Barbara Strupp

Web Bio

Information

Biography

Biographical Statement

Barbara Strupp received her Bachelor's degree in Ethology from Washington University in 1976, and her Ph.D. degree in Psychology in 1982 from Cornell University. She conducted postdoctoral research at the National Institutes of Health (Biological Psychiatry Branch, NIMH) from 1981-1983. She then returned to Cornell University in 1983, funded by a National Institutes of Health New Investigator Research Award. She currently serves as Professor in the Division of Nutritional Sciences, and Adjunct Professor in the Department of Psychology. Professor Strupp is a member of the Graduate Fields of Nutrition, Psychology, and Environmental Toxicology and the Program in Neuroscience. Her research group has been funded by three institutes at the NIH (NICHD, NIDA, and NIMH). These grants have supported research on numerous causes of human cognitive dysfunction (malnutrition, PKU, early lead exposure, prenatal cocaine exposure, Down syndrome, and Fragile X syndrome), as well as the development of novel techniques for detecting and delineating cognitive and affective dysfunction in rodent models.

Strupp's research primarily deals with causes of human cognitive dysfunction, studies that involve both children and rodent models. The goals of the animal studies are to determine the nature and underlying neural basis of the cognitive dysfunction, with implications for therapeutic intervention and for elucidating basic brain-cognition relationships. Current projects (described below) pertain to mouse models of Down syndrome and Fragile X syndrome, rodent and non-human primate models of childhood lead exposure, and a murine model of genetic and dietary alterations in folate status. Two human studies, one ongoing and one in the planning stages, pertain to the lasting cognitive effects of prenatal choline supplementation.

Department Website Summary

More choline for pregnant, nursing women could reduce Down syndrome dysfunction, guard against dementia
The right nutrient can boost baby's brain function

Professional

Current Professional Activities

Cornell University Graduate Field Membership: Psychology; Nutrition; Environmental Toxicology

Editorial Board, Neurotoxicology and Teratology

External Advisory Committee, NIH Program Project grant concerning the Cognitive and Neural Effects of Early Developmental Iron Deficiency; Center for Human Growth and Development, University of Michigan, B. Lozoff, PI, 2003-2013.

Neurobehavioral Teratology Society, Council Member (elected office)

NIH Special Emphasis Panel to review P30 applications in response to RFA-HD-10-022: Intellectual and Developmental Disabilities Research Centers (IDDRC) 2011; June 29-30, 2011 (invited)

Reviewer for following journals:

Genes, brain, and behavior
Neurotoxicology and Teratology
Neurotoxicology

Research

Current Research Activities

Ongoing projects in my lab:

1. Perinatal choline supplementation research: We have recently found that supplementing the maternal diet during pregnancy and lactation significantly improves attention and emotion regulation in a mouse model of Down syndrome. A more circumscribed improvement in attention was also seen in the wildtype (control) littermates. I am currently involved in a collaborative study with investigators at the University of Chicago and NYU to elucidate the neural mechanism(s) that underlies this lasting benefit. We hypothesize that this benefit may reflect protection of cholinergic basal forebrain neurons, which atrophy in these mice with the onset of Alzheimer-like neuropathology. Furthermore we hypothesize that this protection of cholinergic neurons is mediated by improved neurotrophin function. We are in the middle of this project, but thus far the results support these hypotheses. This project should have clinical implications for identifying the choline intake during pregnancy and lactation that is optimal for cognitive functioning throughout the lifespan. Parallel studies with human subjects are also ongoing with two DNS colleagues, (Drs. Caudill and Canfield), In addition, I recently submitted a proposal for a Hatch grant with Dr. Paul Soloway to investigate the epigenetic changes in the offspring of dams supplemented with additional choline during pregnancy and lactation.

2. Succimer chelation of lead-exposed primates: My lab is also collaborating with colleagues at the University of Wisconsin and UC-Santa Cruz on the analysis of

data from a non-human primate study designed to assess the efficacy of succimer chelation for reducing lead-induced neurobehavioral dysfunction. A prior study conducted in my lab several years ago provided the first evidence that succimer chelation could significantly ameliorate lead-induced cognitive dysfunction. This study also provided the first evidence that chelation therapy can produce lasting cognitive deficits if given to individuals without elevated lead levels. This latter finding was pivotal in halting an NIH clinical trial of the efficacy of succimer chelation in treating autistic children. It will be important to assess whether these same effects are seen in non-human primates.

3. The cognitive effects of altered folate status: My lab is also collaborating with Dr. Patrick Stover's lab to assess the effects of altered folate status on cognitive functioning and emotion regulation. Folate status was manipulated in this study by manipulating dietary folate intake, assessed in conjunction with a mutation in the Mthfd1 gene. The $Mthfd1^{+/+}$ mice on the deficient diet exhibited impulsive responding immediately following a change in task characteristics or on trials following an error, indicative of altered regulation of arousal or emotion. In contrast, $Mthfd1^{+/gt}$ mice (regardless of diet) exhibited attentional dysfunction and a blunted affective response to committing an error. Results from a smaller pilot study revealed that the $Mthfd1^{+/gt}$ mice also showed significantly decreased expression levels for genes encoding choline dehydrogenase and the alpha 7 nicotinic cholinergic receptor. The effects of the $Mthfd1^{+/gt}$ mutation on both gene expression and behavior were more pronounced in the mice maintained on the folate sufficient diet (relative to those on the deficient diet), suggesting a compensatory mechanism in the face of the combined genetic and dietary perturbation of folate metabolism. These data demonstrate that common alterations in folate metabolism can produce functionally distinct cognitive and affective changes, and highlight the importance of considering genotype when making dietary folate recommendations.

