CRIME Times

Linking Brain Dysfunction to Disordered/Criminal/Psychopathic Behavior

Volume 9, Number 4, 2003

Developing brains of teens, young adults increase vulnerability to substance abuse

Experts often blame substance abuse by teens and young adults on peer pressure and the stresses of moving from youth to adulthood. A new study, however, argues that young people's vulnerability to drug abuse stems in large part from biological rather than sociological factors.

R. Andrew Chambers and colleagues note that teens and young adults experiment with and become addicted to drugs and alcohol far more often than older adults, and that adult substance abuse generally begins in the teen or early adult years. In addition, they point out, early onset of substance abuse predicts greater severity. The researchers say scientific evidence shows that sociocultural factors cannot fully account for these patterns, which are seen across cultures and are true for both males and females.

The researchers believe that this enhanced vulnerability of adolescents and young adults to substance abuse stems from developmental changes in the brain circuitry underlying motivation, impulsivity, and addictive behavior. Reviewing more than 140 studies on adolescent brain development

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High androgen levels linked to chronic antisocial behavior

A new study adds to evidence linking high levels of androgens ("male" hormones, including testosterone) to chronic antisocial or disruptive behavior.

Noting that the link between elevated androgens and aggression has been documented in male offenders, Athanasios Maras and colleagues decided to see if the same association held true for "at-risk" teenaged boys and girls. (Females also produce androgens, although in much smaller amounts than males.)

> Maras et al. found that boys with persistent aggression and other forms of "externalizing" behavior had the highest levels of plasma androgens.

The researchers tested plasma levels of the two primary androgen metabolites, testosterone and 5-alpha-dihydrotestosterone (DHT), in 87 14-year-olds, 36 of whom were male and 51 of whom were female. Data on the children's levels of externalizing behavior were available from tests conducted when they were 8, 11, and 14 years of age.

Significantly higher levels of both testosterone and DHT were seen in males with high levels of externalizing behavior, Maras and colleagues report. "Moreover," they say, "boys with persistent externalizing behavior exhibited the highest levels of plasma androgens." There was no association between androgen levels and aggression in females.

The researchers conclude, "Due to the findings of higher androgen levels in boys with persistent externalizing behavior, a role of androgens in the development of disruptive or later antisocial disorders can be hypothesized."

The researchers' findings are consistent with an earlier study by Stephanie van Goozen and colleagues (see *Crime Times* Volume 4, Number 2, 1998, pages 6 and 7), who measured levels of the androgen DHEAS in 15 aggressive and antisocial boys diagnosed with conduct disorder, and in 25 controls. All of the subjects in this earlier study were between 8 and 12 years of age, a time during which androgen levels gradually increase. Van Goozen and colleagues found that the boys with conduct disorder had significantly higher levels of DHEAS, and that "DHEAS levels were significantly positively correlated with the intensity of aggression and delinquency as rated by both parents and teachers."

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"Association of testosterone and dihydrotestosterone with externalizing behavior in adolescent boys and girls," A. Maras, M. Laucht, D. Gerdes, C. Wilhelm, S. Lewicka, D. Haack, L. Malisova, and M. H. Schmidt, *Psychoneuroendocrinology*, Vol. 28, No. 7, October 2003, 932-40. Address: Athanasios Maras, Department of Child and Adolescent Psychiatry and Psychotherapy, Central Institute of Mental Health, P.O. Box 122120, 68072 Mannheim, Germany.

FEATURED TOPIC (PAGES 3-6): Depression and Bipolar Disorder

EDITORIAL: Deserving of Justice

Recently, a news station aired a debate on whether or not John Hinckley, the man who shot Ronald Reagan, "deserved" to be allowed to go on unsupervised home visits. The question reveals how little progress we've actually made over the past century in understanding mental illness.

There are many legitimate questions to ask about Hinckley, including: "Is it safe to allow him unsupervised leave?" and "If doctors say he is in remission, how sure are they?" or, "Even if they are sure, should we err on the side of caution?" But it is not legitimate to ask if he *deserves* a particular activity.

