CRIMETimes

Linking Brain Dysfunction to Disordered/Criminal/Psychopathic Behavior

Volume 9, Number 2, 2003

'Body burden': studies reveal ubiquity of toxic chemicals in U.S.

A mericans carry a toxic "body burden" of hundreds of synthetic chemicals and other contaminants, according to a new study by the Centers for Disease Control and Prevention (CDC).

Jim Pirkle and colleagues, who conducted the study, collected urine and blood samples from approximately 2,500 volunteers across the country, testing participants for the presence of 116 different contaminants. They found all 116 of the chemicals (which included toxic metals, combustion byproducts, and byproducts of pesticides, insect repellents and herbicides) present in at least some portion of the study subjects, with many present in more than half of the people they tested. (See related articles on pages 1 and 2 of this issue.)

Their data, the researchers say, paint "a mixed picture, some encouraging findings and some of concern." Among their findings:

- While the number of children aged one to five with very high lead levels has dropped from 4.4 to 2.2 percent since the early 1990s, more than 400,000 young children in America still have dangerously high levels.
- More than half of nonsmokers over the age of three have detectable levels of continued on page 2

New research links pesticides to ADHD, other ills

Salk Institute researchers say a single gene variant could predispose many people to develop a range of neurological disorders, ranging from attention deficit hyperactivity disorder (ADHD) to Gulf War syndrome, following exposure to certain pesticides. The researchers say their findings could explain anecdotal evidence of a link between pesticide exposure and neurological dysfunction.

Carrolee Barlow, Christopher Winrow and colleagues found that in mice, exposure to organophosphates (a group of chemicals that include nerve gas and certain pesticides) inhibits the activity of a gene called neuropathy target esterase, or NTE. Mice bred to lack the NTE gene died before birth, but those with only one copy of the gene exhibited marked ADHD-like behavior when exposed to organophosphates. Mice with two functional NTE genes also exhibited behavior similar to ADHD after organophosphate pesticide exposure, but their symptoms were far milder.

The researchers note that the mice with only one copy of the NTE gene had a 40 percent decrease in the enzyme produced by the gene, which is active in areas of the brain controlling movement (including the hippocampus, cerebellum, and spinal cord).

Barlow says, "NTE is a large gene. It's possible that we all have slightly different forms of the NTE enzyme, which may explain why some may get ADHD when they're exposed at young ages, and why some may get Gulf War syndrome at a later age, or why some of us have no symptoms at all. It appears to be a case of delayed toxicity, inhibiting the function of NTE."

The findings of Barlow and Winnow follow earlier research by Elizabeth Guillette and colleagues (see *Crime Times* Vol. 4, No. 3, 1998, page 1) linking pesticide exposure

Barlow says variations in the NTE gene "may explain why some may get ADHD when they're exposed [to organophosphates] at young ages, and why some may get Gulf War syndrome at a later age, or why some of us have no symptoms at all."

to neurodevelopmental problems. Guillette et al. compared Yaqui Indian children exposed to high or low levels of agricultural pesticides, and found that the children with higher pesticide exposure showed learning disabilities, behavior problems, impaired memory, motor problems, and other signs of significant brain dysfunction.

"Loss of neuropathy target esterase in mice links organophosphate exposure to hyperactivity," Christopher J. Winrow, Matthew L. Hemming, Duane M. Allen, Gary B. Quistad, John E. Casida, and Carrolee Barlow, Nature Genetics, advance online publication, March 17, 2003. Address: Carrolee Barlow, carrolee_barlow@merck.com.
—and—

"Genetic link may tie together pesticides, ADHD, Gulf War syndrome and other disorders," press release, Salk Institute, March 17, 2003.

'Body burden:' Americans exposed to hundreds of toxins (continued from page 1)

cotinine, a byproduct of tobacco smoke, in their bloodstreams. Levels are more than twice as high in children as they are in adults, and higher in African-Americans than in other groups. Overall, however, levels have dropped by 50 percent or more in nonsmokers in every age group.

