COLLOQUIA ERUDITA 2026; 1: 39–40

PROCEEDINGS ABSTRACT

The translational research potential of Iraq's native fauna: the desert gerbil (*Psammomys obesus*) as a non-genetic model of diet-induced metabolic syndrome progression

Noor Dheyaa Kadhim, Apostolos Zarros, Hussam W. Al-Humadi, and Rafal J. Al-Saigh

Received: 25 April 2026; Revised: 09 June 2026; Accepted: 21 June 2026; Published: 23 June 2026

The growing global burden of the metabolic syndrome and of its complications necessitates the development of high-fidelity animal models that can accurately replicate the associated human pathophysiology. To this end, transgenic models frequently fail to capture the complex environment–gene interactions characteristic of the human disease. We herein review recent findings and highlight the translational research importance of the fat sand rat *Psammomys obesus* (a desert gerbil species that is indigenous to the arid environments of Iraq) as a non-genetic model of diet-induced metabolic syndrome progression. *Psammomys obesus* maintains a lean phenotype when in its natural habitat, as it has adapted to survive on a nutrient-poor, low-calorie halophytic diet; however, when this species is transitioned to a standard, high-calorie laboratory diet, it exhibits a cascade of metabolic derangements. This response is believed to mirror that of human populations undergoing nutritional transition as part of the adoption of a (more) Westernized lifestyle, thereby offering a diet-induced, polygenic platform for studying human diseases such as type 2 diabetes mellitus, non-alcoholic fatty liver disease (NAFLD), cardiovascular remodelling, and the metabolic syndrome at large. Our review synthesizes established and emerging evidence detailing the multi-organ effects of dietary interventions in *Psammomys obesus* [1]. The metabolic syndrome-simulating pathophysiological trajectory in this species follows a well-characterized progression from an early compensatory hyperinsulinaemia to a terminal pancreatic β-cell exhaustion and an absolute insulin deficiency [2]. Notably, *Psammomys obesus* can develop "clinical" complications that are notoriously difficult to reproduce in genetically modified rodent models. For example, within the cardiovascular system, the diet-induced stress has been shown to precipitate arterial wall thickening, profound myocardial fibrosis, and severe endothelial dysfunction. Concurrently, the liver of this desert gerbil species is capable of undergoing a rapid diet-induced progression toward advanced NAFLD, that is characterized by aggressive steatosis and irreversible fibrosis. Studies have also highlighted the model's importance for the simulation and elucidation of metabolic syndrome-associated microvascular pathologies, including spontaneous diabetic retinopathy (marked by retinal microvascular permeability) and diabetic nephropathy (featuring significant glomerular basement membrane thickening and overt albuminuria). This unique suscep-

Kadhim N. D., Zarros A., Al-Humadi H. W., Al-Saigh R. J.: The translational research potential of Iraq's native fauna: the desert gerbil (*Psammomys obesus*) as a non-genetic model of diet-induced metabolic syndrome progression. *Colloq. Erud.* 1: 39–40 (2026).
https://doi.org/10.5281/zenodo.20811441

This proceedings abstract is published in *Colloquia Erudita – Volume 1: Proceedings of the 3rd International Babylon Conference on Clinical and Experimental Pharmacological Research (Hillah; June 29–30, 2026)*; a volume of proceedings edited by Rafal J. Al-Saigh, Argyro Kyriakaki, and Apostolos Zarros.

tibility of *Psammomys obesus* to diet-induced metabolic collapse is believed to be driven by uncontrolled oxidative stress and the aberrant expression of key glucose transporters. In fact, unlike traditional laboratory rodents, this species possesses an inherently limited endogenous antioxidant capacity; therefore, when *Psammomys obesus* is exposed to a high-calorie diet, this inherent deficit leads to an accumulation of reactive oxygen species as well as to an accelerated mitochondrial dysfunction across many of the species' tissues. By capturing these diverse systemic manifestations alongside this molecular vulnerability, the diet-induced *Psammomys obesus* model provides a comprehensive representation of the metabolic syndrome's progression that closely simulates clinical reality and offers an invaluable experimental platform for the validation of cytoprotective and metabolism-modulating agents [3]. From a regional perspective, the harnessing of the translational research potential of *Psammomys obesus* may hold strategic importance for the Iraqi scientific community. As a species that is native to Iraq, it represents an accessible and cost-effective biological resource that circumvents many of the logistical and financial burdens associated with importing and maintaining specialized transgenic models. The systematic use of this species in Iraq as a non-genetic model of diet-induced metabolic syndrome progression could facilitate, amongst other translational research applications, the pharmacological screening of bioactive compounds deriving from native flora, thereby strengthening the country's position as a drug discovery hub in the Middle East.

Keywords

desert gerbil; diabetes; Iraq; metabolic syndrome; translational research

Acknowledgements

None.

Conflicts of interest statement

None to declare.

References

[1] Kaiser N., Cerasi E., Leibowitz G.: Diet-induced diabetes in the sand rat (*Psammomys obesus*). *Methods Mol. Biol.* 933: 89–102 (2012). https://doi.org/10.1007/978-1-62703-068-7_7

[2] Shafrir E., Ziv E., Kalman R.: Nutritionally induced diabetes in desert rodents as models of type 2 diabetes: *Acomys cahirinus* (spiny mice) and *Psammomys obesus* (desert gerbil). *ILAR J.* 47(3): 212–224 (2006). https://doi.org/10.1093/ilar.47.3.212

[3] Kalman R., Ziv E., Shafrir E., Bar-On H., Perez R.: *Psammomys obesus* and the albino rat -- two different models of nutritional insulin resistance, representing two different types of human populations. *Lab. Anim.* 35(4): 346–352 (2001). https://doi.org/10.1258/0023677011911949

Affiliation(s)

College of Pharmacy, University of Babylon, Hillah, Iraq *(NDK, AZ, HWA)* ▪ Department of Clinical Laboratory Sciences, College of Pharmacy, University of Babylon, Hillah, Iraq *(RJA)*

Correspondence

Noor Dheyaa Kadhim
College of Pharmacy
University of Babylon
Hillah, Iraq
pha282.noor.dheyaa@student.uobabylon.edu.iq