CRIME Times

Linking Brain Dysfunction to Disordered/Criminal/Psychopathic Behavior

Volume 11, Number 4, 2005

FEATURE ISSUE:
The Effects of Toxins on Behavior and Learning

Seafood during pregnancy: analyzing benefits and risks

Pregnant women should eat fish but avoid seafood high in mercury levels, according to a new study showing that maternal fish consumption during pregnancy is linked to better cognitive skills in infants, while high mercury levels are linked to poorer cognitive performance.

Emily Oken and colleagues studied the relationship of seafood, mercury, and infant cognition using subjects from Project Viva, a large-scale prospective study of pregnancy and infant development. The researchers acquired data about the dietary habits of 135 pregnant women and measured the women's mercury levels using hair samples. They then tested the women's infants at six months of age, using a visual recognition memory (VRM) test that is highly predictive of later IQ.

The researchers report, "For each additional weekly fish serving, offspring VRM score was 4.0 points higher. However, an increase of 1 ppm [part per million] in

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Mounting evidence links mercury, neurological impairment

Many children have elevated mercury levels because they live near mercury-emitting industrial plants, eat diets high in mercury-containing fish, or have been exposed to medications and vaccines containing mercury. Mounting evidence indicates that these children are at increased risk of brain impairment and associated learning and behavior problems.

At a 2005 Congressional policy meeting, Susan West Marmagas of Physicians for Social Responsibility summarized research findings about methylmercury (the form of mercury that contaminates fish), saying that "exposure to methylmercury in the womb can cause adverse developmental and cognitive effects in children, even at low doses that do not result in effects in the mother.... Recent epidemiologic studies have [also] found that children exposed to even low levels of mercury before birth experience subtle symptoms of neurologic damage. Specific effects include poor performance on neuro-behavioral tests, particularly on tests of attention, fine motor function, language, visual-spatial abilities (e.g. drawing) and memory."

In research published last year, Philippe Grandjean et al. followed up on children involved in a long-term study in the Faroe Islands. Many children in these islands, off the coast of Denmark, are exposed to high levels of methylmercury because their diet is high in meat from pilot whales. An earlier study of the children when they were 7 years old, reported in 1998, found that "increased prenatal mercury exposure was associated with deficits in several brain functions including attention, language, verbal memory, spatial function and motor speed."

In their 2004 follow-up, Grandjean and colleagues evaluated the same children at age 14. The researchers' evaluation included brainstem auditory evoked potential (BAEP) testing, which measures the brain's electrical response to a stimulus—in this case, a signal transmitted from the acoustic nerve through several "relays" to the brainstem. In children exposed to higher levels of prenatal mercury, the researchers found, this response lagged significantly compared to the response of children with lower mercury exposure. "This was true at both 7 and at 14 years," Grandjean says, "suggesting that this effect of mercury on the developing

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> Richard Deth et al., who found that very low doses of ethylmercury inhibit biochemical pathways involved in methylation, say, "During the first years of life, networks of neurons that represent the matrix for learning are being developed in the brain. Methylation and the development of neuronal cells to create these networks are critical during this time. If the process is interrupted, the ability to learn and pay attention would naturally be impaired."

Mercury: endangering children's brains? *(cont. from page 1)*

brain is irreversible." Delays in a different component of signal transmission also were detected and appear to be due to postnatal mercury exposure.

Grandjean commented, "It is noteworthy that these children at age 14 had an average exposure that was similar to the exposure limit used by the U.S. Environmental Protection Agency, and that 95 percent of them had exposures below the level which has previously been considered safe by the Food and Drug Administration. Yet, at these exposure levels, we saw a steady slope of increasing delays of the electrical signals, the higher the mercury exposure."

Grandjean concluded, "The importance of brain functions means even a small deficit, whether measured as a decrease in IQ points or otherwise, is likely to impact on an individual's quality of life, academic success and economic prospects in life. Even though the children we examined were all basically normal, we have documented detectable deficits that appear to be permanent."

