Philippe Busquin, the research commissioner, backs the idea of such a group. "A network of pan-European research infrastructures can become a major component of the European Research Area," Busquin told *Nature* (see *Nature* 405, 873; 2000). "I am happy about the high level of agreement reached at this important meeting."

Busquin says that he also welcomes the steering group's recommendation that the commission increase its financial support for building and running common research facilities and giving scientists access to them.

Busquin: backs proposed panel.

This would be good news for two European life-science facilities, the European Mouse Mutant Archive (EMMA) near Rome and the European Bioinformatics Institute (EBI) near Cambridge. Both are in financial difficulties because the rules of the EU's current Fifth Framework Programme exclude such costs from funding.

"The Sixth Framework Programme [FP6], starting in 2003, will certainly contain elements which will help solve the problems of facilities such as EMMA and EBI," predicts Busquin.

Indeed, a proposal to increase infrastructure funding reflects the commission's intention to revise its whole approach under FP6. According to one official, the commission proposes to give less money to small, scattered projects which are often of questionable effectiveness (see *Nature* 404, 695; 2000). The money freed up could then be spent on infrastructure, he says.

"But the commission's role in the funding of infrastructure can only be that of a minority partner, who can hopefully catalyse initiatives," says Busquin, adding that the commission has neither the money nor the power to set up large research facilities without support from member states.

Speaking at last week's meeting, government representatives welcomed proposals to make greater use of the 'variable geometry' approach, under which countries can choose whether to participate in funding particular research facilities.

But an official from the German research ministry points out that governments' enthusiasm for collaboration on research infrastructure is likely to vanish if the costs of new facilities exceed the benefits to the national science base. ■

► http://www.cordis.lu/improving/src/ari_conf_res_infr.htm

The drugs do work: research suggests that early medication helps the immune system fight HIV.

UK faces rethink on AIDS treatment

Karen Birmingham, London

Britain might have to reassess its policy for treating AIDS in the light of new results published this week.

Research at Massachusetts General Hospital and Harvard Medical School in Boston suggests that starting aggressive drug therapy as soon as HIV infection is confirmed is the most effective way to combat the virus. This strategy, say the researchers, allows the patient to develop specialized immune cells that attack the virus (see page 523).

Unlike the United States and most of Europe, Britain delays such treatment until the number of CD4 cells — white blood cells involved in the immune response — drops to below 350 cells per cubic millimetre. This can take several years after initial infection.

The study looked at patients who began taking a cocktail of drugs known as highly active antiretroviral therapy (HAART) within 180 days of infection with HIV. The treatment increased levels of specialized CD4 cells that can destroy HIV-infected cells, and the patients were able to stop taking the medication for up to a year and still suppress HIV levels in their blood. Patients who did not start on HAART until their infection had become 'chronic' showed no signs of this effect.

The case for early treatment is now compelling, says Bruce Walker, director of the Partners AIDS Research Center at Massachusetts General Hospital and one of the paper's co-authors. He is calling for tests that can detect the acute stages of HIV infection to be licensed by the US Food and Drug Administration. This would allow diagnosis roughly two weeks earlier than is possible using current tests.

But Walker and his colleagues stress that their results do not mean that patients can

stop taking their medication without supervision. "It is the drugs that help the immune system to generate the correct CD4 cells," Walker says. "More fine tuning is needed to know how long you need to treat people before you [can] interrupt therapy, and how long that interruption [can] last."

Mike Saag, an AIDS researcher at the University of Alabama at Birmingham, says that the results establish 'proof of principle' that HIV-specific CD4 cells are critical to the body's ability to fight the disease.

"This CD4 cell help is lost very early in the course of infection — within 6 to 12 weeks — and the implication is that early use of antiretrovirals helps preserve this function," he says. "Once preserved, it seems to perpetuate itself even after antiretroviral therapy is stopped for up to a year."

The success of HAART drugs has led to a dramatic fall in AIDS mortality in the western world, but Britain has resisted giving otherwise healthy people these powerful drugs. Over the past two years, as toxic side effects of the long-term use of HAART have surfaced, such caution has seemed justified. Patients can suffer disfiguring body-fat redistribution that might be associated with diabetes and cardiovascular diseases.

Although the new data suggest that immediate treatment could reduce the time that patients spend on the drugs, Jonathan Weber, professor of communicable diseases at Imperial College School of Medicine in London, and a proponent of delaying therapy, urges caution.

"HIV is a long illness, and just because someone looks great at two years, it doesn't tell you what will happen 14 years later," he says. But he admits that "the time is right to start a randomized clinical trial of when it is best to start treatment." ■