

mechanisms that contribute to the workings of the circadian clock. Elucidation of abnormalities of the circadian system has also led to the discovery of new clinical disorders that can now be identified and treated.

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Letter to the Editor

Ghrelin-induced obesity

The July issue of *Growth, Genetics & Hormones* (Vol. 17, p 34-35) contains a discussion of the ability of this 28 amino acid peptide to induce body fat accumulation in rodents.

But of great importance to students of human obesity is the observation that the lean weight of these obese animals was probably less, certainly not greater, than that of the controls. This finding puts such ghrelin-treated animals clearly at odds with the human state, for the latter usually have an increase in lean weight, most certainly not a decrement.¹ The only clearly documented exceptions to this rule are patients with the Prader-Willi syndrome^{2,3} or Cushing's syndrome. With respect to body composition the human state differs from obesity induced by experimental hypothalamic lesions, from that of the "ob/ob" mouse, and the Zucker rat, all of which are characterized by a subnormal lean weight. Obviously, such animals, and those treated with ghrelin, cannot serve as models for human obesity.

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Editor's Response: Dr. Forbes in his talented analytical way has added significantly to the Abstract, Ghrelin: A Gastrointestinal and Hypothalamic Peptide Affecting Hormone Secretion and Fat Metabolism which dealt with studies in rats and not humans. With his astute commentary he reminds us that we should not necessarily project data obtained in rodents to humans. Neither of the Editors commenting on this article were so astute as to mention this most poignant point.

Thanks very much, Dr. Forbes. The Editorial Board eagerly invites each reader to write and comment on pertinent points, ask questions or query us concerning what is published in *Growth, Genetics & Hormones*.

Robert M. Blizzard, MD
Editor-in-Chief

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