5I J QIFNJTUSZ PG)VOHFS 4UJNVMBUJOH)PS $B *O)ZQPUIBMBNVT *T #OFGJDJBM

Mahjabi d [2], M Salahuddin [3]*, Md. Sayfullah [4], Deluwer Hossain [5], MA Momin [6], M. Abu Sayed [7], Jay Prakash Sah [8] and Sanjay K Sh

[1]College o ine ge and Hospital, Mymensingh, University of Dhaka, Bangladesh
[2]Departme iote ring, Faculty of Life Science, Mawlana Bhashani Science and Technology University, Tangail-1902, Bangladesh
[3]Faculty o ine n Road, Hongkong
[4]College o cine llege Hospital, Bogra, University of Rajshahi, Bangladesh
[5]Departme Phar ersity, Mymensingh-2202, Bangladesh
[6]Departme Micr tural University, Mymensingh-2202, Bangladesh
[7]Departme Bioc mmad Danesh Science and Technology University, Dinajpur-5200, Bangladesh
[8]Departme Med llied Sciences, Pokhara University, Lekhnath -12 Kaski, Nepal
[9]Departme Med a Study, Satodobato, Lalitpur, Nepal

Ab

hu ner stimulate to food intake in body system. At present a Q ding the mechanisms by which various hormones and gh od intake where Plasma ghrelin levels are inversely co s. It is a natural ligand of the growth hormone (G highly expressed in the hypothalamus, but is als eripheral tissues. Ghrelin is still recognized D

Keywords: ich leading to the inhibition of ACC activity and Neuroscience CoA levels and nally resulting in increased tivation of carnitine-palmitoyl transferase

Editorial

Ghrelin, the hung by the ghrelinergic ce stomach & a lesser exte in the CNS. Besides regu role in regulating distribution a as G-protein coupled receptor secretagogue receptor (GHSR) or g acids and containing an n-octanoyl g position 3 [1-6]. It is reported that ghrelin is various types of tissues such as duodenum, je heart, pancreas, kidney, testis, pituitary, and hyp system, moreover in the CNS system it expressed a

Recent ndings revealed that administration of g induces food intake and reduction of energy expenses major physiological and biological function of ghrelin includes hormone secretion, stimulation of food intake, gastric acid sec regulation of motility and the regulation of the endocrine and exocri pancreatic secretions.

A er crossing the blood-brain barrier ghrelin reaches in brainstem [17], and transmits its signal through the vagal nerve [18]. In hypothalamus, it activates the arcuate nucleus (ARC), paraventricular nucleus (PVN), dorsomedial region, central nucleus of amygdala, and the nucleus of solitary tract [19-20]. By stimulating the activity of NPY/AGRP neurons and decreasing the activity of POMC and CART neurons, ghrelin increases appetite and food intake [21-23]. AMPK is regulates the fuel availability by stimulating ATP producing pathways and inhibiting ATP consuming pathways [24]. A er ATP depletion, AMP rises and induces the activation of AMPK by phosphorylation [25]. Activated AMPK then induces the phosphorylation of acetyl-CoA

*Corresp
of Hongk
ssdin23@gm

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From the literature review it is reported that, SIRT1 and p53 are required for ghrelin induced AMPK activation [28]. mTOR is regulated by the cellular AMP/ATP ratio; mTOR activity decreases when AMP/ ATP is increases. On the other hand, mTOR activity increases when AMP/ATP ratio decreases [29] and is activated by AMPK [30]. Activated mTOR phosphorylates S6-kinase-1 (S6K1), S6 ribosomal protein (S6), and initiation factor 4E-binding protein (4E-BP1) [31-32]. It has been shown that hypothalamic mTOR signaling mediates the orexigenic action of ghrelin [33-34]. en ghrelin-mediated mTOR activation induces the increase of CREB-pCREB, FoxO-pFoxO1, and BSX transcription factors which in turn activate NPY and AGRP synthesis and nally leading to food intake in body systems [35] (Figure 2).

Summary

It is to be concluded that in the eld of neuroscience ghrelin has attracted tremendous interest in research. is is the key hormone in regulation of energy homeostasis in human body. Current evidences show that ghrelin a ects GH release, food intake, energy and glucose homeostasis, gastrointestinal, cardiovascular and immune functions, cell proliferation and di erentiation, and cognitive behavior. Ghrelin is still recognized as a potential drug target for weight regulation. For its unique molecular structure, in near future it's possible to nd out a breakthrough in the regulation of hunger stimulate, weight control and proper management of food intake in obese and anorexia patient by understanding its biochemical and pathophysiological mechanism.

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