CRIME Times

Linking Brain Dysfunction to Disordered/Criminal/Psychopathic Behavior

Volume 12, Number 1, 2006

FEATURE ISSUE: ADHD and Other Childhood Behavior, Learning, and Attention Problems
(see pages 1-5)

Researchers reverse learning problems in mice with gene disorder

A common cholesterol-lowering drug appears to reverse learning and attention deficits in mice with neurofibromatosis type 1 (NF1). Alcino Silva and colleagues, who announced the discovery, say their finding is "exciting from a clinical perspective," both because it will benefit NF1 patients and because the treatment may also help individuals with other forms of learning disabilities.

NF1 is a disorder in which tumors grow on nerves throughout the body. The condition affects approximately one million people worldwide, with about half of them exhibiting deficits in attention, motor coordination, and the ability to learn new information. Additional effects can include behavioral and emotional problems. The genetic disorder is caused by the overactivity of Ras, a protein that regulates cell growth and proliferation. Excess Ras activity also inhibits long-term potentiation, which is the ba-

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Gene variant linked to higher risk for early antisocial behavior

A specific gene variant greatly elevates the risk for antisocial behavior in at-risk children, according to a new study.

Anita Thapar and colleagues recruited 240 children diagnosed with attention deficit hyperactivity disorder (ADHD), and evaluated them for symptoms of conduct disorder. Thapar and colleagues then performed genetic testing to determine which variants of a particular gene—the catechol *O*-methyltransferase (COMT) gene—each child possessed. Because early-onset antisocial behavior is also linked to an adverse prenatal environment, the researchers obtained data on the children's birth weights as well.

In one variant of the COMT gene, methionine (met) is substituted for valine (val) in one section. Research indicates that individuals possessing two "val" variants of the gene perform more poorly on tasks measuring prefrontal cortical function than do individuals with two "met" variants or a "met/val" combination. Poor prefrontal cortical function is also associated with antisocial behavior.

"Given the links between prefrontal cortical deficits and antisocial behavior and between COMT and

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Fatty acids again shown to aid children with behavior problems

Researchers reported last year that supplementation with essential fatty acids can dramatically accelerate learning and reduce behavior problems in children with developmental coordination disorder (see *Crime Times* Volume 11, Number 3, 2005, page 1). A newer study indicates that these nutrients can benefit children with attention deficit hyperactivity disorder (ADHD) as well.

In the new study, Kalpana Joshi and colleagues used flax oil, which is high in the fatty acid alpha linolenic acid (ALA)—a precursor to the essential fatty acid docosahexaenoic acid (DHA). The researchers gave flax oil supplements to 30 children with ADHD along with supplemental vitamin C, which inhibits harmful fatty acid peroxidation. They report, "There was significant improvement in the symptoms of ADHD reflected by reduction in total hyperactivity scores of ADHD children derived from ADHD rating scales." The children's social functioning and learning improved, and they exhibited marked reductions in impulsivity, restlessness, inattention, self-control problems, and psychosomatic problems.

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"Supplementation with flax oil and vitamin C improves the outcome of attention deficit hyperactivity disorder (ADHD)," Kalpana Joshi, Sagar Lad, Mrudula Kale, Bhushan Patwardhan, Sahebrao P. Mahadik, Bindu Patni, Arti Chaudhary, Sheila Bhave, and Anand Pandit, *Prostaglandins, Leukotrienes and Essential Fatty Acids*, November 25, 2005 (epub ahead of print publication). Address: Kalpana Joshi, Interdisciplinary School of Health Sciences (ISHS), University of Pune, Ganeshkhind, Pune-411007, Maharashtra, India, kalpana@unipune.ernet.in.

Lovastatin: new hope for treating NF1, other causes of "irreversible" learning disabilities?

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sis for remembering learned information.

Silva, whose team first linked NF1-associated learning disabilities to overactive Ras, realized that the statin drugs routinely used to lower cholesterol could affect Ras levels as well. Statin drugs lower cholesterol by blocking the effects of certain fats—and because these same fats are needed by Ras, statin drugs lead to reduced Ras activity.

