ing the notion that these "garbage disposal" pathways may be important in organ formation, says the study's lead author, developmental biologist and pediatric cardiologist Deepak Srivastava of the University of Texas Southwestern Medical Center at Dallas.

"This is a major breakthrough," says developmental biologist Paul Krieg of the University of Texas, Austin. "It opens up a whole new area of research in heart development, because it links a clinical syndrome to a new and exciting pathway in cell biology." Others are more cautious, arguing that it's still possible that other genes also contribute. "This is beautiful work," says Christine Seidman, a cardiologist and geneticist at Harvard Medical School. "But I think it's not yet possible to attach the DiGeorge syndrome to a single gene."

Researchers already knew that in 90% of DiGeorge patients, chromosome 22 is missing a large chunk of DNA—about 3 megabases. This presumably causes the syndrome by eliminating one or more crucial genes, and human geneticists have been trying to pin them down. Srivastava, however, plucked out the key gene not through clinical studies but through basic research—in mice. He and colleagues were studying a transcription factor called dHAND, which turns on an array of genes crucial to the development of the mouse heart. Notes Krieg, "This is a nice example of how basic research can yield clinical answers."

As the heart takes shape, so-called cardiac neural crest cells migrate from the neural fold (the spinal cord precursor) into specific niches in various tissues. These neural crest cells form the connection between the heart chambers and nearby vessels (see figure)—which are precisely the regions affected in DiGeorge syndrome.

In mice lacking the gene for dHAND, these cells did not develop properly. The researchers picked out a dozen genes normally activated by dHAND by looking for messenger RNAs found in normal mice but absent when dHAND was shut down. One corresponded to a gene called *Ufd1* (for ubiquitin fusion degradation), which was infamous for being one of 25 or so genes known to lie within the DiGeorge deletion site.

The link between *Ufd1* and the syndrome tightened when they studied the distribution of its protein product in mouse tissues. "*Ufd1* showed up in virtually all tissues that were affected by the DiGeorge syndrome," says Srivastava, including structures that give rise to the thymus and facial bones. Srivastava then turned to humans and found that of 182 DiGeorge patients, all were missing the gene for *UFD1*. The team also came across one patient who had all the classic symptoms, yet, like 10% of all DiGeorge patients, had no apparent genomic deletion. But after more detailed analysis, the team found a minideletion affecting only two genes, *UFD1* and a cell cycle control gene called *CDC45*. Although he admits that *CDC45* cannot be formally ruled out, Srivastava says that "together this indicates that *UFD1* is the cause for the 22q11 deletion phenotype."

Not quite, cautions cardiologist Seigo Izumo of the Beth Israel Deaconess Medical Center in Boston. "*UFD1* is the most attractive candidate," he says, but "it could still be a combined effect of *UFD1* and *CDC45*." Indeed, the DiGeorge syndrome is probably a game of several players, comments Beverly Emanuel, a human geneticist at the University of Pennsylvania in Philadelphia. "It's clear that *UFD1* contributes, but this is not the complete answer," she says. She notes that there are patients who have genetic disruptions at the suspect region, but seem to have an intact *UFD1* gene. "They need to be explained. Clearly there are other things going on at this locus," she says.

Srivastava, however, is already seeking the proteins that *Ufd1* normally helps degrade. Their untimely accumulation when one copy of the gene is missing might somehow cause the developmental problems, he suggests. And Izumo thinks the discovery may even eventually brighten the outlook for afflicted infants, many of whom must currently undergo open heart surgery. "New studies may eventually lead to a better treatment and perhaps even preventive interventions" for those whose hearts need a little help to be made whole.

–MICHAEL HAGMANN

CREDITS: WILLIAM MCCOY/RAINBOW/PNI

VIROLOGY

Virus Suspect Identified In Elephant Deaths

When Kumari, the first elephant ever born at the Smithsonian Institution's National Zoological Park in Washington, D.C., was just months old, the youthful pachyderm would frolic for adoring crowds, splashing in the pool or playing with the pumpkins she got on Halloween. But the good times didn't last for Kumari: On a sunny spring day in 1995, after a 5-day bout with a mysterious illness, the 16-month-old Asian elephant lay down and died. At the time, zoo scientists had no idea what had killed the 1000-pound youngster.

