Siemens Competition
Math : Science : Technology
Regional Finalist

Names: William Crugnola and Katie Mazalkova
High School: Jericho High School and Valley Stream Central High School
Mentor: Dr. Jodi Evans
Project Title: *Aortic derived mesenchymal stem cells: A novel target for atherosclerosis cessation* (Genetics)

A primary cause of heart disease is atherosclerosis, caused by the buildup of foam cells beneath the endothelium. Macrophage/aortic derived MSCs (mAo's) interaction may promote endothelial foam cell accumulation. The study aims were to investigate the effect and mechanism of the mAo/macrophage interaction. Phagocytosis Assay measured BMMϕ LDL uptake; ELISA quantified pro-inflammatory cytokine TNF-α; Griess test measured NO concentration, and gene assay quantified up-regulation of adhesion molecules that may mediate mAo/macrophage interaction. Using a One-way ANOVA, the ELISA and Griess were deemed statistically significant. In the phagocytosis assay, the mAo/BMMϕ (70% 16+p) had greater phagocytic activity than the BMMϕ alone (17% 16+p), suggesting mAo's promote macrophage LDL phagocytosis. In the ELISA, TNF-α concentration was higher in mAo+BMMϕ (35.413 ng/mL) than in BMMϕ alone (32.26 ng/mL, $p<.05$). In the Griess test, the NO concentration was higher in the mAo+BMMϕ (25.672μM) than in the mAo (6.45μM) and BMMϕ alone (4.76 μM, $p<.05$). Higher TNF- α and NO in the mAo+BMMO suggests mAo's promote M1. E-Selectin (110.47x), P-Selectin (62.10x) and CCL5 (413.33x) were up-regulated by the mAo/macrophage interaction, insinuating their involvement in the mAo/macrophage interaction. Knocking out these genes in a future investigation would verify their role in the mAo/macrophage interaction.