# Infant growth: A sporadic phenomenon

Anthropologist Michelle Lampl, trained as a physician and growth researcher, recalls learning that healthy young children experience "a perfectly regular rate of growth, with no breaks or spurts." Indeed, she says, she recently listened to a growth researcher tell pediatricians that any child who does not grow over a 30-day period must be dead.

But Lampl's own findings challenge the notion of smooth, consistent growth, and instead suggest that children grow in sporadic fits and starts.

Conventional wisdom holds that very young children gain an average of about one-half millimeter in body length per day. But Lampl's measurements of 32 healthy infants and one adolescent indicate that growth occurred in a random series of roughly 1-centimeter spurts, each apparently lasting less than 24 hours. During the two to approximately 60 days that separated successive spurts, she says, absolutely no increases in body length occurred.

Lampl, of the University of Pennsylvania in Philadelphia, described her new study in Chicago this week at a meeting of the American Association for the Advancement of Science.

Over periods of four to 18 months, she visited the homes of the youngsters. With the help of a parent, she stretched out and measured each child – four of them daily, 18 twice-weekly and 11 at weekly intervals. Her daily measurements "provide the most precise description of growth yet reported," she says.

The daily data document long quiescent periods of no growth, suddenly punctuated by a permanent lengthening of 0.5 to 1.8 cm over a 24-hour period. Lampl describes these nonperiodic, stepwise changes in the growth curve as "saltatory," or abruptly jumping. Because saltatory spurts of similar magnitude showed up in children who were measured weekly or twice-weekly, she suspects that these growth changes occurred over a 24-hour period.

While conceding she used a very small study population, Lampl says she saw no signs of a correlation between infant size and the total number of discrete growth episodes. However, she notes, "there was a distinct correlation between fussiness and [increased] hunger at the time of the growth episodes." Parents also reported signs of increased sleepiness right before growth spurts, she adds.

At the same meeting, Michael Hermanussen of the University of Kiel in Germany described a study of lower-leg length in healthy schoolchildren. He found evidence of weekly changes, with growth spurts following no-growth periods that sometimes lasted more than 60 days (including occasional intervals of shrinkage). "I was not aware of saltatory [changes in these data]," he says. However, he adds, "I'm aware that I might have missed them."

That's not surprising, says Lampl, because until now, growth researchers have lacked mathematical models for stepwise changes that are nonperiodic. Without such models, they have attempted to fit their growth data points – usually collected weeks or months apart – to a smooth curve. But Lampl found that such a curve didn't really fit her detailed data.

For help in finding a better curve, she turned to biophysicist Michael L. Johnson, handing over her data on a 13-year-old boy whose height she had measured on about 400 consecutive days. Johnson, of the University of Virginia in Charlottesville, reported at the Chicago meeting that a stepwise, saltatory model fits these data better than any previous model. Without Lampl's daily data on dormant periods and growth pulses, the flaws in the old approach remained unrecognized, he adds.

"I would never have imagined pulsatile growth," comments Mark L. Hartman of the University of Virginia School of Medicine, who studies factors affecting growth-hormone secretion and its relationship to human growth.

The body's pulsed secretion of growth hormone can trigger metabolic changes, such as increased protein synthesis, Hartman notes. And since his group has recently shown that growth-hormone pulses occur frequently throughout the day, sometimes at intervals of just 30 seconds, some of his colleagues suspected that these pulses contribute to slow, incremental daily growth. But Lampl's results certainly confound that picture, he adds.

"My mind is going 100 miles a minute trying to explain the new data," Hartman told SCIENCE NEWS. "I think I will have to talk to my colleagues and see if we can generate some new ideas to explain these new findings." *– J. Raloff*

# Ozone concerns prompt phaseout fury

Reacting to last week's news that an ozone hole could open over North America, President Bush announced this week that the United States will halt production of ozone-depleting chemicals by the end of 1995, four years ahead of schedule. But a loophole in Bush's proposed policy would allow significant production of damaging chlorofluorocarbons (CFCs) and other chemicals after that date.

