

Weekly Colloquium Tuesday, 3/6/2018, 12:30pm, Billings Building – Rosedale Conference Room

"Timing, Training, & Tinctures – Reorganization & Recovery After Stroke"

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Publications:

S.R. Zeiler, R. Gubbard, E. Gibson, T. Zheng, K. Ng, R. O'Brien, J.W. Krakauer. Paradoxical Motor Recovery From a First Stroke After Induction of a Second Stroke: Reopening a Postischemic Sensitive Period. (2015). Sagepub.com/journalsPermissions.nav. DOI:10.1177/1545968315624783.

S.R. Zeiler, J.W. Krakauer. The Interaction between training and plasticity in the poststroke brain. Curr Opin Neurol (2013), 26:000-000. DOI:10.1097/WCO.00000000000025.

K.L. Ng, E.M. Gibson, R. Hubbard, J. Yang, B. Caffo, R.J. O'Brien, J.W. Krakauer, **S.R.** Zeiler. Fluoxetine Maintains a Stat of Heightened Responsiveness to Motor Training Early After Stroke in a Mouse Model. (2015) stroke.ahajournals.org/lookup/suppl/DOI:10.1161/STROKEAHA.115.010471/-/DCI.







Nice were trained to perform a skilled prehension task to an asymptolic level of performance (I1) after which they underwent photocoagulation-induced stroke in the CFA. After a 7 day post-stroke delay (12), the mice were then retrained for 19 days. A second photocoagulation-induced stroke was then induced in either ipsilesional walk orders (8). The mice were re-trained after only a one-day delay (ie. 48 hours later) and ascrificed at 13. Each group had n=6. (C) Prehension performance at time points 11, I2, and 13. A repeated-measures ANOVA showed a significant interaction between group and time points 11, I2 and 13 (p = 0.15). Asterisks indicate significant post-hoc differences compared using Sidak's multiple comparisons tet (* < 0.001; * < 0.0001). (D, E) Drawings of prehension task and training anoardus

Abstract:

Studies in humans and nonhuman animal models show that most recovery from impairment occurs in the first 1–3 months after stroke as a result of both spontaneous reorganization and increased responsiveness to enriched environments and training. Improvement from impairment is attributable to a short-lived sensitive period of postischemic plasticity defined by unique genetic, molecular, physiological, and structural events. Data suggests that there are hree important variables that determine the degree of motor recovery from impairment all else being equal: (i) the timing, intensity, and approach to training with respect to stroke onset, (ii) the unique post-ischemic plasticity milieu, and (iii) The extent of cortical reorganization. I will present data regarding both the biology of the brain's post-stroke sensitive period and the difficult question of what kind of interventions best exploit this period. I will describe limitations of current post-stroke rehabilitation methods and suggest novel nterventions, which incorporate robotics, video-gaming, and pharmacological interventions including SSRIs and Cerebrolysin. Of import, Cerebrolysin has allowed us for the first time to model spontaneous recovery in an animal model of motor stroke.

