

Speculating in Precious Computronium

A new computer embodies an architecture that—to its creators—mimics the structure and dynamics of physical reality

IT TAKES AN OFFBEAT PERSPECTIVE TO THINK of the evanescent electronic fuss going on inside a computer as a sort of primordial stuff from which almost anything can take shape, from crystals to stars. But that's how physicists and computer builders Norman Margolus and Tommaso Toffoli of the Massachusetts Institute of Technology view their computer child, CAM-8 (for Cellular Automaton Machine 8), a prototype of which is due to get its first breaths of electronic life next month.

Then again, CAM-8 is an offbeat computer. Its specialty will be running so-called cellular automata models—computer simulations that portray the world as consisting of lots of discrete, locally interacting pieces—molecules of air in a gaseous slam dance, grains in a windblown sand pile, or species of animals sharing an ecosystem. While simulation programs on conventional computers are generally based on equations or algorithms describing the collective behavior of those pieces, cellular automata focus on the pieces themselves and the simple rules by which they interact: how one air molecule or sand grain nudges the next, for example, or how individual predators and their prey play their game of cat and mouse. As the computer traces these local interactions over time, the collective behavior of the system emerges.

That's a more direct and potentially much more powerful way to simulate many physical systems, Margolus and Toffoli say. What's more, while general-purpose computing Schwarzeneggers such as the Cray X-MP and the Connection Machine 2 can run cellular automata models, computers tailor-made for them might be able to do so millions of times faster. CAM-8 is meant as a modest step toward that goal.

But Margolus explains that he and Toffoli had something more than faster computer simulations in mind when they designed it. The models of reality provided by cellular automata, he says, "may ultimately turn out to be truer than the conventional equations of physics." Thus the researchers claim half-seriously that they are creating a versatile new form of matter, which they call programmable matter or computronium. "In programmable matter, the same cubic meter

of machinery can become a wind tunnel at one moment, a polymer soup at the next; it can model a sea of fermions [elementary particles], a genetic pool, or an epidemiology experiment at the flick of a console key," they wrote this year in the Dutch journal *Physica D*.

Some physicists aren't convinced that CAM-8 and its ilk offer a route to faster simulations, let alone a better description of physical processes. Cellular automata, they say, are curiosities with little relevance to their own work. MIT theoretical physicist Felix Villars comments, "I have not seen a demonstration that convinced me that cellular automata machines can do anything that I cannot do without them." Other researchers, more impressed with the potential of cellular automata, still worry about the difficulty of finding local rules that correspond to real natural systems.

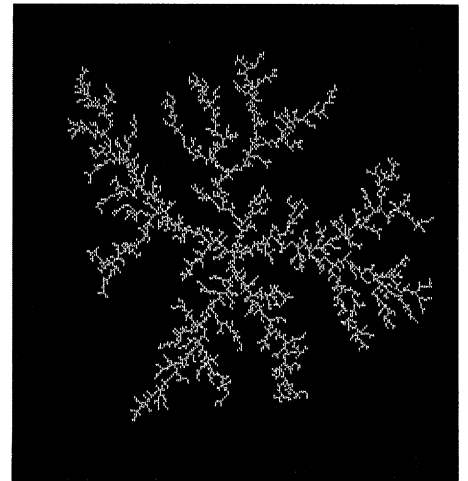
Margolus and Toffoli hope CAM-8 will convince some of the doubters. "We want the machine to make it attractive to people to look for cellular automata models of physical phenomena," Margolus says. Although the concepts behind cellular automata date back to the work of mathematicians and computer pioneers John von Neumann and Stanislaw Ulam in the 1940s, conventional computers are ill-suited to exploring cellular automata models.

That's why Toffoli set about building the first CAM in his garage 9 years ago. He and Margolus refined that mother of all CAMs several times, and in the mid and late 1980s, they even collaborated with a company to try marketing one of their creations, CAM-6, by modifying it into a board that could plug into personal computers. That effort faltered—largely because it could not meet consumer demand for the product, according to Toffoli—but a new company, Automatrix, Inc., in Rexford, New York, headed by electrical engineer Robert C. Tatar, is giving the idea another, more committed, whirl. In June, the company began selling CAM-PC, another personal-computer add-on based on CAM-6. But that will seem like a toy compared to the more specialized and powerful CAM-8.

