Curriculum Vitae

Mary (Molly) Margot C. Maleckar

Schweigaards gate 61A
0656 Oslo, Norway
+47 474 82 159
mmaleck@simula.no

P.O. Box 134
1325 Lysaker, Norway
+47 67 82 82 70

EDUCATION

2008 **Ph.D.**, Biomedical Engineering
Johns Hopkins University, Baltimore, MD

2002 **B.Sc.**, Biomedical Engineering
Tulane University, New Orleans, LA

POSITIONS AND EMPLOYMENT

2015 – present **Senior Scientist,** Computational Cardiac Modeling, Simula Research Laboratory, Oslo, Norway
Training Coordinator, AFib-TrainNet EU MCSA ITN
Coordinator, SysAFib ERA CoSysMed European Project

2012 – present **Representative, Simula Research Laboratory**
Center for Cardiological Innovation (SFI) Board of Directors

2012 – 9/2015 **Director**
Simula School of Research and Innovation, Oslo, Norway

2011 - 2012 **Deputy Director for Simulation and Modeling**
Center for Cardiological Innovation (CCI), a Norwegian Center for Research Innovation (SFI)

2011 – 2012 **Research Department Head**, Computational Cardiac Modeling
Simula Research Laboratory, Oslo, Norway

2010 **Research Group Leader**
Computational Cardiac Modeling and Center for Biomedical Computing, Simula Research Laboratory, Oslo, Norway

2009 **Postdoctoral Research Fellow**
Center for Biomedical Computing, Scientific Computing
Simula School of Research and Innovation, Oslo, Norway

PEER-REVIEWED JOURNAL PUBLICATIONS

S. Kallhovd, S.U. Gerald, J. Saberniak, K. Haugaa, **MM Maleckar**.
Localization and not Extent of Fibrofatty Infiltration is the Primary Factor Determining Conduction Disturbance in a Computational Model of Arrhythmogenic Cardiomyopathy.
Proceedings IEEE e-Health and Bioengineering 2015. EHB 2015, November 19-21, 2015, Iasi, Romania.

Maleckar MM, Lines GT, Koivumäki J, Cordeiro JM, Calloe K.
NS5806 partially restores action potential duration but fails to ameliorate calcium transient dysfunction in a computational model of canine heart failure, 2014 Nov;16 Suppl 4:iv46-iv55.

Koivumäki JT, Clark RB, Belke D, Kondo C, Fedak PW, **Maleckar MM**, Giles WR.
Na(+) current expression in human atrial myofibroblasts: identity and functional roles.
Front Physiol. 2014 Aug 7;5:275. doi: 10.3389/fphys.2014.00275. eCollection 2014.

Frisk M, Koivumaki J, Norseng PA, **Maleckar MM**, Sejersted OM, Louch WE. Variable t-tubule organization and Ca2+ homeostasis across the atria. Am J Physiol Heart Circ Physiol. 2014 Jun 20.

Koivumäki JT, Seemann G, **Maleckar MM**, Tavi P. In silico screening of the key cellular remodeling targets in chronic atrial fibrillation. PLoS Comput Biol. 2014 May 22;10(5):e1003620. doi: 10.1371/journal.pcbi.1003620. eCollection 2014 May.

Yuan L, Koivumäki JT, Liang B, Lorentzen LG, Tang C, Andersen MN, Svendsen JH, Tfelt-Hansen J, **Maleckar M**, Schmitt N, Olesen MS, Jespersen T. Investigations of the Navß1b sodium channel subunit in human ventricle; functional characterization of the H162P Brugada syndrome mutant. Am J Physiol Heart Circ Physiol. 2014 Apr 15;306(8):H1204-12. doi: 10.1152/ajpheart.00405.2013. Epub 2014 Feb 21.

Li P, Lines GT, **Maleckar MM**, Tveito A. Mathematical Models of Cardiac Pacemaking Function. Frontiers in Physics, 1(20): 2013 http://www.frontiersin.org/Journal/10.3389/fphy.2013.00020/abstract

Wilhelms M, Hetmann H, **Maleckar MM**, Koivumäki J, Dossel O, Seeman G. Benchmarking electrophysiological models of human atrial myocytes, Frontiers in Physiology 3(487), 2013.

