

Biochemical and Biophysical Research Communications

Peter (P.W.) Andrews

Department of Biomedical Science
University of Sheffield
Sheffield, UK

Wolfgang Baumeister

Abteilung Molekulare Strukturbioogie
Max-Planck-Institut für Biochemie
Martinsried, Germany

Ernesto Carafoli

Editor-in-Chief
Venetian Institute of Molecular
Medicine (VIMM)
University of Padova, Italy

Chin Ha Chung

School of Biological Sciences
College of Natural Sciences
Seoul National University
Seoul
Republic of Korea

Zengyi Chang

Biochemistry and Molecular Biology
School of Life Sciences
Center of Protein Science
Peking University Beijing, China

Vitaly Citovsky

Department of Biochemistry and
Cell Biology
State University of New York
Stony Brook, NY, USA

Bengt Fadeel

Division of Molecular Toxicology
Institute of Environmental Medicine
Karolinska Institutet Stockholm, Sweden

Barry Halliwell

Biochemistry Department
National University of Singapore
Singapore, Singapore

Cecilia Hidalgo

Faculty of Medicine
University of Chile Santiago, Chile

Hans Jornvall

Department of Medical Biochemistry
and Biophysics
Karolinska Institutet
Stockholm, Sweden

Claude Klee

Laboratory of Biochemistry
National Cancer Institute
National Institutes of Health
Bethesda, Maryland, USA

Guido Kroemer

INSERM, U848
Institut Gustave Roussy
Villejuif, France

William J. Lennarz

Department of Biochemistry and
Cell Biology
State University of New York
at Stony Brook
Stony Brook, New York, USA

Michael Lichten

Laboratory of Biochemistry and
Molecular Biology, Center for Cancer Research,
National Cancer Institute, US

Anders (A.H.) Lund

Biotech Research and Innovation Centre
University of Copenhagen
Copenhagen, Denmark

Carlos Martínez-A

Department of Immunology and Oncology
National Center for Biotechnology
Campus Universidad Autonoma
28049 Madrid, Spain

Hisao Masai

Director of Center for Basic Technology
Research, Genome Dynamics Project
Department of Genome Medicine
Tokyo Metropolitan Institute of Medical
Science, Tokyo, Japan

Satyajit Mayor

Cellular Organization and Signalling Group
National Centre for Biological Science
(NCBS), UAS-GKVK Campus
Karnataka, India

Katsuhiko K. Mikoshiba

RIKEN
Laboratory for Development
Neurobiology, Japan

Davis Ng

Temasek Life Sciences Laboratory
National University of Singapore
Singapore, Singapore

James M. Ntambi

Departments of Biochemistry and
Nutritional Sciences
University of Wisconsin-Madison
Madison WI 53706, USA

Sten Orrenius

Institutet of Environmental Medicine
Karolinska Institutet
Stockholm, Sweden

Jacques Pouyssegur

UMR 6543 CNRS
Centre Antoine Lacassagne
Nice, France

Igor Stagljar

Department of Molecular Genetics and
Biochemistry
University of Toronto
Toronto, Ontario, Canada

Bing Su

Department of Immunology and
Microbiology, Shanghai Institute of
Immunology, China

Kiyoshi Takatsu

Department of Immunology
Institute of Medical Science
University of Tokyo, Tokyo, Japan

Naoyuki Taniguchi

Systems Glycobiology Research Group
RIKEN Global Research Cluster
Wako, Japan

Anna Tramontano

Department of Physics
Sapienza University of Rome
Rome, Italy

Olga Troyanskaya

Department of Computer Science and
Lewis-Sigler Institute for Integrative
Genomics Princeton University,
New Jersey, USA

Isaac P. Witz

Tel Aviv University
Tel Aviv, Israel

Correspondence regarding production may be sent to:

Biochemical and Biophysical Research Communications, Elsevier Inc.

525 B Street, Suite 1800, San Diego, California 92101-4495, USA

Telephone +1 (619) 699-6857, Fax +1 (619) 699-6859, E-mail bbr@elsevier.com



Cover photo. Hypothetical model of the mechanisms involved in AIF processing and release. Exposure of NSCLC cells to the protein kinase C inhibitors, staurosporine or PKC412, results in a hyperpolarization of the plasma membrane. As a consequence, the hyperpolarization-activated HCN2 channel opens and permits Ca^{2+} to enter the cell. Both plasma membrane hyperpolarization and the activation of HCN2 channel are inhibited by Cs^+ . The resulting Ca^{2+} elevation in the cytosol also translocates to the intermembrane space of the mitochondria and results in the activation of calpain as well as enhanced ROS formation. The calcium chelator, BAPTA is able to inhibit both calpain activation and ROS accumulation, whereas only the latter is inhibited by NAC and Trolox. AIF is cleaved by mitochondrial calpain-I. This cleavage is prevented by PD150606, a selective calpain inhibitor. Cleaved AIF is released into the cytosol and translocates to the nucleus, where it contributes to chromatin condensation and highmolecular weight DNA fragmentation. Nuclear translocation of AIF can be inhibited by binding to Hsp70 in the cytosol. (BBRC Volume 396, pages 95–100). It is reproduced by kind permission of the authors – Sten Orrenius, et al.