

1. Record Nr.	UNINA9910715034203321
Autore	Osman Borhan
Titolo	A negotiated end to the Afghan conflict : the Taliban's perspective // Borhan Osman
Pubbl/distr/stampa	Washington, DC : , : United States Institute of Peace, , 2018 ©2018
Descrizione fisica	1 online resource (26 pages) : one illustration
Collana	Peaceworks ; ; no. 137
Soggetti	Peace-building - Afghanistan - Public opinion Public opinion - Afghanistan Diplomatic negotiations in international disputes - Public opinion Afghanistan Politics and government 2001-2021 Afghanistan
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Nota di bibliografia	Includes bibliographical references (page 26).

2. Record Nr.	UNINA9910416108503321
Autore	Kim Cheorl-Ho
Titolo	GM3 Signaling / / by Cheorl-Ho Kim
Pubbl/distr/stampa	Singapore : , : Springer Nature Singapore : , : Imprint : Springer, , 2020
ISBN	981-15-5652-0
Edizione	[1st ed. 2020.]
Descrizione fisica	1 online resource (VII, 138 p. 32 illus., 26 illus. in color.)
Disciplina	378.1662
Soggetti	Molecular genetics Cancer Genetics Biochemistry Biological transport Cell membranes Molecular Genetics Cancer Biology Genetics and Genomics Membrane Trafficking
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Nota di contenuto	1. History of sialic acids, gangliosides and GM3 -- 2. Synthesis of GM3 -- 3. Molecular localization of GM3 in cells -- 4. Basic function of GM3 as an interacting molecule -- 5. GD3 mimetics with a neurite forming capacity -- 6. GM3 as a pathogenic infection receptor -- 7. GM3 and related gangliosides prevent inflammation and atherosclerosis -- 8. GM3 has an anti-tumor capacity -- 9. GM3 suppresses tumor angiogenesis -- 10. Interaction between EGFR and GM3 -- 11. Membrane ganglioside-specific neuraminidase 3 (NEU3) regulates GM3 signaling -- 12. Regulation of GM3-mediated EGFR signaling by NEU3 sialidase -- 13. VEGFR-GM3 interaction in angiogenesis -- 14. GM3, competing with GM1, interaction with urokinase plasminogen activator receptor (uPAR) in endothelial caveolar-lipid rafts inhibits angiogenesis -- 15. GM3 interacts with TGF Rs in the epithelial-mesenchymal transition (EMT) during posterior capsular opacification (PCO) formation

-- 16. Galectin-1 promotes tumor growth and escapes immune surveillance -- 17. GM3-HGFR, FGFR and PDGFR cancer cell behavior, and IGF-1R in diabetic wound healing -- 18. GM3, caveolin-1 and insulin receptor in insulin resistance -- 19. GM3 suppresses arthritis -- 20. GM3 protects cochlear hair cells and hearing from corti degeneration -- 21. GM3 increases osteoclast differentiation via direct GM3 cooperation with RANKL and IGF-1 -- 22. GM3 in leukemic cells into terminal differentiation -- 23. 2,3-Sialyllactose (3SL) or 2,6-sialyllactose (6SL) of GM3 glycan in innate immunity. .

Sommario/riassunto

This book reviews recent progress in understanding of the signaling and biochemistry of GM3 ganglioside in eukaryotic cells. GM3 is the simplest of the gangliosides and the precursor of other gangliosides. It is expressed in the outer leaflet of plasma cell membranes and has roles in the recognition, interaction, binding, adhesion, and motility of cells. In addition, GM3 has been documented to have functional roles in cell migration, proliferation, senescence, and apoptosis. The full range of topics of interest are addressed in the book. The early chapters discuss the synthesis of GM3, its molecular localization in cells, and its basic function as an interacting molecule. The ways in which GM3 exerts its effects via various growth factor receptors are fully explored. Current knowledge of the part played by GM3 in health and disease is discussed in depth. For example, its roles in preventing inflammation, inhibiting tumor angiogenesis and tumor growth, and suppressing arthritis are highlighted, and attention drawn to the significance of GM3 as a driver of impaired wound healing in diabetics. The book will be of interest to all who want a comprehensive update on research in this field.