The effects of prenatal methylmercury exposure are still a subject of controversy, because another large-scale study—this one of children in the Seychelles, where fish is a staple of the diet—showed no adverse effects related to elevated prenatal methylmercury levels. A third large-scale study, this one in New Zealand in 1998, was consistent with the Faroe study, finding subtle psychological and academic decrements in children exposed to elevated levels of mercury in utero. The differences between the findings of the Seychelles study and those of the Faroe and New Zealand studies may be due to different measurement techniques, psychological testing methods, or population groups, or to other factors.

Grandjean notes that his own work has been replicated in other areas, saying, "We have also seen adverse effects in studies of mercury-exposed children from Brazil and Madeira, where developmental exposure levels were determined from current hair-mercury concentrations." He also notes that a second group of Faroese children, recruited in 1994, exhibited "mercury-associated decreases in the neonatal Neurological Optimality Score and in postnatal growth." The findings of Grandjean and the New Zealand researchers are of particular importance in light of an analysis suggesting that as many as one in every six pregnant women may have elevated mercury levels (see *Crime Times* Volume 10, Number 2, 2004, page 7).

> "Given the concerns about mercury... it is critical that we err on the side of caution."
> —Congressman Dave Weldon, M.D. (R-FL)

While these studies focused on methylmercury, other research is investigating the effects of ethylmercury. Once considered to be safer than methylmercury, and widely used in vaccines and other medicinal products until recent years, ethylmercury is now being investigated as a possible factor in rising rates of autism and learning disabilities (see book review on page 5). While epidemiological studies have reached conflicting results, clinical studies are causing increasing concern. Among the findings:

—In a 2005 study, Thomas Burbacher and colleagues gave injected ethylmercury and oral methylmercury to newborn monkeys, and found that thimerosal was cleared from the monkeys' systems faster than methylmercury and that brain concentration of total mercury was three times lower in the monkeys exposed to ethylmercury. However, Burbacher et al. found that ethylmercury rapidly crossed the blood-brain barrier, where it was converted to inorganic mercury incapable of leaving the brain. The result: compared to methylmercury, ethylmercury exposure may result in twice as much mercury being trapped in the brain.

—Mady Hornig and colleagues report that low-level exposure to ethylmercury can cause behavioral changes resembling autism in mice vulnerable to autoimmune disorders. Hornig et al. exposed both normal mice and autoimmune disease-susceptible mice to thimerosal (a vaccine preservative that is 50 percent mercury) in doses calibrated to be comparable to those received by children in vaccines, and found that the susceptible mice showed increased brain size and abnormalities in brain structure, particularly in areas involved in emotion and cognition. They also exhibited abnormal responses to new surroundings, a limited behavioral repertoire, and reductions in exploratory behavior.

—Richard Deth and colleagues found that thimerosal inhibits biochemical pathways involved in methylation, a process crucial to "turning off" or "turning on" genes at the proper times. Deth et al. found that thimerosal disrupted methylation at doses 100 times lower than children receive in a single dose of a thimerosal-containing vaccine. (Children received multiple vaccines containing thimerosal through the late 1990s until the FDA recommended its discontinuation, and stockpiles of thimerosal-containing vaccines are still being used.)

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Smokers put children at risk for learning, behavior woes

The evidence that parental smoking harms children continues to grow, with new research showing a correlation between prenatal or childhood tobacco exposure and a range of behavioral and learning problems.

In one study, Tanya Button and colleagues compiled data from questionnaires sent to families of nearly 2,000 pairs of identical or fraternal twins. Because identical twins share twice as many genes as fraternal twins, twin studies help researchers differentiate between genetic and environmental influences. At the time of the study, all of the twins were between 5 and 18 years old. The researchers asked about symptoms of antisocial behavior or attention deficit hyperactivity disorder (ADHD), and about the number of cigarettes the mothers smoked while pregnant.

Button et al. report, "Maternal prenatal smoking contributed small but significant amounts to the variance of ADHD and of antisocial behavior." Both behaviors were significantly and independently associated with maternal prenatal smoking, with average scores for both ADHD and antisocial behavior increasing with the number of cigarettes the mothers smoked.

In a second study, Kimberly Yolton and colleagues measured levels of cotinine, a tobacco byproduct and an indicator of tobacco smoke exposure, in the blood of 4,399 children between 6 and 16 years of age. Only children whose cotinine tests indicated environmental exposure, rather than first-hand use of tobacco products, were included in the study.

