The Effects of Long-term Ritalin (Methylphenidate) Use

Krystle A. Cole

Abstract

Ritalin, or methylphenidate, is often used to treat attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD). Some of the many long-term effects of Ritalin use are reduced cerebral blood flow, increased energy consumption in many areas of the brain, permanent loss of brain tissue, life-long increased sensitivity to cocaine, and life-long increased rates of depression and anxiety.

Introduction

Ritalin, or methylphenidate, is commonly prescribed as treatment for attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD). It is estimated that 750,000 to 1 million school children are receiving 20 million prescriptions for stimulant medications and the figure is still growing (IMS Health 2002).

Many of the long-term effects of Ritalin use stay with the patient for life. This is primarily because of the fact that Ritalin is most often used during childhood and adolescence while the brain is still developing.

Constricts Blood Flow to the Brain

Investigators from the Brookhaven National Laboratory used PET scans to study the effects of Ritalin on overall cerebral blood flow. They measured the effect of clinical doses of Ritalin on blood flow in normal volunteers. They found that Ritalin decreased the overall flow of blood into the brain. The loss was large: 23-30% in all areas of the brain, including the higher brain centers in the frontal lobes, as well as in the basal ganglia deeper in the brain. The changes were a sufficiently dramatic to be grossly

apparent in the before and after photos. The reduction in blood flow is most likely caused by construction of the blood vessels related to the drug's impact on dopamine (Wang et al. 1994).

Increases the Brain's Energy Consumption

The brain uses glucose to meet its energy needs. The affects of Ritalin occur throughout many parts of the brain and especially those affected by dopaminergic nerves. Scientists at the National Institute of Mental Health studied the effects of Ritalin in the brains of conscious rats. They found that there were significant dose dependent alterations in metabolic activity in numerous areas of the brain. Energy consumption was increased in areas of the brain that are central to both motor activity and mental function like the frontal cortex, mediodorsal thalamus, nucleus accumbens, and substantia negra (Porrino et al. 1987).

Loss of Brain Tissue

A study at Ohio State University found that over 50 percent of the 24 young adults being studied had atrophy, or loss, of brain tissue. They were all treated with stimulants since childhood for hyperactivity. The researchers concluded that cortical atrophy may be a long term effect of taking stimulant medications (Nasrallah 1986).

Increased Sensitivity to Cocaine

A study at the Chicago Medical School examined how low doses of Ritalin affect dopamine cells in the brains of adolescent rats. Dopamine is a brain chemical that has been implicated in natural rewards, such as food and sex, as well as in drug abuse and addiction. The study showed that the rats experienced brain cell changes that subsequently made them more sensitive to the rewarding effects of cocaine (NIH 2003).

Another thirty year study at the University of California followed around 400 kids with ADHD that were treated with stimulant drugs during childhood. When those kids

reached their mid to late 20s, researchers discovered they took up cigarette smoking earlier, smoked more heavily, and were more likely to abuse cocaine and other stimulants as adults (Lambert 1999).

This research also suggests a "sensitization hypothesis" based on animal studies showing that early exposure to amphetamine and methylphenidate predisposes rats to the reinforcing impact of cocaine. The same sensitization may occur in humans if exposure to stimulants such as methylphenidate predisposes children to the stimulating effects of tobacco, cocaine, and amphetamines (Lambert 1999).

Rates of Depression and Anxiety Increase

A study at the Harvard Medical School looked at how pre-adolescent exposure to methylphenidate affected certain behaviors in rats when they reached adulthood. They found that early exposure to twice-daily injections of methylphenidate increased behaviors that could indicate depression (NIH 2003).

Another study at the University of Texas Southwestern Medical Center assessed certain behaviors of adult rats that were given methylphenidate prior to adolescence. They found that compared to drug-naive rats, those chronically exposed to methylphenidate were less responsive to natural rewards, such as sugar and sex, and more sensitive to stressful situations. The animals that were exposed to methylphenidate also had increased anxiety-like behaviors and enhanced blood levels of stress hormones (NIH 2003).

Conclusion

Ritalin, or methylphenidate, is often used to treat attention deficit hyperactivity disorder (ADHD) and attention deficit disorder (ADD). Some of the many long-term effects of Ritalin use are reduced cerebral blood flow, increased energy consumption in many areas of the brain, permanent loss of brain tissue, increased sensitivity to cocaine, and life-long increased rates of depression and anxiety.

References

IMS Health. 2002. IMS Health Reports. http://www.imshealth.com

Lambert, N. 1999. Ritalin and its Cousins: Rx or Gateway Drugs? *The Regents of the University of California*. Vol. 27, No. 34.

Nasrallah, H., J. Loney, S. Olson, M. Mccalley-whitters, J. Kramer, C. Jacoby. 1986. Cortical atrophy in young adults with a history of hyperactivity in childhood. *Psychiatry Research*. 17: 241-246

NIH. 2003. http://www.nih.gov/news/pr/dec2003/nida-08.htm

Porrino, L.J., G. Lucignani. 1987. Different patterns of local brain energy metabolism associated with high and low doses of methylphenidate. *Biological Psychiatry.* 22**:** 126-138

Wang, G.J., N.D. Volkow, J.S. Fowler, R. Ferrieri, D.J. Schyler, D. Alexoff, N. Pappas, J. Lieberman, P. King, D. Warner, C. Wong, R.J. Hitzemann, A.P. Wolf. 1994. Methylphenidate decreases regional cerebral blood flow in normal human subjects. *Life Science*. 54: PL143−PL146.