When it comes to running cellular automata models, the TV-sized CAM-8 pro-

tototype, containing a fraction of the circuitry of conventional supercomputers, should outpace them by a factor of as much as 10, Margolus and Toffoli expect. And they think a full-blown CAM-8, containing as much hardware as a Cray or Connection Machine, would be able to perform a trillion or more cellular automata computations per second, boosting its advantage to a factor of about 1000. In the long run, a machine built of multiple CAM modules might multiply that figure even further, to several million.

The secret of this postulated speed lies in the close match of CAM-8's internal architecture to the kind of computations required by cellular automata models. Like some existing supercomputers—the Connection Machines, for example—CAM-8 has multiple processors, working in parallel.

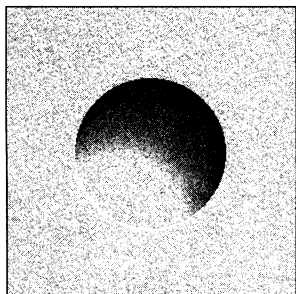


Fake window frost. CAM-6 simulates particles diffusing and aggregating into dendritic structures.

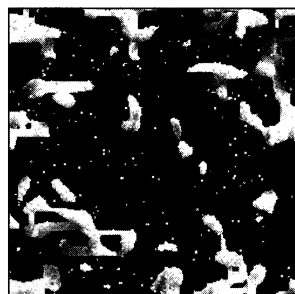
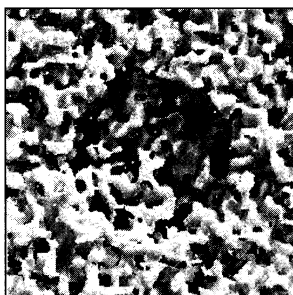
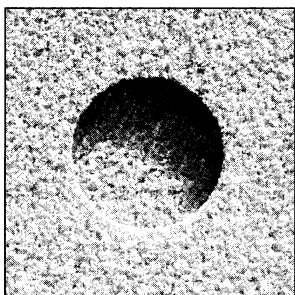
But instead of all being interlinked, each processor in CAM-8 talks only to neighboring ones, cutting down greatly on time-consuming long-distance communications as the processors calculate the states of millions of "cells" residing in high-capacity memory chips. Thus "CAM-8 is doing something simple all the time as fast as it can," notes Charles Bennett, a physicist at IBM's Thomas J. Watson Research Center in Yorktown Heights, New York, who studies the physics and limits of computation.

"It's a beautiful exercise in novel computation," adds Brosi Hasslacher, a physicist at Los Alamos National Laboratory. Hasslacher is looking much further ahead, envisioning palm-sized CAM-like supercomputers in which the cells might reside in nanometer-scale "quantum dot" structures and the local rules might be embodied in quantum mechanical interactions between electrons confined within neighboring dots. Margolus shares a similar vision: a cellular automata computer with components on the atomic or

Toffoli and Margolus



Materials on the move. A cellular automaton depicts how an input of energy alters a three-dimensional distribution of material.



T. Cloney

molecular scale. "Such a machine would be a kind of computing crystal with all parts participating in the computation," Margolus says—a lump of "pure computronium."

To turn any CAM into a simulacrum of a specific physical system, Toffoli explains, an investigator interprets cells in the computer's memory banks as, say, atoms in a lump of gold, molecules crystallizing from a vapor, bacteria in a Petri dish, smoke particles in a wind tunnel, volumes of atmosphere in a thunderstorm, or parcels of interstellar hydrogen collapsing into a star. Each cell includes information about the state of the atom, molecule, living cell, or dust particle—whether it is moving, say, or electrically charged. "We are synthesizing universes here," Toffoli says like a man with a mission.