Koivumäki J, Christ T, Seemann G, and **Maleckar MM**. Divergent action potential morphology in human atrial cells vs. tissue: underlying ionic mechanisms, In: Computing in Cardiology, ed. by Alan Murray, vol. 39, pp. 121-124, Alan Murray (ISBN: 978-1-4673-2076-4), 2012. Refereed proceedings.

Rose RA, Belke DD, **Maleckar MM**, Giles WR. Ca2+ Entry Through TRP-C Channels Regulates Fibroblast Biology in Chronic Atrial Fibrillation. Circulation 126(17): 2039-41, 2012.

Tveito A, Lines GT, Edwards AG, **Maleckar MM**, Michailova A, Hake J, McCulloch A. Slow Calcium-Depolarization-Calcium waves may initiate fast local depolarization waves in ventricular tissue. Prog Biophys Mol Biol 110(2-3): 295-304, 2012.

Tveito A, Lines G, Rognes ME, and **Maleckar MM**. An analysis of the shock strength needed to achieve defibrillation in a simplified mathematical model of cardiac tissue. International Journal of Numerical Analysis and Modeling 9(3): 644-57, 2012.

Tveito A, Lines G, and **Maleckar MM**. Note on a possible pro-arrhythmic property of anti-arrhythmic drugs aimed at improving gap-junction coupling. Biophys J 102(2): 231-37, 2012.

Niederer SA, Kerfoot E, Benson A, Bernabeu MO, Bernus O, Bradley C, Cherry EM, Clayton R, Fenton FH, Garny A, Heidenreich E, Land S, **Maleckar M**, Pathmanathan P, Plank G, Rodríguez JF, Roy I, Sachse FB, Seemann G, Skavhaug O and Smith NP. N-Version Benchmark Evaluation of Cardiac Tissue Electrophysiology Simulators. Philosophical Transactions of the Royal Society VPH Issue. Philos Transact A Math Phys Eng Sci. 369(1954): 4331-51, 2011.

McDowell K, Arevalo H, **Maleckar MM**, and Trayanova NA. Susceptibility to reentry in the infarcted heart depends on active fibroblast density. Biophysical Journal 101(6): 1307-15, 2011.
Tveito A, Lines G, Skavhaug O, and **Maleckar MM**. Unstable eigenmodes are possible drivers for cardiac arrhythmias. Journal of the Royal Society Interface. 8(61): 1212-6, 2011.

Tveito A, Lines G, Artebrant R, Skavhaug O, and **Maleckar MM**. Existence of excitation waves for a collection of cardiomyocytes electrically coupled to fibroblasts. Mathematical Biosciences 230(2): 79-86, 2011.

Maleckar MM, Greenstein JL, Giles WR, and Trayanova NA. Electrotonic coupling between human atrial myocytes and fibroblasts alters excitability and repolarization. Biophysical Journal 97(8): 2179-2190, 2009.

Maleckar MM, Greenstein JL, Giles WR, and Trayanova NA. Repolarization in the human atrial myocyte – rate-dependent changes in the action potential waveform. Am J Physiol Heart Circ Physiol 297(4): 1398-1410, 2009.

Maleckar MM, Greenstein JL, Trayanova NA, and Giles WR. Mathematical simulations of ligand-gated and specific cell-type effects in the human atrium. Prog Biophys Mol Biol 98: 161-70, 2008.

Maleckar MM, Woods MC, Sidorov VY, Holcomb MR, Mashburn DN, Wikswo JP and Trayanova NA. Polarity reversal lowers activation time during diastolic field stimulation of the rabbit ventricles: Insight into mechanisms. Am J Physiol Heart Circ Physiol 295(4):H1626-33, 2008.