- Levels of chlorpyrifos, an organophosphate pesticide widely used in residential settings until 2001, are twice as high in children as in adults. Levels of DDE, a metabolite of DDT (a pesticide banned in 1973), are three times higher in Mexican Americans than in other groups. "Additionally," the researchers say, "DDE levels were clearly measurable in people aged 12 to 19 years, even though people in this age group were born after DDT was banned in the United States." However, they note, population levels of DDE are far lower than before 1990.
- Phthalates, found in many cosmetics, household products and soft plastics, are present in a large percentage of the population. Levels of one particular phthalate, found in plastic products such as infant toys, were highest among children. These chemicals are believed to interfere with normal hormonal metabolism, increasing the risk of cancer or behavioral changes.
- Levels of banned PCBs and dioxins were too low to detect in most people.

The researchers say that the reductions in levels of lead, cotinine, PCBs, and DDE are encouraging evidence that environmental regulation and education efforts are leading to drops in toxic exposure. However, other researchers say that overall, the CDC data are alarming. Says pediatrician and toxins expert Philip J. Landrigan,

M.D., "The bottom line... is that a whole raft of synthetic chemicals that simply did not exist 40 or 50 years ago is now in the bodies and in the bloodstreams of most Americans."

In a related report, a study by the Environmental Working Group and Mt. Sinai's School of Community Medicine found 167 chemical contaminants in the urine and blood of nine study participants. Of these, the researchers say, 94 are toxic to the brain and nervous system, and 79 can cause abnormal development.

They add, "Subjects contained an average of 91 compounds, most of which did not exist 75 years ago....

Both studies [this one and the CDC study] reveal disturbing gaps in scientific understanding of environmental contaminants and in our system of regulatory safeguards."

National Report on Human Exposure to Environmental Chemicals, J. Pirkle et al., Centers for Disease Control and Prevention, January 31, 2003, http://www.cdc.gov. See also: "CDC releases most extensive assessment to date of Americans' exposure to environmental chemicals," http://www.cdc.gov.

-and-

"Body burden," Environmental Working Group, January 2003, http://www.ewg.org.

—and—

"Proof of burden," Ben Harder, Science News, Vol. 163, February 22, 2003.

Prenatal mercury linked to cognition, language delays

The Centers for Disease Control and Prevention (CDC) issued a report several years ago warning that ten percent of women in America are at risk of having infants with neurological disorders due to prenatal exposure to high levels of environmental mercury. A new study adds to the evidence supporting that warning, showing that children exposed to mercury in utero have a high rate of language and cognitive problems.

The new study involved residents of Tagum, an area of the Philippines where residents are exposed to high levels of mercury due to mining activity. G. B. Ramirez et al. analyzed 48 Tagum children's cord blood and meconium mercury levels, head circumference at birth, and duration of breastfeeding, and then measured their cognition and linguistic ability at age 2 using the CAT/CLAMS (a combination of the Cognitive Adaptive Test and the Clinical Linguistic Auditory Milestone Scale). The subjects were compared to chil-

dren from Saranggani, another area of the Philippines.

"The mercury level in cord blood was negatively correlated with CAT/CLAMS at two years," the researchers say. They found that the mercury-exposed Tagum children scored lower than control children on both the CAT and the CLAMS, and had lower scores in both expressive language and full-scale development. "Fifteen percent of Tagum subjects had global delay," they note, "versus 5.48 percent in Saranggani controls." Length of breastfeeding did not correlate with developmental scores in this study.

"Tagum study II: follow-up study at two years of age after prenatal exposure to mercury," G. B. Ramirez, O. Pagulayan, H. Akagi, A. Francisco Rivera, L. V. Lee, A. Berroya, M. C. Vince Cruz, and D. Casintahan, *Pediatrics*, Vol. 111, No. 3, March 2003, e289-95. Address: G. B. Ramirez, Research Development Office, Philippine Children's Medical Center, Quezon City, Philippines, rdo@hiss.pcmc.org.ph.

Low DHA again linked to symptoms of depression

L acids are strongly linked to depression, bipolar disorder, and learning and attention problems (see *Crime Times* Vol. 5, No. 1, 1999, p. 1, and Vol. 8, No. 2, 2002, p. 2). A new study adds to this evidence, showing a powerful link between depression and low adipose tissue levels of the omega-3 fatty acid DHA (docosahexaenoic acid).