Extension

Education

Courses

Courses Taught

NS 7030 Graduate Seminar in Nutrition

NS 4010 Empirical Research in Nutrition

NS 4990 Honors Research in Nutrition

Psych 4700 Undergraduate Research in Psychology

Bio 2990 Undergraduate Research in Biology

Bio 4990 Undergraduate Research in Biology

Websites

Related Websites

Psychology Web Page

Program in Neuroscience

Graduate Field of Environmental Toxicology

Administration

Publications

Selected Publications

Stangle, DE., Strawderman M, Smith D., Kuypers M, & Strupp BJ. Repeated regimens of Succimer show different treatment efficacy in brain versus blood in a rodent model of childhood lead exposure, *Environmental Health Perspectives*, 112:302-308, 2004 (*online, Oct. 31, 2003).*

Gendle MH, White TL, Strawderman M, Mactutus CF, Booze RM, Levitsky DA, and Strupp BJ. Enduring Effects of Prenatal Cocaine Exposure on Selective Attention and Reactivity to Errors: Evidence from an Animal Model. *Behavioral Neuroscience*, 118(2): 290-297, 2004.

Driscoll LL; Carroll JC; Moon J-S; Crnic LS, Levitsky DA; Strupp BJ. Impaired Sustained attention and error-induced stereotypy in the aged Ts65Dn mouse, a mouse model of Down syndrome and Alzheimer disease. *Behavioral Neuroscience*, 2004, 118 (6): 1196–1205.

Strupp BJ & Beaudin S. Assessing the neurobehavioral effects of early toxicant exposure: A perspective from animal research. In: Bellinger D (ed.), Human Developmental Neurotoxicology, New York, NY: Taylor & Francis Group, 2006: 415-445.

Moon J, Beaudin AE, Crnic L, Levitsky, DA, Strupp BJ. Impairments in inhibitory control, arousal regulation and sustained attention in the *fmr1* mouse model of Fragile X syndrome. *Behavioral Neuroscience*, 2006 Dec;120(6):1367-79.

Grantham-McGregor S, Cheung YB, Cueto S, Glewwe P, Richter L, Strupp B; International Child Development Steering Group. Developmental potential in the first 5 years for children in developing countries. *Lancet*. 2007 Jan; 369(9555):60-70.

Stangle DE, Smith D, Beaudin SA, Strawderman MS, Levitsky DA, and Strupp BJ. Succimer chelation improves cognition and arousal regulation in lead-exposed

rats but produces lasting cognitive impairment in the absence of lead exposure. *Environ Health Perspect.* 2007 Feb;115(2):201-9. Epub 2006 Oct 30.

Beaudin SA, Stangle DE., Strawderman M, Levitsky, DA, and Strupp BJ. Succimer chelation normalizes emotion regulation in rats exposed to lead early in life: Evidence from an olfactory conditional discrimination task with periodic omission of an expected reward. *Neurotox. Teratol* 29: 188–202 (2007) [Online 12 November 2006].

McNaughton, C. H., Moon, J., Strawderman, M. S., Maclean K. N., Evans, J., Strupp, B. J. (2008). Evidence for social anxiety and impaired social cognition in a mouse model of Fragile X syndrome. *Behav. Neurosci, 2*008 Apr;122(2):293-300.

Moon J, Ota KT, Driscoll LL, Levitsky DA, Strupp BJ (2008). A mouse model of Fragile X syndrome exhibits heightened arousal and/or emotion following errors or reversal of contingencies. *Developmental Psychobiology,* 2008 Jul;50(5):473-85.

Bushnell PJ, Strupp BJ. Assessing Attention in Rodents. In: Methods of behavioral analysis in neuroscience (2nd ed.). Buccafusco, Jerry J. (Ed.); Boca Raton, FL, US: CRC Press, 2009. pp. 119-143.

Moon J., Chen M, Gandhy SU, Strawderman M, Levitsky DA, Maclean KN, and Strupp BJ. Perinatal choline supplementation improves cognitive functioning and emotion regulation in the Ts65Dn mouse model of Down syndrome. *Behavioral Neuroscience,* 2010, 124 (3):346–361.

Beaudin SA, Gendle MH, Strupp BJ. Lasting attentional and affective dysfunction produced by prenatal cocaine exposure in a rodent model: Gender effects. Chapter 4 in: Gender Differences in Effects of Prenatal Substance Exposure", Lisa Kestler and Michael Lewis (Eds), Washington, D.C., American Psychological Association, 2011.