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PRESS RELEASE

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CAN CHILDHOOD STRESS MAKE ONE STRONGER?

Childhood stress, adversity, or trauma are acknowledged risk factors for detrimental impact on adult health. But research in Germany reveals that adverse early life experiences might actually help some individuals adapt, enabling them to cope better with adversity as adults.

Experiencing adversity in childhood can generate long-lasting brain changes — affecting learning and memory, or leading to various disorders. "Until now, human studies indicated that early life stress generally correlates with increased risk for psychiatric disorders such as depression," noted **Dr Mathias Schmidt** of the Max Planck Institute of Psychiatry in Munich. "But not everyone who experiences early adversity develops a mood disorder. Some individuals are more stress-resilient than others." So additional factors may need to be taken into account, he pointed out — including genetic risk, and adult living or work environments in relation to early life conditions.

Speaking today (7 July) at the at the FENS Forum of Neuroscience, Dr Schmidt described research in mice finding that genetic predisposition may determine whether stressful early experiences can be adaptive in some, rather than detrimental.

Scientists know that life experiences can modify how genes are expressed in individuals. "We wanted to learn how genetic and environmental components interact over time, and together shape risk of developing disorders," said Dr Schmidt. "We asked what long-term cascades are activated by early life stress in relation to adult stress that may result in differences between stress-resilient and susceptible individuals."

His team's research in mice examined 'mismatched' early and adult life environmental stress conditions, as well as 'matched' adversity during development and adulthood. "One of the most-known environmental causes of psychiatric disease is stress," said Dr Mathias Schmidt. "So we examined what long-term effect stress has on different individuals. Studying mice enabled us to see how this may work over a lifetime."

Each situation was tested with mice expressing specific genetic risk factors. Moderate early life stress was created by providing some mothers with less nesting materials, generating unpredictability affecting young mice. Adult stress was created by controlling social contact in various ways.

Behavioural screening and molecular examination indicated that mice with certain genetic factors adapted better to adult stress; moderate adversity during development resulted in increased stress resilience in adulthood. But those less genetically prone to adaptation were more affected by repeated adversity.

The results indicate that early stressful environments and experiences indeed shape adult emotional and cognitive health — but effects are influenced by genes. Individual outcomes depend on genetic predisposition, intensity and extent of childhood exposure, and adult environments.

"Together, our data call for a balanced view on the consequences of early life adversity," said Dr Schmidt. Genetically predisposed individuals facing early stress might have increased risk for later psychiatric disorders, he explained. But others genetically predisposed to be less affected might be better equipped to deal with similar adult challenges. "So a rough childhood may or may not make one more resilient to stress in adult life," he noted. "Within a range, there can be adaptive results."

Dr Schmidt hopes this research might eventually point towards enhanced therapies for adult humans already exhibiting disorders induced by early life stress; and better identifying people at higher risk for stress-related mood disorders, predicting more effective treatments.

"There is no magic bullet to take away early life stress," said Dr Schmidt. "But at least in mice, we are beginning to know how this works in the brain." His continuing research targets brain regions altered molecularly, structurally, or functionally by early stress, which express behaviour changes — and investigates adjusting and reversing some of these effects in the brain.

END

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Symposia S30: Ramping up resilience: from (epi) genetics, to optogenetics and imaging.

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NOTES TO EDITORS

The 9th FENS Forum of Neuroscience, the largest basic neuroscience meeting in Europe, organised by FENS and hosted by the The Società Italiana di Neuroscienze (SINS) (Italian Society for Neuroscience) will attract an estimated 5,500 international delegates. The Federation of European Neuroscience Societies (FENS), founded in 1998, aims to advance research and education in neuroscience, representing neuroscience research in the European Commission and other granting bodies. FENS represents 42 national and mono-disciplinary neuroscience societies with close to 23,000 member scientists from 32 European countries. http://fens2014.neurosciences.asso.fr/

Further Reading (Schmidt)

Evidence supporting the match/mismatch hypothesis of psychiatric disorders. S Santarelli, SL Lesuis, XD Wang, KV Wagner, J Hartmann, C Labermaier, SH Scharf, MB Müller, F Holsboer, MV Schmidt. *European Neuropsychopharmacology*. June 2014; Volume 24, Issue 6: 907–918. DOI: 10.1016/j.euroneuro.2014.02.002

Mismatch or cumulative stress: Toward an integrated hypothesis of programming effects. E Nederhof, MV Schmidt. *Physiology & Behavior*. July 2012; Volume 106, Issue 5: 691–700. DOI: 10.1016/j.physbeh.2011.12.008

Animal models for depression and the mismatch hypothesis of disease. MV Schmidt. *Psychoneuroendocrinology.* April 2011; Volume 36, Issue 3: 330-338. DOI: 10.1016/j.psyneuen.2010.07.001