Resource Summary Report

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Cellular Open Resource

RRID:SCR_008022
Type: Tool

Proper Citation

Cellular Open Resource (RRID:SCR_008022)

Resource Information

URL: http://cor.physiol.ox.ac.uk/

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Description: Cellular Open Resource is a Microsoft Windows environment for cellular modeling that is built around CellML (except for reactions and metadata which are not supported). It offers, through CellML, an "out of the box" access to a large database of single cell models. COR was among the early adopters of this standard, eventually forming the first publicly available CellML-based modeling and collaboration environment. From the onset, COR was designed to provide an environment that could not only be used by experienced modelers, but also by experimentalists, teachers and students. It therefore tries to combine a user-friendly interface with a computationally efficient numerical engine. In this paper, we introduce the philosophy behind COR, explain its user interface and current functionality, including the editing and running of CellML files, highlight lessons learned from user feedback and problems experienced during the development of COR and conclude by exploring future development potential. Sponsors: This study has been supported by a grant from the UK Biotechnology and Biological Sciences Research Council (BB/E024955/1). Keyword: Cell, Model, Cellular, Modeling, Open resource, Microsoft, Environment, Database, Experimentalist, Teacher, Student, Modeler, Computationally, Development,

Synonyms: COR

Resource Type: data or information resource, database, software resource

Resource Name: Cellular Open Resource

Resource ID: SCR_008022

Alternate IDs: nif-0000-10187

Record Creation Time: 20220129T080245+0000

Record Last Update: 20260729T044216+0000

Ratings and Alerts

No rating or validation information has been found for Cellular Open Resource.

No alerts have been found for Cellular Open Resource.

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 15 mentions in open access literature.

Listed below are recent publications. The full list is available at nidm-terms .

Zaniboni M, et al. (2018) Short-term action potential memory and electrical restitution: A cellular computational study on the stability of cardiac repolarization under dynamic pacing. PloS one, 13(3), e0193416.

Gemmell P, et al. (2016) Rabbit-specific computational modelling of ventricular cell electrophysiology: Using populations of models to explore variability in the response to ischemia. Progress in biophysics and molecular biology, 121(2), 169.

Garny A, et al. (2015) OpenCOR: a modular and interoperable approach to computational biology. Frontiers in physiology, 6, 26.

Aronsen JM, et al. (2015) Hypokalaemia induces Ca²? overload and Ca²? waves in ventricular myocytes by reducing Na?,K?-ATPase ?? activity. The Journal of physiology, 593(6), 1509.

Cacciani F, et al. (2015) Chronotropic Modulation of the Source-Sink Relationship of Sinoatrial-Atrial Impulse Conduction and Its Significance to Initiation of AF: A One-Dimensional Model Study. BioMed research international, 2015, 496418.

Savi M, et al. (2014) Titanium dioxide nanoparticles promote arrhythmias via a direct interaction with rat cardiac tissue. Particle and fibre toxicology, 11, 63.

Neal ML, et al. (2014) A reappraisal of how to build modular, reusable models of biological systems. PLoS computational biology, 10(10), e1003849.

Solovyova O, et al. (2014) The cardiac muscle duplex as a method to study myocardial heterogeneity. Progress in biophysics and molecular biology, 115(2-3), 115.

Gemmell P, et al. (2014) Population of computational rabbit-specific ventricular action potential models for investigating sources of variability in cellular repolarisation. PloS one, 9(2), e90112.

Maltsev VA, et al. (2013) Numerical models based on a minimal set of sarcolemmal electrogenic proteins and an intracellular Ca(2+) clock generate robust, flexible, and

energy-efficient cardiac pacemaking. Journal of molecular and cellular cardiology, 59, 181.

Moreau A, et al. (2013) Sodium overload due to a persistent current that attenuates the arrhythmogenic potential of a novel LQT3 mutation. Frontiers in pharmacology, 4, 126.

Yaniv Y, et al. (2013) Mechanisms of beat-to-beat regulation of cardiac pacemaker cell function by Ca²? cycling dynamics. Biophysical journal, 105(7), 1551.

Noble D, et al. (2012) How the Hodgkin-Huxley equations inspired the Cardiac Physiome Project. The Journal of physiology, 590(11), 2613.

Corrias A, et al. (2010) Arrhythmic risk biomarkers for the assessment of drug cardiotoxicity: from experiments to computer simulations. Philosophical transactions. Series A, Mathematical, physical, and engineering sciences, 368(1921), 3001.

Noble D, et al. (2007) From the Hodgkin-Huxley axon to the virtual heart. The Journal of physiology, 580(Pt 1), 15.