Under the President's plan, companies could continue producing the banned chemicals for "essential uses and for servicing certain existing equipment." The Alliance for Responsible CFC Policy in Arlington, Va., estimates that, to provide for existing equipment, production would have to continue at 15 percent of its 1986 level. If the President's policy allows this production level, the new controls would speed the phaseout process by only one year. Current U.S. law requires companies to limit their production to 15 percent of 1986 levels by the end of 1996.

The Alliance, which represents companies that produce and use CFCs, praised the President's policy for balancing environmental and economic concerns. It estimates that by 1996, the United States will have $135 billion in equipment that relies on CFCs.

Liz Cook, with Friends of the Earth in Washington, D.C., calls the exemption "a big loophole." Last year, a coalition of U.S. environmental groups called for a total ban on production of CFCs by the end of 1994, with an immediate phaseout of halons and phaseout of other chemicals by the end of 1992.

Negotiators will meet later this year to discuss strengthening the Montreal Protocol – an international treaty governing the phaseout of ozone-depleting chemicals. Like the U.S. regulations, the Montreal Protocol requires a decrease to 15 percent of 1986 production levels by the end of 1996, with a complete phaseout by 2000. *– R. Monastersky*

# Myotonic dystrophy: A short gene is best

For years, neurologists have noticed a strange phenomenon: Patients afflicted by myotonic muscular dystrophy, a muscle-stiffening disorder, often have children with a more severe form of the disease. And their children's children, in turn, are usually affected even more severely, and at a younger age.

What causes this distressing genetic generation gap? Last week, three groups of researchers studying myotonic dystrophy came closer to answering that question in simultaneous findings that they say should allow for better screening tests for the inherited disease.

The three groups of geneticists found that people with myotonic dystrophy have extra bits of DNA at a specific spot on the long arm of chromosome 19. Moreover, they discovered that those with the worst symptoms have the most extra DNA.

In the Feb. 6 NATURE, the researchers speculate that the extra DNA pieces somehow disrupt an as-yet-unidentified gene, one possibly involved in controlling muscle tone. The three teams – led by Duncan J. Shaw of the University of Wales

College of Medicine in Cardiff, Keith Johnson of Charing Cross and Westminster Medical School in London and Pieter J. de Jong of the Lawrence Livermore (Calif.) National Laboratory – are now attempting to isolate and characterize the gene.

Myotonic dystrophy, the most common form of muscular dystrophy affecting adults, strikes roughly one in every 8,000 persons worldwide. Symptoms of the disorder – which usually emerges in adolescence or early adulthood – include muscle spasms and wasting, particularly in the head and neck. While mildly affected people may simply have difficulty unclenching a fist, those with more severe forms of myotonic dystrophy cannot walk and have difficulty swallowing. The disorder's other symptoms include cataracts, premature balding, shrunken ovaries or testicles, and mental retardation.

To uncover the genetic defect, the three research groups used enzymes to chop DNA taken from myotonic dystrophy patients into tiny pieces. After sorting the pieces by size on a gel, all three groups found that myotonic patients had longer DNA pieces than did healthy individuals. They also found that the affected children of people with the disorder had even longer pieces than their parents.

Myotonic dystrophy is only the third genetic disease that scientists have associated with longer-than-normal DNA segments. Last year, U.S. and Dutch researchers found that people with fragile X syndrome – the most common inherited cause of mental retardation – bear repetitive DNA segments in a gene they named *FMR*-1, for fragile X mental retardation-1 (SN: 6/8/91, p.359). Such enlarged genetic segments have also been discovered in spinal-bulbar muscular atrophy, a rare inherited muscle-wasting syndrome.

Myotonic dystrophy may be an example of "genetic imprinting," in which the same gene produces a different effect, depending on which parent provides the gene (SN: 5/20/89, p.312). Mothers with myotonic dystrophy tend to have babies severely affected by the disease from birth.

The new finding should "permit new approaches to understanding the molecular pathology [of myotonic dystrophy]," Johnson says. He adds that some medical laboratories can now use the same technique as his research team to tell whether the children of myotonic dystrophy patients will also suffer from the disease, and if so, how severely it may affect them.

