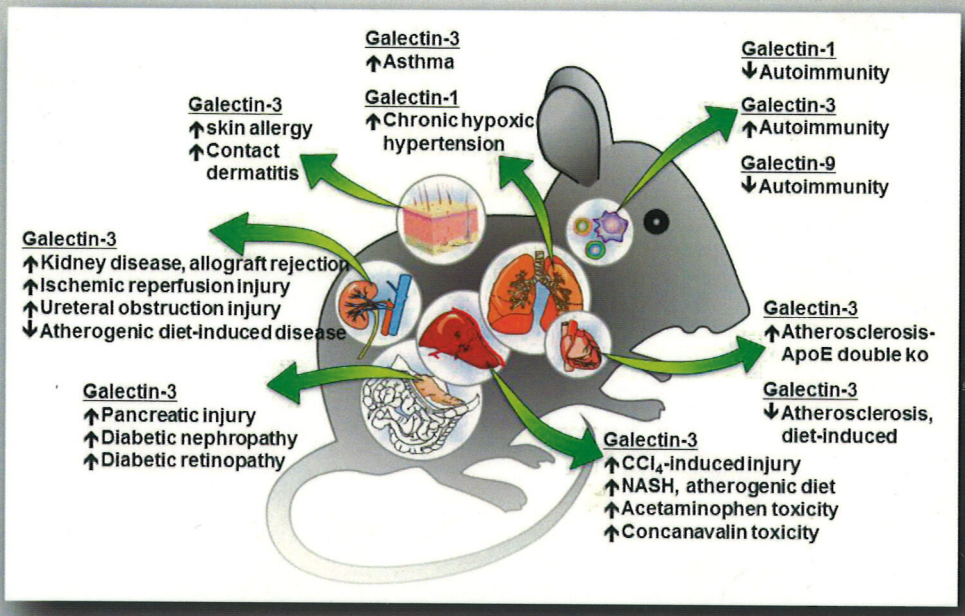
ACS SYMPOSIUM SERIES 1115

Galectins and Disease Implications for Targeted Therapeutics



EDITED BY

Anatole A. Klyosov and Peter G. Traber

Galectins and Disease Implications for Targeted Therapeutics

ACS SYMPOSIUM SERIES **1115**

Galectins and Disease Implications for Targeted Therapeutics

Anatole A. Klyosov, Editor
Galectin Therapeutics, Inc.
Norcross, Georgia

Peter G. Traber, Editor
Galectin Therapeutics, Inc.
Norcross, Georgia

American Chemical Society, Washington, DC

Distributed in print by Oxford University Press

Library of Congress Cataloging-in-Publication Data

Galectins and disease implications for targeted therapeutics / Anatole A. Klyosov, editor, Galectin Therapeutics, Inc., Newton, Massachusetts, Peter G. Traber, editor, Galectin Therapeutics, Inc., Newton, Massachusetts.
pages cm. -- (ACS symposium series ; 1115)
Includes bibliographical references and index.
ISBN 978-0-8412-2880-1
1. Lectins--Therapeutic use. I. Klesov, A. A. (Anatolii Alekseevich) II. Traber, Peter G.
QP552.L42G354 2012
572'.69--dc23

2012042318

The paper used in this publication meets the minimum requirements of American National Standard for Information Sciences—Permanence of Paper for Printed Library Materials, ANSI Z39.48n1984.

Distributed in print by Oxford University Press

PRINTED IN THE UNITED STATES OF AMERICA

Foreword

The ACS Symposium Series was first published in 1974 to provide a mechanism for publishing symposia quickly in book form. The purpose of the series is to publish timely, comprehensive books developed from the ACS sponsored symposia based on current scientific research. Occasionally, books are developed from symposia sponsored by other organizations when the topic is of keen interest to the chemistry audience.

Before agreeing to publish a book, the proposed table of contents is reviewed for appropriate and comprehensive coverage and for interest to the audience. Some papers may be excluded to better focus the book; others may be added to provide comprehensiveness. When appropriate, overview or introductory chapters are added. Drafts of chapters are peer-reviewed prior to final acceptance or rejection, and manuscripts are prepared in camera-ready format.

As a rule, only original research papers and original review papers are included in the volumes. Verbatim reproductions of previous published papers are not accepted.

ACS Books Department

Contents

Galectin Mechanisms and Therapeutic Approaches in Inflammation and Fibrosis

Indexes

Chapter 9

The Role of Galectin-3 in Malignant Melanoma

Gordana Radosavljevic,[1] Ivan Jovanovic,[1] Jelena Pantic,[1] Nada Pejnovic,[1] Nebojsa Arsenijevic,[1] Daniel K. Hsu,[2] and Miodrag L. Lukic*,[1]

[1]Center for Molecular Medicine and Stem Cell Research, Faculty of Medicine Science, University of Kragujevac, Svetozara Markovica 69, 34000 Kragujevac, Serbia
[2]Department of Dermatology, School of Medicine, University of California Davis, Sacramento, California 95816, United States
***E-mail: miodrag.lukic@medf.kg.ac.rs**

Mechanisms by which Galectin-3 participate in regulation of innate and acquired immune responses include adhesion of neutrophils, chemoattraction of monocytes/macrophages and activation of mast cells and T-cell survival. We have demonstrated proinflammatory effects of Galectin-3 in several experimental models of T-cell-mediated inflammatory and autoimmune diseases. Galectin-3 is also expressed in malignant cells and is involved in tumor development and progression as well as tumor immune escape. Using Galectin-3-deficient mice and malignant melanoma model we recently showed that the expression of Galectin-3 in the host cells facilitate establishment of metastasis in the lung. Mechanisms contributing to resistance to melanoma in Galectin-3-deficient mice appear to be the lower tumor cell adhesion and modulation of immune responses. Galectin-3-deficient mice had higher serum levels of IFN-gamma and IL-17 and a decrease in the percentage and total number of regulatory $CD4^+Foxp3^+$T cells compared to wild-type mice. Protective effects of Galectin-3 deficiency on metastatic melanoma spread was dependent on NK cells and associated with an enhanced cytotoxic activity of splenic NK cells *in vitro* and increased frequency of effective

ISBN 978-0-8412-2880-1

PUBLISHED BY THE AMERICAN CHEMICAL SOCIETY