77

## Southern Chinese patients with systemic lupus erythematosus in Hong Kong have low vitamin D levels

MY Mok[1], TJ Yao[2], Y Lo[1], S Tam[3]
[1]Department of Medicine, Queen Mary Hospital, The University of Hong Kong, Hong Kong
[2]Clinical Trials Centre, The University of Hong Kong, Hong Kong
[3]Departments of Pathology and Clinical Biochemistry, Queen Mary Hospital, The University of Hong Kong, Hong Kong

**Background:** Vitamin D insufficiency has been linked to pathogenesis of autoimmune diseases. This study aimed to measure serum 25(OH)D level in patients with systemic lupus erythematosus (SLE) in Hong Kong and to evaluate association between serum 25(OH)D level and disease activity.

**Methods:** Serum 25(OH)D level was measured by radioimmunoassay in SLE patients and healthy controls. Lupus disease activity was determined by SLE disease activity index (SLEDAI), serum anti-dsDNA antibody, C3 and C4 levels.

**Results:** Fifty-two SLE patients with mean ± standard deviation disease duration of 15.5±8.6 years were recruited. Five patients had active lupus disease. Five (9.6%) patients had serum 25(OH)D levels <30 nmol/L. Serum 25(OH)D level was significantly lower in SLE patients compared to age- and sex-matched controls (n=52) [45.5±12.3 vs 51.1±12.6 nmol/L, P=0.02]. Serum 25(OH)D levels were not found to be related to SLEDAI, elevated anti-dsDNA antibody, low C3 or C4 levels or medications. One vitamin D insufficient patient had low serum albumin-corrected calcium. Serum 25(OH)D levels were found to correlate negatively with estimated glomerular filtration rate ($r$= –0.30, P=0.03) but was not different between patients who had normal and impaired renal function (P=0.38).

**Conclusion:** SLE patients in Hong Kong were found to have low serum 25(OH)D level despite its subtropical location.

78

## Functional consequences of overexpressing the gap junction Cx43 in the cardiogenic potential of pluripotent human embryonic stem cells

JC Moore[1,2,3], SY Tsang[2,3], SN Rushing[1,2,3], D Lin[2,3], HF Tse[1,4], CW Chan[1,2,3], RA Li[1,2,3,4]
[1]Stem Cell & Regenerative Medicine Program, HBHA & Department of Medicine, The University of Hong Kong, Hong Kong
[2]Department of Cell Biology, UC Davis, Shriners Hospital, Sacramento, United States
[3]Department of Human Anatomy, UC Davis, Shriners Hospital, Sacramento, United States
[4]Division of Cardiology, Department of Medicine, The University of Hong Kong, Hong Kong

Gap junctions, encoded by the connexin (Cx) multi-gene family, couple adjacent cells and underlie cell-cell communications. Previous mouse studies suggest that Cxs play an important role in development but their role in human cardiogenesis is undefined. Human embryonic stem cells (hESC) provide a unique model for studying human differentiation. Lentivirus-mediated stable overexpression of Cx43 in hESC (Cx43-hESC) did not affect colony morphology, karyotype and expression of pluripotency genes such as Oct4 but completely suppressed the formation of spontaneously beating, cardiomyocyte-containing clusters in embryoid bodies (EBs). Unlike control hEBs, the transcripts of several mesodermal markers (kallikrein, delta-globin, and CMP), ventricular myosin light chain and cardiac troponin I were absent or delayed. Transcriptomic and pathway analyses showed that 194 genes crucial for movement, growth, differentiation and maintenance were differentially expressed in Cx43-hESC. We conclude that Cx43 mediates the expression of an array of genes involved in human cardiogenesis, in addition to intercellular communication.