The Biology of Addiction Risk Looks Like Addiction

Reports new study in Biological Psychiatry

Philadelphia, PA, July 1, 2014 – Research suggests that people at increased risk for developing addiction share many of the same neurobiological signatures of people who have already developed addiction. This similarity is to be expected, as individuals with family members who have struggled with addiction are over-represented in the population of addicted people.

However, a generation of animal research supports the hypothesis that the addiction process changes the brain in ways that converge with the distinctive neurobiology of the heritable risk for addiction. In other words, the more one uses addictive substances, the more one’s brain acquires the profile of someone who has inherited a risk for addiction.

One such change is a reduction in striatal dopamine release. Dopamine is a key brain chemical messenger involved in reward-related behaviors. Disturbances in dopamine signaling appear to contribute to reward processing that biases people to seek drug-like rewards and to develop drug-taking habits.

In the current issue of Biological Psychiatry, researchers at McGill University report that individuals at high risk for addiction show the same reduced dopamine response often observed in addicted individuals, identifying a new link between addiction risk and addiction in humans.

Dr. Marco Leyton and his colleagues recruited young adults, aged 18 to 25, who were classified into three groups: 1) a high-risk group of occasional stimulant users with an extensive family history of substance abuse; 2) a comparison group of occasional stimulant users with no family history; and 3) a second comparison group of individuals with no history of stimulant use and no known risk factors for addiction. Volunteers underwent a positron emission tomography (PET) scan involving the administration of amphetamine, which enabled the researchers to measure their dopamine response.

The authors found that the high-risk group of non-dependent young adults with extensive family histories of addiction displayed markedly reduced dopamine responses in comparison with both stimulant-naïve subjects and non-dependent users with no family history.

“This interesting new parallel between addiction risk and addiction may help to focus our attention on reward-related processes that contribute to the development of addiction, perhaps informing prevention strategies,” said Dr. John Krystal, Editor of Biological Psychiatry.

Leyton, a Professor at McGill University, said, “Young adults at risk of addictions have a strikingly disturbed brain dopamine reward system response when they are administered amphetamine. Past drug use seemed to aggravate the dopamine response also but this was not a sufficient explanation. Instead, the disturbance may be a heritable biological marker that could identify those at highest risk.”

This finding suggests that there are common brain mechanisms that promote the use of addictive substances in vulnerable people and in people who have long-standing habitual substance use.

Better understanding this biology may help to advance our understanding of how people develop addiction problems, as well as providing hints related to biological mechanisms that might be targeted for prevention and treatment.

Notes for editors
Full text of the article is available to credentialed journalists upon request; contact Rhiannon Bugno at +1 214 648 0880 or Biol.Psych@utsouthwestern.edu. Journalists wishing to interview the authors may contact Dr. Marco Leyton at +1 514 398 5804 or marco.leyton@mcgill.ca.

The authors’ affiliations, and disclosures of financial and conflicts of interests are available in the article.

John H. Krystal, M.D., is Chairman of the Department of Psychiatry at the Yale University School of Medicine, Chief of Psychiatry at Yale-New Haven Hospital, and a research psychiatrist at the VA Connecticut Healthcare System. His disclosures of financial and conflicts of interests are available here.

About Biological Psychiatry
Biological Psychiatry is the official journal of the Society of Biological Psychiatry, whose purpose is to promote excellence in scientific research and education in fields that investigate the nature, causes, mechanisms and treatments of disorders of thought, emotion, or behavior. In accord with this mission, this peer-reviewed, rapid-publication, international journal publishes both basic and clinical contributions from all disciplines and research areas relevant to the pathophysiology and treatment of major psychiatric disorders.

The journal publishes novel results of original research which represent an important new lead or significant impact on the field, particularly those addressing genetic and environmental risk factors, neural circuitry and neurochemistry, and important new therapeutic approaches. Reviews and commentaries that focus on topics of current research and interest are also encouraged.

Biological Psychiatry is one of the most selective and highly cited journals in the field of psychiatric neuroscience. It is ranked 4th out of 135 Psychiatry titles and 13th out of 251 Neurosciences titles in the Journal Citations Reports® published by Thomson Reuters. The 2012 Impact Factor score for Biological Psychiatry is 9.247.

About Elsevier
Elsevier is a world-leading provider of information solutions that enhance the performance of science, health, and technology professionals, empowering them to make better decisions, deliver better care, and sometimes make groundbreaking discoveries that advance the boundaries of knowledge and human progress. Elsevier provides web-based, digital solutions — among them ScienceDirect, Scopus, Elsevier Research Intelligence and ClinicalKey — and publishes nearly 2,200 journals, including The Lancet and Cell, and over 25,000 book titles, including a number of iconic reference works.

The company is part of Reed Elsevier Group PLC, a world-leading provider of professional information solutions in the Science, Medical, Legal and Risk and Business sectors, which is jointly owned by Reed Elsevier PLC and Reed Elsevier NV. The ticker symbols are REN (Euronext Amsterdam), REL (London Stock Exchange), RUK and ENL (New York Stock Exchange).

Media contact
Rhiannon Bugno
Editorial Office
+1 214 648 0880
Biol.Psych@utsouthwestern.edu