**Genetic variations of the bicarbonate secreting anion exchanger SLC26A6 are not associated with chronic pancreatitis**

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**Introduction:** Pancreatic ductal $HCO_3^-$ secretion is critically dependent on the cystic fibrosis transmembrane conductance regulator chloride channel (CFTR) and the solute-linked carrier 26 member 6 anion transporter (SLC26A6). Deterioration of $HCO_3^-$ secretion is observed in chronic pancreatitis (CP), and *CFTR* mutations increase CP risk.

**Aims:** Therefore, *SLC26A6* is a reasonable candidate for a CP susceptibility gene, which has not been investigated in CP patients so far.

**Patients & methods:** As a discovery cohort, 96 subjects with CP and 99 control subjects with no pancreatic disease were recruited from the Hungarian National Pancreas Registry. In 30 non-alcoholic CP cases the entire *SLC26A6* coding region was sequenced. Variants c.616G>A (p.V206M) and c.1191C>A (p.P397=) were further genotyped by restriction fragment length polymorphism analysis. A German replication cohort of 321 CP cases and 171 controls was analyzed by sequencing.

**Results:** Sequencing of the *SLC26A6* coding region revealed four common variants: intronic variants c.23+71_103del, c.183-4C>A, c.1134+32C>A, and missense variant c.616G>A (p.V206M) which were found in linkage disequilibrium indicating a conserved haplotype. The distribution of the haplotype did not show a significant difference between patients and controls in the two cohorts. A synonymous variant c.1191C>A (p.P397=) was detected in a single case.

**Conclusion:** Our data show that *SLC26A6* variants do not alter the risk for the development of CP.