

been perfect sites for the mosquitoes that transmit the disease to breed in, and also that the growing human populations that agriculture permitted would have provided an abundance of convenient hosts.

Dr Volkman and her team tested this idea by looking at the range of genetic variation found in *P. alci arum*. In general, the more variation there is in a gene in a population, the longer it is since that population's individuals shared an ancestor. Such variation can therefore act as a "molecular clock" from which the age of the common ancestral gene can be deduced.

The best genes for this purpose are so-called neutral genes: those that are not subject to strong selective pressures that will stop random mutations from accumulating them. Averaged over a sufficient period, such random changes accumulate at a constant rate in neutral genes. But previous studies of genetic variation in *P. alciparum* have concentrated on those genes that help the parasite to evade the human immune system, or protect it from insecticides. These are clearly subject to strong selective pressures, and therefore make poor molecular clocks.

When Dr Volkman looked at nine neutral genes, she found little variation across widely separated parasite populations, suggesting the common ancestors of those genes were recent. And when she applied estimates of the speed of mutation to her results, the ages coincided neatly with the Neolithic agricultural revolution.

Dr Tishkoff and her colleagues studied the frequency of variants in the human G6PD (

glucose-6-phosphate-dehydrogenase) gene, which confers resistance to malaria. G6PD carries the instructions for making an enzyme that is essential for the transport of oxygen in the bloodstream. Mutations in this gene may have adverse consequences for a person's health but rather like the mutation of the haemoglobin gene that causes sickle-cell anaemia, they can also protect that individual against malaria.

Again like the sickle-cell gene some G6PD mutations have spread in parts of the world in which malaria is endemic. Dr Tishkoff reconstructed the recent history of G6PD by examining the variants present in more than 450 Africans. She then simulated the way that the mutations spread to find out how long the process took. And, just as Dr Volkman did, she hit the Neolithic.

How much light all this sheds on the risks of new infections is unclear. But there is a respectable argument that the cutting of tropical forests for farmland and timber, by bringing people into close proximity with wild animals that they would not previously have encountered, may encourage the spread of new diseases. Perhaps things have not changed so much in the past few thousand years, after all. m

Bacteria and ore-formation

Goldbugs

Gold mines may owe their origins to microbes

MEDIEVAL alchemists found in the end that they could not create gold. Modern geochemists have a similar problem. They find it hard to understand how natural gold deposits form. There is much handwaving about gold-rich fluids from deep in the earth, and chemical precipitation, but the physics does not add up. The answer may be that what is happening is not geochemical at all, but biochemical. And a casual experiment by a bacteriologist may hold the key.

Derek Lovley, of the University of Massachusetts Amherst has been studying "metal-eating" bacteria for two decades. These bacteria make their living by converting (or "reducing") the dissolved ions of metallic elements from one electrical state to another. This reduction releases energy, which the bacteria extract for their own purposes.

Unsurprisingly, such bacteria tend to prefer common metals such as iron and manganese for lunch, though some species are able to subsist on such exotica as uranium. A few months ago, though, as "a bit of a lark" Dr Lovley decided to put some of his bacteria into a solution of gold chloride. He was fully prepared for nothing to happen, as gold compounds are generally toxic to bacteria. Instead the test tube containing the solution turned a beautiful shade of purple, the colour of metallic gold when it is dispersed very finely in water.

Bacteria are already known to be involved in the formation of an iron ore

called limonite and Dr Lovley has argued that they are also involved in the creation of certain ores of uranium. His jokey experiment, reported in the July issue of Applied and Environmental Microbiology, opens up the possibility that gold deposits, too may have a bacterial origin, with the microbes acting as the agent that concentrates gold from sources such as volcanic springs into a form that people can mine.

Dr Lovley has some support among geologists. According to Francis Chapelle, of the US Geological Survey's branch in South Carolina his hypothesis would neatly explain the origin of some of that state's gold deposits. The rocks of the Carolina slate belt including the Haile gold mine, contain the metal in an unusual form: rather than appearing in veins and nuggets, it is finely disseminated in a layer of sedimentary rock.

The sediments that form the Carolina slates were once a seabed through which volcanic fluids flowed. According to Dr Chapelle, metal-reducing micro-organisms may have extracted gold ions from these fluids reduced them, and dropped the waste (ie metallic gold) as a powdery precipitate in the sediments. How that relates to the more traditional deposits of gold in veins and nuggets remains to be seen although the fact that several species of Dr Lovley's gold-eating bacteria prefer high temperatures suggests they might survive well in the hot environments mineral veins are thought to form in.

Dr Chapelle also suspects that Dr Lovley's discovery may have a bearing on the origin of the vast Witwatersrand goldfield in South Africa whose mines produce about one-third of the world's supply. Like South Carolina Witwatersrand has an area of gold that is found not in igneous rocks but in a thin sheet sandwiched among sediments. Perhaps the Boer war was actually triggered by arguments over bacterial excreta.



Microbe droppings?