swift change occurred about 6,000 B.C., with the appearance of high-quality ceramic pottery.

But the Neolithic "revolution" is being reassessed. Recent work at several Near East sites reveals a step-by-step introduction of domesticated plants and animals, beginning around 9,500 B.C., sometimes in the absence of farming villages. And according to a study in the just-released summer JOURNAL OF FIELD ARCHAEOLOGY, the technology required for pottery making formed the basis of an extensive plaster industry long before ceramics gained popularity. Plaster production rapidly expanded between 7,200 B.C. and 6,000 B.C., the investigators report.

"Describing the Neolithic in terms of the invention of pottery, plaster and agriculture is incorrect," says study director W. David Kingery of the University of Arizona in Tucson. "It was rather a period of industry establishment based on much earlier inventions."

Kingery and his colleagues examined 36 samples—typically 1 cubic centimeter or smaller — taken from Neolithic artifacts thought to be made of plaster. The artifacts, including flooring material, containers, sculptures and ornamental beads, are now housed in museums around the world and represent sites throughout most of the Near East.

Although some artifacts were unearthed decades ago, this is the first analysis of their microscopic and chemical makeup. Researchers studied fractured surfaces with a scanning electron microscope, including a procedure to identify the chemical composition of each specimen.

The investigators uncovered the microstructure of two types of plaster: lime and gypsum. Lime artifacts came primarily from sites in what is now Israel and Turkey; gypsum specimens originated in Syria and sites further east. Production of these plasters in quantity requires a number of steps, Kingery notes. Large amounts of wood must be gathered to heat limestone at 800 to 900°C (gypsum at 150 to 200°C) for up to several days. This product is then soaked in water to form a paste. Various substances, such as sand or gravel, are then added, and the paste is applied and shaped for a particular use. Many artifacts examined in the study show evidence of a thin plaster overcoat containing dye, as well as burnishing. This suggests plaster manufacture was a skilled activity conducted by special craftsmen, Kingery says.

In his view, the growth of villages and farming created a need for durable storage vessels demanding less fuel in their production, thus leading to the replacement of plaster containers by ceramics.

The earliest plaster artifacts in the sample — several stone blades with lime plaster used as an adhesive material — date to about 12,000 B.C., near the beginning of the Neolithic. The first evidence of plaster as an architectural material occurs between 10,300 and 8,500 B.C.

"The technological advances in plaster making [uncovered by Kingery and his co-workers] are unanticipated," says Yale University archaeologist Frank Hole, who granted the researchers access to several plaster artifacts. The first written records of job specialization in the Near East occur around 2,500 B.C., shortly after the Neolithic period ended. But there is no solid evidence for social complexity or craft specialization accompanying earlier plaster manufacture, Hole adds.

Kingery disagrees. "As we delve more deeply into the technology of early [Neolithic] times," he says, "specialized societies appear earlier and earlier."

— B. Bower

Well-bred cells: Poor hosts to viruses

Using genetic engineering, scientists have for the first time modified the DNA of mammalian cells to inactivate viral replication within the cells.

The experimental technique, reported this week, dramatically interrupts herpesvirus replication within cultured mouse cells, but has much greater practical potential against other viruses, such as the AIDS-causing HIV, say these and other researchers. Similar experiments with HIV may be completed soon in other laboratories, says Steven L. McKnight, who performed the herpesvirus research with Steven J. Triezenberg and Alan D. Friedman at the Carnegie Institution of Washington in Baltimore.

The novel defense strategy, dubbed "cellular immunization" by molecular biologist and Nobel laureate David Baltimore, represents a new avenue of antiviral research in which scientists program host cells to produce mutant proteins that specifically interfere with viral reproductive machinery.

"Will intracellular immunization really work [against AIDS]? I see no theoretical barriers, only practical questions," Baltimore says in a commentary accompanying the research results in the Sept. 29 NATURE. "I believe intracellular immunization has as good a chance as any other procedure of becoming a real AIDS therapy."

Normally, when a cell becomes infected by a herpesvirus, a viral protein called VP16 enters the host cell along with viral DNA. Once inside the cell, VP16 binds to certain host cell proteins, forming an "activating" complex that can bind to and "turn on" viral genes. These genes in turn regulate transcription — the first stage of viral replication.

McKnight and his colleagues reported this summer that it is possible to disable this self-starting mechanism by chopping off a particular portion of the virus' VP16 protein. In the new research, they inserted into cultured mouse cells a gene that codes for the production of such a truncated version of VP16, causing the cells to build up a supply of the functionally crippled viral protein. When these engineered host cells became infected with herpesviruses, their vast supply of bogus VP16 outcompeted the virus' own VP16 for the limited number of binding sites on the viral DNA, thus blocking viral replication.

Despite success in the laboratory, Triezenberg says, "I don't think this is going to have much applicability for the herpes simplex infection. That virus infects skin cells, and there's no way we'll be able to put a truncated version of VP16 into everybody's skin cells." Rather, he says, the technique is suited to viruses that infect a population of cells whose progenitors are easily located and engineered — such as HIV.

"You could take bone marrow cells that are not yet infected with the HIV . . . and introduce into them some altered version of an HIV activator gene, then reinject those back into the patient so they set up shop back in the bone marrow," says Triezenberg, now at Michigan State University in East Lansing. "Those cells, if all works well, might resist HIV infection."

Carl O. Pabo, of the Johns Hopkins School of Medicine in Baltimore, is one of several researchers experimenting with the primary activator gene in HIV — called *tat* — which is in some ways equivalent to the VP16 gene. He says scientists have identified regions of the *tat* protein that appear critical to its proper functioning, and in theory it is possible to engineer human bone marrow cells to produce appropriately crippled versions of that HIV protein. However, he and David Baltimore note, substantial obstacles — including possibilities of toxicity — must be overcome before genetic therapies of this nature become feasible.

— R. Weiss