But now, on page 1171, a team led by Laura Richman and Gary Hayward of Johns Hopkins School of Medicine in Baltimore and the National Zoo's Richard Montali reports that it has found the killer—a novel herpesvirus distantly related to the virus that causes cold sores in humans. The new virus has killed at least seven other juvenile Asian elephants at zoos.

Exactly how Kumari became infected with the virus is unclear, but it may have been transmitted to her or her mother by an African

ScienceScope

Everglades Summit A trio of prominent ecologists will serenade Interior Secretary Bruce Babbitt in Washington, D.C., next week with concerns about a controversial $8 billion plan to restore Florida's Everglades ecosystem. The 22 February gathering in Washington was arranged after six scientists—including Stuart Pimm of the University of Tennessee, Knoxville, Peter Raven of the Missouri Botanical Garden in St. Louis, and Gordon Orians of the University of Washington, Seattle—wrote Babbitt last month, complaining of the plan's "deep, systematic" scientific failings. They called for a review by an independent body such as the National Academy of Sciences.

That would take too long, say Interior officials, who hope to submit a blueprint to Congress later this year. Instead, officials have suggested a faster, internal study that examines the concerns, which have made headlines in Florida. The letter "obviously touched a raw nerve," says Pimm. Now, he and his colleagues are waiting to see how the department responds to such external stimuli.

Healthy Ties Canada wants to create a "virtual" Canadian Institutes of Health Research. Unveiled this week as part of the government's 1999–2000 budget proposal, the institutes are expected to replace the Medical Research Council (MRC) as Canada's primary mechanism for funding biomedical research at academic centers.

The new structure—conceived by MRC President Henry Friesen as part of a bid to increase federal support for health research (*Science*, 8 May 1998, p. 821)—will also involve an electronic network linking scientists in particular fields. But the research will continue to be carried out at universities around the country, and the new institutes are not expected to have their own labs.

Details of the plan will be worked out over the next year. One unknown is funding. Proponents want $325 million a year on top of the MRC's current budget of $163 million, but it remains to be seen whether Parliament will be so generous.

over his data to a four-member audit committee for investigation.

That committee's report, released on 28 October 1998, didn't mention the alleged mix-up; instead, it acknowledged that experiments with lectin-transgenic potatoes had been carried out, but concluded they did not support the suggestion that the potatoes affected growth, organ development, or immune function in rats. Pusztai, who was forbidden by Rowett to talk to the press, sent copies of the audit report, his own rebuttal to it, and a transcript from the *World in Action* show to dozens of scientists who had asked for them, asking them to review the material.

In the news. Arpad Pusztai's data make headlines.

The responses, collected by protein chemist Edilbert Van Driessche of the Vrije Universiteit in Brussels, were presented along with a statement last week at a press conference in the House of Commons. The statement contends that Pusztai's data do suggest that the transgenic potato affected the rats' immune systems, affected their organs, and slowed their growth. The data in the audit report, it says, "appeared to be arbitrarily selected and biased towards brushing aside the conclusions of the experimental findings."

Pusztai's supporters also point to a follow-up study performed last fall by Stanley Ewen—a pathologist at Aberdeen Royal Hospitals who has worked with Pusztai for 10 years—who examined the guts of the rats from Pusztai's experiments under a microscope. Ewen, who presented the results at an EU-sponsored lectin meeting in Lund, Sweden, in November, found that the animals fed a transgenic diet had symptoms of infection, with white blood cells accumulating in their gut lining. The same reaction didn't occur in rats that had been fed a nontransgenic potato diet spiked with the same lectin. Although it's unclear how the diets could have had different effects, "they are profound changes," says Thorkild Bøg-Hansen, a lectin expert at the University of Copenhagen, "that require further investigation."

