haplotypic frequency of *HLA-G -727C 1074A 1597C 3741_3754ins14 3775C* was higher in the BD patients (p = 0.046) than in the controls (Table I).

HLA-G 3741_3754ins14 variant leads to splice out the 92-bp of exon 8 in 3'-UTR and induces a significantly lower mRNA level and a lower concentration of soluble HLA-G than the corresponding HLA-G mRNA isoform with the deleted 14bp (10).

In conclusion, the *HLA-G 3741_3754ins14* variant and the haplotype carrying this variant are associated with an increased risk for BD. These results suggest that the presence of the HLA-G *3741_3754ins14* variant which is related with lower mRNA level is probably associated with the development of BD.

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A molecular basis for the absence of familial Mediterranean fever in Ethiopian Jews

Sirs,

Familial Mediterranean fever (FMF) is the most prevalent hereditary inflammatory disorder (1). Four ethnic groups are classically affected: Armenians, Arabs, Turks and Jews. However, FMF is not equally shared among Jews. Non Ashkenazi Jews, i.e. Sefardi Jews, are far more affected than Jews from Europe.

Ethiopian Jews have been arriving in Israel in ever increasing numbers since around 1979. Today more than 50,000 Ethiopian Jews are living in Israel (2). In our experience, as well as in the English literature, no case of FMF has been reported in this community

In the present study, we have searched for MEFV mutations (3, 4) in 95 unrelated unaffected Ethiopian Jews.

Ninety five adult Jewish subjects from Ethiopian ancestry (44 males, 51 females) were enrolled. All of them arrived in Israel during the last five years and were living in two immigration centers in the north of Israel. There is no identified Christian Ethiopian community in Israel so we could not include a control group of "non Jewish Ethiopian population" in our study. The subjects had no special medical history except for infections: one active pulmonary tuberculosis, one hepatitis B infection with cirrhosis and one HIV positive. The study received Helsinki committee approval and informed consent in Hebrew and Ethiopian language was obtained from every participant.

No subject was found to be either homozygous or compound heterozygous at the MEFV locus. One mutated allele was identified in 14 individuals. We found none of the most penetrant/severe mutations associated with FMF, i.e. at codons 694 and 680, nor other mutations in exon 10. A common polymorphism was identified, however (P706, 1%). In contrast, we detected a relatively high rate of mutations E148Q (6%), and P369S (1%) (Table I).

Our study suggests a molecular basis for the absence of FMF in Ethiopian Jews.

The present report may contribute to the persistent debate of whether E148Q is a polymorphism or a true mutation (5-7). Recent studies have suggested that this mutation/polymorphism may enhance inflammation non-specifically, of possible advantage during evolution (8).

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Table I. MEFV sequence variants in 95 unaffected Ethiopian Jews.

	N	%	95%CI
E148Q	11	6%	0.03-0.09
P369S	2	1%	0.001-0.03
P706*	1	0.5%	0.001-0.02
Mutations in exon 10	0	0%	0-0.02
All mutations*	14	7.5%	0.04-0.11
Carrier estimation		14%	0.09-0.19
Genetic FMF estimation		0.5%	0.001-0.02

* P706 is considered a polymorphism