G. Mamalakis et al. studied 247 healthy adults (146 men and 101 women), assessing their self-ratings of depressive symptoms. They found that mildly depressed individuals had nearly 35 percent lower levels of DHA in their adipose tissue than participants with no depressive symptoms. For the group, depressive symptoms correlated negatively with amounts of adipose tissue DHA.

"In line with the findings of other studies," the researchers say, "the observed negative relation between adipose tissue DHA and depression in the present study appears to indicate increasing long-term dietary DHA intakes with decreasing depression." Noting that depression is associated with increased production of inflammatory cytokines, and that omega-3 fatty acids inhibit cytokine synthesis, they conclude, "The observed negative relation between adipose DHA and depression, therefore, may stem from the inhibiting effect of DHA on cytokine synthesis."

"Depression and adipose essential polyunsaturated fatty acids," G. Mamalakis, M. Tornaritis, and A. Kafatos, *Prostraglandins, Leukotrienes and Essential Fatty Acids*, Vol. 67, No. 5, Nov. 2002, 311-8. Address: G. Mamalakis, Department of Social and Preventive Medicine, University of Crete, Iraklion, Crete, Greece.

ADHD girls exhibit serious social, behavioral problems

ong considered largely a "boys' problem," attention deficit hyperactivity disorder (ADHD) affects many girls and causes them significant academic and social difficulty, according to a study by Stephen Hinshaw and colleagues.

The researchers recruited 228 6-to 12-year-old girls to attend sixweek summer camps, held over three successive years. Of the girls, 140 were diagnosed with ADHD. The ADHD girls were taken off their medications prior to the camps, so that the researchers could evaluate their natural behavior patterns. Counselors, unaware of which girls were diagnosed as ADHD, observed the interactions and behavior of the campers.

Compared to the non-ADHD controls, Hinshaw says, the ADHD girls "are very impaired, academically and socially." He notes, "The girls with ADHD were less likely to follow the directions of the teacher than the comparison girls. They were also more likely to tease their peers and show aggressive behavior, though not at the same rate as boys with ADHD in previous summer camps. They were also more likely to display social isolation—wandering

and failing to become engaged in activities."

Evaluating the ADHD girls' social relationships, the researchers found that they "had fewer mutual friends and were more likely to have no friends." Overall, they say, "although girls with ADHD were able to make friends to some extent, they differed from comparison girls in terms of the likelihood of doing so, the ability to maintain the friendships that they did form, and the levels of negative features found in their friendships."

When Hinshaw and colleagues administered neuropsychological tests to a group of the ADHD girls, the tests revealed significant deficits in executive function—skills, associated with the brain's frontal lobes, that include self-control, goal-setting, long-range planning, and flexible responses to changing situations. "These functions are crucial for long-term academic, social and occupational success," Hinshaw says. "Deficits in executive functions are seen in other disorders, such as autism, but they may well be the core underlying problems for youth and adults with ADHD."

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Omega-3 supplement aids in recovery from depression

Antidepressant drugs can be highly effective in treating symptoms of depression, but some patients do not respond to the medications and in many others the effects of these drugs wear off over time. An Israeli study, however, indicates that augmenting drug treatment with supplements of omega-3 fatty acids can enhance the effects of antidepressant drugs.

In a four-week double-blind, pla-

cebo-controlled study, Boris Nemets and colleagues gave 20 patients with recurrent major depression either a placebo or capsules containing the omega-3 fatty acid E-EPA (ethyleicosapentaenoic acid). All participants also were taking antidepressant medications, but had continued to experience symptoms despite increased medication doses, good compliance, and supportive psychotherapy.

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Book Review: The Blank Slate, by Steven Pinker

2002, Viking Press, Hardback, \$27.95

When a child fails at school, hurts other children, or takes illegal drugs, the almost-universal tendency is to blame the child's parents. But as Steven Pinker shows in his new book, children are not born with "a clean slate," and parenting has vastly less influence on a child's behavior than do genes.