Bourn DW, **Maleckar MM**, Rodríguez B, Trayanova NA. Mechanistic enquiry into the effect of increased pacing rate on the upper limit of vulnerability. Phil Trans. Royal Soc A, 346:1333-1348, 2006.

Gurev V, **Maleckar MM**, and Trayanova NA. Cardiac Defibrillation and the Role of Mechano-Electric Feedback in Postshock Arrhythmogenesis. The Annals of the New York Academy of Sciences,1080:320-333, 2006.

GRANTS AND FELLOWSHIPS

05.2016 – 2019	SysAFib: Systems medicine for diagnosis and stratification of atrial fibrillation ERA CoSysMed, European Commission and BIOTEK2021 Research Council of Norway
09.2015 – 2019	EU Training Network on Novel Targets and Methods in Atrial Fibrillation (AFib-TrainNet) Marie Skłodowska-Curie Actions, European Commission
06.2015 – 2018	"Risk factors for sudden cardiac death during acute myocardial infarction (MI-RISK" Novo Nordisk Foundation Interdisciplinary Synergy Grant
2014-2015	PREPARE2: Increased science awareness among youth Simula School of Research and Innovation PROFORSK, Research Council of Norway
2014	Expert Advisor Policy Fellowship, The Research Council of Norway Brussels Office
2012-2013	Can Simulation shed light on a complex disease process? Simula Research Laboratory/University of California San Diego IS-BILAT, Research Council of Norway
2011-2019	The Center for Cardiological Innovation Simula Research Laboratory SFI, Research Council of Norway
2003-2007	State of Louisiana Board of Regents Graduate Fellowship Tulane University, New Orleans, LA

LANGUAGES

English (native); Spanish (excellent comprehension, spoken and written); Norwegian (excellent comprehension, spoken and written)

SELECTED PROFESSIONAL AFFILIATIONS

2013 – present	**Associate Editor**, Frontiers in Physics
2011 – present	**Member**, European Society of Cardiology Working Group on Cellular Cardiac Electrophysiology
2012 – present	**Member**, European Society of Cardiology Working Group on eCardiology
2009 – present	**Member**, Heart Rhythm Society

SELECTED CONFERENCES

Maleckar MM; Putting the pieces together: Towards supplementing sparse clinical data with multi physics simulation Foundation Teofilo Rossi di Montelera Forum 2015, December 6-9, 2015, Lugano, Switzerland. Invited talk.

S. Kallhovd, V. Mezzano, S.U. Gerald, J. Saberniak, F. Sheikh, K. Haugaa, **MM Maleckar**.
Localization and not Extent of Fibrofatty Infiltration is the Primary Factor Determining Conduction Disturbance in a Computational Model of Arrhythmogenic Cardiomyopathy.
Proceedings IEEE e-Health and Bioengineering 2015, accepted. EHB 2015, November 19-21, 2015, Iasi, Romania.

Maleckar MM; How many ionic models do we need for modelling of the atria? Atrial Signals 2015, Karlsruhe, Germany, 22.-24. October. Invited talk.

Maleckar MM. Patient-specific modeling: how good do we have to be? TRM Forum 2013, December 1-3, 2013, Lugano, Switzerland. Invited talk.

Maleckar MM, Lines GT, Koivumäki JT, Calloe K, Cordeiro JM. NS5806 partially restores APD but fails to ameliorate Ca2+-transient dysfunction in a computational model of canine heart failure. Cardiac Physiome Workshop, Bar Harbor, ME, October 15-19 2013. Poster.

Maleckar MM, Lines GT, Koivumäki JT, Calloe K, Cordeiro JM. Ca2+-transient dysfunction and ion channel therapy: what can we gather from a computational model of canine heart failure? EHRA Scientific Sessions 2013, 37th Annual Meeting of the ESC Working Group on Cardiac Cellular Electrophysiology, 2013. Poster.

Koivumäki J, Christ T, Ravens U, and **Maleckar MM**. The controversial late INa in human atrial myocytes: a computational analysis of Ranolazine's effects, EHRA Scientific Sessions 2013, 37th Annual Meeting of the ESC Working Group on Cardiac Cellular Electrophysiology, 2013. Poster.

