

# 19<sup>TH</sup> WORLD OBESITY CONGRESS

July 10-11, 2018 Bangkok, Thailand

## Recuperative effect of nesfatin-1 in the spermatogenesis and steroidogenesis in the diabetes mice

Ashutosh Ranjan and Amitabh Krishna  
Banaras Hindu University, India

**Introduction & Aim:** Recent investigation has reinforced and validated the nesfatin-1 as an adipokine principally involved in whole body energy homeostasis. It imparts several physiological roles including puberty onset, sleep, stress regulation, behavior response, reproduction, etc. This study was aimed to evaluate the effect of nesfatin-1 in the regulation of testicular physiology in the diabetic mice.

**Method:** The diabetic mice were treated intraperitoneally for 14 days (SD) with 1.25 nM/gbw nesfatin-1. The treatment produced significant changes in the spermatogenesis and steroidogenesis activity in the diabetic mice.

**Result:** Nesfatin-1 treated diabetic mice showed increased proliferation of germ cells as indicated by increased accumulation of spermatocytes and round spermatid in the seminiferous tubule. Nesfatin-1 treatment increases the testicular expression of Proliferating Cell Nuclear Antigen (PCNA) and B-Cell Lymphoma-2 (BCL-2) expression compared to diabetic control group mice, which further support the importance of nesfatin-1 in germ cell proliferation, their survival and spermatogenesis. The diabetic mice treated with nesfatin-1 showed significant increase in testosterone synthesis compared to diabetic control mice due to stimulatory effect of nesfatin-1 on testicular 3 beta HSD activity and increased expression of Steroidogenic Acute Regulatory protein (StAR) and Luteinizing Hormone (LH-receptor) proteins.

**Conclusion:** In addition, nesfatin-1 treatment also showed increased glucose transport by increasing the expression of glucose transporter (GLUT-8) and Insulin Receptor (IR) proteins in the testis. This study further explored the increased production of testosterone may be mediated via increased production of nitric oxide. Altogether, the study suggests the stimulatory role of nesfatin-1 in the regulation of testicular steroidogenesis and spermatogenesis, including testicular metabolism in diabetic mice.

ashutoshranjan.bhu@gmail.com

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