Upon axonal cleavage, the distal end of the axon will undergo a degenerative process called Wallerian degeneration, which serves to clean the area of axoplasmic debris. This is triggered due to the activation of the enzyme *sterile α and TIR motif containing 1* (SARM1) [1]. This multidomain protein has an autoinhibitory N-terminal domain. Therefore, in a normal, healthy axon, it is in the "off" state because of nicotinamide mononucleotide adenylyl-transferase (NMNAT) and nicotinamide adenine dinucleotide (NAD). The transferase NMNAT is essential in the synthesis of NAD, which is an indispensable coenzyme found in every living cell. When the axoplasm is exposed to extracellular media, NMNAT commences to degrade, which in turn causes the precursor to NAD, nicotinamide mononucleotide (NMN) to accumulate. Degradation of NMNAT and accumulation of NMN stops SARM1 inhibition and starts the Wallerian degeneration process [2] [3]. This process begins several days after an injury in mammals [4]. Despite all the regenerative mechanisms the peripheral nervous system (PNS) has, one of the greatest issues in functional recovery is the reinnervation of the correct target tissue [5]. Therefore, a potential solution to this dilemma is to locally stop Wallerian degeneration and use the distal axon, which already innervates the correct tissue, as an anchor point. This is already done in small lesions, such as neuropraxia, with the generation of bands of Büngner, however a solution for larger lesions has yet to be developed.

Genetically modified induced pluripotent stem cells (iPSC) could provide a possible solution. The iPSC would need to function as a form of neural platelet. Fortunately, research regarding the *ex vivo* development of iPSC platelets is on-going. [6] The surface bound receptors of the artificial platelets could be genetically modified to respond to NAD instead of collagen. NAD is typically found intracellularly, therefore these platelets would respond only in the case of axonal cleavage which would release NAD into the extracellular media. NMN can be found extracellularly and would therefore not be a good candidate. A potential receptor could be found in *Arabidopsis thaliana* [7]. Once NAD binds to these receptors, this iPSC should be modified to release three substances. The first is NMNAT. Local release of this transferase would cause the cleaving of local NMN and the production of NAD. This would then delay the onset of Wallerian degeneration by inhibiting SARM1. The second substance is vascular endothelial growth factor A (VEGF-A). This growth factor stimulates the growth of blood vessels which would assist in providing oxygen to keep the distal axon alive and further inhibit SARM1 and degeneration. The third substance is glial cell line-derived neurotrophic factor (GDNF). Studies show that GDNF is a potent growth factor for axons [8]. The release of this substance would then give the proximal axon a direction in which to regenerate. This study should be tested in an *in vitro* environment with a cell culture of primate/human axons to establish proof of concept.

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