CT scans performed after Hinckley shot the President revealed marked atrophy of the cortex, a common finding in schizophrenia. Hinckley was in fact diagnosed as schizophrenic by his doctors, and he is currently taking the antipsychotic drug Risperdal, which appears to be controlling his symptoms. The development of his schizophrenia followed a classic pattern of deterioration, beginning in adolescence and progressing to delusions, withdrawal, bizarre thinking, and other clear signs of psychosis.

Thus, there is no real debate over the fact that when Hinckley committed his crime, *he was suffering from a brain dysfunction that made him mentally ill.* Not evil—ill. And there is a huge difference. Asking if he now "deserves" unsupervised leaves is as foolish as asking if a heart patient "deserves" to leave the hospital, rather than asking if it's safe to discharge him.

Mental illness is the only disease in which victims are routinely blamed and punished for symptoms over which they have little or no control—a phenomenon that reflects poorly on us as a society. U.S. District Court Judge William Wayne Justice recently noted, "If we reject the moral necessity to distinguish between those who willingly do evil and those who do dreadful acts on account of unbalanced minds, we will do injury to these people. But the ultimate injury is the one we will inflict on ourselves, and on the rule of law."

Although Hinckley's parents reached out for help before he committed his crime, he received almost no effective treatment. Both they and their son were victimized—as were the people Hinckley injured—by a society that failed to recognize and effectively treat his mental illness, and now blames him for its effects.

Similarly, we blame millions of people with undiagnosed ADHD, depression, bipolar disorder, and other mental disorders for their perceived "crimes"—laziness, aggression, defiance—without asking if we instead are to blame for failing to help these individuals before their problems escalate to the point where they ruin their lives or ours.

Perhaps the question we should ask ourselves is this: do we "deserve" to call ourselves an enlightened society, when we treat the victims of undiagnosed and untreated brain diseases as though they are to blame for their illness?

An explanation for "inexplicable" acts?

Anneliese Pontius has written extensively on the phenomenon of senseless crimes committed by previously nonviolent people who generally have no memory of committing their violent acts. Her theory is that these individuals suffer from seizures originating in the limbic system, a "primitive" part of the brain involved in emotion, memory, and survival instincts (see *Crime Times* Volume 2, Number 4, 1996, page 6).

In a new study, Pontius reports that, in many cases, these acts of violence appear to stem from chronic, intermittent stimulation of the vagus nerve occurring in vulnerable individuals. The vagus nerve runs from the brain to the gastrointestinal tract, and studies show that repeated stimulation of the nerve can provoke seizures. (Controlled stimulation, on the other hand, is now being used as a means of reducing seizures in epileptic patients.)

In her new study, Pontius evaluated six unselected, consecutively referred males referred to her after they had received felony convictions for out-of-character aggressive episodes. All of the subjects had histories of head injuries, three had histories of seizures, one had an EEG consistent with seizures, and two exhibited cortical atrophy.

Pontius discovered that five of the six men had histories of recurrent nasopharyngeal infections, and she hypothesizes that these infections caused intermittent mild stimulation of the vagus nerve. "Supportive evidence shows that experimental vagus stimulation has the most excitatory impact on hippocampus and amygdala," she says, "which are also the most susceptible to limbic

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Depression takes huge toll

In a given year, 13 to 14 million Americans—or 6.6 percent of the population—suffer from major depressive episodes, according to a recent report in the *Journal of the American Medical Association*. The study also found that 35 million Americans will suffer from major depression during at least one point in their lives, and that only one fifth of those who develop depression will receive adequate treatment.

The study, by Ronald Kessler and colleagues of Harvard, found that the majority of people affected by depression have severe symptoms, and that their illness takes an enormous toll on their social and professional lives. "These findings," the researchers say, "confirm that depression is an enormous societal problem both in terms of the number of people involved and in terms of clinical severity."

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"The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R)," R. C. Kessler, P. Berglund, O. Demler, R. Jin, D. Koretz, K. R. Merikangas, A. J. Rush, E. E. Walters, and P. S. Wang, *Journal of the American Medical Association*, Vol. 289, No. 23, June 18, 2003, 3095-3105. Address: Ronald C. Kessler, Department of Health Care Policy, Harvard Medical School, Boston, MA 02115, NCS@hcp.med.harvard.edu.