As this carefully researched book (which includes 900 references) clearly reveals, children are born with different talents, temperaments, and cognitive abilities. The greater a child's innate ability to use logic and insight, the more he or she will benefit from good parenting—but, conversely, the less damage poor parenting will elicit. As for the children who murder or rape, Pinker says, "genetics and neuroscience are showing that a heart of darkness cannot always be blamed on parents or society."

Denying that human nature is largely innate and genetically determined, Pinker says, leads to misguided social and political policies. For instance, he says, the money we spend on trying to change troubled children's home environments could be better spent on identifying and understanding genetic influences on dysfunctional behavior.

Pinker, an MIT professor, is widely considered to be one of the world's leading cognitive scientists. His brilliant book is an important addition to a growing body of research that shows that our genes play a far larger role in our behavior than most of us—including the "experts"—realize.

Quotes from The Blank Slate, by Steven Pinker:

"Some faculties may endow us with greed or lust or malice, but others may endow us with sympathy, foresight, self-respect, a desire for respect from others, and an ability to learn from our own experiences and those of our neighbors. These are physical circuits residing in the prefrontal cortex and other parts of the brain, not occult powers of a poltergeist, and they have a genetic basis and an evolutionary history no less than the primal urges."

"When scientific facts come in they rarely conform exactly to our expectations; if they did, we would not have to do science in the first place. So when facts tip over a sacred cow, people are tempted to suppress the facts and to clamp down on debate because the facts threaten everything they hold sacred. And this can leave us unequipped to deal with just those problems for which new facts and analyses are most needed."

"The belief on the left that human nature can be changed at will, and the belief on the right that morality rests on God's endowing us with an immaterial soul, are becoming rearguard struggles against the juggernaut of science."

"'All traits are heritable' is a bit of an exaggeration, but not by much. Concrete behavioral traits that patently depend on content provided by the home or culture are, of course, not heritable at all: which language you speak, which religion you worship in, which political party you belong to. But behavioral traits that reflect the underlying talents and temperaments *are* heritable:

how proficient with language you are, how religious, how liberal or conservative. General intelligence is heritable, and so are the five major ways in which personality can vary: openness to experience, conscientiousness, extroversion-introversion, antagonism-agreeableness, and neuroticism. And traits that are surprisingly specific turn out to be heritable, too, such as dependence on nicotine or alcohol, number of hours of television watched, and likelihood of divorcing."

"[T]he doctrine that the mind is a blank slate has distorted the study of human beings, and thus the public and private decisions that are guided by that research. Many policies on parenting, for example, are inspired by research that finds a correlation between the behavior of parents and the behavior of children.... Parents, remember, provide their children with genes, not just a home environment. The correlations between parents and children may be telling us only that the same genes that make adults loving, authoritative, and talkative make their children self-confident, well-behaved, and articulate."

"[C]onvicted murderers and other violent, antisocial people are likely to have a smaller and less active prefrontal cortex, the part of the brain that governs decision making and inhibits impulses. These gross features of the brain are almost certainly not sculpted by information coming in from the senses, which implies that differences in intelligence, scientific genius, sexual orientation, and impulsive violence are not entirely learned."

Short-term Ecstasy use may lead to long-term grief

ven brief experimentation with the "club drug" Ecstasy (MDMA) could put adolescents and voung adults at risk for lifetime depression, according to a recent study.

Lynn Taurah and Chris Chandler studied 519 subjects including current and past Ecstasy users, people who used other drugs such as cocaine and amphetamines but not Ecstasy. and people who did not use illicit drugs. The researchers asked participants to fill out a questionnaire measuring depressive symptoms, with a score of 25 or more indicating clinical depression. Subjects were divided into three categories: frequent Ecstasy users (more than 20 times), infrequent users (one to 19 times), and nonusers.