Yolton et al. measured the children's cognitive and academic abilities using standardized IQ and achievement tests, and found a significant correlation between higher cotinine levels and declines in reading and reasoning ability—even when cotinine levels were extremely low. In fact, there was an average one-point decline in reading scores for each unit increase in cotinine at levels above 1 ng/ml, while there was a five-point drop for each unit increase in cotinine at levels below 1 ng/ml. The findings remained true when the researchers controlled for demographic and socioeconomic variables and the children's ferritin levels and blood lead concentration. Yolton et al. say their data, which reveal an association between ETS exposure and cognitive deficits even at extremely low levels of exposure, "support policy to further restrict children's exposure."

The new findings are consistent with more than a dozen earlier studies linking prenatal tobacco exposure to learning and behavior problems (see *Crime Times* Vol. 9, No.4; Vol. 8, No. 1; Vol. 7, No. 1; Vol. 6, No. 3; Vol. 5, No. 3; and Vol. 5, No. 2).

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"Relationship between antisocial behaviour, attention-deficit hyperactivity disorder and maternal prenatal smoking," T. M. M. Button, A. Thapar, and P. McGuffin, *British Journal of Psychiatry*, Vol. 187, 2005, 155-60. Address: Tanya M. M. Button, Institute for Behavioral Genetics, 447 UCB, Boulder, CO 80309-0447, tanya.button@colorado.edu.

—and—

"Exposure to environmental tobacco smoke and cognitive abilities among U.S. children and adolescents," K. Yolton, K. Dietrich, P. Auinger, B. P. Lanphear, and R. Hornung, *Environmental Health Perspectives*, Vol. 113, No. 1, January 2005, 98-103. Address: Kimberly Yolton, Cincinnati Children's Environmental Health Center, Cincinnati Children's Hospital Medical Center, Cincinnati, OH 45229-3039, kimberly.yolton@cchmc.org.

Seafood and pregnancy: a risk/benefit analysis

(continued from page 1)

mercury was associated with a decrement in VRM score of 7.5 points." VRM scores were highest in infants whose mothers ate fish more than twice a week but had mercury levels below 1.2 ppm, a finding that remained true when the researchers controlled for a wide range of sociological and biological variables.

"It may seem contradictory that, on the one hand, fish intake raises mercury levels and higher mercury levels lead to worse cognition but, on the other hand, higher fish consumption is associated with better cognition," Oken and colleagues say. "The most likely explanation is that the benefit is conferred by consuming fish types with the combination of relatively little mercury and high amounts of beneficial nutrients."

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"Maternal fish consumption, hair mercury, and infant cognition in a U.S. cohort," Emily Oken, Robert O. Wright, Ken P. Kleinman, David Bellinger, Chitra J. Amarasiriwardena, Howard Hu, Janet W. Rich-Edwards, and Matthew W. Gillman, *Environmental Health Perspectives*, Vol. 113, No. 10, October 2005. Address: Emily Oken, Department of Ambulatory Care and Prevention, Harvard Medical School and Harvard Pilgrim, 133 Brookline Avenue, Boston, MA 02215, emily_oken@harvardpilgrim.org.

"Safe" levels of seafood toxin cause permanent behavior, memory changes in offspring

Pregnant women concerned about the mercury content of fish now have another reason to be cautious about eating seafood: a new animal study shows that maternal exposure to even "safe" levels of a naturally-occurring toxin in seafood, domoic acid, may cause lasting behavioral changes in offspring.

Edward Levin and colleagues injected pregnant rats with varying doses of domoic acid and then measured their offspring's behavior during a variety of tests measuring movement, learning, and memory. They report that exposed rats showed greater initial activity in a maze test than control rats, followed by a more rapid decline in activity levels. While male rats normally outscore females on spatial discrimination learning tasks, this difference was diminished in exposed rats. Also, both male and female rats showed increased vulnerability to scopolamine, a drug that induces amnesia. The researchers say this suggests that the rats "had less functional reserve" to aid in performing tasks that call on memory.