—and—

"Millions of Americans suffer from major depression," news release, Harvard Medical School, June 17, 2003.

Antidepressants may work by "growing" new neurons

Antidepressant drugs appear to accomplish their job by stimulating the growth of new neurons, according to a team of Columbia University researchers.

Research already links long-term stress, anxiety, and depression to atrophy or death of neurons in the hippocampus, a structure in the brain's limbic system. In addition, studies have shown that some antidepressants increase cell genesis in the hippocampus. However, researchers were not sure if this new cell growth was directly responsible for the drugs' behavioral effects.

To find out, Rene Hen and colleagues conducted several experiments. In the first, they treated mice with antidepressants, and found that while the drugs had no short-term behavioral effects, long-term exposure (for a period of four weeks or longer) caused the mice to be less anxious. In addition, the mice exposed to antidepressants for a long period showed a 60 percent increase in a marker of new cell generation in the hippocampus.

To determine if this new cell growth directly changed the mice's behavior, the researchers used x-rays to selectively destroy proliferating cells, reducing neurogenesis by 85 percent. The irradiated mice, they say, failed to respond to antidepressant treatment in the same way as other mice. For instance, normal mice that are chronically stressed exhibit poor grooming behavior, a symptom that improves when the mice are treated with the antidepressant fluoxetine. In the irradiated mice, no improvement in grooming behavior occurred in similarly stressed mice given the drug.

Hen et al. next created mice lacking a gene that codes for a key subtype of serotonin receptor. These mice, unlike regular mice, exhibited no new cell genesis in response to long-term treatment with fluoxetine (which enhances the effects of serotonin), and their behavior was not affected by the drug. However, they did show behavioral responses and new cell growth in response to treatment with tricyclic antidepressants, which affect a different neurotransmitter (norepinephrine).

These findings, Hen and colleagues say, could explain why "drugs like Prozac and Zoloft take a month before acting and people can spend several months trying different drugs before finding the one that works for them."

Sheline study: antidepressants help prevent hippocampal volume loss

In a separate study, Yvette Sheline and colleagues used magnetic resonance imaging (MRI) scans to measure the volume of the hippocampus in 38 women who had suffered an average of five episodes of major depression during their lives. Some of the women had been treated with antidepressants, while others had not. The researchers also interviewed the women to determine how long their episodes of depression had lasted, and how long they had taken antidepressants.

Sheline et al. found that while the depressed women had smaller hippocampal volumes than non-depressed women, the reduction was less marked in the subjects who had received antidepressant treatment. The researchers were able to predict the amount of hippocampal volume loss, based on the ratio of number of days of depression to the number of days on antidepressant treatment. They note that animal studies also

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Studies reveal that antidepressants "grow" new neurons

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show that antidepressants can protect against stress-induced reductions in hippocampal volume.

The researchers say it is not clear why the hipppocampus shrinks in people with depression, but the damage may result from high levels of chemicals such as cortisol (a stress-related hormone), or from damage to the connections between nerve cells.

Sheline and colleagues say that their findings about the neuroprotective effects of antidepressants show the importance of recognizing and treating depression early in the disease process. They also say their data indicate that treatment between episodes of depression could help protect the brain.

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"Requirement of hippocampal neurogenesis for the behavioral effects of antidepressants," L. Santarelli, M. Saxe, C. Gross, A. Surget, F. Battaglia, S. Dulawa, N. Weisstaub, J. Lee, R. Duman, O. Arancio, C. Belzung, and R. Hen, *Science*, Vol. 301, No. 5634, August 8, 2003, 805-9. Address: Rene Hen, Center for Neurobiology and Behavior, Columbia University, New York, NY 10032.

—and—

"Creation of new neurons critical to antidepressant action in mice," news release, National Institute of Mental Health, August 7, 2003.

