

Resource Summary Report

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CellML

RRID:SCR_008061

Type: Tool

Proper Citation

CellML (RRID:SCR_008061)

Resource Information

URL: <http://www.cellml.org/>

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Description: The CellML language is an open standard based on the XML markup language. The purpose of CellML is to store and exchange computer-based mathematical models. CellML allows scientists to share models even if they are using different model-building software. It also enables them to reuse components from one model in another, thus accelerating model building. Although CellML was originally intended for the description of biological models; CellML includes information about model structure (how the parts of a model are organizationally related to one another), mathematics (equations describing the underlying processes) and metadata (additional information about the model that allows scientists to search for specific models or model components in a database or other repository). The CellML team is committed to providing freely available tools for creating, editing, and using CellML models. We provide information regarding tools we are developing internally and links to external projects developing tools which utilize the CellML format. Please let us know if you have an open source CellML tool looking for a home on the internet, as we are able to offer limited hosting services on cellml.org.

Abbreviations: CellML

Synonyms: CellML project, The CellML Project

Resource Type: data or information resource, interchange format, markup language, narrative resource, standard specification

Defining Citation: [PMID:15142756](#), [PMID:18658182](#), [PMID:19564239](#), [PMID:19380315](#), [PMID:18579471](#), [PMID:17947072](#), [PMID:17271569](#)

Keywords: biological model, cell, mathematical model, mathematics, metadata, model structure, model, xml, annotation, mark up language, FASEB list

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International Union of Physiological Sciences: Physiome Project ;
aneurIST ;
NZIMA ;
Foundation for Research Science and Technology ;
Wellcome Trust

Availability: The CellML project is built by an open, Democratic community on an Open unspecified license / free ethic.

Resource Name: CellML

Resource ID: SCR_008061

Alternate IDs: nif-0000-10448

Record Creation Time: 20220129T080245+0000

Record Last Update: 20260821T193833+0000

Ratings and Alerts

No rating or validation information has been found for CellML.

No alerts have been found for CellML.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 153 mentions in open access literature.

Listed below are recent publications. The full list is available at [NIF](#) .

Cestariolo L, et al. (2026) Mathematical model of the zebrafish ventricular cardiomyocyte action potential and calcium transient. The Journal of physiology, 604(13), 5391.

Avezz?? A, et al. (2026) A model for the human fetal ventricular myocyte electrophysiology. PLoS computational biology, 22(1), e1013889.

Barral YHM, et al. (2025) Comparison of in silico predictions of action potential duration in response to inhibition of IKr and ICaL with new human ex vivo recordings. PLoS computational biology, 21(7), e1012913.

Cabo C, et al. (2025) Strategies for prolonging ventricular action potential duration without increasing transmural dispersion of repolarization. Physiological reports, 13(23), e70693.

Zhang Y, et al. (2025) Genotype-specific Digital Twins for Arrhythmia Ablation Targeting in Arrhythmogenic Right Ventricular Cardiomyopathy. Research square.

Balaur I, et al. (2025) FAIRification of computational models in biology. bioRxiv : the preprint server for biology.

Yang Z, et al. (2025) Dominant ionic currents in rabbit ventricular action potential dynamics. PloS one, 20(7), e0328261.

Simitev RD, et al. (2025) A large population of cell-specific action potential models replicating fluorescence recordings of voltage in rabbit ventricular myocytes. Royal Society open science, 12(3), 241539.

Nicastro M, et al. (2025) Bi-allelic variants in POPDC2 cause an autosomal recessive syndrome presenting with cardiac conduction defects and hypertrophic cardiomyopathy. American journal of human genetics, 112(7), 1681.

Trayanova NA, et al. (2024) Computational modeling of cardiac electrophysiology and arrhythmogenesis: toward clinical translation. Physiological reviews, 104(3), 1265.

Athavale ON, et al. (2024) Neural regulation of slow waves and phasic contractions in the distal stomach: a mathematical model. Journal of neural engineering, 20(6).

Verkerk AO, et al. (2024) Effects of Acute Hypernatremia on the Electrophysiology of Single Human Ventricular Cardiomyocytes: An In Silico Study. Reviews in cardiovascular medicine, 25(6), 194.

Verkerk AO, et al. (2023) Injection of IK1 through dynamic clamp can make all the difference in patch-clamp studies on hiPSC-derived cardiomyocytes. Frontiers in physiology, 14, 1326160.

McCormick L, et al. (2023) Long QT syndrome-associated calmodulin variants disrupt the activity of the slowly activating delayed rectifier potassium channel. The Journal of physiology, 601(17), 3739.

Maltsev AV, et al. (2023) A novel conceptual model of heart rate autonomic modulation based on a small-world modular structure of the sinoatrial node. Frontiers in physiology, 14, 1276023.

Gast R, et al. (2023) PyRates-A code-generation tool for modeling dynamical systems in biology and beyond. PLoS computational biology, 19(12), e1011761.

C??mara-Checa A, et al. (2023) A gain-of-function HCN4 mutant in the HCN domain is responsible for inappropriate sinus tachycardia in a Spanish family. Proceedings of the National Academy of Sciences of the United States of America, 120(49), e2305135120.

Verkerk AO, et al. (2023) Human Sinoatrial Node Pacemaker Activity: Role of the Slow Component of the Delayed Rectifier K⁺ Current, IKs. International journal of molecular sciences, 24(8).

Means SA, et al. (2023) Steady-state approximations for Hodgkin-Huxley cell models: Reduction of order for uterine smooth muscle cell model. PLoS computational biology,

19(8), e1011359.

Cabo C, et al. (2023) Multichannel modulation of depolarizing and repolarizing ion currents increases the positive rate-dependent action potential prolongation. *Physiological reports*, 11(9), e15683.