Levin concludes, "Brief, low-dose domoic acid exposure in rats during gestational development results in subtle neurobehavioral impairments that persist into adolescence and adulthood. Furthermore, long-lasting effects on locomotor activity and cognitive function occurred at levels having no clinically evident consequences for the animals." This finding, he says, indicates that "we may need to re-evaluate monitoring of waters, shellfish and fish to make sure that the most sensitive parts of the human population are protected from toxic exposure to domoic acid."

Domoic acid is produced by algae called *Pseudo-nitzchia*, and the toxin accumulates in shellfish that eat the algae. Currently, officials close fisheries when domoic acid levels reach 20 parts per million in animal tissues, the level the FDA designated as unsafe when the toxin was first discovered in the 1990s. However, the rats in Levin's study were exposed to levels lower than the FDA threshold.

> Levin et al. found that even brief, low-level exposure to domoic acid in utero caused lifelong behavioral alterations in rats.

Earlier animal studies using far higher doses of domoic acid revealed that the toxin can cause extensive damage to the hippocampus, a brain region involved in learning and memory. Human adults exposed to high levels of the toxin can suffer "amnesic shellfish poisoning," resulting in a permanent loss of short-term memory.

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"Persisting behavioral consequences of prenatal domoic acid exposure in rats," E. D. Levin, K. Pizarro, W. G. Pang, J. Harrison, and J. S. Ramsdell, *Neurotoxicology and Teratology*, July 26, 2005 (epub ahead of print). Address: Edward Levin, Department of Psychiatry and Behavioral Sciences, Duke University Medical Center, Box 3412 DUMC, Durham, NC 27710.

—and—

"Prenatal exposure to marine toxin causes lasting damage," news release, Duke University Medical Center, September 6, 2005.

Switch to organic diet cuts children's pesticide burden

An encouraging report indicates that switching to an organic diet quickly reduces the levels of organophosphate pesticides in children's bodies. Organophosphates are strongly implicated as a culprit in learning and behavioral problems (see *Crime Times* Volume 4, Number 3, 1998; Volume 4, Number 4, 1998; Volume 6, Number 1, 2000; and Volume 9, No. 2, 2003).

Chensheng Lu and colleagues recruited 23 children between the ages of 3 and 11 for their study, asking parents to feed the children a typical diet for three days and then switch them to an organic diet for five days. After that period, the children returned to their regular diets. The researchers took daily urine samples from each child before and during the intervention to measure pesticide levels.

When children changed to the organic diet, the researchers say, a "dramatic and immediate" drop in pesticide levels occurred. Two common agricultural pesticides, chlorpyrifos and malathion, were detected in all urine samples when children ate a typical diet, but disappeared in most urine samples during the organic-food period.

It is notable, the researchers say, that pesticides were not used in any of the children's homes. Thus, they say, their results "[support] the conclusion made by the National Research Council's 1993 report that dietary intake of pesticides could represent the major source of exposure in infants and young children."

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"Organic diets significantly lower children's dietary exposure to organophosphorus pesticides," Chensheng Lu, Kathryn Toepel, Rene Irish, Richard A. Fenske, Dana B. Barr, and Robert Bravo, *Environmental Health Perspectives*, September 1, 2005 (epub). Address: Chensheng Lu, 1518 Clifton Road, NE, Room 226, Atlanta, GA 30322, clu2@sph.emory.edu.

—BOOK REVIEW—

Evidence of Harm: Mercury in Vaccines and the Autism Epidemic

By David Kirby
St. Martin's Press, 2005
Hardback $26.95; Paperback $14.95

Veteran *New York Times* science reporter David Kirby's new best-seller tackles a highly charged issue: whether or not the vaccine preservative thimerosal, which is 50 percent mercury, is a key culprit in rapidly rising rates of autism and related neurodevelopmental disorders.

While *Evidence of Harm* takes no sides in the controversy, readers will be disturbed by Kirby's unsparing portrayal of the failure of government agencies and drug companies to evaluate the potential health risks of thimerosal—even as the number of childhood vaccines containing the substance soared. (Many children were exposed through vaccines to more than 100 times the amount of mercury the Environmental Protection Agency's safety threshold allowed.) Moreover, both medical professionals and parents will come away with a sense that the government "watchdog" agencies responsible for protecting children's health are too often focused on protecting the bottom line of pharmaceutical companies instead.