—and—

"Untreated depression and hippocampal volume loss," Y. I. Sheline, M. H. Gado, and H. C. Kraemer, *American Journal of Psychiatry,* Vol. 160, No. 8, August 2003, 1516-8. Address: Yvette Sheline, Department of Psychiatry, Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, MO 63110, yvette@npg.wustl.edu.

—and—

"Antidepressant drugs may protect brain from damage due to depression," news release, Washington University School of Medicine, August 1, 2003.

Scans show progressive damage in untreated bipolar disorder

Left untreated, bipolar disorder often worsens over time, with manic and depressive symptoms occurring with increasing frequency. New research indicates that this poor prognosis stems from progressive brain damage caused by the disorder.

Raymond Deicken and colleagues compared 15 non-symptomatic males with familial bipolar disorder to 20 controls, using proton magnetic resonance spectroscopy. They found significantly lower concentrations of N-acetylaspartate (NAA) in the right hippocampus of the bipolar subjects, and those who had suffered from the disease the longest had the lowest levels of NAA. NAA is the second most abundant amino acid in brain tissue, and the researchers note, "Low NAA is an indication that the integrity of neurons and/or axons has been compromised in some way, either by damage, loss, or dysfunction." The decrease in NAA over time in bipolar subjects, they say, indicates that the disease causes progressive damage. Similar decreases in NAA are seen in Alzheimer's disease, multiple sclerosis, Parkinson's disease, and other neurodegenerative diseases.

Deicken et al. say their research also confirms that the hippocampus plays a key role in bipolar disorder. Reduced hippocampal size, they note, is also seen in patients with major depression (see related story on page 3).

The researchers say their findings offer insight as to why lithium is highly effective in treating bipolar disorder. Studies on humans show that lithium increases both the amount of NAA and the amount of gray matter in the brain.

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"Lower concentration of hippocampal N-acetylaspartate in familial bipolar I disorder," R. F. Deicken, M. P. Pegues, S. Anzalone, R. Feiwell, and B. Soher, *American Journal of Psychiatry*, Vol. 160, No. 5, May 2003, 873-82. Address: Raymond Deicken, Magnetic Resonance Unit and Psychiatry Service, 116-N, Veterans Affairs Medical Center-San Francisco, 4150 Clement Street, San Francisco, CA 94121, deicken@itsa.ucsf.edu.

—and—

"Study suggests bipolar disorder may cause progressive brain damage," news release, University of California at San Francisco, May 6, 2003.

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Diet matters: Reduced levels of folic acid, other nutrients may increase depression risk

Two new studies indicate that low folic acid levels play a strong role in depression, and other research suggests that nutritional deficiencies are common in depressed patients.

Tommi Tolmunen and colleagues assessed depressive symptoms in more than 2,600 men between the ages of 42 and 60, and then divided them into three groups according to their dietary intake of folic acid.

The researchers report, "Those in the lowest third of energy-adjusted folate intake had a higher risk of being depressed than those in the highest folate intake third," a finding that remained true after the researchers adjusted for a wide range of other lifestyle and socioeconomic factors. "These results," Tolmunen et al. say, "indicate that nutrition may have a role in the prevention of depression."

In a related study, M. S. Morris and colleagues examined the relationship between depression and folic acid levels in a multi-ethnic group of nearly 3,000 subjects between the ages of 15 and 39. After adjusting for other factors, the researchers found that "subjects who met criteria for a lifetime diagnosis of major depression had folate concentrations in serum and red blood cells that were lower than those of subjects who had never been depressed." Low folic acid levels were most common in subjects who had recently recovered from depressive episodes, leading the researchers to suggest that "folate supplementation may be indicated during the year following a depressive episode."

Deficiencies of other nutrients also are linked to the development or exacerbation of depression. Physician Rebecca Kirby recently conducted a chart review of 12 randomly selected patients with depression, and reports finding "overtly deficient plasma vitamin C levels in 25 percent." In addition, levels of riboflavin, niacin, and vitamin B6 were low in many depressed patients, and half had low levels of magnesium. Levels of zinc and chromium were reduced in one quarter of depressed patients, and 40 percent had low levels of the omega-3 fatty acid eicosapentaenoic acid (EPA). (See related articles in *Crime Times* Volume 5, Number 1, 1999, pages 1, 2, and 6.)

