Abstract: Recent evidence demonstrates a novel metabolic syndrome among HIV-infected patients, including altered lipid metabolism, substrate flux, fat distribution, and insulin resistance. Increased visceral adiposity and subcutaneous fat atrophy are most prominent. Significant progress has been made in recent years to understand this syndrome. Increased lipolysis and flux of fatty acids to the liver and muscle contribute to insulin resistance. Protease inhibitors have been shown to affect PPAR signaling, adipocyte differentiation, and apoptosis. At the same time, NRTIs, through inhibition of mitochondrial DNA polymerase gamma, impair fatty acid oxidation, resulting in lipid accumulation in both the liver and muscle and repartitioning away from the subcutaneous compartment. Furthermore, reduction in critical cytokines, including adiponectin, impairs fatty acid oxidation.

Recent data suggest significant clinical consequences of insulin resistance in HIV-infected patients, including increased cardiovascular disease. Recent data also suggest potentially beneficial effects of insulin sensitizing agents, such as the thiazolidinediones, to increase adiponectin and subcutaneous adipogenesis while inhibiting lipolysis and reducing hepatic and intramuscular fat accumulation. The HIV lipodystrophy syndrome is a novel metabolic syndrome in which to understand the mechanisms of insulin resistance and fat redistribution. The proposed symposium will cover critical pathophysiologic mechanisms of altered lipid metabolism and nutrient trafficking, highlighting the most recent clinical and molecular data on the effects of protease inhibitors and nucleoside reverse transcriptase inhibitors on these processes.

PATHOLOGICAL CALCIFICATION: CRYSTALLIZATION, INFECTION, OR CELL TRANSDIFFERENTIATION

Sunday, April 2, 2006 – 3:15 to 5:15 pm

Room 274/276 – Moscone Convention Center

Organizer Name: Virginia M. Miller, PhD
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Abstract: It is becoming increasingly clear that the pathophysiology of calcification within diseased human tissues is complex. Inflammation appears to be associated with many calcific processes, including advanced atherosclerosis, nephrolithiasis, and calciphylaxis of end-stage renal disease. In all three instances characteristic cellular responses could potentially either mediate or ameliorate the calcific response. Increasingly, microorganisms are being identified as an unexpected cause of disease, with a recent well-known example being the association of *H. pylori* and peptic ulcer. The possibility that microorganisms contribute to pathologic calcification and the associated inflammation is, however, controversial. This symposium will examine evidence for three possible but not mutually exclusive mechanisms of pathologic calcification: (1) processes that drive and influence *inorganic* crystallization; (2) the mechanisms by which cells mediate crystallization, including cell transdifferentiation; and (3) evidence for the presence of calcifying microorganisms within diseased human kidneys and arteries. The program will conclude with a panel discussion led by a moderator who will comment on divergent points from the perspective of a pathologist.