But setting a model universe in motion takes more than just mapping its pieces onto the cells in the computer. A universe needs laws—and in a cellular automaton model that means local rules. Discovering rules that successfully describe natural phenomena remains the central challenge of the field. Hasslacher and colleagues in France have scored one notable success by developing transition rules that enable cellular automata to model many problems in fluid dynamics, such as the propagation of sound waves and the flow of liquid through a pipe. But so far cellular automata enthusiasts haven't come up with a good general method for finding such rules. Until they do, prospects for cellular automata will remain uncertain.

Still, the sight of a cellular automaton simulation evolving on a computer monitor is striking enough for witnesses to think they are watching the inner workings of reality, says Tatar of Automatrix. Consider a model of vapor molecules assembling into a crystal on a surface, like frost growing on a window: "The seed crystal stays fixed as gaseous molecules seem to jiggle and bounce around. When the gas molecules touch the seed, it looks as if a chemical reaction takes place and they stick to the seed. In a few minutes, you see a dendritic structure emerge," he says.

The promise of running more compli-

cated models much faster, Toffoli and Margolus think, surely will help CAM-8 and its progeny earn a place in the computational toolbox of scientists and engineers. But they see a deeper message in those compelling screen displays—a hint that physical reality is more like a cellular automaton, made up of tiny pieces interacting in discrete time steps with their neighbors,

atoms, or subatomic particles—so too might space and time, the two researchers suggest.

Not everyone buys that line. Villars of MIT, for one, feels that Margolus and Toffoli have been too quick to claim philosophical weight for cellular automata. But in the end, says IBM's Bennett, "it's probably a matter of taste and aesthetics." "If you have a lurking feeling that there is a discrete underlying structure for our physics," he says, you may be ready to accept program-mable matter as something more than a catchy metaphor.

■ IVAN AMATO

New Clue Found to Alzheimer's

Alzheimer's is a puzzling disease, raising a host of questions for which answers are only now beginning to accumulate. A central paradox is whether the protein called β -amyloid, which forms the core of the abnormal plaques that stud the patients' brains, actually causes the nerve cell degeneration of Alzheimer's or is merely the result of that degeneration. Now, a team of Boston researchers has given a big boost to the idea that β -amyloid causes the nerve cell degeneration by showing for the first time that the peptide can produce neuronal damage, similar to that seen in Alzheimer's, in the brains of living animals.

What's more, the research team, led by Bruce Yankner of Children's Hospital in Boston and Neil Kowall of Harvard's Massachusetts General Hospital, showed that the degeneration can be blocked by another peptide—substance P—which is one of the chemicals the body uses to transmit nerve signals. Taken together, the findings provide "an important insight for drug development," says Zaven Khachaturian, who oversees Alzheimer's research for the National Institute on Aging (NIA). Not only do they suggest that substance P or related compounds might be useful for Alzheimer's therapy, but the work may also provide an animal model for testing the effectiveness of potential therapeutic drugs.

According to Yankner, the new results, published in the 15 August issue of the *Proceedings of the National Academy of Sci-*

ences, are an outgrowth of previous research in which he and his colleagues showed that β -amyloid causes nerve cells growing in culture to degenerate. Now they've injected pure, synthetic β -amyloid directly into two areas of rat brains, the hippocampus and cortex, that are severely affected in human Alzheimer's. "We tried to create an experimental plaque," Yankner says. The result? "The animals showed profound neuronal degeneration around the plaques."

Even more intriguing, antibody studies indicated that the neurons surrounding the experimental plaques contain a protein known as tau, which is a major component of the neurofibrillary tangles, a second characteristic feature of Alzheimer's pathology. Although plaques and tangles are found together throughout Alzheimer's brains, nobody knows whether the two are linked in any way. This work suggests they are—that β -amyloid deposition might be somehow involved in inducing tangle formation.

But if substance P is given at the same time or a little before the β -amyloid injections, Yankner says, formation of both plaques and tangles can be prevented. And that's important for a couple of reasons. For one thing, a great deal of evidence suggests β -amyloid deposition alone doesn't cause the nerve cell death of Alzheimer's. Yankner and Kowall's results suggest that substance P deficiency might be one of the other factors.

And then, of course, there are the therapeutic implications. According to Khacha-