The researchers found that people who did not use Ecstasy, including subjects who took other drugs, had average depression scores of about four points. In contrast, Ecstasy users—including those who had used the drug only one or two times—had scores as high as 16 or 17, and chronic users had scores as high as

Taurah says, "People often think taking Ecstasy just once or twice won't matter, but what we're seeing is evidence that if you take Ecstasy a couple of times you do damage to your brain that later in life will make you more vulnerable." She adds, "[W]e've got a group taking every other kind of drug, including amphetamines, ketamine and cocaine, and they haven't got these depression scores." This, she says, argues against the idea that the depressive symptoms of Ecstasy users predated drug use.

The researchers' findings support those of laboratory scientists who are reporting that Ecstasy damages neurons which produce the neurotransmitter serotonin. Serotonin abnormalities are linked to depression as well as to other psychiatric problems including impulsive aggression and suicide attempts. In addition, the drug appears to damage neurons involved in the production of the neurotransmitter dopamine. Among recent findings:

Researcher Andy Parrott reports

"Experimental studies report that

MDMA's neurotoxic effects on

serotonin neurons in primates are

extremely long-lasting and may be

permanent," say Una McCann and

colleagues.

that a review of the literature on Ecstasy shows that "repeated doses of MDMA cause serotonergic neurotoxicity in laboratory animals, and there is extensive evi-

dence for long-term neuropsychopharmacological damage in humans." He notes that even after stopping their use of the drug, regular Ecstasy users "often display reduced levels of 5-HT, 5-HIAA, tryptophan hydroxylase and serotonin transporter density," all evidence of impaired serotonin system function. In addition, he says, studies of Ecstasy users show deficits in learning and memory, higher cognitive processing, sleep, appetite, psychiatric well-being, and sexual desire.

- Using PET scans, R. Buchert and colleagues in Germany found that compared with non-drug users, Ecstasy users showed significant alterations in serotonin transporter distribution in several brain regions. Tests of past drug users indicated that these changes may be reversible to some degree.
- Johns Hopkins researchers reported that primates exposed to several doses of Ecstasy "developed severe brain dopaminergic neurotoxicity, in addition to less pronounced serotonergic neurotoxicity." They concluded, "MDMA users may unwittingly be putting themselves at risk, either as young adults or later

in life, for developing neuropsychiatric disorders related to brain dopamine and/or serotonin deficiency."

Una McCann and colleagues caution, too, that reports that Ecstasy damage may be at least partially reversible should not lead to a false sense of security. "Axonal regeneration in the adult brain may lead to

> abnormal, dysfunctional circuitry," they say, adding, "Experimental studies report that MDMA's neurotoxic effects on serotonin neu-

rons in primates are extremely longlasting and may be permanent." McCann and George Ricaurte cite research showing that in the dorsal neocortex, the density of serotonin axons remains markedly reduced for up to seven years following exposure to Ecstasy.

Research by Lynn Taurah and Chris Chandler reported at the annual conference of the British Psychological Society, March 15, 2003. Quotes appear in "Single Ecstasy tablet could lead to brain damage and depression," L. McDougall, Sunday Herald, March 16, 2003, and "Ecstasy makes users depressed for life," S. Goodchild and K. Johnson, U. K. Independent, March 16,

—and—

"Long-term effects of 'Ecstasy' use on serotonin transporters of the brain investigated by PET," Journal of Nuclear Medicine, Vol. 44, No. 3, March 2003, 375-84. Address: R. Buchert, Department of Nuclear Medicine, University Hospital Hamburg-Eppendorf, Hamburg, Germany.

—and—

"Assessing long-term risks of MDMA (Ecstasy)," George Ricaurte and Una McCann, The Lancet, Vol. 358, No. 9296, December 1, 2001, 1831-2. Address:

Dopamine gene variant linked to externalizing behavior

One variant of a gene that affects dopamine use by the brain is linked to externalizing behavior problems in children, according to a recent study.

The term "externalizing behavior" describes chronic aggression, delinquency, and destructive, oppositional, and impulsive behavior. Children who exhibit such behavior are often treated with drugs such as methylphenidate (Ritalin), which alter the function of the dopamine transporter (DAT). This transporter's job is to terminate the activity of dopam-

ADHD girls exhibit serious social, behavioral problems (continued from page 3)

Although little long-term research has been conducted on females with ADHD, ADHD in males is a risk factor for delinquency, criminality, and social, academic, and career problems (see *Crime Times* Vol. 3, No. 3, 1997, p. 1).

