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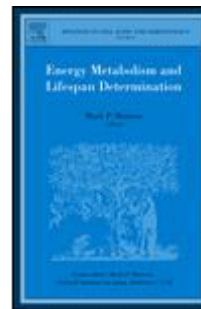
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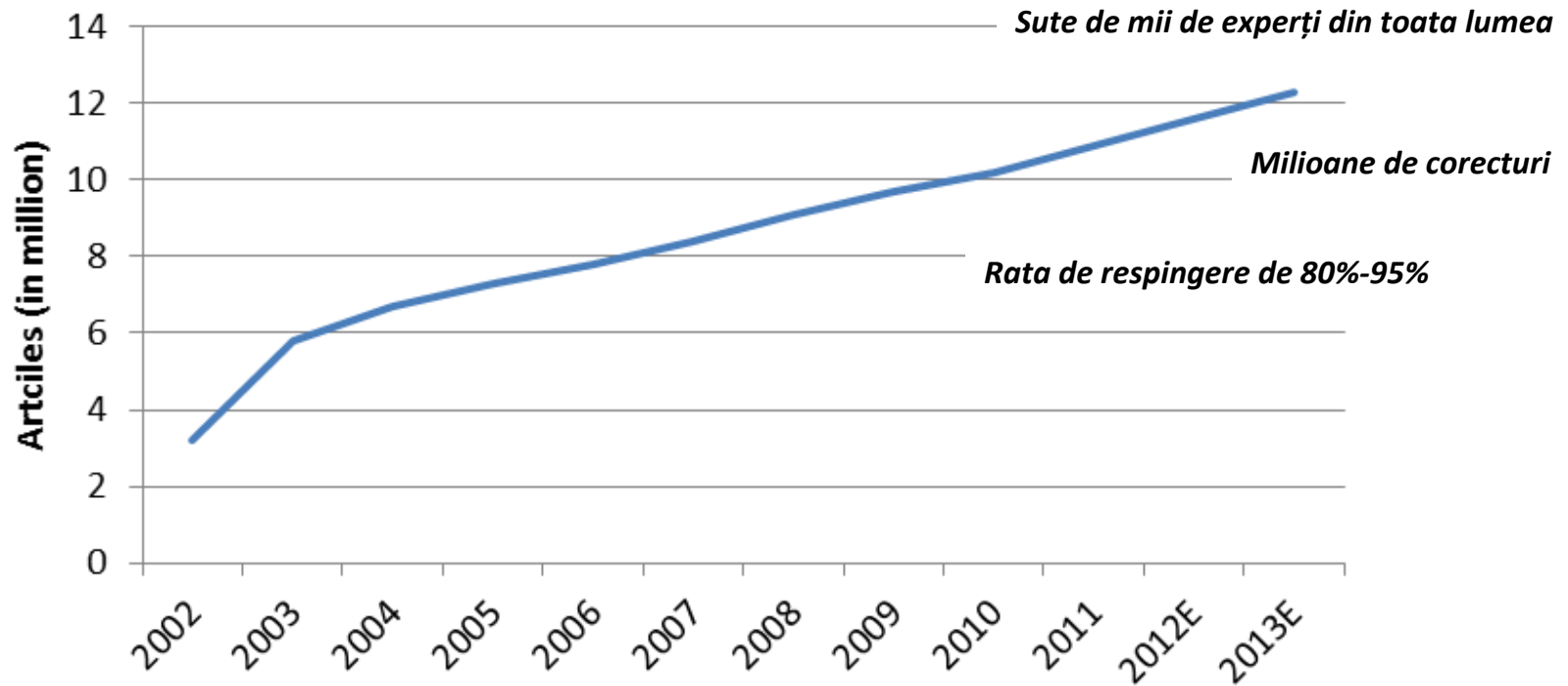
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Artificial Intelligence in Medicine
Volume 46, Issue 1, May 2009, Pages 1-10
Artificial Intelligence in Medicine

