



The Role of Stat1 in Retinoic Acid-Induced Myelomonocytic Differentiation of Human Leukemia Cells

BY

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Role Of Stat1 In Retinoic Acidinduced Myelomonocytic Differentiation Of Human Leukemia Cells

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Role Of Stat1 In Retinoic Acidinduced Myelomonocytic Differentiation Of Human Leukemia Cells:

The Role of Stat1 in Retinoic Acid-Induced Myelomonocytic Differentiation of Human Leukemia Cells Anna Dimberg,2002

Vitamin A and Retinoids: An Update of Biological Aspects and Clinical Applications Maria A. Livrea,2012-12-06

Rapid progress has been made in our understanding of the molecular mechanisms of cell growth and oncogenesis during the past decade Special attention has been given to the presentation of the frequently neglected close correlation between changes in signal transduction and metabolic pathways during oncogenesis This book advances the knowledge of mechanisms regulating metabolism and functioning of vitamin A and offers the most recent results of research on the clinical efficiency of retinoids in skin disorders and cancer The book presents recent findings on the regulation of cell growth in normal and neoplastic tissues by growth factors including hormones and by the activation and inactivation of oncogenes and tumor suppressor genes respectively It also offers a survey of the molecular and cell biochemistry of retinoids Basic researchers in biochemistry pharmacology and cell biology as well as clinicians will find this book very informative and up to date This book advances the knowledge of mechanisms regulating metabolism and functioning of vitamin A and offers the most recent results of research on clinical efficiency of retinoids in skin disorders and cancer Basic researchers in biochemistry pharmacology cell biology and clinicians will find this book very informative and up to date The chapters organized in six sections are contributed by leading scientists who have been working in the retinoid field for decades Their experience and competence is acknowledged worldwide *Antiangiogenic Agents in Cancer Therapy* Beverly A. Teicher,1998-12-28 Beverly Teicher and a panel of distinguished investigators survey the state of the art of antiangiogenesis research from the lab bench to clinical trials Timely and authoritative the contributors summarize our current understanding of tumor growth and its dependence on vascular development as well as the present status of antiangiogenic agents in preclinical and clinical development In addition the book also examines what is known about the mechanisms by which these therapeutic agents interfere with tumor vasculature and grapples with the problem of establishing criteria by which to assess their clinical efficacy *Antiangiogenic Agents in Cancer Therapy* offers a unique cutting edge compendium of antiangiogenic research taking stock of what has been accomplished where the experimental therapeutics of antiangiogenic agents is going and the continuing evolution of their role in cancer treatment and novel drug development *Acta Universitatis Upsaliensis* ,2002

Index Medicus ,2001-08 Vols for 1963 include as pt 2 of the Jan issue Medical subject headings *Abridged Index Medicus* ,1997-10 *Cumulated Index Medicus* ,2000 **Overcoming the Differentiation Block** Holly A. Jensen,2015 Differentiation induction therapy aims to reform rather than retaliate like conventional chemotherapeutic methods The most successful differentiation therapy has been treatment of t 15 17 positive acute promyelocytic leukemia APL patients with retinoic acid RA an embryonic morphogen and derivative of vitamin A Broad application of RA induced differentiation therapy is currently limited by na ve emergent RA resistance cell type specific responsivity and the ambiguous mechanism of RA

action This dissertation provides an introduction to RA differentiation therapy and myeloid leukemia cell lines in Chapter 1 Using patient derived HL60 myeloblastic FAB M2 leukemia cells where all trans retinoic acid RA induces granulocytic differentiation we developed two sequentially emergent RA resistant HL60 cell lines which are characterized by loss of RA inducible G1 G0 arrest CD11b expression oxidative metabolism and signaling factor expression reviewed in Chapter 2 All materials and methods are described in Chapter 3 We found that the Src family kinase inhibitor PP2 can rescue differentiation markers in RA resistant HL60 cells Chapter 4 Investigating additional kinase inhibitors targeting an interrelated kinase network revealed that the c Raf inhibitor GW5074 also rescues RA induced differentiation and clarified that a highly correlated RA induced c Raf Lyn module is uncoupled from traditional downstream events like Akt and ERK activation Chapter 5 Focusing on the lineage selection aspects of RA granulocytic vs vitamin D3 monocytic induction differentiation in bipotent HL60 cells we discovered that RA resistant HL60 display a progressive reduced response to D3 treatment Chapter 6 Finally we compared phenotype and transcription factor expression in wild type and RA resistant HL60 to the myeloid cell lines NB4 U937 and K562 and assessed the fundamental lineage specific induction mechanisms in cells of varying hematopoietic maturity Chapter 7 RA resistant cells provide a means to test combination treatment efficacy and comparatively can elucidate the crucial mechanisms required for the wild type response RA resistance can be combated with kinase inhibitors which should be selected in context based on their anti proliferative and pro differentiative capability Another differentiation inducing agent vitamin D3 cannot necessarily abrogate temporally segregated early or late RA resistance defects but D3 can induce significant if not complete monocytic differentiation in RA resistant cells Despite divergence of original isolates and limitations of monoculture a panel of cell lines can clarify fundamental mechanisms and responses to differentiation induction agents Potential future directions and closing remarks are offered in Chapter 8

