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Infection-Induced Coronary Dysfunction and Systemic Inflammation in Piglets Are Dampened in Hypercholesterolemic Milieu

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Introduction: The synergism of infection with conventional cardiovascular risk factors in early life atherosclerosis is much debated. We hypothesized that coronary arterial injury correlates with infection recurrence and pathogen burden, and is further aggravated by hypercholesterolemia.

Methods: Forty-two Göttingen minipigs were assigned to repeated intra-tracheal inoculation of either saline, Chlamydia pneumoniae (Cpn), or both Cpn and influenza virus at 8, 11, and 14 weeks of age. Animals were fed either standard or 2% cholesterol diet (chol-diet). At 19 weeks of age coronary vasomotor responses to acetylcholine (Ach) and adenosine were assessed in vivo, and blood and tissue samples were collected. Nonparametric tests were used to compare the groups.

Results: In cholesterol fed animals, total cholesterol/HDL was significantly increased in infected animals ($P = 0.01$ vs. non-infected). C-reactive protein (CRP) rose in infected animals ($P < 0.01$ vs. non-infected) without significant difference between the mono- and co-infected groups. Among co-infected animals, both CRP and haptoglobin were lower in those fed chol-diet compared to animals fed standard diet ($P < 0.05$). The vasoconstricting response to Ach was most prominent in co-infected animals ($P = 0.03$ vs. non-infected, and $P = 0.07$ vs. mono-infected). Among mono-infected animals, similar to CRP, a trend for less vasoconstriction was observed in those fed chol-diet ($P = 0.08$).

Conclusion: Co-infection of piglets appears to be associated with more pronounced coronary muscarinic vasomotor dysfunction. In mono-infected animals, use of chol-diet seems to dampen both coronary dysfunction and systemic inflammation induced by infection.