**Objective**

Iron regulatory hormone hepcidin is possible to be possibly involved in the physiological function of heart and pathogenesis of heart disease. The current paper reports the establishment of heart specific Hepc1 transgenic mice and analysis of the effect of Hepc1 on the development of dilated cardiomyopathy in the cTnTR141W transgenic mice.

**Methods**

The heart specific Hepc1 transgenic mice were generated by the method of microinjection and the target gene was under the control of α-MHC promoter. The expression level of the gene Hepc1 was determined by RT-PCR and slot blotting. The Hepc1 × cTnTR141W double transgenic mice were generated by breeding the Hepc1 transgenic mice with cTnTR141W transgenic mice. The structural and functional changes of the transgenic heart were analysed with M-mode echocardiography. Survival data were recorded from 1 month to 7 months (the 1st to the 7th month) postnatally. The histological and subcellular changes were observed under light microscope and transmission electronic microscope. The accumulation of phos-ERK1/2 and ERK1/2 in wild type, Hepc1, cTnTR141W and Hepc1 × cTnTR141W transgenic mice were determined by Western blotting. Phos-ERK1/2 expression was also detected in H9c2 cell line, H9c2 cell line treated with desferrioxamine (50 μmol/l), and H9c2 cell lines expressing Hepc1 protein and H9c2 cell lines expressing Hepc1 protein treated with ferric ammonium citrate (100 μmol/l).

**Results**

2 heart specific Hepc1 transgenic mice lines were established. Hepc1 transgenic mice demonstrated no signs of cardiomyopathy or lethality up to 7 months of age. The immature death rate was 18% before 7 months of age in the cTnTR141W mice, while Hepc1 × cTnTR141W transgenic mice showed a lower immature death (10%). Hepc1 × cTnTR141W transgenic mice showed a smaller ventricular chamber, improved left ventricular systolic function, and ameliorated interstitial fibrosis, compared with (to) cTnTR141W transgenic mice. ERK1/2 phosphorylation was strongly decreased in cTnTR141W transgenic mice compared with (to) WT mice, while it was significantly increased in the Hepc1 × cTnTR141W transgenic mice compared with (to) cTnTR141W transgenic mice. Both desferrioxamine, an iron chelator and the gene Hepc1 significantly increased ERK1/2 phosphorylation in H9c2 cell line.

**Conclusions**

Hepc1 showed a marked improvement on the pathologic phenotype of DCM in cTnTR141W transgenic mice, in which ERK1/2 signal pathway may play an important role. Hepc1 may be a new target in the treatment of human dilated cardiomyopathy.

**Objective**

Lung cancer is the most common cause of cancer deaths worldwide; it is a complex disease that many genes involved (involves many genes) on chromosomes. Recently, several genome-wide association studies have identified chromosomal region 15q24-25.1 as a hotspot for lung cancer risk. We therefore aimed to explore the association of CHRNA3 gene rs1051730 and rs8034191 polymorphisms of this region with lung cancer via meta-analysis.

**Methods**

Random-effects model was performed irrespective of the between-study heterogeneity. Data and study quality were assessed in duplicate. Publication bias was evaluated using the fail-safe number.

**Results**

Overall, five studies involving nine populations were identified for both rs1051730 (cases: controls = 7594:8784) and rs8034191 (9553:7953) polymorphisms. Genotype frequencies of two polymorphisms satisfied the Hardy–Weinberg law in controls. Presence of rs1051730 A allele was significantly associated with 52% increased risk of lung cancer (95% CI 1.25 to 1.39; p < 0.00001). Contrastingly, rs8034191 C allele only conferred a marginal association yielding 18% increased risk (95% CI 0.99 to 1.42; p = 0.07). Under assumption of dominant and recessive modes, risk magnitude was strengthened for rs1051730 with an increased risk of 39% (95% CI 1.50 to 1.48; p < 0.00001) and 48% (95% CI 1.25 to 1.76; p < 0.00001) for lung cancer, respectively. As for rs8034191, there was marginal or null association for the dominant (pooled OR =1.28; p = 0.06) and recessive (pooled OR =1.26; p = 0.15) modes. The fail-safe number at the level of 0.05 supported these significant associations.

**Conclusions**

Our results demonstrated that the rs1051730 A allele was significantly associated with an increased lung cancer risk and the effect of rs8034191 polymorphism was moderate.