Silva and colleagues administered the statin drug lovastatin to mice bred with the NF1 mutation. These mice exhibit learning disabilities, attention problems, and coordination deficits similar to those seen in humans with NF1. The researchers then compared the mice to NF1 mice receiving a placebo, using three tests measuring attention, spatial learning, and coordination. They report that lovastatin treatment "reversed [the mice's] spatial learning and attention impairments," actually leading to better performance on two of the tests than that seen in normal mice.

"The Ras pathway is central to memory and learning," says Silva, "and I believe Ras is connected to either the problem or the solution in many other learning disabilities, directly or indirectly."

Because lovastatin is already approved for use in humans, three clinical trials are already underway to determine if the drug will have similar effects on children or adults with NF1. In addition, Silva and colleagues believe lovastatin may prove to be an effective treatment for other groups of learning disabled individuals. "The Ras pathway is central to memory and learning," says Silva, "and I believe Ras is connected to either the problem or the solution in many other learning disabilities, directly or indirectly."

Editor's note: A different research group announced recently that in fruit flies, a substance called MPEP appears to reverse behavioral problems associated with Fragile X syndrome, another common genetic cause of behavior and learning problems. Such findings show the tremendous potential that gene research holds for understanding and treating learning and behavior problems once thought to be untreatable.

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"The HMG-CoA reductase inhibitor lovastatin reverses the learning and attention deficits in a mouse model of neurofibromatosis type 1," W. Li, Y. Cui, S. A. Kushner, R. A. Brown, J. D. Jentsch, P. W. Frankland, T. D. Cannon, and A. J. Silva, *Current Biology*, Vol. 15, No. 21, November 8, 2005, 1961-7. Address: Alcino Silva, Department of Neurobiology, University of California at Los Angeles, Los Angeles, CA 90095.

—and—

"Recreating 'Flowers for Algernon' with a happy ending," news release, University of California Los Angeles, November 7, 2005.

—and—

"Common drug cures learning disability," Gaia Vince, *New Scientist*, November 7, 2005.

Combination of additives, food colorings toxic to brain cells

The common food additives MSG and aspartame can become highly toxic to brain cells when combined with certain artificial colorings, according to new research. The findings support claims that food additives and colorings are culprits in attention deficit hyperactivity disorder (ADHD) and other learning and behavioral disorders.

Karen Lau and her colleagues created two combinations of food additives—MSG combined with the food coloring "brilliant blue," and aspartame combined with the food coloring "quinoline yellow"—and studied the effects of the mixtures on nerve cells, using mouse neuroblastoma cells as a model. The researchers report that while each of the four substances in isolation could harm cells at high enough concentrations, the combinations exhibited a powerful synergistic effect, stunting nerve cell growth to a far greater degree than individual toxicities would suggest—up to four times as much for MSG/brilliant blue, and up to seven times as much for aspartame/quinoline yellow. The researchers note that the concentrations of additives and colorings found to be toxic in combination were "theoretically achievable in plasma by ingestion of a typical snack and drink."

Lau et al. found that their additive/coloring mixtures disrupted cell growth and signalling at concentrations theoretically consistent with amounts in a single snack and soda.

The effects of such toxic combinations of additives and colorings on brain development during early childhood, the researchers say, could be profound. "Cell proliferation, migration, differentiation and synapse formation progress in a tightly programmed and orderly fashion," they note. "Interference with any stage of this

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Iron: Too little, too much a risk for behavioral problems?

Iron deficiency is associated with attention deficit hyperactivity disorder (ADHD), with a study last year by Eric Konofal and colleagues revealing that one-third of children with ADHD but only 3% of non-ADHD children have extremely low iron levels (see *Crime Times* Volume 11, Number 1, 2005, page 4). A new study by the same researchers indicates that correcting iron deficiency can dramatically reduce ADHD symptoms—but another research group's findings suggest that too much iron during prenatal development can lead to behavior problems.