"Patterns of friendship among girls with and without attention-deficit/hyperactivity disorder," D. R. Blachman and S. P. Hinshaw, Journal of Abnormal Child Psychology, Vol. 30, No. 6, December 2002, 625-40; and, "Preadolescent girls with attention-deficit/ hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification," S. P. Hinshaw, E. T. Carte, N. Sami, J. J. Treuting, and B. A. Zupan, Journal of Consulting and Clinical Psychology, Vol. 70, No. 5, October 2002, 1099-111; and, "New studies of girls with ADHD led by UC Berkeley professor reveal overlooked and serious situation," press release, University of California at Berkeley, October 1, 2002.

Address for all: Stephen Hinshaw, Department of Psychology, University of California, Berkeley, CA 94720-1650. ine by transporting it from the synapses—the spaces between neurons—back to the cells that released it.

Susan Young et al. investigated the role of a particular allele (variant) of the DAT1 gene, the 9-repeat allele, on children's externalizing behaviors. The 790 participants in the study were recruited through the Colorado Longitudinal Twin Study and the Colorado Adoption Project.

Evaluating data provided by parents on the children's behavior at 4, 7, and 9 years of age, Young et al. found that the 9-repeat variant of the DAT1 gene "is a significant risk allele for externalizing behavior at ages 4 and 7 years." While the association was nonsignificant at age 9, the researchers say that across the three ages, "the overall association of the 9-repeat DAT1 allele with externalizing behavior is well supported."

Some studies have also suggested that the 9-repeat DAT1 allele is a risk factor for alcohol dependence and substance abuse. A different variant of the DAT1 allele, the 10-repeat variant, is tentatively linked to attention deficit hyperactivity disorder (ADHD).

Young et al. conclude, "These findings encourage continued investigation of the role of the dopamine system in the biological basis of problem behavior."

"Dopamine transporter polymorphism associated with externalizing behavior problems in children," Susan E. Young, Andrew Smolen, Robin P. Corley, Kenneth S. Krauter, John C. DeFries, Thomas J. Crowley, and John K. Hewitt, *American Journal of Medical Genetics*, Vol. 114, No. 2, 2002, 144-9. Address: Susan E. Young, Institute for Behavioral Genetics, Campus Box 447, University of Colorado, Boulder, CO 80309.

Omega-3 fatty acids aid in treatment of depression

(continued from page 3)

After four weeks, the researchers found, six of ten study participants receiving E-EPA, but only one of ten people receiving the placebo, had significantly reduced symptoms. Symptoms of depression, guilt, worthlessness, and insomnia all were reduced in the E-EPA-treated group.

These findings support other reports showing that omega-3 fatty acids are an effective treatment for depression (see related story on page 3, and previous stories in *Crime Times* Vol. 8, No. 3, 2002, page 2, and Vol. 5, No. 1, 1999, page 1).

"Addition of omega-3 fatty acid to maintenance medication treatment for recurrent unipolar depressive disorder," Boris Nemets, Ziva Stahl, and R. H. Belmaker, *American Journal of Psychiatry*, Vol. 159, No. 3, March 2002, 477-9. Address: Boris Nemets, Faculty of Health Sciences, Ben Gurian University of the Negev, Beer Sheva, Israel.

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Animal study: do high levels of the hormone progesterone create bad dads?

Testosterone is the hormone most often linked to male dominance and aggression, but a new study indicates that the hormone progesterone may play a bigger role in how males react to their offspring.

Adult male mice rarely show nurturing behavior toward their offspring, and frequently attack and kill them shortly after the babies are born. Studies have shown that testosterone levels, correlated with adult intermale aggression in animals, do not correlate reliably with paternal nurturing or aggression in mice. Looking for another explanation, Johanna Schneider et al. investigated the effects of progesterone a hormone produced in large amounts by females, and in much smaller amounts by males—on the parenting behavior of male mice.

