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Autore	DEL VECCHIO, Mario <1958- >
Titolo	Guidare le aziende sanitarie nella crisi : il ruolo dei direttori generali : l'esperienza della Regione Lazio / Mario Del Vecchio, Anna Romiti
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Autore	Charles Dudley Mills
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Nota di contenuto	Macrophages at the fork in the road to health or disease / Charles D. Mills, Laurel L. Lenz and Klaus Ley -- Introduction to Macrophage Biology and Functions -- Metabolism via arginase or nitric oxide synthase: two competing arginine pathways in macrophages / Meera Rath, Ingrid Müller, Pascale Kropf, Ellen I. Closs and Markus Munder -- Evolution of innate immunity: clues from invertebrates via fish to mammals / Kurt Buchmann -- Evolutionary roots of arginase expression and regulation / Jolanta Maria Dzik -- Macrophage: SHIP of Immunity / Charles D. Mills, Anita C. Thomas, Laurel L. Lenz and Markus Munder -- From monocytes to M1/M2 macrophages: phenotypical vs. functional differentiation / Paola Italiani and Diana Boraschi -- Spatial, temporal, and functional aspects of macrophages during “The Good, the Bad, and the Ugly” phases of inflammation / Robert A. Harris -- “Of mice and men”: arginine metabolism in macrophages / Anita C. Thomas and Joshua T. Mattila -- Macrophage Influences in Different Diseases / A. Wounds and Cancer: Stark Examples of the Two-Edged Sword Nature of Macrophage Responses -- Rethinking regenerative medicine: a macrophage-centred approach / Bryan N. Brown, Brian M. Sicari and Stephen F. Badylak -- Hepatic localization of macrophage phenotypes during fibro genesis and

resolution of fibrosis in mice and humans / Leonie Beljaars, Marlies Schippers, Catharina Reker-Smit, Fernando O. Martinez, Laura Helming, Klaas Poelstra and Barbro N. Melgert -- Functional relationship between tumor-associated macrophages and macrophage colony-stimulating factor as contributors to cancer progression / Damya Laoui, Eva Van Overmeire, Patrick De Baetselier, Jo A. Van Ginderachter and Geert Raes -- Frontiers in Immunology March 2015 M1/M2 Macrophages: The Arginine Fork in the Road to Health and Disease -- Depletion of tumor-associated macrophages slows the growth of chemically induced mouse lung adenocarcinomas / Jason M. Fritz, Meredith A. Tennis, David J. Orlicky, Hao Yin, Cynthia Ju, Elizabeth F. Redente, Kevin S. Choo, Taylor A. Staab, Ronald J. Bouchard, Daniel T. Merrick, Alvin M. Malkinson and Lori D. Dwyer-Nield -- B. Macrophages in Infections -- Phenotypic diversity and emerging new tools to study macrophage activation in bacterial infectious diseases / Mignane B. Ka, Aurélie Daumas, Julien Textoris and Jean-Louis Mege -- The role of myeloid cell activation and arginine metabolism in the pathogenesis of virus-induced diseases / Kristina S. Burrack and Thomas E. Morrison -- C. Macrophage Responses in Atherosclerosis and Other Non-Pathogen-Induced Inflammatory Conditions-- Classical and alternative activation and metalloproteinase expression occurs in foam cell macrophages in male and female ApoE null mice in the absence of T and B lymphocytes / Elaine Mo Hayes, Aikaterini Tsaousi, Karina Di Gregoli, S. Rhiannon Jenkinson, Andrew R. Bond, Jason L. Johnson, Laura Bevan, Anita C. Thomas and Andrew C. Newby-- Cholesterol efflux pathways regulate myelopoiesis: a potential link to altered macrophage function in atherosclerosis / Andrew James Murphy, Dragana Dragoljevic and Alan Richard Tall -- Dynamic aspects of macrophage polarization during atherosclerosis progression and regression / Michael Peled and Edward A. Fisher -- Functions of arginase isoforms in macrophage inflammatory responses: impact on cardiovascular diseases and metabolic disorders / Zhihong Yang and Xiu-Fen Ming -- Role of alveolar macrophages in chronic obstructive pulmonary disease / Ross Vlahos and Steven Bozinovski -- Macrophage polarization in obesity and type 2 diabetes: weighing down our understanding of macrophage function? / Michael James Kraakman, Andrew James Murphy, Karin Jandeleit-Dahm and Hélène L. Kammoun -- Are “resting” microglia more “M2”? / Jonathan D. Cherry, John A. Olschowka and M. Kerry O’ Banion -- M1/M2 macrophage polarity in normal and complicated pregnancy / Mary B. Brown, Maria von Chamier, Ayman B. Allam and Leticia Reyes -- Ontogeny and polarization of macrophages in inflammation: blood monocytes versus tissue macrophages / Adwitia Dey, Joselyn Allen and Pamela A. Hankey-Giblin -- Frontiers in Immunology March 2015 M1/M2 Macrophages: The Arginine Fork in the Road to Health and Disease -- Regulation of Macrophage Differentiation and Functions -- Molecular mechanisms that influence the macrophage M1–M2 polarization balance / Nan Wang, Hongwei Liang and Ke Zen -- SOCS proteins in macrophage polarization and function / Heather M. Wilson -- Myeloid colony-stimulating factors as regulators of macrophage polarization / Thomas A. Hamilton, Chenyang Zhao, Paul G. Pavicic Jr. and Shyamasree Datta -- Regulation of macrophage polarization by RON receptor tyrosine kinase signalling / Amitabha Chaudhuri -- Complement, C1q, and C1q-related molecules regulate macrophage polarization / Suzanne S. Bohlson, Sean D. O’Conner, Holly Jo Hulsebus, Minh-Minh Ho and Deborah A. Fraser -- Purinergic and calcium signalling in macrophage function and plasticity / Bimal N. Desai and Norbert Leitinger -- Nitric oxide synthase: non-canonical expression patterns / Joshua T. Mattila and