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Position paper

The coming of age of artificial intelligence in medicine ☆

Vimla L. Patel^a, Edward H. Shortliffe^{a, b}, Mario Stefanelli^c, Peter Szolovits^d, Michael R. Berthold^e, Riccardo Bellazzi^c, Ameen Abu-Hanna^f

^a Department of Biomedical Informatics, Arizona State University, ABC1, 425 North Fifth Street, Phoenix, AZ 85004, USA
^b Department of Basic Medical Sciences, University of Arizona College of Medicine, ABC1, Room 227, 425 North Fifth Street, Phoenix, AZ 85004-2157, USA
^c Dipartimento di Informatica e Sistemistica, University of Pavia, Via Ferrata 1, 27100 Pavia, Italy
^d Computer Science and Artificial Intelligence Laboratory, Massachusetts Institute of Technology, The Stata Center, 32-254, 32 Vassar Street, Cambridge, MA 02139, USA
^e Department of Computer and Information Science, University of Konstanz, BOX M712, 78457 Konstanz, Germany
^f Department of Medical Informatics, Academic Medical Centre, University of Amsterdam, Meibergdreef 15, 1105 AZ Amsterdam, The Netherlands

<http://dx.doi.org/10.1016/j.artmed.2008.07.017>, How to Cite or Link Using DOI
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Summary

This paper is based on a panel discussion held at the Artificial Intelligence in Medicine Europe (AIME) conference in Amsterdam, The Netherlands, in July 2007. It had been more than 15 years since Edward

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6. The system of reasoning
 - 6.1. The development of critical explanations for dressing performance
 - 6.2. A worked example of the system of reasoning



Volume 38, issue 5, October 2001, Pages 533-545

The palliative management of fungating malignant wounds – generalising from multiple-case study data using a system of reasoning

Patricia Grocott  , Sarah Cowley

Florence Nightingale School of Nursing and Midwifery, King's College London, James Clerk Maxwell Building, 57 Waterloo Road, London SE1 8WA, UK

Abstract

The project focused on individual experiences, from 45 participants, of living with a fungating wound and

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Management of Malignant Fungating Wounds in Advanced Cancer

2006, Seminars in Oncology Nursing

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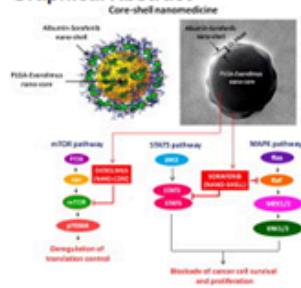
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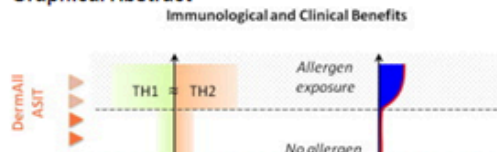
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 Parvathy Chandran, Neha Gupta, Archana Payickattu Retnakumari, Giridharan Loganathan Malarvizhi, Pavithran Keechilat, Shantikumar Nair, Manzoor Koyakutty
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3 **Implications for oxidative stress and astrocytes following 26S proteasomal depletion in mouse forebrain neurones** Original Research Article Open Access
 Pages 1930-1938
 Jamal Elkharaz, Aslihan Uqun-Klusek, Dumitru Constantin-Teodosiu, Karen Lawler, R John Mayer, Ellen Billett, James Lowe, Lynn Bedford

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Abstract

Neurodegenerative diseases are characterized by progressive degeneration of selective neurones in the nervous system, but the underlying mechanisms involved in neuroprotection and neurodegeneration remain unclear. Dysfunction of the ubiquitin proteasome system is one of the proposed hypotheses for the cause and progression of neuronal loss. We have performed quantitative two-dimensional fluorescence difference in-gel electrophoresis combined with peptide mass fingerprinting to reveal proteome changes associated with neurodegeneration following 26S proteasomal depletion in mouse forebrain neurones. Differentially expressed proteins were validated by Western blotting, biochemical assays and immunohistochemistry. Of significance was increased expression of the antioxidant enzyme peroxiredoxin 6 (PRDX6) in astrocytes, associated with oxidative stress. Interestingly, PRDX6 is a bifunctional enzyme with antioxidant peroxidase and phospholipase A₂ (PLA₂) activities. The PLA₂ activity of PRDX6 was also increased following 26S proteasomal depletion and may be involved in neuroprotective or neurodegenerative mechanisms. This is the first in vivo report of oxidative stress caused directly by neuronal proteasome dysfunction in the mammalian brain. The results contribute to understanding neuronal–glial interactions in disease pathogenesis, provide an in vivo link between prominent disease hypotheses and importantly, are of relevance to a heterogeneous spectrum of neurodegenerative diseases.