The researchers created progesterone receptor "knock-out" (PRKO) mice, missing the gene that encodes these receptors (meaning that the mice are not affected by progesterone). Lead researcher Jon Levine reports, "In male knockout mice we noticed something quite startling. They behaved differently, and the most obvious changes were a complete lack of aggression toward infants and the emergence of active paternal care. These animals are terrific dads."

While 74 percent of male control mice killed their offspring, none of the PRKO mice committed infanticide. Moreover, the PRKO mice were more nurturing, frequently touching their pups and returning them to their nests.

In another experiment, the researchers administered a drug that blocked progesterone receptors in normal mice. These mice, they found, behaved much like the knock-out mice, becoming more nurturing and less aggressive toward their offspring.

Interestingly, both the progesterone knock-out mice and the mice receiv-

Levine says the progesterone knockout mice "behaved differently.... These animals are terrific dads."

ing the progesterone-blocking drug continued to show typical levels of aggression toward other adult male mice. Thus, the aggression-enhancing effects of progesterone appear limited to behavior toward infant mice. While Levine notes that further research is needed to determine if these results are applicable to other species including humans, he says, "At least in the case of mice, this appears to be an important neurochemical switch that can increase paternal behavior and decrease aggressive behavior toward infants."

The researchers note that while females have higher levels of progesterone than males, studies show that "female responses to progesterone are both qualitatively and quantitatively different from those observed in males." In females, high progesterone appears to inhibit responsiveness toward infants and to increase aggression toward adult male intruders, but not to increase violence against infants.

"Progesterone receptors mediate male aggression toward infants," Johanna S. Schneider, Marielle K. Stone, Katherine E. Wynne-Edwards, Teresa H. Horton, John Lydon, Bert O'Malley, and Jon E. Levine, *Proceedings of the National Academy of Sciences USA*, Vol. 100, No. 5, March 4, 2003, 2951-2956. Address: jlevine@northwestern.edu.

-and-

"Progesterone regulates male mouse behavior," press release, Northwestern University, February 24, 2003.

Even brief Ecstasy use linked to long-term depression (continued from page 5)

George Ricaurte, Johns Hopkins School of Medicine, Baltimore, MD 21224.

-and-

"'Ecstasy' and serotonin neurotoxicity," U. McCann, G. Ricaurte, and M. Molliver, Archives of General Psychiatry, Vol. 58, October 2001, 907-8. Address: Una D. McCann, Department of Psychiatry and Behavioral Science, Johns Hopkins University School of Medicine, 725 N. Wolfe Street, Baltimore, MD 21205.

—and—

"Human psychopharmacology of Ecstasy (MDMA): a review of 15 years of empirical research," A. C. Parrott, *Human Psychopharmacology*, Vol. 16, No. 8, December 2001, 557-77. Address: A. C. Parrott, Department of Psychology, University of East London, Romford Road, London, U.K. E15 4L2.

—and—

"Severe dopaminergic neurotoxicity in primates after a common recreational dose regimen of MDMA ('Ecstasy')," G. A. Ricaurte, J Yuan, G. Hatzidimitriou, B. J. Cord, and U. D. McCann, *Science*, Vol. 297, September 2002, 2260-3. Address: George Ricaurte, Johns Hopkins University School of Medicine, Baltimore, MD 21224.

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QUOTABLE "Mind and body, psychologists and neurologists now agree, aren't that different. The brain is just another organ, albeit more intricate than the rest. The thoughts and emotions that seem to color our reality are the result of complex electrochemical interactions within and between nerve cells. The disembodied voices of schizophrenia and the feelings of worthlessness and self-hatred that accompany depression, although they seem to be based on reality, are no more than distortions in brain electrochemistry. Researchers are learning how these distortions arise, how to lessen their severity and, in some cases, how to correct them."

Michael Lemonick, in the Time cover story, 1/20/03, on "Your Mind, Your Body"

"Francis Crick and I brought together chemistry and biology. The next century will bring together biology and psychology. In the past, I never wanted to learn psychology because I didn't think its proponents had a solid basis for what they claimed. Now we're going to begin to understand behavior from a genetic point of view."

James Watson, co-discoverer of the form of DNA, writing in Time, 2/17/03

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