The audit committee's chairman, Rowett senior scientist Andrew Chesson, says he stands by his report but doesn't want to discuss the reviewers' findings, to avoid a debate about raw data in the press. Pusztai should publish his results in a scientific journal, Chesson says: "If the data are sound, I don't think he'd have any problem publishing them."

The new analyses of Pusztai's data immediately led Simpson to demand a "complete moratorium" on genetically modified food—a measure British Prime Minister Tony Blair said he wasn't ready to take, as he strongly believed the new food was safe. Simpson also says, "If the data are now being corroborated, someone has to explain the basis upon which his research was suppressed." Several MPs expressed suspicions about the government's role in the affair, which were stirred up even further when a newspaper revealed on 16 February that science minister Lord Sainsbury once had a financial interest in a company that owns a patent on the cauliflower mosaic promoter, a gene often used in plant genetic modification. Conservative MPs said Sainsbury was a biotech "advocate" and demanded his resignation. But Chesson says his institute was not influenced by the government or the industry and has "never ever" attempted to suppress any results. "The sooner the data get into the scientific journals, the happier we'll be," he says.

Whatever the fate of the findings, most parties agree on at least one thing: The affair has been an outstanding example of how not to communicate scientific findings to an already confused and worried public.

–MARTIN ENSERINK

CREDITS: (TOP) DAVE CHESKIN/PRESS ASSOCIATION NEWS; (BOTTOM) P. BAGLA

COSMOLOGY

Superheavy Particles From the Big Bang?

CHICAGO—Pity the poor Wimps. Although theorists have proposed that these Weakly Interacting Massive Particles—hypothetical slow-moving, exotic relics of the big bang—could account for much of the mass of the universe, no one has conclusively observed a Wimp. Worse, in their bid for the title as the unseen "dark matter" that astronomers believe our galaxy must contain in large amounts, they have to compete with big, brawny lumps of common stuff—stones or gas—that go by the acronym Machos. As if all that were not bad enough for the effete Wimps, a formidable new rival has just emerged: a Godzilla of a particle called the Wimpzilla.

Described here last month at a gathering of the world's leading cosmologists,* Wimp-

* The Pritzker Symposium on the Status of Inflationary Cosmology, University of Chicago, 29 to 31 January, with a closely related workshop from 1 to 3 February.

ScienceScope

Crops Chief Moves On After just 18 months on the job, Shawki Barghouti (below) has resigned as head of the struggling International Crops Research Institute for the Semi-Arid Tropics (ICRISAT) in Patancheru, India. The Jordanian agronomist says he has successfully steered the institute in "a new direction" and that it is time to move on.

Founded in 1972, ICRISAT is part of a global network of 16 centers aimed at improving agriculture in the developing world (*Science*, 2 January 1998, p. 26). Barghouti says he eliminated his institute's $5 million deficit by cutting spending by 20% and by coaxing donors to add $3 million to a $25 million budget. "Not a single research program was hacked in my tenure," Barghouti claims. But not everybody is convinced that ICRISAT is out of the woods. Yeshwant

Nene, an ex-deputy director-general of the institute, fears the current "peaceful" period could end with yet another changing of the guard. Barghouti plans to leave on 1 September.

Accelerated Recycling A dismantled Dutch linear accelerator will find new life in Russia. At the end of last year, particle physicists shut down the 20-year-old Medium Energy Accelerator and the 7-year-old Amsterdam Pulse Stretcher in response to the government's decision to cut back on high-energy physics. Now, the Netherlands' only linear accelerator—a 180-meter-long pipe that fires electrons into a 68-meter-diameter storage ring—will be recycled into a synchrotron radiation source at the Joint Institute for Nuclear Research in Dubna, Russia.

Though the Dutch machine is free, the Russian institute must raise millions of dollars for reassembly, a 4-year project. "Fortunately we have a building which just fits," says Dubna chief engineer Igor Meshkov.

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