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Yue, Guangzhi Di, Yueyun Yu, Wei Wang, Huanli Shi

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Media of Language & Linguistics (Second Edition), 2006, Pages 518-523
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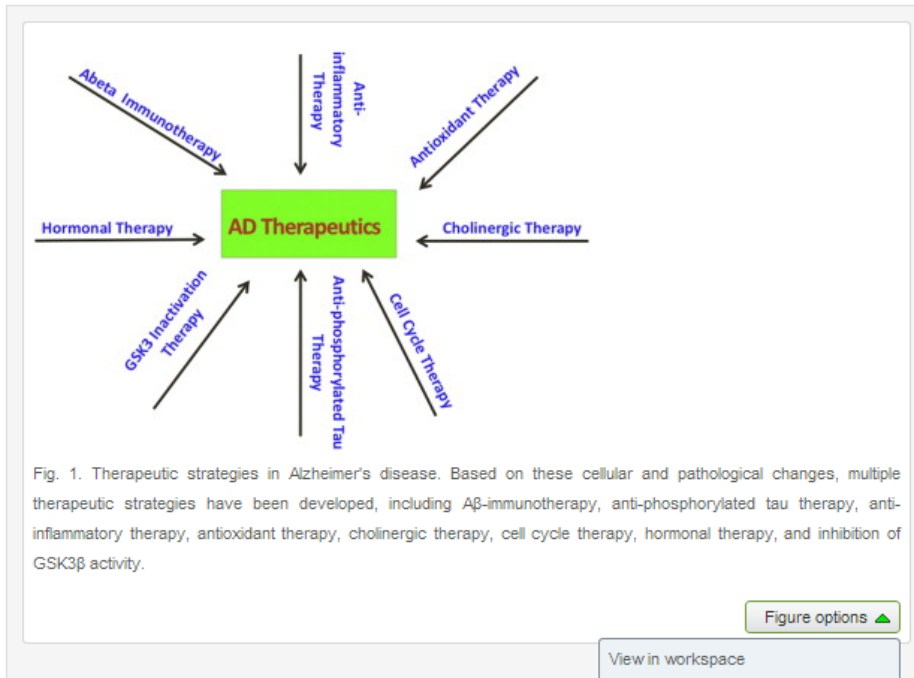
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[40], [42] and [30], and inhibition of GSK3 β activity [13], [14], [15], [16] and [17] (Fig. 1). Although tremendous progress has been made in understanding the AD progression and pathogenesis, and in developing therapeutic strategies, we still not have agents or drugs that can slow or prevent AD progression.



The purpose of this article is to review the latest developments of GSK3 β in AD pathogenesis, particularly its association with mitochondria in causing mitochondrial dysfunction. We also review and discuss GSK3 β involvement in phosphorylation of tau protein and its interaction with hexokinases in AD neurons.

