Severe Neuropathy and Erectile Dysfunction After Androgen Deprivation and cavernous Nerve Injury Are Improved with Testosterone Administration
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INTRODUCTION
After radical prostatectomy (RP), many men develop erectile dysfunction (ED) due to injury to the major pelvic ganglia (MPG) and cavernous nerve (CN). The incidence of ED increases when androgen deprivation therapy (ADT) is used to shrink the prostate before RP. Testosterone (T) is vital to maintenance of neuronal health and function. Without T, nerves are vulnerable to injury. Following ADT, recovery of some T is often slow or incomplete. Although T supplementation in survivors has been considered taboo, T has the potential to both repair nerves and improve erectile function in RP-induced nerve injury.

OBJECTIVES
To determine if ADT before CN injury increased the severity of both neuronal damage and ED, and assess if supplementary T restored neuronal health and erectile function.

METHODS
Male Sprague-Dawley rats (12 weeks, n=8/group)
12 wks 16 wks 18 wks
Control (CON) CAST
Castrated (CAST) CAST + BCNI (C+B)
Cavernosal Smooth Muscle Function - Myograph
Cavernous nerve (CN) was stimulated at 2V, 4V and 6V at 6Hz, 0.5ms duration, and 30s pulse width for 1 min with 4 min rest. Intracavernosal pressure (ICP) and mean arterial pressure (MAP) were recorded.

RESULTS
Testosterone increases peak ICP after CAST + BCNI
Testosterone normalizes penile contraction following CAST + BCNI

RESULTS
NO donor and NANC penile relaxation improves with testosterone supplementation

RESULTS
Nitricergic neurons are markedly reduced with CAST + BCNI and restored with testosterone

CONCLUSIONS
The combination of ADT and BCNI caused severe ED and markedly impaired neuronal health. Low T leaves post-injury nerves highly susceptible to increased apoptosis and Schwann cell activation. T supplementation restored erections and neuron health after the increased impact of nerve injury during ADT. This work highlights the ability of T to improve erections and pelvic neuron health.

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