The genotype and allelic frequencies and carriage rates of five investigated single nucleotide polymorphisms (SNPs) can be defined as

\[
PJI = G_A 0.31\ G Interleukin CC A G Healthy Controls 0.55 0.85 0.85 correction after TJA were involved into the study Nucleotide 0.38 C G GA (SNPs) 0.25 0.52 0.83 0.04 0.72 A GENE POLYMORPHISM WITH PROSTHETIC JOINT INFECTION T the TJA patients with PJI A to pathogens 0.21 G G 0.09 without TJA. All patients/controls 1 0.17 GA G TJA without PJI of AA 0.31 G 0.91 0.56 A, Mr genes A 0.81 Single 0.44 CC using A 0.39 GG G 0.46 GA 0.91 GC T T groups G G 0.33 0.2 0.32 AA C G 0.84 variants 0.04 GG 0.96 0.07 for development is determined by comparison with the patients 0.84 G located A between T

Cytokines & PJI

Proinflammatory cytokines play an important role namely in innate immune response to pathogens potentially causing PJI.

Production and regulation of cytokines is affected by functional variants (polymorphisms) of cytokine genes.

We hypothesized that individual susceptibility /resistance to PJI development is determined by specific combinations of polymorphic markers in cytokine genes.

**Aim**

To determine whether functional gene polymorphisms in proinflammatory cytokines are associated with PJI after TJA.

**Results**

Distribution of genotypes for all investigated polymorphisms conformed to Hardy–Weinberg equilibrium (HWE) using the chi-squared test.

The frequencies of variants were compared between the patients with PJI and both control groups using the standard chi-squared test with Bonferroni correction.

We nominate a functional variant in the gene encoding cytokine IL-1beta as a possible genetic factor which may contribute to susceptibility to PJI in Czech population.

Our data are of preliminary character and should be independently replicated; possible functional role of IL1B-511 gene variant in PJI remains to be elucidated.

**Patients and Methods**

**Study groups:**

Eighty nine patients with PJI after TJA were involved into the study together with two control groups: 1) TJA control - 214 patients with TJA that did not develop PJI at least 6 yrs. after the surgery, and 2) population control - 168 healthy control subjects without TJA. All patients/controls were unrelated and of Czech ethnicity.

**Genotyping:**

Five Single Nucleotide Polymorphisms (SNPs) located in the genes for:

- *Interleukin-1beta* (IL1B-511)
- *Tumour necrosis factor alpha* (TNF-308, -238)
- *Interleukin-6* (IL6-174, nt565)

Genotyping was performed by PCR–SSP using the “Heidelberg” kit.

**Statistical methods:**

Distribution of genotypes was tested for conformity with Hardy–Weinberg equilibrium (HWE) using the chi-squared test.

The frequencies of variants were compared between the patients with PJI and both control groups using the standard chi-squared test with Bonferroni correction.

**Conclusion**

We nominate a functional variant in the gene encoding cytokine IL-1beta as a possible genetic factor which may contribute to susceptibility to PJI in Czech population.

Our data are of preliminary character and should be independently replicated; possible functional role of IL1B-511 gene variant in PJI remains to be elucidated.