Anita C. Thomas -- Metabolic reprograming in macrophage polarization -- References.

Sommario/riassunto

Macrophages have unique and diverse functions necessary for survival. And, in humans (and other species), they are the most abundant leukocytes in tissues. The Innate functions of macrophages that are best known are their unusual ability to either “Kill” or “Repair”. Since killing is a destructive process and repair is a constructive process, it was stupefying how one cell could exhibit these 2 polar – opposite functions. However, in the late 1980’s, it was shown that macrophages have a unique ability to enzymatically metabolize Arginine to Nitric Oxide (NO, a gaseous non – specific killer molecule) or to Ornithine (a precursor of polyamines and collagen for repair). The dual Arginine metabolic capacity of macrophages provided a functional explanation for their ability to kill or repair. Macrophages predominantly producing NO are called M1 and those producing Ornithine are called M2. M1 and M2 – dominant responses occur in lower vertebrates, and in T cell deficient vertebrates being directly driven by Damage and Pathogen Associated Molecular Patterns (DAMP and PAMP). Thus, M1 and M2 are Innate responses that protect the host without Adaptive Immunity. In turn, M1/M2 is supplanting previous models in which T cells were necessary to “activate” or “alternatively activate” macrophages (the Th1/Th2 paradigm). M1 and M2 macrophages were named such because of the additional key findings that these macrophages stimulate Th1 and Th2 – like responses, respectively. So, in addition to their unique ability to kill or repair, macrophages also govern Adaptive Immunity.All of the foregoing would be less important if M1 or M2 – dominant responses were not observed in disease. But, they are. The best example to date is the predominance of M2 macrophages in human tumors where they act like wound repair macrophages and actively promote growth. More generally, humans have become M2 – dominant because sanitation, antibiotics and vaccines have lessened M1 responses. And, M2 dominance seems the cause of ever - increasing allergies in developed countries. Obesity represents a new and different circumstance. Surfeit energy (e.g., lipoproteins) causes monocytes to become M1 dominant in the vessel walls causing plaques. Because M1 or M2 dominant responses are clearly causative in many modern diseases, there is great potential in developing the means to selectively stimulate (or inhibit) either M1 or M2 responses to kill or repair, or to stimulate Th1 or Th2 responses, depending on the circumstance. The contributions here are meant to describe diseases of M1 or M2 dominance, and promising new methodologies to modulate the fungible metabolic machinery of macrophages for better health.