



-Protective effects of camel whey protein against scrotal heat mediated damage and infertility in the mouse testis through YAP/Nrf2 and PPAR-gamma signaling pathways

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Abstract:

Elevation of scrotal temperature is one of the most important causes of impaired spermatogenesis and male infertility, but the exact mechanism remains controversial. The present study investigated the impact of camel whey protein (CWP) on the mechanisms of heat stress (HS)-mediated testicular damage in male mice. Exposure to HS was associated with significant increase in the testicular tissues' oxidative stress. Mechanistically, exposure to HS resulted in upregulation of P53 and Nrf2 expressions; downregulation of Bcl2 and PPAR- α expressions; and induction of testicular Leydig cell hyperplasia. Because Leydig cells produce testosterone up on stimulation with Luteinizing hormone (LH), HS mice also exhibited significant reduction in the serum testosterone levels followed by significant reduction in the percentages of progressively motile sperm and higher percentages of immotile sperm, when compared with those of control mice. Interestingly, treatment of HS mice with CWP significantly restored the levels of ROS and the activities of antioxidant enzymes in the testicular tissues nearly to those observed in control mice. Furthermore, CWP supplemented HS mice exhibited complete restoration of Bcl2, P53, Nrf2, and PPAR- α expressions; testicular Leydig cell distribution; significant higher levels of testosterone levels; and hence higher percentages of progressively motile sperm and lower percentages of immotile sperm as compared to HS mice. Our findings reveal the protective effects of CWP against testis injury and infertility induced by exposure to HS by rescuing functional Leydig cells. Additionally, the present study has shed light on the molecular mechanisms underlying improved testicular damage following CWP treatment. This article is protected by copyright. All rights reserved.

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