Australian site yields early human dates

Scientists have identified Australia's oldest known human settlement, dating to approximately 50,000 years ago. The finding leads them to suggest that the initial peopling of Australia occurred around 60,000 years ago.

Until now, the oldest date for a human occupation site in Australia was nearly 40,000 years. In New Guinea, researchers had similarly dated a site containing stone hand axes to at least 40,000 years ago — a time when a land bridge connected New Guinea and Australia (SN: 12/13/86, p.374).

The new dates for humans in Australia arise from an archaeological site, initially excavated in the 1970s, that lies at the foot of a sloping cliff leading to the Arnhem Land plateau in the northern part of the continent. On returning to the site in 1988, Richard G. Roberts of the University of Wollongong, Australia, and his colleagues unearthed more than 1,500 artifacts of human design from the lowest occupation levels. Artifacts included stone flakes, red and yellow ochres, a grindstone and pieces of human-altered quartzite or white quartz.

The team applied the thermoluminescence (TL) dating technique to quartz grains taken from nine progressively deeper locations in the sediment. A pair of TL samples that sandwiched the deepest, earliest occupation level dates to between 45,000 and 61,000 years ago, with an average margin of error of 11,000 years. A TL sample from within that occupation level dates to 52,000 years ago, with the same margin of error, the team reports in the May 10 NATURE.

TL dating compares the decay of radioactive elements in buried objects with radioactive decay in the ground that surrounds them. Researchers heated quartz grains from artifacts at the Australian site and measured the radioactive energy emitted in the form of light. They also determined the TL dates of sediments lying within 1 foot of each TL-measured artifact.

Although some scientists have questioned the accuracy of the technique at other sites, Roberts and his co-workers defend their TL dates as reliable. Not only do the nine TL dates become older in deeper sediment layers, but TL dates of up to 40,000 years agree with previous carbon-14 dates for the same site. Moreover, they say, the artifacts show no signs of having been moved or otherwise disturbed by streams in prehistoric times.

The investigators conclude that humans from southeast Asia probably reached northern Australia about 60,000 years ago and arrived at the base of the Arnhem Land plateau a few thousand years later. — *B. Bower*

Drug-resistance gene saves mouse marrow

Most people don't talk about their p-glycoproteins, but everybody's got them. Scattered upon the surfaces of cells in the colon, liver, kidneys and a few other organs, these proteins act like microscopic sump pumps, bailing out the occasional poisonous molecule absorbed by these cells from food.

Unfortunately, many tumor cells also sport these pumps, which grant them the ability to spit out potent anticancer drugs before the treatments have a chance to work. While higher drug doses or longer treatment periods could probably overcome the cells' bailing capacity, such intense regimens have a life-threatening side effect: They can wipe out the bone marrow, birthplace of oxygen-carrying red blood cells and immune-enhancing white cells.

Michael Gottesman and Ira Pastan of the National Cancer Institute (NCI) in Bethesda, Md., now say they have successfully spliced the human p-glycoprotein gene into mouse embryos to make strains of mice whose marrow cells can resist a wide variety of anticancer drugs. The accomplishment provides a convenient living model for analyzing this mechanism of chemotherapy resistance and for designing and testing potentially "irresistible" drugs that could dismantle or bypass the pumps. The work also hints of a future in which genetic engineers might splice the p-glycoprotein gene into cancer patients' bone marrow cells, thus affording these cells protection during the course of intensive chemotherapy, the researchers say.

One strain of the altered mice expressed the human gene only in their bone marrow cells, Gottesman reported in Tokyo this week at the 13th Bristol-Myers Squibb Symposium on Cancer Research. Experiments *in vivo* indicate the marrow cells survive chemotherapy treatments that would decimate normal mouse marrow. In cell cultures, the researchers have also managed to switch the transplanted gene on and off at will, Gottesman told SCIENCE NEWS. That suggests scientists may someday gain enough control over the gene to regulate its activity differently in different tissues, Pastan adds.

Drug designers may be the first to make use of the new mouse model, but cancer patients might ultimately derive lifesaving benefits from p-glycoprotein genes inserted directly into their marrow cells. Such gene therapy "may seem a little science-fictiony, but we take it seriously," Pastan says. — *R. Weiss*

Tick protein tapped to attack blood clots

Low on the list of nature's most popular animals creeps the tick. Described by one entomologist as an "ugly bag of skin," this squatty, slow-crawling relative of the spider has an unwavering thirst for fresh blood and is a common carrier of disease, including Rocky Mountain spotted fever and Lyme disease. But if a scientific hunch proves correct, thousands of people threatened by blood clots may someday have kinder words for ticks.

From an extract of the "soft" tick *Ornithodoros moubata,* researchers have purified a peptide, or small protein, which they speculate may have therapeutic value as an anticoagulant. Physicians often use anticoagulant drugs to prevent blood clots in patients recuperating from heart attacks, stroke or major surgery, but not without risking serious side effects. For instance, heparin — the anticoagulant most commonly prescribed for heart attack and stroke patients — can cause abnormal bleeding, bruises, rashes, aching bones or burning feet. And in a small fraction of patients, lethal clots may form despite heparin treatment.

Like heparin, the newly isolated tick anticoagulant peptide (TAP) interrupts the chain of enzyme reactions that produces fibrin, an insoluble mesh of protein that gives structure to a blood clot. But "unlike other clot preventers, such as heparin, this isolated tick anticoagulant acts only on [clotting] factor Xa, and not on other enzymes in the clot chain or on platelets," biochemist George P. Vlasuk told SCIENCE NEWS. "Hence, there might be fewer side effects if this anticoagulant could ever be used clinically."

Vlasuk and his colleagues at Merck Sharp & Dohme Research Laboratories in West Point, Pa., describe their work in the May 4 SCIENCE.

"One interesting sidelight," adds Vlasuk, is that "ticks cause Lyme disease . . . . If we knew more about how they naturally use a blood anticoagulant to feed, we might be able to control ticks by developing an antibody to a tick anticoagulant." Researchers have already developed such "tick vaccines" for livestock, derived from anticoagulants other than TAP (SN: 3/25/89, p.186).

"But it's a leap of faith at this point to extrapolate from soft ticks to the deer [Lyme disease] tick," cautions Richard Endris of Merck Sharp & Dohme in Rahway, N.J., who served as an entomological consultant on the study. Noting that this species feeds much more slowly than *O. moubata,* he says, "The same [anticoagulant] mechanism might not occur in the deer tick." — *W. Stolzenburg*