Presentation Key Highlights
Coronary Heart Disease, Atherosclerosis and Applied Clinical Vascular Biology

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Day one. 1.30-3.20:

- There are no diseases only processes. If you can understand the process you can understand all diseases and the common, underlying denominators.
- Vascular disease is the result of an imbalance between vascular injury and vascular repair. There are three finite vascular responses to an infinite number of insults. These are: oxidative stress, inflammation and immune dysfunction.
- Endothelial dysfunction (ED) is the earliest event in vascular disease that eventually leads to functional and structural changes within the blood vessels. ED precedes atherosclerosis and cardiovascular events by decades, making early detection critical for prevention.
- Treatments are required to:
  - Increase eNOS and nitric oxide (NO): through its ability to increase the intracellular signalling molecule cyclic AMP (cAMP), NO is our primary endogenous vasodilator and possesses an important regulatory function in maintaining the health of the endothelium. NO can be increased via boosting endogenous production such as through citrulline or L-arginine supplementation (the precursor to NO) and providing co-factors and co-substrates for its production (e.g. vitamin B2, B6, folate, thiols etc.). It can also be increased through exogenous intake from dietary nitrates/nitrates (e.g. dark green, leafy vegetables).
  - Increasing NO availability: The enzyme asymmetric di-methyl arginine (ADMA) competes with arginine, thereby reducing NO. ADMA is increased by elevated homocysteine, obesity, insulin resistance, inflammation, native or oxidised LDL. Thus a spiralling effect occurs with high endothelial LDL levels causing greater ADMA values, which in turn inhibit NO production needed to promote vasodilation.
  - Reducing Endothelin (ET-1), Angiotensins and Aldosterone: Endothelin is a precursor to Angiotensin-II which is a potent vasoconstrictor, thrombogenic, pro-oxidant, pro-inflammatory hormone. Aldosterone mimics the effects of angiotensin-II.
  - Balance oxidative stress and antioxidant defences: Oxidative stress with inadequate oxidative defences amplifies ED via converting NO into a more-potent free radical – peroxynitrate. What’s more increased reactive oxygen species within the endothelium, triggers the production of vascular adhesion molecule, induces inflammation and oxidises LDL, triggering atherosclerotic plaque formation.
  - Reduce overactive immune system activation and autoimmune responses: reduce endothelial activation by leukocyte recruitment and inflammatory cytokines such as with NAC, GSH, thiols, sulfhydryls, omega-3, phytonutrients and through increasing NO.
- Understand and treat the mechanism, the processes and the “why”, not the manifestation. Emphasis should be on the pathophysiology of vascular disease and addressing the metabolic and functional medicine causes.
- A summary of prevention and treatment options for ED and CVD are provided within the manual, pages 61-63.

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Dr. Mark Houston has an extensive clinical and academic background in chemistry, internal medicine and human nutrition and currently holds a wealth of highly esteemed positions, including Associate Clinical Professor of Medicine, Vanderbilt University School of Medicine and Director of the Hypertension Institute and Vascular Biology. Dr. Houston specialises in hypertension, lipid disorders, prevention and treatment of cardiovascular diseases, nutrition and age management. The Consumer Research Council has four times in recent years selected Dr. Houston as the Outstanding Physician in Hypertension in the U.S.