In the new study, Konofal and colleagues conducted a single-case experiment to see if restoring iron levels to normal could lead to a reversal of ADHD symptoms. Their subject, a three-year-old boy, exhibited marked impulsivity, inattention, hyperactivity, and sleep problems. Testing revealed low iron stores, although the boy was not anemic.

The researchers began supplementing the boy with 80 mg of ferrous sulfate per day, and after four months of treatment, his iron levels normalized. At that point, parents and teachers reported only mild behavioral improvement, although the boy's sleep patterns did improve markedly. However, after eight months of iron treatment, the boy's parents described him as "transformed," and teachers reported that he was far more organized and attentive, much better at relating to other children, and less forgetful and impulsive. On the Conners' Parent and Teacher Rating Scales for hyperactivity, his scores dropped significantly, from 30 and 32 respectively before treatment to 19 and 13 afterward.

Noting that a 1997 study (Sever et al.) reported only modest improvements in boys with ADHD receiving iron supplementation, Konofal and colleagues point out that none of the boys in the earlier study were actually iron-deficient and that treatment continued for only 30 days compared to eight months in the current study. "In animal models," Konofal et al. say, "it has been reported that iron supplementation in iron deficient rats restored blood hemoglobin levels faster than brain iron levels; improvement in learning deficits lags behind blood hemoglobin by at least two months."

While iron treatment appears to be remarkably beneficial for a number of ADHD children, a research group in Australia is reporting that non-iron-deficient mothers who take iron during pregnancy may be putting their children at risk for future behavior problems.

S. J. Zhou and colleagues initially set out to investigate the possibility that children whose mothers took iron supplements might have higher IQs or exhibit better behavior than other children. Their investigation was based on the fact that offspring of iron-deficient rats consistently show behavioral deficits.

However, when the researchers evaluated 300 children of non-iron-deficient mothers, half of whom took iron during pregnancy, they discovered that there were no IQ differences but that a higher percentage of children of mothers in the iron group exhibited hyperactivity and other behavior problems. The findings appear to suggest that iron supplementation during pregnancy is appropriate only for mothers who are iron-deficient.

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"Effectiveness of iron supplementation in a young child with attention-deficit/hyperactivity disorder," Eric Konofal, Samuele Cortese, Michel Lecendreux, Isabelle Arnulf, and Marie Christine Mouren, *Pediatrics*, Vol. 116, No. 5, November 2005, e732-4 (epub ahead of print publication). Address: Eric Konofal, Service de Psychopathologie de l' Enfant et de l' Adolescent, 48 Boulevard Serurier, 75019 Paris, France, eric.konofal@rdb.ap-hop-paris.fr.

—and—

"Behavioral effects of prenatal iron supplementation in children: long term follow up of a randomized controlled trial," S. J. Zhou, M. Makrides and R. A. Gibson, *Asia Pacific Journal of Clinical Nutrition*, Vol. 14 (Supplement), November 2005, S46. Address: Maria Makrides, Child Health Research Institute, 72 King William Road, North Adelaide, South Australia 5006, maria.makrides@cywhs.sa.gov.au.

—see also—

"Iron 'risk' warning for mothers-to-be," Louise Gray, *The Scotsman*, October 27, 2005.

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Excess manganese implicated as cause of impaired childhood intellectual functioning

The human body needs trace amounts of manganese, but excess levels are toxic to brain cells. Evidence suggests that elevated manganese levels can contribute to violent behavior (see *Crime Times* Volume 2, Number 2, 1996, page 3), and a new study indicates that high manganese intake can impair intellectual function in children.

Gail Wasserman and colleagues evaluated 142 ten-year-old children in Bangladesh who consumed well water containing varying amounts of manganese. The researchers controlled for levels of arsenic, because an earlier study linked arsenic in drinking water to impaired intellectual functioning.

