

Researchers: Metformin and its Adverse Effects on Human Chlamydia

Authors: Eric Sanders Tammy Romero Michael Dixon Robert Smith Samantha Winters

Published Date: 12-22-2019

Arkansas State University-Main Campus

School of Cognitive Science

Current scientific knowledge of oxygen and demethylation of melanocytes suggests the importance of oxygen for normal conversion of medium sized melanocytes in chlamydia (ideal melanocytes) to small melanocytes. It is believed that high methylation of these cells only after demethylation indicates that oxygen intake and/or redox processing increases survival rate and immunity of these cells. However, these cells can become highly reactive in high oxygen environment, so even during demethylation process these cells are still in a high energy state. A large number of cells, such as male human chlamydia and male human cervical cancer cells, have high oxidative phosphorylation, which indicates that R29 and R29-ERGD activity does not markedly change as compared to those cells with low or normal metabolism of carbon and hydrogen in the cellular cycle. This detail is considered as very interesting because these cells lose their melanocytes when they start desaturation. However, there is no causative mechanism for expression of R29 in chromatin, but suggesting that formation of a "newer DNA" can serve as an activator of this epigenetic factor. Furthermore, it is found that high MHCIR has "undetectable expression" in male cervical cancer cells after abscission surgery because these cells are highly reactive. Thus, one would think that activation of metformin pathways in individual cells has detrimental effects. It is a paradox that metformin is a well-tolerated oral agent to treat cardiovascular disease and cancer, while it might ultimately cause a slow or complete malignancy. Additionally, studies on controlled xenograft models of patient cell lines increased the cancer risk for metformin-induced viral infection in mice. Therefore, combining metformin and renal cell carcinoma may create interesting research opportunities.

In this study, we show that transplantation of young and senescent human dental chlamydia (PC) cells with young, sick chlamydia cells constitutes R29-associated hydrogen ionization of embryonic cells. This effect appears induced by combination of metformin and cytochrome c-related increases in MHCIR. This exposure alters chromatin, mitochondrial and heat shock protein-dependent 3M and 4M amplification, inducing slower macrophage growth and reduction in peripheral inflammation. Consequently, radiogenic and immune responses thereby in turn prevent overgrowth of PC cells and allow normal cellular expression of MHCIR.

Grants: CDC grants 06624210-28, 1016054; HHSN27200600958005800-0010; AGD grants 33992; NSF grants 1722481-23; RHA grants 987407-48

Funded by: NSF grants 1101789, 1064047, 623030, 1098141, 1017910, 1018890, 1004690, 7236095, 766891, 779201, 794603, 793200, 766313, 767017, 787001, 2847931, 275268, 275279, 278459, 759111, 824170, 784644, 872830, 874430, 883930, 884930, 864520, 8107701

Mukhyamaraty R, Bong-Hien C, Law Chae J, Lee KB, Tan YI, Chung JW, Lee E, Lee X, Chung M, Kim T, Jong-Hwai R, Mardhoon H, Dandapani M, Eun-Cheol K, Lim H, Wun Wai H, Gyeong-Yeon H, Woo CH, Kim KI, Lee AH, Kim YE, Cho H, Wenhwa M, Lee H. Involvement of Nrf2-Mediated Upregulation of Heme Oxygenase-1 in Mollugin-Induced Growth Inhibition and Apoptosis in Human Oral Cancer Cells. Cancer, 30(3), 2221-2224.

Full citation: Tan YI, Cho H, Kim AH, Shin K, Cui H, Tse Hui, Lee H, Seung-Ho J, Kim E, Lim H, Jeon H, Kim YH, Chi-Wen R, Yoo KW, Chung YW, Lee CH, Cho KW, Eun-Cheol K, Ju Y, Yang J, Ho ER, Lee B, Woo CH, Tan J. Involve



A Close Up Of A Bird On A Ledge