EXCLI Journal 2022;21:647-650 – ISSN 1611-2156
Received: February 03, 2022, accepted: March 16, 2022, published: March 24, 2022

Supplementary information to:

Letter to the editor:

HDAC11: A NOVEL INFLAMMATORY BIOMARKER IN HUNTINGTON'S DISEASE

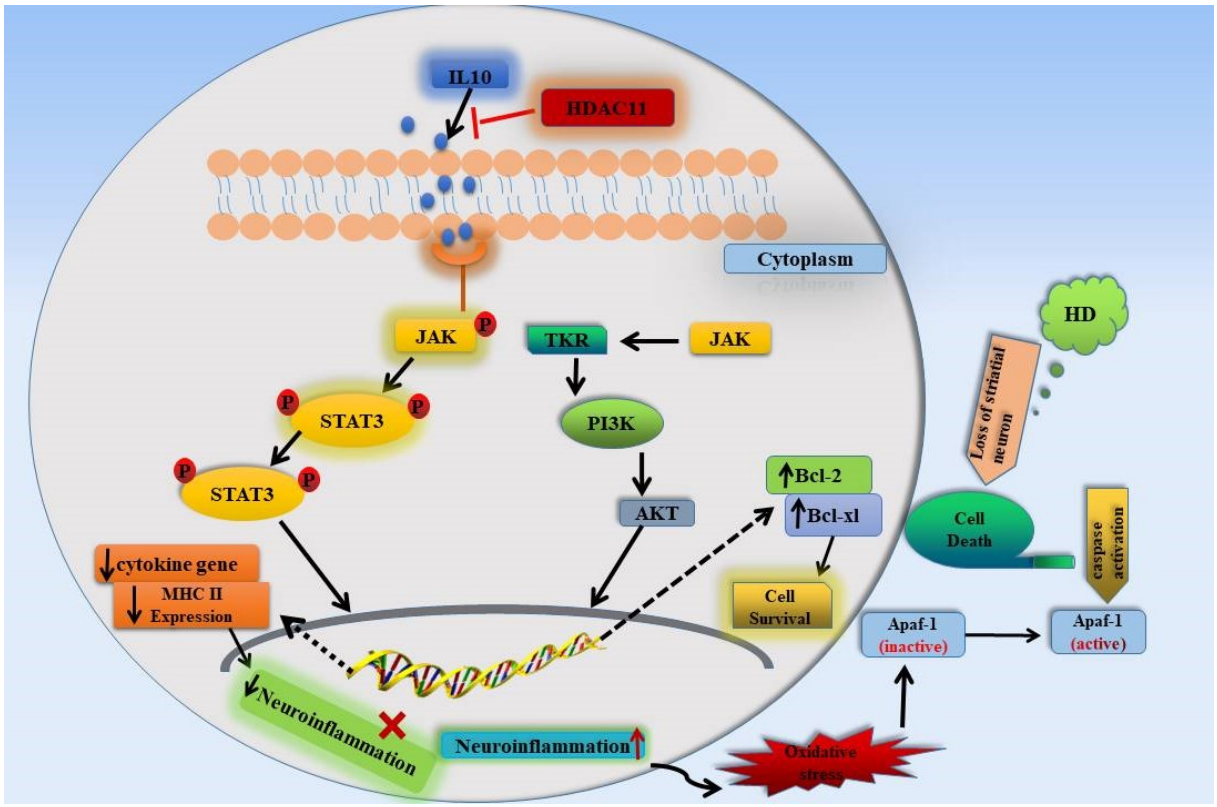
Vishal Kumar[a], Simranjit Kaur[a], Lakshay Kapil[a], Charan Singh[b], Arti Singh[a*]

[a] ISF College of Pharmacy, Department of Pharmacology, Moga-142001, Punjab, India
[b] ISF College of Pharmacy, Department of Pharmaceutics, Moga-142001, Punjab, India
[a+b] affiliated to IK Gujral Punjab Technical University, Jalandhar, Punjab, India

[*] **Corresponding author:** Dr. Arti Singh, ISF College of Pharmacy, Department of Pharmacology, Moga-142001, Punjab, India. E-mail: artiniper@gmail.com

https://dx.doi.org/10.17179/excli2022-4741

EXCLI Journal 2022;21:647-650 – ISSN 1611-2156
Received: February 03, 2022, accepted: March 16, 2022, published: March 24, 2022



Supplementary Figure 1: Mechanistic representation of HDAC11 mediated HD pathogenesis. IL-10 acts through the JAK-STAT pathway migrates to the cell nucleus and lowers cytokine levels, resulting in the prevention of inflammation. In addition, IL-10 inhibits apoptosis and acts through the PI3K/AKT pathway to increase production of Bcl-2 and Bcl-xl by decreasing caspase-3 expression and increasing cell survival. HDAC11 acts on IL-10 and inhibits its anti-inflammatory activity, leading to increased inflammation and ROS production, which can further lead to neuronal cell death via caspase signaling pathway activation.