Kirby concludes that his research uncovered substantial evidence implicating thimerosal as a cause of the current autism epidemic, but that "evidence of harm is not proof of harm." One form such proof could take, he says, would be a drop in autism rates now that thimerosal is being phased out of vaccines—a trend that is now being seen in California, where the Department of Developmental Disabilities is reporting significant declines in new cases of autism after a steep and consistent rise for more than a decade.

Kirby's book is an important read for parents and professionals alike. With rates of ADHD, depression, and learning disabilities rising rapidly along with rates of autism, *Evidence of Harm* should be a wake-up call for America to demand more extensive and honest research—and more accountability from public and private agencies—when it comes to the the exposure of our children to toxins that could alter their brains and behavior for a lifetime.

Quotes from *Evidence of Harm* by David Kirby:

Mercury is a recognized neurotoxin that can destroy cells in key centers of the brain and nervous system. It is especially hazardous to fetuses and small infants,whose vital organs are still developing. Mercury is known to halt cell division and migration within the forming brain, and has been shown to bind to DNA, interrupting chromosomal reproduction and blocking several essential proteins.

Sensitivity to mercury ranged widely among individuals. In fetuses and developing infants, there was a ten thousandfold increase in sensitivity as compared to adults. What's more, boys were four times more likely to be mercury sensitive than girls—the same ratio found in cases of autism. It was also roughly the same ratio for ADD, tics, speech delay, and most of the other neurological developmental disorders associated with increased thimerosal exposure by the CDC itself.

The Mad Hatter in *Alice in Wonderland* was believed to be modeled on a syndrome resulting from occupational exposure to mercury vapor used in millinery, called "Mad Hatter's disease." The affliction struck a certain percentage of hatmakers in centuries past. People with Mad Hatter's disease suffered from depression, sluggishness, acute anxiety, and irrational fears. They grew nervous and timid. They blushed readily, were uncomfortable in social situations, and sought to avoid people. "Mad Hatters" were easily upset, had trouble with movement and coordination, and were prone to agitation, irritability, and aggression.

(T)he thimerosal debate has compelled the scientific community, however reluctantly, to consider an environmental component to [autism], rather than looking for a purely genetic explanation. Autism, by most accounts, is epidemic. And there is no such thing as a genetic epidemic.

For attention deficit disorder, [researchers] found a statistically significant, dose-dependent response for exposure at six months of age. The increased relative risk for each microgram of ethylmercury was 1.006—or 0.6 percent. Therefore, a child who received 62.5 micrograms by six months of age was 30 percent more likely (RR 1:30) to develop ADD than a child who received 12.5 micrograms.

If thimerosal is one day proven to be a contributing factor to autism, and if U.S.-made vaccines containing the preservative are now being supplied to infants the world over, the scope of this potential tragedy becomes almost unthinkable.

Even minor elevations in lead levels reduce children's IQ

In recent decades the federal government has gradually reduced the threshold for acceptable blood lead levels from 60 micrograms to 10 micrograms per deciliter. However, a new study concludes that even lead levels below the 10-microgram threshold can have adverse effects on children's intelligence.

Bruce Lanphear and colleagues pooled data from seven large-scale studies in four different countries, involving more than 1,300 children, and analyzed the effects of increasing levels of lead on full-scale IQ. After adjusting for a variety of socioeconomic variables, the researchers found reductions in IQ as lead levels rose, both in children with levels considered toxic and in children with levels considered safe.

In fact, the researchers say, in accord with previous research (see *Crime Times* Volume 7, Number 3, 2001, page 2), the declines they saw in IQs as lead level rose were steeper in children with lower lead levels. Overall, the researchers found that IQ decreased 6.9 points as lead levels rose from 2.4 (the lowest level in their sampling) to 30 micrograms per deciliter. Breaking their numbers down into dose-level increments, the researchers discovered that there was a 3.9-point drop as levels rose from 2.4 to 10 micrograms; an additional 1.9-point drop as lead levels rose from 10 to 20 micrograms; and a 1.1-point drop as lead levels rose from 20 to 30 micrograms.