Koivumäki J, **Maleckar MM**, and P. Tavi. Mechanisms Promoting Chronic Atrial Fibrillation: Role of Remodelled Intracellular Calcium Handling and Cellular Hypertrophy, Gordon Research Conference on Cardiac Arrhythmia Mechanisms, 2013. Poster.

Maleckar MM. Towards Modeling Arrhythmogenic Cardiomyopathy – Can Simulation Shed Light on a Complex Disease Process? Cardiac Physiome Workshop, San Diego, October 30 – November 2, 2012. Invited talk. Poster.

Koivumäki J, Christ T, Seemann G, and **Maleckar MM**. Divergent action potential morphology in human atrial cells vs. tissue: underlying ionic mechanisms, Computing in Cardiology Conference, 2012. Poster.

Koivumäki J, Christ T, Seemann G, and **Maleckar MM**. Divergent action potential morphology in human atrial cells vs. tissue: underlying ionic mechanisms, EWGCCE meeting, Nantes, France, 2012. Poster.

Maleckar MM. Modeling the effects of rotigaptide in atrial tissue: a cautionary tale. 9th International Conference of Numerical Analysis and Applied Mathematics, September 19-25, 2011. Invited talk.

Tveito A, Lines G, **Maleckar MM**, Skavhaug O. Simplified mathematical models of defibrillation. Virtual Physiological Human Scientific Sessions, 30.09-01.10 2010, Brussels, p. 367-369. Poster.

McDowell K, Arevalo H, **Maleckar MM**, Blake RC, and Trayanova NA. Fibroblast-myocyte coupling induces alterations in potassium currents that trigger regional action potential duration (APD) prolongation in infarcted myocardium. Heart Rhythm, 7(5S): S163-164, 2010. Poster.

McDowell K, Arevalo H, **Maleckar MM**, Blake RC, Plank G, Trayanova NA. Effects of fibroblast-myocyte coupling in the infarcted rabbit heart. Heart Rhythm 6(5S): S295-296, 2009. Poster.

Maleckar MM, Greenstein JL, Giles WR, Trayanova NA. Electrotonic coupling between human atrial myocytes and fibroblasts alters excitability and repolarization. Gordon Research Conference on Cardiac Arrhythmia Mechanisms, Il Ciocco, Italy, February 2009. Poster.

Maleckar MM, Greenstein JL, Giles WR, Trayanova NA. Coupling of Human Atrial Myocytes and Myofibroblasts Can Lead to Conduction Disturbances. Heart Rhythm 5(5S): S57, 2008. Abstract talk.

Maleckar MM, Blake RC, Trayanova NA. Fibrosis Decreases Activation Time During Defibrillation Shocks. Heart Rhythm 3(5): S187, 2006. Poster.

Woods MC, **Maleckar MM**, Sidorov VY, Holcomb MR, Mashburn DN, Trayanova NA, Wikswo JP. Polarity Reversal Lowers Activation Time During Diastolic Field Stimulation of the Rabbit Heart. Heart Rhythm 3(5S):S181, 2006. Poster.

Maleckar MM, Bourn DW, Rodríguez B, Trayanova NA. Mechanistic insight into the increase in the upper limit of vulnerability for rapid pacing. Heart Rhythm 2(5S):S219, 2005. Poster.

Maleckar MM, Bourn DW, Rodriguez B, and Trayanova NA. Mechanistic insight into the increase in the upper limit of

vulnerability for rapid pacing. Gordon Research Conference on Cardiac Arrhythmia Mechanisms, Santa Inez Valley, CA. February 2005. Poster and presentation.

Maleckar MM and NA Trayanova. Negative VEP affects activation during diastolic stimulation in the rabbit ventricles. Heart Rhythm 1(5):S225, 2004. Poster.

Maleckar MM and NA Trayanova. Paradoxical activation time during diastolic stimulation in the rabbit ventricles. 2003 BMES Annual Fall Meeting. Poster.