



UNIVERSITÀ
DI TRENTO

Dipartimento di
Biologia Cellulare, Computazionale e Integrata - CIBIO

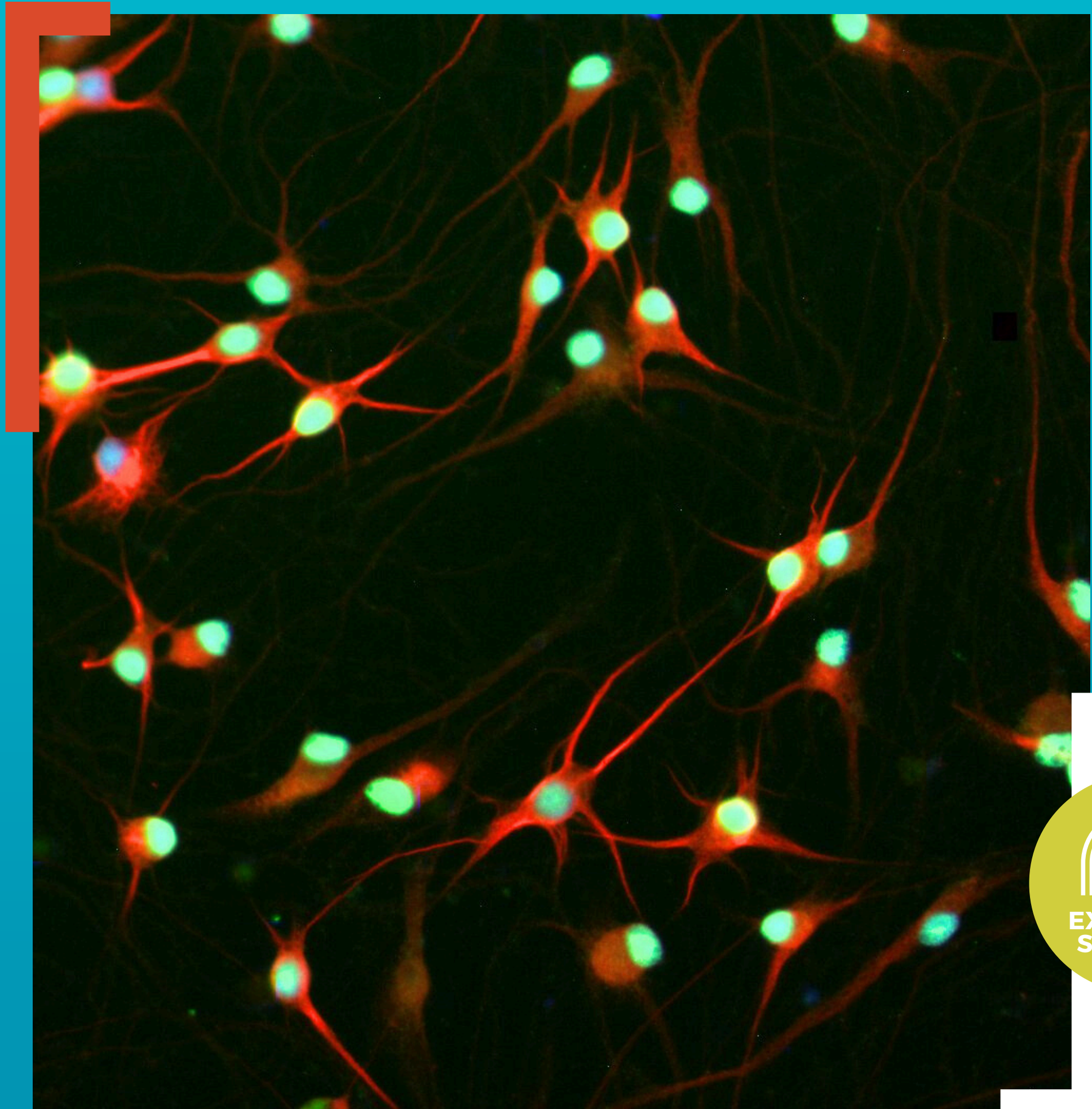
12 SEPTEMBER

11.30 A.M.

ROOM A106 - POVO 1

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CRTD, TECHNISCHE UNIVERSITÄT DRESDEN, GERMANY



- ● IDENTIFYING NOVEL STRATEGIES TO
- ● PROTECT AXONS AGAINST FUS-ALS
- ● PATHOGENESIS USING IPS CELLS

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease where motor neurons (MNs) degenerate in a “dying-back” pattern, with axons affected before somas. Juvenile ALS (<25 years) is often caused by FUS mutations. To investigate axonal mechanisms, we generated isogenic FUS-eGFP iPSCs, differentiated them into spinal MNs, and isolated axons using microfluidic chambers.

We found that mutant FUS axons are highly vulnerable, likely due to aberrant FUS accumulation in ribonucleoprotein (RNP) granules, impairing local translation. Preliminary data suggest that specific short RNAs can block FUS recruitment to RNPs, potentially alleviating ALS phenotypes.