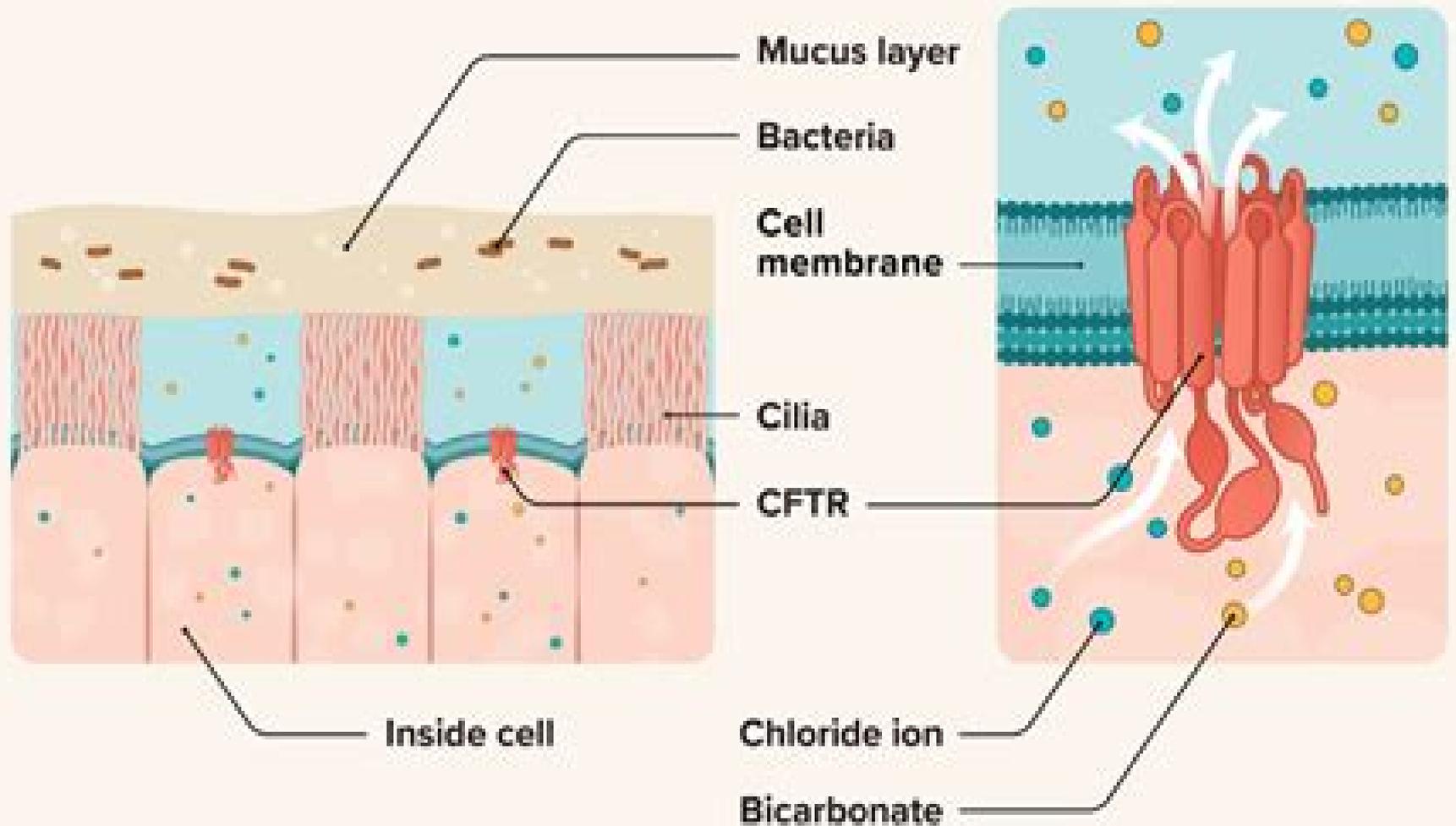


# Location and Function

## Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) Protein



# Cystic Fibrosis Transmembrane Conductance Regulator

**Raffaella Di Napoli**



## **Cystic Fibrosis Transmembrane Conductance Regulator:**

**The Cystic Fibrosis Transmembrane Conductance Regulator** Kevin L. Kirk, David C. Dawson, 2003-10-31 The CFTR chloride channel is one of the most well studied transport proteins in biology Yet there remain many mysteries about the functional properties and biological roles of this ABC transporter The Cystic Fibrosis Transmembrane Conductance Regulator addresses a select series of hot topics that relate to the function of CFTR and the links between CFTR dysfunction and human disease i e cystic fibrosis The timeliness of these topics distinguishes this collection from previous volumes of this type Given the general interest in CFTR this collection will appeal to a broad readership with interests in CFTR cystic fibrosis ion channels and ABC transporters *Studies of the Cystic Fibrosis Transmembrane Conductance Regulator Protein* M. M. C. Pereira, 1994 **Regulation of Expression of the Human Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) Gene** Rebecca Kate Rowntree, Ann Harris, 2001 **Cystic Fibrosis Methods and Protocols** William R. Skach, 2008-02-02 Since the cloning of the cystic fibrosis transmembrane conductance re lator CFTR nearly a decade ago cystic fibrosis CF research has witnessed a dramatic expansion into new scientific areas Basic researchers clinicians and patients increasingly rely on fundamental techniques of genetics molecular biology electrophysiology biochemistry cell biology microbiology and immunology to understand the molecular basis of this complex disease Research into the pathophysiology of CF has established numerous paradigms of ion channel dysfunction that extend from inflammation and infection in the airways of patients to basic mechanisms of protein processing and regulation in intracellular components With these rapid advances has come an increasing need for research scientists to understand and utilize a growing array of basic laboratory tools This volume of *Methods in Molecular Medicine Cystic Fibrosis Methods and Protocols* satisfies that need by providing detailed protocols for the laboratory techniques used throughout CF research From electrophysiology and cell biology to animal models and gene therapy the comprehensive set of methods covered here provide step by step instructions needed for investigators to incorporate new approaches into their research programs Contributions have been chosen to reflect the rich diversity of techniques and to provide a cohesive framework for understanding challenges that are currently at the forefront of CF research It is hoped that this volume will serve as a valuable reference that will not only foster interdisciplinary investigations into current problems encountered in CF but also facilitate the translation of new scientific discoveries into clinical solutions *Function and Regulation of the Cystic Fibrosis Transmembrane Conductance Regulator* Matthew P. Anderson, 1993 *Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) Modulators and Their Mechanistic Basis* Louise Clare Pyle, 2009 *Regulation and Function of the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) [microform]* Ilana Kogan, 2003 **Regulation of Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) by Rab GTPases** Simarna Kaur, 2008 Defining a Role for the Cystic Fibrosis Transmembrane Conductance Regulator in the Heart Zachary M. Sellers, 2010 While cystic fibrosis CF is commonly thought