The researchers also found that current blood lead levels, or average lifetime estimates of lead exposure, were better predictors of lead-linked IQ deficits than were early childhood blood lead levels. "The stronger effects of concurrent and lifetime measures of lead exposure may be due to chronicity of exposure," they say.

The researchers conclude, "Although blood lead concentrations less than 10 micrograms per deciliter in children are often considered 'normal,' contemporary blood lead levels in children are considerably higher than those found in preindustrial humans. Moreover, existing data indicate that there is no evidence of a threshold for the adverse consequences of lead exposure." They note that these adverse effects include learning problems and delinquency. "Collectively," they say, "these data provide sufficient evidence to eliminate childhood lead exposure by banning all nonessential uses of lead and further reducing the allowable levels of lead in air emissions, house dust, soil, water, and consumer products."

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"Low-level environmental lead exposure and children's intellectual function: an international pooled analysis," Bruce P. Lanphear et al., *Environmental Health Perspectives*, Vol. 113, No. 7, July 2005 (online). Address: Bruce P. Lanphear, Cincinnati Children's Hospital Medical Center, 3333 Burnet Avenue, Mail Location 7035, Cincinnati, OH 45229-3039, bruce.lanphear@cchmc.org.

Mercury implicated in children's learning, behavior problems

(continued from page 2)

Deth comments, "During the first years of life, networks of neurons that represent the matrix for learning are being developed in the brain. Methylation and the development of neuronal cells to create these networks are critical during this time. If the process is interrupted, the ability to learn and pay attention would naturally be impaired." Thus, he suggests, thimerosal exposure could contribute to rising rates of ADHD, as well as to autism.

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Testimony of Susan West Marmagas, Director, Environment and Health Program, Physicians for Social Responsibility, April 19, 2005, at a Senate Democratic Policy Committee Hearing.

—and—

"Delayed brainstem auditory evoked potential latencies in 14-year-old children exposed to methylmercury," K. Murata, P. Weihe, E. Budtz-Jorgensen, P. J. Jorgensen, and P. Grandjean, *Journal of Pediatrics*, Vol. 144, No. 2, 2004, 177-83. Address: Philippe Grandjean, Dept. of Environmental Health, Harvard School of Public Health, Landmark Center, 3-110, 401 Park Drive, Boston, MA 02215, pgrand@hsph.harvard.edu.

—and—

Philippe Grandjean, testimony at the Mercury MACT Rule Hearing, Maine State House, Augusta, Maine, March 1, 2004.

—and—

"Comparison of blood and brain mercury levels in infant monkeys exposed to methylmercury or vaccines containing thimerosal," Thomas Burbacher, Danny Shen, Noelle Liberato, Kimberly Grant, Elsa Cernichiari, and Thomas Clarkson, *Environmental Health Perspectives*, April 21, 2005 (epub). Address: Thomas Burbacher, Department of Environmental and Occupational Health Sciences, Box 357234, University of Washington, Seattle, WA 98195.

—and—

"Neurotoxic effects of postnatal thimerosal are mouse strain dependent," M. Hornig, D. Chian, and W. I. Lipkin, *Molecular Psychiatry*, June 8, 2004 (epub). Address: Mady Hornig, Jerome L. and Dawn Greene Infectious Disease Laboratory, Department of Epidemiology, Mailman School of Public Health, Columbia University, 722 W. 168th St., New York, NY 10032.

—and—

"Thimerosal, found in childhood vaccines, can increase the risk of autism-like damage in mice," news release, *Molecular Psychiatry*, June 8, 2004.

—and—

"Activation of methionine synthase by insulin-like growth factor-1 and dopamine: a target for neurodevelopmental toxins and thimerosal," M. Waly, H. Olteanu, R. Banerjee, S. W. Choi, J. B. Mason, B. S. Parker, S. Sukumar, S. Shim, A. Sharma, J. M. Benzecry, V. A. Power-Charnitsky, and R. C. Deth, *Molecular Psychiatry*, January 27, 2004 (epub). Address: Richard C. Deth, Department of Pharmaceutical Sciences, Northeastern University, Boston, MA 02115.

—and—

"Study suggests vaccine, autism link," Salynn Boyles, WebMD, February 5, 2004.

