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TITLE

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Regulated protein degradation by the human proteasome is essential for cellular homeostasis, and the proteasome is therefore widely recognized as an important target in several human diseases, including cancer, inflammatory and neuro-inflammatory disorders. To date, all clinically relevant proteasome-based therapeutics affect global proteasome-dependent proteolysis and function via a competitive mechanism involving their covalent attachment to the active sites within the 20S catalytic core. Consequently, the use of competitive proteasome inhibitors in clinical applications has been limited primarily to cancer treatment, in part due to toxic side effects associated their pan-proteomic perturbations.

In contrast to competitive inhibitors, non-competitive modulators bind to the target protein at a site other than the catalytic site and often display high target selectivity, as well as unique alterations in enzymatic specificity. The work presented here will focus on the orally available small molecule, TCH-013, which regulates proteasome activity via a non-competitive mechanism. Consequently, this new class of proteasome modulators, inhibit NF- κ B mediated cytokine production in vivo, and exhibit potent anti-arthritis and anti-cancer properties, with no apparent toxicity to the host. These results indicate the feasibility of non-competitive proteasome modulators as potential new class of therapeutics for the treatment of various inflammatory disorders and cancer.

Biography

Luz María Sánchez Perera has completed his Ph.D at the age of 35 years from Havana Agrarian University and postdoctoral studies from Valencia University School of Pharmacy, Spain; Sao Francisco University, Brasil; Autonomy University of Mexico. She is the director of Phytochemistry Lab, a Cuban Educational Minister organization. She has published more than 40 papers in reputed journals and serving as an editorial board member of repute.