Wasserman and colleagues asked the children to perform six intellectual tests (similarities, digit span, picture completion, coding, block design, and mazes) and used the results to calculate verbal, performance, and full-scale intelligence scores. The tests were adapted from a standard IQ test, in order to be relevant to the rural Bangladeshi children.

The researchers divided the children into four groups based on manganese exposure via drinking water. and found significant reductions in verbal, performance, and full-scale intelligence scores for children in the highest-intake group (manganese higher than 1,000 micrograms per liter), compared to those in the lowest-intake group (manganese lower than 200 micrograms per liter). Smaller decrements in performance and full-scale intelligence scores were detected in children in the intermediate-manganese groups.

The researchers say their findings are relevant to children in the United States, because about 6% of domestic wells contain manganese concentrations higher than 300 micrograms per liter, a level at which some impairment was detected in the Bangladeshi children. They also say, "It is interesting to note that although breast milk contains between 3 and 10 micrograms of manganese per liter, infant formulas have been reported to contain as much as 50 to 300 micrograms per liter. Our findings, coupled with the absence of reports of manganese deficiency in young children, led us to conclude that the possible consequences in children of excess exposure to manganese from water, diet, and gasoline additives deserve further attention."

Wasserman et al. say their findings are relevant to children in the United States, because about 6% of domestic wells contain manganese concentrations higher than 300 micrograms per liter, a level at which some impairment was detected in the Bangladeshi children.

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"Water manganese exposure and children's intellectual function in Araihazar, Bangladesh," Gail Wasserman, Xinhua Liu, Faruque Parvez, Habibul Ahsan, Diane Levy, Pam Factor-Litvak, Jennie Kline, Alexander van Geen, Vesna Slavkovich, Nancy J. Lolacono, Zhongqi Cheng, Yan Zheng, and Joseph Graziano, *Environmental Health Perspectives*, Volume 114, Number 1, January 2006, 124-9. Address: Gail A. Wasserman, New York State Psychiatric Institute, 1051 Riverside Drive, Unit 78, New York, NY 10032, wassermg@childpsych.columbia.edu.

Combination of additives, colorings toxic to neurons

(continued from page 2)

cascade of events may alter normal progression of subsequent stages and short-term disruptions may have long-term effects later in life."

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"Synergistic interactions between commonly used food additives in a developmental neurotoxicity test," Karen Lau, W. Graham McLean, Dominic P. Williams, and C. Vyvyan Howard, *Neurotoxicology*, December 13, 2005. Address: Karen Lau, Developmental Toxicopathology Unit, Department of Human Anatomy and Cell Biology, University of Liverpool, Sherrington Buildings, Liverpool L69 3GE, UK.

—and—

"Combining food additives may be harmful, say researchers," Felicity Lawrence, *The Guardian*, December 21, 2005.

Why Crime Times?

The more we learn about the brain dysfunction that underlies much delinquency and criminal behavior, the more successful we will be in truly rehabilitating offenders and preventing at-risk children from turning to lives of crime. The purpose of *Crime Times*, a free publication sponsored by the Wacker Foundation, is to foster this effort by reporting state-of-the-art worldwide research on biological causes and treatment of aberrant behavior. It is our hope that physicians, researchers, educators, law enforcement professionals, and parents can use the information in *Crime Times* to build a better, safer future for at-risk children and for the communities in which they live.

Heavy alcohol exposure before birth a risk for impaired "moral maturity," delinquency

Children exposed to large amounts of alcohol before birth exhibit a lower level of "moral maturity" than non-exposed peers, a recent study shows.

Amy Schonfeld and colleagues compared 27 children with heavy prenatal alcohol exposure to 29 children with no history of such exposure. The children ranged in age from 10 to 18, and were matched for age, gender, handedness, socioeconomic status, and ethnicity. The alcohol-exposed group included children both with and without Fetal Alcohol Syndrome (FAS), a specific pattern of alcohol-related physical and mental abnormalities.