The anticipated discovery of the gene disrupted in myotonic dystrophy may also lead to a treatment for the disorder, says Leon Charash, chairman of the medical advisory committee of the Muscular Dystrophy Association. He predicts that researchers will identify the gene underlying myotonic dystrophy "in the very, very near future . . . possibly within the next six months." – *C. Ezzell*

# Light, chemicals modify silicon's glow

With the first reports of silicon luminescence not yet two years old, scientists have scrambled to modify and harness this property to make faster computers and new optoelectronic devices (SN: 12/14/91, p.399). Now chemists have discovered another way to exploit silicon's glow, while physicists and engineers report progress in controlling and understanding this property.

Light or electricity will make porous silicon emit light. But the addition of organic compounds to the surface of this silicon, made porous by the acid-etching, will temporarily change or stop that luminescence, says Michael J. Sailor, a chemist at the University of California, San Diego. Different solvents change the silicon's reddish luminescence in characteristic ways, he and his research team will report in the Feb. 26 JOURNAL OF THE AMERICAN CHEMICAL SOCIETY.

"[Porous silicon] is a reversible chemical sensor," says Sailor, "and it's incredibly sensitive." For example, an etched wafer can distinguish benzene from toluene – similar molecules that differ by one carbon and two hydrogen atoms, adds Sailor.

In addition, Sailor and his San Diego colleague Vincent V. Doan have used light to create patterns of luminescence in a silicon wafer, they report in the Feb. 3 APPLIED PHYSICS LETTERS.

Scientists make silicon porous by placing it in acid and passing a low current across it. Doan and Sailor modified the process by projecting a high-contrast pattern – and, later, someone's picture – onto the silicon during etching.

Light creates a small current in silicon, and varying the current during etching alters the color of the luminescence. "We realized there was a way to direct the photochemistry," Sailor says. The light projected onto the silicon wafer adds to, or subtracts from, the current passing across the wafer in the acid bath, depending on the type of silicon used.

Where bright light hits phosphorus-doped silicon, red luminescence results; dim light leads to an orange glow; darkened parts do not glow at all. "You are not limited to black and white," Sailor says. "You have a range of colors that allow you to pack more information into this thing."

This approach is simpler than other lithographic techniques for patterning silicon chips, says Sailor. The 20-micron resolution Sailor and Doan achieved does not come close to the single-micron resolution possible in integrated circuits. But this technique could prove useful for connecting adjacent computer chips via optical fibers instead of copper, for faster computing, he notes.

Steven Shih and his colleagues at the University of Texas at Austin took a different approach to controlling luminescence in silicon samples. By exposing silicon wafers to oxygen and then heating them before etching, these electrical engineers changed the range of colors emitted, they report in the Feb. 3 APPLIED PHYSICS LETTERS. After etching, unoxidized silicon glows with a wide range of colors and looks red. Oxidation narrows that range, making the glow more orange, the Texas researchers note.

Sailor/U Calif. San Diego

*A face projected onto silicon during etching made a wafer glow in this image.*

In addition, Michael A. Tischler, a physicist, and his colleagues at the IBM Thomas J. Watson Research Center in Yorktown Heights, N.Y., investigated why silicon's glow changes over time when the wafer is exposed to air. They put a wafer in a chamber and flowed nitrogen, hydrogen or oxygen over it. The first two gases had little effect, but oxygen in the presence of light caused the luminescence to fade by two orders of magnitude, they report in the Feb. 3 APPLIED PHYSICS LETTERS.

These results strengthen the idea that hydrogen – which links with silicon during etching – plays an important role in a wafer's optical properties, says Reuben T. Collins, a physicist who works with Tischler. Hydrogen's role is to tie up open-ended bonds – typically found in silicon – that could otherwise take up the extra energy that forms the basis for luminescence. That energy comes from the light or electrical current, which excites silicon's electrons. The electrons then give off light energy to calm down again.

Oxidation disrupts the connections between silicon atoms and leads to the recreation of open-ended bonds. These dangling bonds allow the electrons to return to their original state without emitting light, so the luminescence disappears, says Collins. – *E. Pennisi*