Role of Retinoic Acid in Cell Differentiation of Acute Promyelocytic Leukemia Cells Saurabh Shyam Malpani, Program in Cell Biology and Anatomy. Graduate School of Basic Medical Sciences. New York Medical College, New York Medical College. Graduate School of Basic Medical Sciences, New York Medical College. Graduate School of Basic Medical Sciences. Program in Cell biology and Anatomy, 2011 Signal Regulation by CD38, Lyn, and MAPK-associated Molecules During All-trans Retinoic Acid-induced Differentiation of Acute Myelogenous Leukemia Cells Johanna Congleton, 2012 All trans retinoic acid ATRA is successful in treating acute promyelocytic leukemia but has shown limited success with other cancers Identifying signaling pathways that confer ATRA responsiveness and compounds that enhance the effects of the drug could expand its therapeutic efficacy The leukocyte antigen CD38 is an ectoenzyme and receptor that propels MAPK signaling and ATRA induced differentiation when overexpressed CD38 enzymatic activity regulates calcium flux and NAD metabolism while its receptor functions drive phosphorylation of ERK c Cbl and p85 PI3K We found that crippling enzymatic activity with inhibitors or a point mutation CD38 E226Q did not affect ATRA induction while a cytosolic truncation CD38 DELTA 11 20

that prevented CD38 membrane expression crippled ATRA induced differentiation This indicated that CD38 receptor function is important for ATRA induction In contrast we found CD38 DELTA 11 20 cells responded to the monocytic inducer 1 25dihydroxyvitamin D3 similar to wild type cells suggesting CD38 signaling is important for granulocytic but not monocytic maturation We also found that CD38 may sustain ERK phosphorylation to maintain a cellular precommitment memory associated with ATRA exposure Since CD38 receptor functions appeared necessary for ATRA induction we identified CD38 interaction partners These included SLP 76 Vav1 and Lyn and we found that this complex was interrupted by inhibiting Lyn Lyn inhibition also blocked ATRA and CD38 stimulated phosphorylation of c Cbl and p85 PI3K suggesting some aspects of CD38 signaling are dependent on Lyn kinase activity Finally we found that the Lyn inhibitors PP2 and dasatinib enhanced ATRA induced differentiation Co treatment with ATRA plus either inhibitor upregulated total Lyn and c RafpS259 and increased associations between Lyn c Raf and cRaf ERK This was consistent with increased c Raf C terminus phosphorylation that may be regulated by ERK propelled feedback signaling within a KSR1 scaffold PP2 and dasatinib had different effects on Lyn activity suggesting that Lyn acts as an adaptor scaffold independent of its kinase function These results provide valuable insight as to how ATRA regulated molecules including CD38 Vav1 SLP 76 Lyn and the Raf MEK ERK axis may enhance the differentiation of acute myelogenous leukemia cells This report also identifies new therapeutic targets and compounds that may be used in combination with ATRA to improve its clinical efficacy

Identification of Changes in Mouse Embryonic Stem Cell Gene Expression During Retinoic Acid Induced Differentiation Harl Ray Tolbert,1996 *Role of Granulocyte Colony-stimulating Factor Receptor in WEHI-3B Myelomonocytic Leukemia Cell Differentiation* Debbie C. Koay,1997

Retinoic Acid Signaling in Acute Promyelocytic Leukemia Julien Ablain,2012

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