FIRST POSTER SESSION PLASTICITY/PHYSIOLOGY

POSTER **ABSTRACTS**

CLINICAL-TRANSLATIONAL RESEARCH SYMPOSIUM

RIT1 is required for IGF1-mediated Neurogenesis

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genesis. Using a genetic strategy to express activated RIT1 in the treatment of a range of neurological disorders. dentate gyrus (DG), we observed robust stem cell proliferation and neurogenesis in the adult dentate gyrus. In vitro studies

The formation of new neurons (neurogenesis) persists in the using cultured hippocampal neuronal precursor cells (HNPCs) postnatal and adult mammalian hippocampus dues to the exist- demonstrate that IGF-1 promotes a RIT1-dependent increase in ence of neural stem cells (NSCs). As with other somatic stem Sox2 transcription factor activity, resulting in pro-neural gene cells, adult NSCs are found within specialized niches that are expression and increased neurogenesis. When compared to wild organized to facilitate stem cell self- renewal and differentia- -type NPCs, knockdown of Rit impairs IGF-1-dependent Akt sigtion, under the control of extracellular growth factor signaling. naling, and neuronal differentiation. Collectively, these data While circulating insulin-like growth factor I (IGF1) is known to provide new insight into mechanisms that enable IGF-1 signaling promote neurogenesis, its mechanisms of action remain incom- and in stem-cell maintenance, embryogenesis and developpletely characterized. Here, we identify the RIT1 GTPases as ment, and suggest that activation of RIT1 has broad therapeutic playing a central role in IGF-1R dependent hippocampal neuro- potential in the setting of traumatic brain injury and for the

An examination of corticomotor plasticity in individuals with and without chronic ankle instability

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cle using Transcranial Magnetic Stimulation(TMS) assessment in then normalized to Mmax. patients with chronic ankle instability(CAI), indicating a potential reorganization in the supraspinal sensorimotor system. Mapping of the motor cortex may further quantify this potential reorganization by identifying shifting and/or expansion of the area of the motor cortex associated with selected muscle.

for the FL muscle in patients with and without CAI.

Design: Single-blinded case control study.

Setting: Research laboratory. Patient or Other Participants: Sev-CAI enteen patients (23.5±3.7years,179.17±7.3cm,73.19±11.5kg) and 16 Healthycontrols(HC) (21.1±2.2years, 168.6±10.4cm, 66.5±10.2kg) volunteered.

Interventions: Peripheral nerve stimulation was applied over the superficial peroneal nerve to determine the average of three maximum muscle response (Mmax) of the FL. The average

Context: Previous literature has found decreased excitability of three motor evoked potentials(MEP) at Active Motor Threshwithin the corticospinal pathway of the fibularis longus(FL) mus- old(AMT) intensity was recorded for each scalp site and was

Main Outcome Measures: Corticomotor plasticity was determined through 1) size of the corticomotor map area and 2) volume of the corticomotor map. The map area is expressed as the mean number of sites from which normalized MEPs were evoked. The map volume was calculated as the sum of normal-Objective: Compare differences of the corticomotor map output ized MEP amplitude across scalp sites. Independent T-tests were used to assess group differences and Cohen's d effect sizes along with 95% confidence intervals(CI) were calculated.

> **Results:** CAI patients exhibited less map volume (P=0.018,CAI:8.3±3.3vs.HC:11.3±3.8,d=0.85,CI:0.12,1.54) and map area (P=0.046, CAI:12.8±6.0vs.HC:17.37±6.8, d=0.70,CI:0.02,1.38) compared to HC

> Conclusions: The smaller map area and volume suggest a more concentrated area of motor cortical cells associated with the FL muscle in patients with CAI. This may explain altered movement strategies that lead to ankle re-injury.