of as a lung disease since its first description 70 years ago we have come to understand that loss of CFTR cystic fibrosis transmembrane conductance regulator function affects numerous tissues and systems throughout the body Concerning the heart right ventricular dysfunction secondary to pulmonary abnormalities has long been recognized However the issue as to whether loss of CFTR function causes primary disturbances in the heart has remained an unsolved and often debated question Thus the goal of the research presented herein was to answer two fundamental questions 1 0 Does CFTR play a physiological role in heart function 0 and 2 0 What direct impact does loss of CFTR activity have on heart function 0 These are particularly timely questions now as CF patients are living longer and the development of CFTR modulators is being pursued with increased vigor Thus an understanding if CF patients will be at increased risk for heart disease and whether new therapies may affect heart function are of direct relevance to the care of these individuals To begin to answer the questions presented we have examined how modulation of CFTR affects neonatal ventricular cardiomyocyte contraction rate We found that CFTR is involved in the regulation of both baseline and beta adrenergic stimulated contraction rate Additionally loss of CFTR activity whether acutely or chronically leads to modulation of intracellular signaling molecules and other Cl channels which in some cases can completely compensate for this loss while in other cases cannot Finally in vivo examination of heart function in CFTR KO mice has elucidated disturbances in heart structure and function which may translate into problems experienced by CF patients In sum this work presents a significant expansion of our understanding regarding the role of CFTR in heart function and highlights the need to further pursue a more in depth analysis of the health of the heart in CF patients

**The Cystic Fibrosis Transmembrane Conductance Regulator**, 1993 **Cystic Fibrosis in the 21st Century** Andrew Bush, 2006 Cystic fibrosis used to be thought of as a respiratory and digestive disease with a uniformly and rapidly fatal outcome The spectrum of the disease has broadened into the mild atypical case presenting in middle age with the potential for complications in virtually every system of the body In the past few years there has been an explosion of knowledge of the basic science of the defect The editors have therefore invited the leading scientists and clinicians in the field of cystic fibrosis to describe the recent advances in this disease Although there are many Recent Advances texts previous books have been selective in their choice of topics This book is the first to cover the entire field of this complex disease and encompasses the rapidly moving topics of the basic molecular and cellular biology as well as the recent multi system multi disciplinary advances in the clinical care of patients The authors have been charged with writing only about new developments and not to rehash old literature The bulk of the references is therefore less than five years old This book addresses all professionals who treat cystic fibrosis and want to have an up date of new findings in the field particularly of those outside their immediate specialisation It will also be useful for basic researchers interested in related scientific areas and the clinical context of their work *Cumulated Index Medicus*, 1999 **Study of the Cystic Fibrosis Transmembrane Conductance Regulator (Cftr) Protein in Human and Mouse Vas Deferens** Ingrid

Reynaert,1998-12-01      **Cystic Fibrosis Transmembrane Conductance Regulator** Lisa Sue Smit,1993      **CMR** ,2020  
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of January 2020      Cystic Fibrosis Transmembrane Conductance Regulator Simarna Kaur,2009-08 Cystic fibrosis CF is  
caused by the dysfunction of cystic fibrosis transmembrane conductance regulator CFTR The predominant mutation found in  
CF patients delta F508 results in a mislocalized channel which is unable to reach the plasma membrane Rab proteins are  
small GTPases involved in vesicle transport The regulation of CFTR by Rab GTPases 4 and 27a was evaluated in multiple cell  
systems the colorectal HT 29 cells bronchial epithelial IB3 1 and S9 cells Over expression of Rab proteins led to inhibition of  
CFTR activity by reducing its cell surface level without affecting the total CFTR pool GDP locked and GTPase deficient  
mutants of Rabs 4 and 27a were found to regulate CFTR at varying degrees by binding to the channel Signaling mechanisms  
that control modulation of CFTR activity by Rab proteins include the PI3 kinase and MAP kinase cascades Proposed models  
showcase a possible role for Rab4 in mediating the recycling of CFTR or delta F508 from the plasma membrane to the  
microsomes inside the cell Rab27a can potentially be a key player in regulating the trafficking of CFTR or delta F508 towards  
the lysosomes for degradation which is reversed by effector proteins      "Code of Massachusetts regulations, 2016" ,2016  
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David Zeltwanger,1998      *NIH Repository of Human and Mouse DNA Probes and Libraries* American Type Culture  
Collection,1989

## The Enigmatic Realm of **Cystic Fibrosis Transmembrane Conductance Regulator**: Unleashing the Language is Inner Magic

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