Kirby concludes that an out-of-balance biochemistry caused by nutritional deficiencies "can cause mood swings and depression to become so extreme that they interfere with normal activities."

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"Dietary folate and depressive symptoms are associated in middle-

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DEPRESSION AND BIPOLAR DISORDER: SOME STATISTICS

Depressive is more common, and is starting earlier in life, than in past generations.

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One study found that only 3.5 percent of newly diagnosed depressed patients received appropriate treatment.

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The mortality rate for untreated manic-depressive illness is higher than that for most types of heart disease. Fifteen to 25 percent of people with untreated or poorly treated bipolar illness attempt suicide.

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Major depressive disorder is the leading cause of disability in the United States.

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In 2000, Steven Pliszka and colleagues reported that 42 percent of juvenile offenders they studied suffered from depression, mania, or bipolar disorder. S. L. McElroy and colleagues reported in 1999 that 61 percent of male sex offenders they studied suffered from a mood disorder, with 36 percent having bipolar disorder. In 1990, J. J. Collins and colleagues studied 1,140 incarcerated male felons and found "evidence of a direct relationship between a lifetime diagnosis of dysthymia [minor depressive symptoms] and an arrest or incarceration history for robbery as well as with multiple incidents of fighting since age 18." They also found that recurrent depression was significantly associated with a history of incarceration for robbery, and that "depression symptoms (regardless of whether a disorder diagnosis was made) were associated with multiple incidents of fighting since age 18."

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Eighty percent of depressed individuals who do not receive treatment have poor outcomes. In addition to increased risk of suicide, they are at high risk for lost jobs, marital strife, and academic failure.

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Depressive illnesses cost the U.S. approximately $44 billion annually. Of this, $24 billion is lost on absenteeism and low productivity.

RESEARCH IN BRIEF: DEPRESSION TREATMENTS

Omega-3 fatty acids

More evidence of the beneficial effects of omega-3 fatty acids on depression was recently reported by researchers in Taiwan. K. P. Su and colleagues conducted an eight-week double-blind, placebo-controlled trial of omega-3 polyunsaturated fatty acids (PUFAs) on 28 patients with major depressive disorder. (Patients also continued their regular treatment regimens during the trial.) The researchers report, "Patients in the omega-3 PUFA group had a significantly decreased score on the 21-item Hamilton Rating Scale for Depression [compared to] those in the placebo group." (See related story in *Crime Times* Volume 5, Number 1, 1999, pages 1, 2, and 6.)

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"Omega-3 fatty acids in major depressive disorder. A preliminary double-blind, placebo-controlled trial," K. P. Su, S. Y. Huong, C. C. Chiu, and W. W. Shen, *European Neuropsychopharmacology*, Vol. 13, No. 4, August 2003, 267-71. Address: K. P. Su, Department of Psychiatry, China Medical College Hospital, No. 2, Yuh-Der Road, Taichung 404, Taiwan.

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Testosterone gel

Researchers measuring testosterone levels in 54 depressed men who failed to respond to antidepressant treatment discovered that 24 of the men had low or borderline testosterone. They recruited 22 of the men for a second study, in which 12 applied a testosterone gel every day for eight weeks, while 10 received a placebo. At the end of the study, the researchers report, all of the men receiving testosterone treatment reported feeling significantly better, when compared to those who did not receive active treatment. "These preliminary findings," the authors say, "suggest that testosterone gel may produce antidepressant effects in the large and probably underrecognized population of depressed men with low testosterone levels."

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"Testosterone gel supplementation for men with refractory depression: A randomized, placebo-controlled trial," H. G. Pope, G. H. Cohane, G. Kanayama, A. J. Siegel, and J. I. Hudson, *American Journal of Psychiatry*, Vol. 160, No. 1, January 2003, 105-11. Address not listed.

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St. John's wort

A recent study of children and teens with major depression, by R. L. Findling and colleagues, found that 25 of 33 showed a significant positive response to treatment with the herb St. John's wort. St. John's wort appears to work in a manner similar to Prozac and related drugs, by inhibiting the reuptake of serotonin from synapses.

