Delayed union of femoral fractures in older rats:decreased gene expression

ABSTRACT

The fracture was found to up-regulate all genes in both age groups. This meant that the older rats did not heal quickly, and it may be due to the lack of expression of these genes. However, the mRNA gene expression returned to normal levels before healing took place in the elderly rats, which may explain why they didn't union earlier. No genes were overly upregulated in them. The older rats did not show a positive-feedback increase in the mRNA expression of stimulatory cytokines, despite their slower healing response.

INTRODUCTION

Type 1 diabetes mellitus (T1DM) is a disease that involves multiple organs, including humans. It has an important genetic component, but it is predominantly caused by the MHC located on the short arm of chromosome 6, which is also affected by several other inherited phenotypes. A range of methods has been employed to pinpoint T1DM susceptibility regions, including case-control studies of candidate genes, combined linkage and association-based studies, and systematic total genome searches in addition to analyses of individual chromosomal regions. Immunogenetic predisposition to T1DM differs markedly from country to country, and disease incidence also appears to vary along with these discrepancies. For instance, the incidence of T01DM is comparable in Southern India (10.4/100000 cases per year) to the number of cases reported in Asian children in the UK and white children of European descent. The presence of an MHC component in T1DM susceptibility is apparent in Southern India, but no correlation has been observed with the insulin gene or ILIR1 in case-control studies. This implies that there may be differences in the non-MHC T1/DM component between Southern Indians and Caucasians of European descent. An association with the insulin gene has been reported universally in the latter population, and some Northern Europeans have reported an IL1R1 association to T1DM. Allelic variation in VDR also increases the susceptibility of Indian Asians, Germans and Taiwanese to T1DM. In the VDR locus, there are six known polymorphisms: FokI restriction enzyme detects an initiation codon polymer (exon 2), BsmI, Tru9I and ApaI ("reflection fragment length polymers") between exons 8 and 9 in the vector flyer type (VDR) loci, and a poly A polyphenystym downstream of the 3' untranslated region. The FokI polymorphism does not seem to have a significant impact on the BsmI, ApaI and TaqI (the three major immune depressive genes): in Japanese patients with T1DM, we studied the exon 2 initiation codon (VDR-FokI) gene polyfomi and found no association with GAD65 antibody (Ab) status.

CONCLUSION

In vivo experiments have revealed an osmotic stress-dependent serine phosphorylation of the eukaryotic histidine kinase homologue DokA. The phosphatation is not dependent on the conserved histoidine residue, which is crucial for two-component systems and is unlikely to occur through autophosphorousion. This supports the notion that eukaryotic homologues of bacterial signal transduction systems could be involved in serine/threonine kinases-related signaling pathways.