Using a test called the Socio-moral Reflection Measure-Short Form, the researchers found that the alcohol-exposed children typically operated at Stage 2 (responses reflecting a concern with avoiding negative consequences or benefiting oneself), while non-exposed children operated at Stage 3 (concern for others and what is socially accepted). While the overall maturity deficit of the exposed children could be explained by their lower verbal IQs, the researchers found that specific deficits in moral reasoning about helping family and friends remained significant even after controlling for verbal IQ scores. Schonfeld and colleagues say, "This lends additional support to the idea that impaired socialization and interpersonal relationship skills represent a core deficit following prenatal alcohol exposure beyond the influence of depressed IQ scores."

Not surprisingly, alcohol-exposed children were more likely to be delinquents than non-exposed children, with impaired moral reasoning about affiliation, property, and law significantly predicting delinquency. Half of the alcohol-exposed children without FAS exhibited conduct disorder, while none of the children with FAS did—a finding consistent with previous research. The children's verbal IQ scores did not explain this difference.

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"Moral maturity and delinquency after prenatal alcohol exposure," Amy M. Schonfeld, Sarah N. Mattson, and Edward P. Riley, *Journal of Studies on Alcohol*, Vol. 66, No. 4, July 2005, 545-54. Address: Amy Schonfeld, University of California-Los Angeles Neuropsychiatric Institute and Hospital, Dept. of Child and Adolescent Psychiatry, 760 Westwood Plaza, Room 58-242, Los Angeles, CA 90024, aschonfeld@mednet.ucla.edu.

Genes help determine which kids thrive despite abuse

While abused children are at heightened risk for conduct disorder, not all children who suffer abuse develop behavior problems. A new study suggests that genes can be the key to why one maltreated child "goes bad" while another thrives.

Evaluating data from more than a thousand British twin pairs, Sara Jaffee and colleagues identified children who had definitely or possibly suffered abuse. The researchers used parent and teacher evaluations to determine which of the maltreated children showed symptoms of conduct disorder, such as persistent lying, bullying, violence, physical cruelty, and stealing. They then compared monozygotic (identical) twins, who share 100% of their genes, to dizygotic (fraternal) twins, who are no more alike genetically than non-twin siblings. This allowed the researchers to determine what role genes played in determining which children developed conduct disorder and which did not. A substantial role of genes would be indicated, they note, if the identical twins whose co-twins exhibited conduct disorder had a significantly elevated rate of CD, while identical twins whose co-twins had no symptoms of conduct disorder had a low rate, with fraternal twins exhibiting similar but weaker patterns.

In line with previous studies, Jaffee and colleagues detected a strong genetic influence on conduct disorder. Moreover, they report, "The experience of maltreatment was associated with an increase of 2% in the probability of a conduct disorder diagnosis among children at low genetic risk for conduct disorder but an increase of 24% among children at high genetic risk."

The researchers say these findings are consistent with their earlier research (see *Crime Times* Volume 8, Number 4, 2002, page 1), which found that 85% of severely abused subjects with a low-activity variant of a gene that affects activity of monoamine oxidase (MAOA) developed some form of antisocial behavior. In contrast, participants with the high-activity variant of the gene almost never exhibited aggressive or criminal behavior in adulthood, even if they had been severely abused as children.

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"Nature x nurture: genetic vulnerabilities interact with physical maltreatment to promote conduct problems," Sara R. Jaffee, Avshalom Caspi, Terrie B. Moffitt, Kenneth A. Dodge, Michael Rutter, Alan Taylor, and Lucy A. Tully, *Development and Psychopathology*, Vol. 17, 2005, 67-84. Address: Sara Jaffee, Department of Psychology, University of Pennsylvania, 3720 Walnut Street, Philadelphia, PA 19104, srjaffee@psych.upenn.edu.