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"An open-label pilot study of St. John's wort in juvenile depression," R. L. Findling, N. K. McNamara, M. A. O'Riordan, M. D. Reed, C. A. Demeter, L. A. Branicky, and J. L. Blumer, *Journal of the American Academy of Child and Adolescent Psychiatry*, Vol. 42, No. 8, August 2003, 908-14. Address: R. L. Findling, Department of Psychiatry, Case Western Reserve University/University Hospitals of Cleveland, Cleveland, OH.

OUR THANKS TO Frank Schmalleger, author of the textbook *Criminology Today* (3rd Edition), for including *Crime Times* in the book's list of criminology references (see http://wps.prenhall.com/chet_schmalleger_crimtoday_3/0,8087,906959-site_search_frame,00.html). We are pleased to be described as "one of the Web's best sources for reviews and information about research on biological causes of criminal, violent, and psychopathic behavior."

NEW INSTITUTE TO UNCOVER ROLES OF BRAIN GENES

Paul Allen, co-creator of Microsoft, is contributing 100 million dollars to a scientific project to map the genes that affect brain development and function. Says Mark Boguski, Director of the new Allen Institute for Brain Science, the Human Genome Project identified all of the genes in the human genome, but did not reveal the function of each. "It's like opening a box filled with parts to build two tables and there are 30,000 parts and no instructions," he says." The Institute plans to determine the function of each of the approximately 20,000 genes believed to be involved in the development and function of the brain.

Diet matters in depression

(continued from page 5)

aged Finnish men," T. Tolmunen, S. Voutilainen, J. Hintikka, T. Rissanen, A. Tanskanen, H. Viinamaki, G. A. Kaplan, and J. T. Salonen, *Journal of Nutrition*, Vol. 133, Number 10, October 2003, 3233-6. Address: Jukka T. Salonen, Inner Savo Health Centre, Suonenjoki, Finland.

—and—

"Depression and folate status in the U.S. population," M. S. Morris, M. Fava, P. F. Jacques, J. Selhub, and I. H. Rosenberg, *Psychotherapy and Psychosomatics*, Vol. 72, No. 2, March-April 2003, 80-7. Address: M. S. Morris, Jean Mayer USDA Human Nutrition Research Center on Aging, Tufts University, 711 Washington Street, Boston, MA 02111.

—and—

"Mood swings and depression: let's get to the bottom of it," Rebecca K. Kirby, *Health Hunter*, Vol. 17, No. 9, October 2003, page 1. Address: Rebecca Kirby, Center for the Improvement of Human Functioning International, 3100 North Hillside Avenue, Wichita, KS 67219.

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Are young brains more vulnerable to substance abuse?

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and related research, the researchers conclude that "particular sets of brain circuits involved in the development of addictions are the same ones that are rapidly undergoing change during adolescence. Normally, these processes cause adolescents to be more driven than children or adults to have new experiences. But these conditions also reflect a less mature neurological system of inhibition, which leads to impulsive actions and risky behaviors, including experimentation and abuse of addictive drugs."

> A major implication of their theory, Chambers and colleagues say, "is that substance use disorders constitute neurodevelopmental disorders."

Developmental changes that can increase vulnerability to substance abuse, the researchers say, include:

• Greater activity of the dopamine system, which promotes exploratory behavior, than the serotonin system, which helps to inhibit impulsive behavior.

• Changes in sex steroid levels. The researchers note that sex steroid receptors are highly expressed in the hippocampus, a brain region involved in the detection of and behavioral response to novel situations.

"A major implication of this model," Chambers and colleagues say, "is that substance use disorders constitute neurodevelopmental disorders." They suggest that while "psychiatrically compromised" teens are at the highest risk for substance abuse, all adolescents are vulnerable to some degree. "Here we have a phenomenon," say Chambers et al., "where a neurodevelopmental stage common to virtually everyone regardless of genetic make-up confers enhanced neurobiological vulnerability to addiction."