Hyperbaric oxygen therapy: new hope for children damaged by fetal alcohol exposure?

Children whose brains are damaged by fetal alcohol exposure suffer from global intellectual impairments, learning disabilities, memory problems, attention deficits, poor problem-solving skills, and social and behavioral difficulties. Doctors typically consider these problems to be untreatable, but a startling new study suggests that a simple, safe intervention can dramatically reduce symptoms of FASD (Fetal Alcohol Syndrome Disorders)—even years after the damage occurs.

In a single-case study, Kenneth Stoller treated a 15-year-old boy with Fetal Alcohol Syndrome (FAS, the most severe form of FASD) using hyperbaric oxygen, a treatment commonly used for diabetic wounds, diving-related "bends," and brain injuries. The boy underwent 40 treatments, followed seven months later by an additional 33 treatments, undergoing 60 minutes of low-pressure oxygen therapy during each treatment. Before and after intervention, Stoller administered a specialized neuropsychological test battery. He reports that:

—After the first 40 treatments, the boy showed improvement in all six categories on the test battery.

—The boy maintained his gains in verbal memory and continued to exhibit reductions in impulsive behavior at a six-month follow-up after treatment.

—After 33 additional treatments, "[the subject's] verbal memory was 95 percent (pretreatment 55 percent), visual memory was 57 percent (pretreatment 38 percent), reaction time was 0.64 second (pretreatment 1.03 second), visual motor speed score was 20.1 (pretreatment 18.6 [higher score on this test is better]) and all previously reported symptoms resolved."

Stoller says his findings add to growing evidence that "it is time to revise the old concept that brain injury is a condition for which there is no treatment other than supportive measures." He notes that deterioration can continue for years following a brain injury and that, conversely, scientists now know that stem cells in the adult brain can cause neural regeneration—a process, he notes, that is oxygen-dependent.

Stoller says his findings add to growing evidence that "it is time to revise the old concept that brain injury is a condition for which there is no treatment other than supportive measures."

The retinal damage sometimes associated with hyperbaric oxygen therapy should not be an issue in treating alcohol-damaged individuals, he says, because this damage appears to be triggered initially by hypoxia, which is not relevant to FASD.

Stoller concludes, "Low-pressure hyperbaric oxygen therapy is a therapy with an extremely low risk profile and relatively low cost, with potential benefits that seem to be significant and measurable for a condition considered incurable."

Stoller notes that the treatment could help vast numbers of children, because "the prevalence of Fetal Alcohol Syndrome and alcohol-related birth defects combined is at least 10 cases per 1,000 births, or 1 percent of all births." Earlier research (see *Crime Times* Volume 1, Number 1; Volume 3, Number 1; Volume 6, Number 1; Volume 6, Number 4; and Volume 10, Number 4) reveals the high societal cost of fetal alcohol exposure, which greatly increases the risk of delinquency (Stoller's own subject had already been charged with a crime), criminality, aberrant behavior, and academic, social, and vocational failure. FASD also is the leading non-hereditary cause of mental retardation and may be the leading preventable cause of learning disabilities.

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"Quantification of neurocognitive changes before, during, and after hyperbaric oxygen therapy in a case of fetal alcohol syndrome," Kenneth P. Stoller, *Pediatrics*, Vol. 116, No. 4, October 2005, e586-91. Address: Kenneth P. Stoller, 404 Brunn School Road #D, Santa Fe, NM 87505, hbotnm@netzero.net.

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.

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QUOTABLE

"THE FINDING that an environmental toxin can permanently reprogram a heritable trait also may alter our concept of evolutionary biology. Traditional evolutionary theory maintains that the environment is primarily a backdrop on which selection takes place, and that differences between individuals arise from random mutations in the DNA. The work by Skinner and his group raises the possibility that environmental factors may play a much larger role in evolution than has been realized before."

—From a June 2, 2005 news release from Washington State University describing research by Michael Skinner and colleagues, whose recent paper in the journal Science *described how exposing pregnant rats to environmental toxins altered the male offspring's fertility and sperm counts—as well as those of males in every generation therafter. The cause appears to be "epigenetic inheritance," in which chemical modifications to genes—including modifications induced by environmental toxins—can permanently alter the way the genes work.*

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