Autoinflammatory diseases are a group of disorders characterized by seemingly unprovoked inflammation in the absence of high-titer autoantibodies or antigen-specific T cells. Familial Mediterranean fever (FMF) is an autosomal recessive disorder and the archetypal autoinflammatory disease. It is characterized by recurrent self-limiting episodes of fever and painful polyserositis. FMF is prevalent in specific ethnic groups namely, non-Ashkenazi Jews, Armenians, Turks, and Arabs. The gene responsible for FMF, *MEFV*, was identified in 1997.

There seems to be a distinctive clinical picture in Arab patients with FMF, and the range and distribution of *MEFV* mutations is different from that noted in other commonly affected ethnic groups.

The aim of this study was to delineate the distribution of *MEFV* mutations amongst an Arabic FMF patient cohort and to assist the genotype-phenotype correlation in these patients. We collected DNA samples from 406 FMF patients (from Qatar, Jordan, Algeria and Palestine) who have been clinically diagnosed with FMF. We designed primers to cover the entire genomic region of *MEFV*. Mutation detection is done by resequencing the entire coding sequence and splice sites then the rest of the genomic region and the promoter will be sequenced as a second tier.

So far we have identified 283 (out of 676) mutant alleles by sequencing exon 10, the main hot spot for *MEFV* mutations (M694V, V726A, M694I, M680IGC, M680IGA, R653H, A744S and R761H). In addition, four novel variations were identified in our cohort in exons 3, 5, 2 & 10, and we are currently investigating the phenotypic significance of these novel variations.

The spectrum of *MEFV* mutations in Arabs seems different from other ethnic groups commonly affected by FMF. The identifiable disease causing alleles are the lowest amongst the commonly affected ethnic groups. The low number of identified alleles suggests the presence of mutations within unexamined regions, such as conserved intronic sequences or the involvement of modifier genes.