The hypothesis of this PhD programme was based on assumption that vitamin D is involved in the regulation of EF via NO production. It is well established that NO initiates and maintains vasodilation in the endothelium, and vitamin D may have an important role in regulating eNOS activity, which enhances the availability of NO. Vitamin D involved in vascular smooth muscle (VSM) function by modulating the regulation of growth and proliferation of VSM cells.

Fat mass and ageing also modulates of these physiological processes. Individuals with excess body weight tend to have lower vitamin D concentrations - possibly due to the sequestration of 25OHD in adipose tissue and/or volumetric dilution of 25OHD. Ageing is associated with decreased NO generation mediated by the reduction of the eNOS activity. This will lead to endothelial cell senescence due to less responsive of hemodynamic shear stress. As vessels age, blood flow were reduced as resulted by less infectiveness of heart function. Furthermore, as people aged, the endothelial cells become less responsive to shear stress, resulting in a reduction of NO generation. I therefore postulated that vitamin D status is associated positively with EF and with biomarkers of NO activity. In addition, I postulated that fat mass is a significant confounder of this association with a greater association seen in lean compared to obese individuals. Similarly, I predict that ageing was a significant confounding factor between vitamin D, EF and stronger associations being observed in older compared to younger individuals. Further analyses also explored whether the associations between ageing, vitamin D and whole body NO production.