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During development, neurons extend axons across great distances to form a neural network. However, the length of axons makes them vulnerable to injury and disease. Following injury, axons degenerate and must be regrown to restore function.

In the central nervous system, regeneration fails completely. By contrast, in the peripheral nervous system, neurons activate a regenerative response that is capable of achieving limited regrowth. In severe cases, however, this regeneration is insufficient for functional recovery. If the regenerative program could be induced or sustained pharmacologically, functional recovery would be more obtainable.

The DiAntonio laboratory has previously demonstrated that Dual Leucine Zipper Kinase (DLK), a stress-induced MAP triple kinase, is required for activation of the regenerative program following injury. However, the regulatory mechanisms governing DLK remain poorly understood.

Using an *in vitro* assay of DLK activity, we demonstrated that ROCK inhibitors (ROCKi) could block cytoskeletal stress-induced, DLK-dependent regenerative signaling. Because DLK signaling plays an important role in other aspects of the neuronal injury response, including axon degeneration and cell death, we examined the ability of ROCKi to influence these responses. Treatment with the inhibitors did not affect cell death or axon degeneration, suggesting that their capacity to regulate DLK activity is restricted to a regenerative context.

Ultimately, our work has identified novel pharmacological regulators of DLK, a kinase required for activation of the regenerative program, and interrogated their mechanism. An expanded understanding of the program's regulatory mechanisms will provide therapeutic targets to combat neurodegenerative disease and injury.