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"Developmental neurocircuitry of motivation in adolescence: a critical period of addiction vulnerability," R. Andrew Chambers, Jane R. Taylor, and Marc N. Potenza, *American Journal of Psychiatry*, Vol. 160, No. 6, June 2003, 1041-52. Address: R. Andrew Chambers, Connecticut Mental Health Center, 34 Park Street Third Floor, New Haven, CT 06508, robert.chambers@yale.edu.

—and—

"Adolescents are neurologically more vulnerable to addictions," press release, Yale University, June 18, 2003.

Pontius: an explanation for "inexplicable" acts?

(continued from page 2)

seizure kindling by intermittent subthreshold stimuli."

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"Aggression in temporal lobe epilepsy and limbic psychotic trigger reaction implicating vagus kindling of hippocampus/amygdala (in sinus abnormalities on MRIs)," Anneliese A. Pontius and Marjorie J. LeMay, *Aggression and Violent Behavior*, Vol. 8, No. 3, May-June 2003, 245-57. Address: Anneliese A. Pontius, Harvard Medical School, 25 Shattuck Street, Boston, MA 02115.

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Studies show cumulative effects of biological insults

Two recent studies illustrate the additive effects of biological insults on brain function.

In one study, Maryse Bouchard et al. compared manganese workers who drank heavily with those who drank lightly, and found that workers with the highest alcohol consumption and the highest blood levels of manganese, a toxic metal, displayed the highest scores for mood disorder. In addition, they found that "in the lower [blood] manganese category, those in the higher alcohol consumption group did not have higher scores than the others." The interactive effects of alcohol and high manganese were most significant for depression, anger, fatigue, and confusion.

In a separate study, Robert Kahn et al. found that hyperactivity and oppositional behavior were linked to a specific allele of the dopamine transporter (DAT) gene, but only in children whose mothers smoked during pregnancy. Neither the DAT allele nor prenatal smoke exposure alone was significantly associated with elevated behavior problems.

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"Blood manganese and alcohol consumption interact on mood states among manganese alloy production workers," M. Bouchard, D. Mergler, M. Baldwin, M. P. Sassine, R. Bowler, and B. MacGibbon, *Neurotoxicology*, Vol. 24, No. 4-5, August 2003, 641-7. Address: M. Bouchard, CINBIOSE, University of Quebec in Montreal, Montreal, Quebec, Canada.

—and—

"Role of dopamine transporter genotype and maternal prenatal smoking in childhood hyperactive-impulsive, inattentive, and oppositional behaviors," R. S. Kahn, J. Khoury, W. C. Nichols, and B. P. Lanphear, *Journal of Pediatrics*, Vol. 143, No. 1, July 2003, 104-10. Address: R. S. Kahn, Div. General and Community Pediatrics, Cincinnati Children's Hospital Medical Center, Cincinnati, OH 45229.

QUOTABLE

"Law enforcement and corrections officers will tell you that they are at ground zero of our country's mental health crisis, 24/7. Here are just a few of the grisly statistics:

"25% to 40% of mentally ill individuals become involved in the criminal justice system;

"In July 1999, the Department of Justice issued a Special Report announcing that at least 16% of [the population of] state jails and prisons, or 260,000 people, are individuals with severe mental illness. That is more than four times the number of people currently in state mental hospitals;

"The American Jail Association estimates that 600,000 to 700,000 bookings each year involve individuals with mental illness;

"On any given day, at least 284,000 schizophrenic and manic depressive individuals are incarcerated, and 547, 800 are on probation;

"By default, L.A. County Jail is now the largest mental institution in the United States, holding an estimated 3,300 mentally ill inmates on any given night.

"These statistics represent countless backwards steps that have been made in the name of progress. They remind me of what the governor of Virginia said when he expressed dismay that he was 'forced to authorize the confinement of persons with mental illnesses in the Williamsburg jail, against both his conscience and the law,' because of lack of appropriate services. That was in 1773."

U.S. Representative Ted Strickland, at a 2000 Congressional hearing on "The Impact of Mentally Ill Offenders on the Criminal Justice System."

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