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Review

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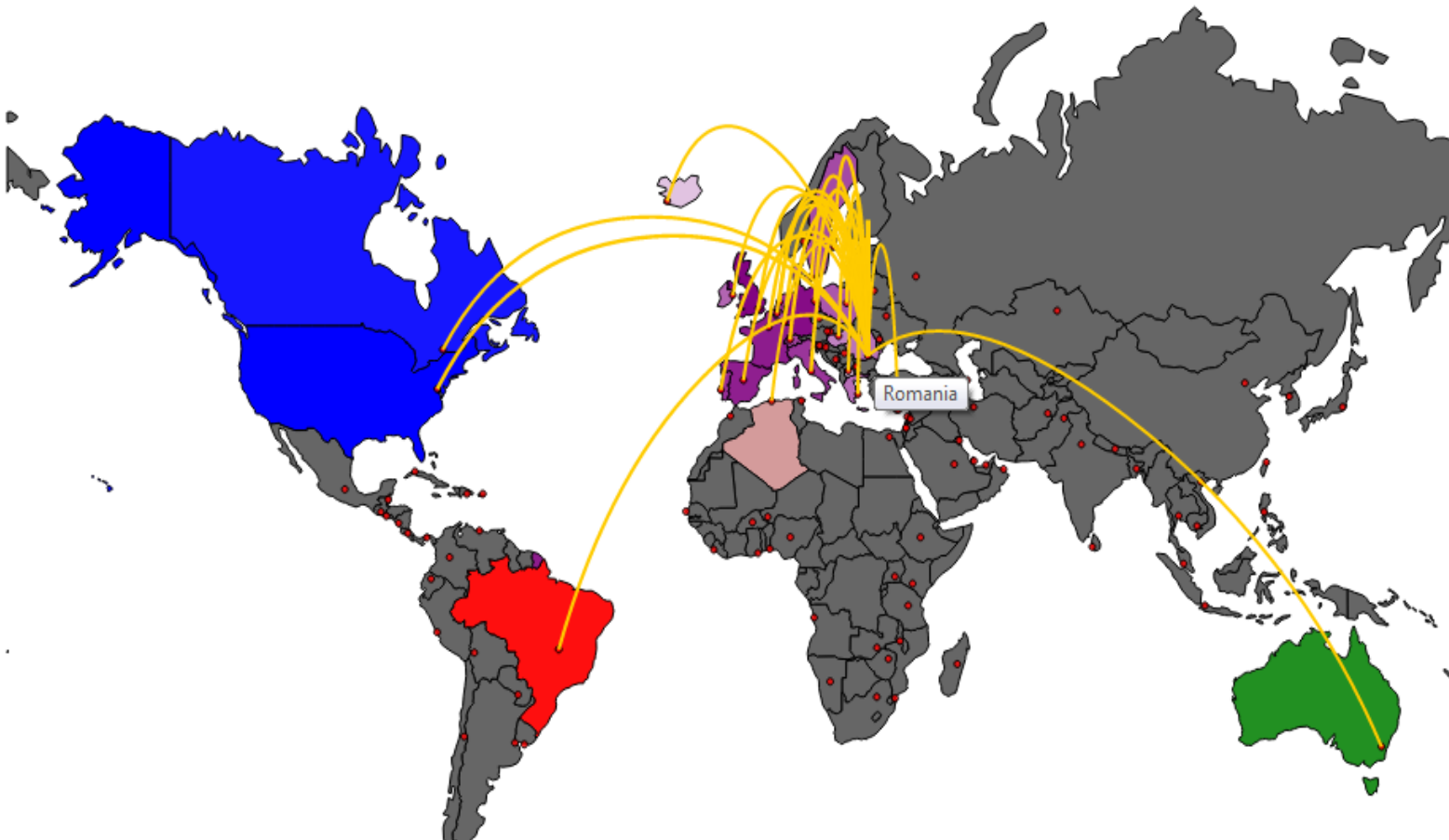
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A (Very) Brief History of Artificial Intelligence

Bruce G. Buchanan

■ In this brief history, the beginnings of artificial intelligence are traced to philosophy, fiction, and imagination. Early inventions in electronics, engineering, and many other disciplines have influenced AI. Some early milestones include work in problems solving which included basic work in learning, knowledge representation, and inference as well as demonstration programs in language understanding, translation, theorem proving, associative memory, and knowledge-based systems. The article ends with a brief examination of influential

niz and Blaise Pascal designed calculating machines that mechanized arithmetic, which had hitherto been the province of learned men called "calculators," but they never made the claim that the devices could think. Etienne Bonnot, Abbé de Condillac used the metaphor of a statue into whose head we poured nuggets of knowledge, asking at what point it would know enough to appear to be intelligent.

Science fiction writers have used the possibility of intelligent machines to design the

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