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Role of pyrroloquinoline quinone and resveratrol in survival and regeneration of cerebellar granular neurons

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Axonal damage and degeneration are key events following stroke, and injuries to brain and spinal cord. Under such critical conditions, degenerated axons do not have the ability to regenerate and their capacity to re-establish lost connections is limited, which can lead to long term disability. The inability of central nervous system (CNS) neurons to regenerate following axonal injury can be attributed to several factors including (i) loss of cells due to apoptosis, (ii) the extrinsic inhibitory environment, (iii) the limited intrinsic potential of neurons to regenerate. Experimental evidence has shown that fostering the intrinsic potential of damaged neurons to regenerate and sprout may be a promising therapeutic approach.

Pyrroloquinoline quinone (PQQ), a cofactor of several oxidoreductases, was previously reported for its neuroprotective effect and potential to promote peripheral nerve regeneration. Here, we investigated the ability of PQQ to induce neurite re-growth in a wound healing model on cultured CNS neurons. A scratch injury was applied to a culture of cerebral granular neurons (CGNs) and axonal re-growth into the scratch area was quantified. The neuroprotective effect of PQQ was examined utilizing the K+ deprivation-induced apoptosis model in CGNs. Resveratrol (RVT), a naturally occurring phytoalexin and an established compound promoting neuroprotection was also investigated alone and in combination with PQQ to establish possible synergistic effects and to manipulate some of the pathways targeted by PQQ and RVT that are known to affect the intrinsic potential of CNS neurons to re-grow following injury.

Our results showed that the capacity to induce neurite re-growth in the wound healing model was lower for PQQ than for RVT. PQQ alone and in combination with RVT showed a significant neuroprotective effect in the K+ deprivation-induced apoptosis model. The combination of both models allows the identification of novel promoters of neuroregeneration after injury.

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