—BOOK REVIEW—

HARDWIRED BEHAVIOR: What Neuroscience Reveals About Morality

By Laurence Tancredi
Cambridge University Press, 2005
Hardback $19.13

Laurence Tancredi is uniquely qualified to write about the neural roots of human morality. As a lawyer, Tancredi has consulted in many legal cases involving the effects of toxins on brain function and behavior, as well as criminal cases involving assault, rape, and homicide. In addition, he is a noted physician and Professor of Psychiatry at New York University School of Medicine, as well as the author of several books on law, ethics, and psychiatry.

In this new book, Tancredi poses such questions as: Are the brain and the mind separate? How does the physical brain work to develop moral decisions? What is the biology of mental illness? Are specific moral rules innate? What is the impact of hormones on psychosexual development? How important is free will? The answers to these questions, as Tancredi shows, are rapidly emerging as our understanding of the brain evolves—and those answers are challenging our most basic ideas about good, evil, and free will.

Hardwired Behavior is written to be easily understood by lay readers, but will also be of great interest to behavioral researchers and legal professionals. Each chapter of the book has between 19 and 75 references, providing additional resources.

We strongly recommend this book for anyone involved in the study of immoral behavior in such disparate areas as money, deception, sex, or criminal activity.

Quotes from *HARDWIRED BEHAVIOR* by Laurence Tancredi:

Our view of morality has already been altered by new understanding of brain biology, and at the rate that new discoveries are being made, that view will change even more in the future. With these changes will come the understanding that we can intervene at the most fundamental biological levels to affect moral development.

(R)ecent neuroimaging and genetic studies have revealed specific brain images that correlate with discrete gene dysfunction to produce a child who is very likely to become highly violent and antisocial as an adult.

Since the late 1980s, positron emission tomography (PET) studies have been conducted on violent and aggressive offenders. These have shown correlations between brain metabolism and the potential for violent behavior. PET studies of repetitively violent offenders revealed decreased cortical blood flow and hypometabolism in their nondominant frontal and temporal lobes, compared to control subjects. Some even showed involvement into the prefrontal region, which affects cognitive understanding.

(H)ow many of us would accept the idea that our personal choices in life are influenced, even determined, by brain biology? We resist this notion even if we've known older people, perhaps in our own families, who have suffered stroke or a serious disease such as Alzheimer's, and we've seen how such physical brain injuries can affect not only their ability to move but their ability to think rationally.

(N)euroscience is forcing us to rethink the extent of our personal control over our choices, and the implications of limits on personal control over our choices are nothing short of mind-boggling.

Imaging studies of true psychopaths, who lack empathic abilities, are demonstrating structural and functional abnormalities in some of these key areas of the brain, particularly the prefrontal cortex and the limbic system.

(E)ach of us holds our own position on the spectrum of being influenced by neurochemicals and brain changes from the prenatal phase of our development. We may not be able to control gender or sexual preferences, as these appear to be shaped prenatally and during the early years of development, but most of us can exert some control over our behavior. Nonetheless, the *degree* of that control is largely determined by biological forces.

Understanding how parts of the brain work to affect our thinking and behavior may eventually transform our formerly sacrosanct beliefs about personal identity and free will.

Our objective should be to use neuroscientific information—including diagnostic measures such as imaging technologies—to address rationally the responsibility of those who commit "bad" or criminal acts.

Wired to lie: Pathological liars exhibit structural differences in prefrontal white, gray matter

The brains of people who are pathological liars differ structurally from the brains of normal individuals, according to a magnetic resonance imaging (MRI) study.

Yaling Yang and colleagues obtained their study subjects from a group of 108 volunteers in a temporary employment pool, using a series of psychological tests and interviews to identify 12 people with histories of repeated lying. The researchers compared MRI scans of these individuals to scans of 21 normal controls. Because pathological lying is often associated with antisocial personality disorder, the study included a second control group of 16 people with a history of antisocial personality disorder but no history of pathological lying. The researchers also controlled for age, ethnicity, verbal-performance IQ discrepancy scores, full-scale IQ, psychopathy, or a history of conduct disorder.

