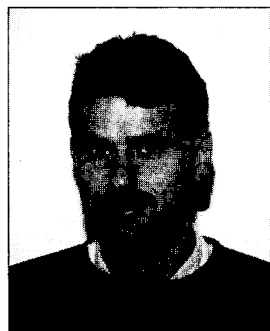


What the new breed of vitamin C researchers are attempting to do is dissociate themselves from that fringe. One researcher in the field thinks they're on the way to doing that—but they're not there yet. "There are some things that are new and exciting," says vitamin researcher Mark Levine of the National Institute of Diabetes, Digestive, and Kidney Diseases. But Levine adds that the field is still in a state of transition. A lot of low-quality work, left over from the old days, is still being done: poorly controlled experiments and physiological measurements of vitamin C levels that use outdated and unreliable assays. "The only way to get at vitamin C is to do the basic stuff that has never been properly done at all," Levine says, pointing out for starters that



***"There was no oxidation damage [to LDL] as long as vitamin C was around."***

—BALZ FREI

no one really knows for sure what blood levels you get from taking a gram of vitamin C, or 10 grams, or 20, leaving the meaning of megadoses completely unknown.

Levine is gearing up to address the question of tissue levels of the vitamin using a newly developed and more reliable assay in human volunteers. Once tissue levels are known, Levine proposes to analyze vitamin C systematically, to deduce the optimum

level for various functions in the body, using whole cell or organ preparations. "You have to go after this and ask these questions," says Levine. "You can't just assume that more is better." Levine's approach, which he has begun to apply in several tissues, has won praise from a variety of quarters, but not from old-time nutritionists like Olson. "He is doing all kinds of research in vitro, and claiming that it applies to the intact body, which is nonsense," Olson fumes.

It's not surprising that Levine's work calls forth sharply divided opinions. Almost everything about vitamin C still elicits much stronger reactions than most research subjects—reactions that can only be considered appropriate, given how recently the topic was far outside the mainstream of science. But, like many scientific ideas once thought to be absurd that later appear in the guise of orthodoxy, vitamin C seems to be creeping closer and closer to winning the respect that Rodney Dangerfield never quite seems to get.

■ MARCIA BARINAGA

## Duesberg Vindicated? Not Yet

A surprising—some say shocking—result from an AIDS vaccine trial in monkeys has forced researchers to reconsider some of their approaches to developing an AIDS vaccine for humans. Writing in the 3 October issue of *Nature*, E. James Stott and his colleagues at the National Institute for Biological Standards and Control in Hertfordshire, England, found that a vaccine made from uninfected human T-cells—intended as a control—was at least as effective in protecting animals from infection with SIV (the simian version of the AIDS virus) as a vaccine that was made with the attenuated virus itself grown in the same T-cell line.

But if researchers are uncertain what to make of the new findings, they had a very different reaction to a *Nature* article (26 September, p. 297), written by the journal's editor John Maddox, about the implications of the research. Maddox concluded that Stott's study, together with results recently published in *Science* (6 September, p. 1138) suggesting that AIDS shares features with autoimmune diseases, supported the controversial thesis espoused by virologist Peter Duesberg: namely, that HIV is not the cause of AIDS.

"In general, I'm pretty mild mannered, and I was furious" about Maddox's article, says David D. Ho, director of the Aaron Diamond AIDS Research Center at the New York University School of Medicine. Ho isn't alone. Numerous other researchers contacted by *Science* failed to see any con-

nection whatsoever between Stott's work or the *Science* study and the stand taken by Duesberg, who is a professor at the University of California at Berkeley. That goes for Duesberg, too: "Those studies have nothing to do with [my position]," he says.

What Stott's work does do is undermine some of the assumptions about how previous vaccines have protected animals that have been challenged with injections of SIV. Animals will mount an immune response when presented with a foreign substance, called an antigen. "Everyone had assumed that [the immune response] had something to do with a response to SIV antigens," says Ronald C. Desrosiers of the New England Regional Primate Center in Southborough, Massachusetts. But Stott's experiments suggest that the protection is caused by some component of the T-cell line that the animals were vaccinated with—which would be unprecedented, says Desrosiers. "It's going to be scientifically interesting and important to sort out what's going on here."

But even Stott acknowledges that his work does not invalidate the hypothesis that HIV causes AIDS. Since the attenuated virus vaccine also afforded protection against subsequent challenge with SIV, "our findings do not, of course, exclude the possibility of a virus-specific component in protection," Stott writes. He adds that studies with viral antigens made from recombinant DNA technology—not grown in cell culture—may ultimately explain exactly how the vac-

cines are producing protection.

In the other study cited by Maddox as supporting Duesberg, physicist-turned-microbiologist Geoffrey W. Hoffmann and microbiologist Tracy A. Kion at the University of British Columbia showed that mice that had never been exposed to HIV could nevertheless produce antibodies to HIV antigens. In one case, animals from a strain that suffers from an autoimmune disease developed antibodies to HIV antigens spontaneously. In another, mice developed antibodies to HIV after they were exposed to cells from different mouse strains. Hoffmann's hypothesis is that certain HIV antigens resemble mirror images of normally occurring cell surface antigens that control response to foreign tissue. In some way, these mirror-image look-alikes trigger an autoimmune disease that produces the symptoms that resemble AIDS and even shares the same antibodies. But just because an AIDS-like disease *can* occur without HIV doesn't mean that it typically does, he asserts. "We have nothing in common with [Duesberg]'s idea that HIV has nothing to do with AIDS," says Hoffmann.

So how does Maddox explain why he leapt to that conclusion? "I'm not for a minute saying Duesberg is right in all points," says Maddox. "But I feel sorry that *Nature* has not done more to give his view prominence. It would have hastened the process by which the scientific community is coming around to the view that the pathogenesis of AIDS is more complicated than the baby-talk stories we were all given a few years ago."

■ JOSEPH PALCA