Hominid bones show strides toward walking

Animal fossils stored in a box since their excavation 15 years ago at an underground cave in South Africa have yielded an unexpected discovery—four foot bones that fit together to form the left instep of a hominid that lived about 3.5 million years ago. The bones provide the first clear fossil evidence that members of the human evolutionary family evolved in stages from climbing in trees and moving about on all fours to walking upright.

"This partial foot provides a locomotor missing link in the hominid fossil record," asserts Phillip V. Tobias, an anthropologist at the University of the Witwatersrand Medical School in Johannesburg. "It combines a weight-bearing heel used for two-legged walking with a chimplike big toe capable of grasping."

Tobias and Witwatersrand colleague Ronald J. Clarke unveil the foot fossils in the July 28 SCIENCE.

In conjunction with an excavation last year of the oldest fossil-bearing sediment in South Africa's Sterkfontein cave, Clarke examined material that had been recovered from the same soil layer in prior fieldwork and stored in boxes. One such container, thought to hold only the bones of nonhominids, disclosed the new foot specimens.

Until now, the oldest hominid remains at Sterkfontein dated to between 2.6 million and 2.8 million years ago, based on analyses of animal fossils that lay near them and magnetic reversals in the caves' sediment layers. These hominid finds have been assigned to the species *Australopithecus africanus*.

The new fossils come from a deeper soil layer that probably dates to about 3.5 million years ago, according to Tobias and Clarke. The bones most likely belong to a species of *Australopithecus*, perhaps the earliest known *A. africanus*, the scientists contend.

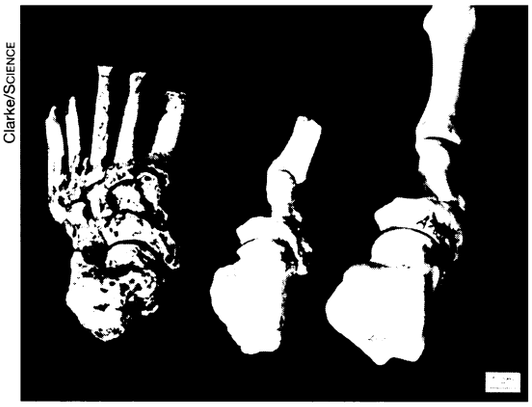
The heel bone, fully capable of supporting body weight during two-legged walking, closely resembles that of modern humans, Tobias says. The remaining three bones become progressively more apelike as they move away from the heel. The bone that forms the base of the big toe angles away from the other toes and shows evidence of considerable mobility, as in chimpanzees. And a large muscle that helped the hominid grasp objects was once attached to a deep groove in that bone, Tobias holds.

Anthropologists have long debated whether early hominids favored two-legged walking or balanced their walking with considerable tree climbing. Bones from a 2.5-million-year-old Sterkfontein hominid (SN: 4/22/95, p.253), as well as an analysis of hominid inner-ear bones (SN: 4/9/94, p.231), have recently indicated that an upright gait emerged gradually.

Some investigators assert that the approximately 3.6-million-year-old hominid footprints preserved in volcanic ash at the Laetoli site in East Africa, which were discovered in 1978, indicate a strikingly human foot anatomy and gait. A reconstruction of the entire Sterkfontein foot, derived partly from other early hominid foot specimens found in East Africa, closely matches the Laetoli footprints, Tobias contends.

"This is an exciting find because it helps to confirm the belief that hominids came down from the trees at some point and that there was an intermediate stage on the way to modern human feet," notes Michael Day, an anthropologist at the British Museum in London. Day examined the Sterkfontein foot bones last week on a visit to South Africa.

Day argues, however, that Clarke and Tobias' foot reconstruction is probably not accurate. East African specimens cannot provide a reliable picture of toe length or other features of the South African hominid, in his opinion.



Clarke/SCIENCE

New Sterkfontein find (center) has more apelike big toe than East African Homo habilis *(left) or modern human (right).*

Feet like those of the Sterkfontein hominid could not have made the Laetoli prints, which look like the footprints of modern humans who habitually walk barefoot, Day maintains.

"Our foot reconstruction may change, but the important point is the transitional nature of the bones we have now," Tobias remarks. *— B. Bower*

Immune factor inhibits the spread of HIV

After infection with HIV, the AIDS-causing virus, people usually experience a period of good health before a devastating decline of immune function results in a host of opportunistic infections. Some people, however, remain asymptomatic for a remarkably long time (SN: 3/18/95, p.172).

Researchers are just now beginning to determine which aspects of the immune system keep HIV at bay. A team of San Francisco researchers has pinpointed a substance secreted by certain immune cells that prevents the virus from replicating. The finding could lead to new strategies for controlling HIV infection.

The substance, known as cell antiviral factor (CAF), suppresses the virus' ability to spread from cell to cell through the immune system. "Understanding how CAF inhibits the virus could give us insights into new therapies for HIV," says immunologist Jay A. Levy of the University of California at San Francisco.

Levy's group identified CAF in the mid-1980s while trying to grow HIV. The researchers found they could isolate large amounts of the virus from white blood cells, or T cells, of late-stage AIDS patients, but not from the blood of newly infected or asymptomatic patients.

To identify what was inhibiting HIV production in asymptomatic patients, the team separated the T cells into CD4 cells, which help trigger immune responses and which form the target of HIV infection, and CD8 cells, which destroy infected cells. Populations of HIV-infected CD4 cells produced large amounts of HIV, whether they came from asymptomatic or late-stage AIDS patients, but adding CD8 cells from long-term survivors or uninfected people shut down HIV production. CD8 cells of late-stage AIDS patients had no effect.

Further studies indicated that the CD8 cells showing antiviral activity secreted the substance researchers named CAF. Researchers also found that CD8 cells from long-term survivors of HIV infection maintain the ability to stymie the virus. Still, no one knew at what stage CAF shuts down HIV.

New information presented by Levy's colleague Carl E. Mackewicz at the Ninth International Congress of Immunology in San Francisco now indicates that CAF inhibits HIV replication within CD4 cells at an early stage, preventing the virus from making copies of itself. However, CAF doesn't disrupt the normal functioning of infected CD4 cells. Because the cells continue to perform immune functions while CAF suppresses the virus, Levy sees hope for a therapeutic strategy against HIV.

Mackewicz and Levy both point out that they still don't know what CAF is. "The factor is produced in such small amounts that isolating it has been and will be extremely difficult," says Mackewicz.

Immunologist Alan L. Landay of Rush-Presbyterian–St. Luke's Medical Center in Chicago agrees that therapeutic uses for CAF hinge upon knowing its molecular makeup, but he notes that the San Francisco team's elucidation of the way CAF inhibits HIV replication will help in developing new ways to fight HIV.

— L. Seachrist