Normal individuals show increased bilateral activation in the prefrontal cortex when they tell lies. In this study, the researchers report, "Liars showed a 22 to 26% increase in prefrontal white matter and a 36 to 42% reduction in prefrontal gray/white ratios compared with both antisocial controls and normal controls." Liars exhibited a 14.2% decrease in prefrontal gray matter compared to normal controls.

Study co-author Adrian Raine notes that these differences may increase the ability to lie easily, while impairing brain processes involved in moral restraint. He explains, "Lying takes a lot of effort. It's almost mind reading. You have to be able to understand the mindset of the other person. You also have to suppress your emotions or regulate them because you don't want to appear nervous…. Our argument is that the more networking there is in the prefrontal cortex, the more the person has an upper hand in lying. Their verbal skills are higher. They've almost got a natural advantage." The coexisting deficit in prefrontal gray matter seen in pathological liars, he says, may mean that "they are less likely to care about moral issues or are less likely to be able to process moral issues."

The researchers note that autistic individuals typically are not good at lying, and they say, "intriguingly, brain neurodevelopmental studies of autism show the converse pattern of gray/white ratios to that shown by the liar group." They note, too, that as the brains of normal children develop a greater volume of white matter, the children become more adept at lying. These converging lines of evidence indicate, the researchers say, that "the prefrontal cortex is centrally involved in the capacity to lie."

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"Prefrontal white matter in pathological liars," Yaling Yang, Adrian Raine, Todd Lencz, Susan Bihrle, Lori Lacasse and Patrick Colletti, *British Journal of Psychiatry*, Vol. 187, 2005, 320-5. Address: Yaling Yang, Department of Psychology, University of Southern California, Los Angeles, CA 90089-1061, yalingy@usc.edu.

—and—

"Liars' brains wired differently," news release, University of Southern California, August 29, 2005.

Gene variant linked to elevated risk of antisocial behavior in at-risk kids (continued from page 1)

prefrontal cortical functioning, we hypothesized that the [val/val] variant would be associated with antisocial behavior," the researchers say. "We specifically set out to examine the subtype of antisocial behavior purported to have the strongest neurobiological and heritable origins, that is, childhood-onset conduct disorder symptoms accompanied by ADHD."

As predicted, both the "val/val" genotype and low birth weight were independent risk factors for conduct disorder in the ADHD children. Moreover, the researchers found significant evidence for an interaction between val/val status and prenatal environment.

"These results are of considerable interest," Thapar and her colleagues say, "because they suggest not only that COMT genotype and birth weight influence antisocial behavior in this high-risk group but also that those with the val/val genotype are particularly susceptible to the effects of lower birth weight."

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"Catechol *O*-methyltransferase gene variant and birth weight predict early-onset antisocial behavior in children with attention-deficit/hyperactivity disorder," Anita Thapar, Kate Langley, Tom Fowler, Frances Rice, Darko Turic, Naureen Whittinger, John Aggleton, Marianne Van den Bree, Michael Owen, and Michael O'Donovan, *Archives of General Psychiatry*, Vol. 62, No. 11, November 2005, 1275-8. Address: Anita Thapar, Department of Psychological Medicine, Cardiff University, Heath Park, Cardiff, Wales, thapar@cardiff.ac.uk.

Crime Times is interested in hearing from readers conducting research pertaining to biological influences on criminality and psychopathology. Reprints of research papers are appreciated.

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QUOTABLE "[P]eople are not blank slates upon which culture-specific experiences inscribe our personalities; rather, we all come into the world with a preprogrammed set of innate mechanisms and personal proclivities. Using a diverse array of methods, including survey research, computer simulations, brain imaging, and population and molecular genetics, researchers have shown that personality traits are highly heritable, replicable across a wide range of cultures and even species, largely stable across the life span, and linked (albeit weakly) to specific genes, hormones, neurotransmitters, and brain activation patterns."

—Richard W. Robins, in Science, *October 7, 2005*

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