Materials and Methods We present data from the ENEStnd study. In ENEStnd, a phase 3, multicentre, open-label, randomised study, patients treated with nilotinib demonstrated higher and faster rates of major molecular response (MMR), more profound molecular response (MR), and complete cytogenetic responses (CCyR) compared with imatinib by 12 and 24 months. 282 adult patients were randomly assigned to receive nilotinib 500 mg twice daily, 281 to receive nilotinib 400 mg twice daily and 283 to receive imatinib. Patients were eligible if they had been diagnosed with chronic phase, Philadelphia chromosome-positive CML within the previous 6 months.

Results By 24 months after the start of treatment, significantly more patients had a MMR with nilotinib than with imatinib (201 with nilotinib 500 mg twice daily, 187 with nilotinib 400 mg twice daily and 124 with imatinib; p < 0.0001 for both comparisons). Significantly more patients in the nilotinib groups achieved a complete molecular response at any time than did those in the imatinib group (74 with nilotinib 500 mg twice daily, 59 with nilotinib 400 mg twice daily and 29 with imatinib; p < 0.0001 for nilotinib 500 mg twice daily vs. imatinib, p = 0.0004 for nilotinib 400 mg twice daily vs. imatinib).

Conclusions Nilotinib continues to demonstrate superiority over imatinib with faster and more profound molecular responses. These results support nilotinib as a first-line treatment option for patients with newly diagnosed Philadelphia chromosome-positive and chronic myeloid leukaemia.

No conflict of interest.

Results The study included a total of 40 patients. The mean age of the patients was 38.66 ± 13.98 years and 61.1% were female. The distribution for genotypes was 81.6% AA, 15.8% GA and 2.6% GG. Significant correlation wasn’t found between genotypes or alleles of this polymorphism and clinical response to infliximab. Instead, statistically significant differences were shown for approximately 6 months of treatment when comparing patients with genotypes GG and GA/AA and a positive response (p = 0.047) when considering the biological response. Similarly patients with a G allele had a more frequent negative response than those with the A allele (p = 0.045). On the other hand, significant correlation was found between patients carrying the A allele and the positive response, at 3, 6 and 12 months based on biological response distribution.

Conclusions The results of this study show an association of this polymorphism with response to infliximab. Worst response rates are observed in patients carrying allele G diagnosed with CD. We need more studies on this polymorphism and with a larger sample size to confirm these findings.

No conflict of interest.

Background Sunitinib (SU) is an oral, small-molecule, multitargeted tyrosine kinase receptor inhibitor that is approved for the treatment of renal cell carcinoma (RCC). However, several patients either do not respond to treatment, or they do, but they experience significant toxicity.

Purpose To find genetic markers of toxicity and efficacy using a commercially available DNA microarray genotyping system.

Materials and Methods 25 patients with newly-diagnosed metastatic RCC were evaluated prospectively from January 2010 to May 2011. Patients received SU in repeated 6-week cycles of 50 mg/day orally for 4 weeks, followed by 2 weeks off treatment. A total of 92 single nucleotide polymorphisms (SNPs) in 34 genes in the pharmacokinetic and pharmacodynamic pathways of drugs were analysed using the Drug inCode pharmacogenetic service. This test is performed from a saliva sample and uses a DNA microarray system. Polymorphisms in candidate genes, together with clinical characteristics, were tested by univariate analysis for association with the number of days of sunitinib treatment until the first reduction of dose, progression free survival (PFS) and overall survival (OS).

Results Patients with CYP1A2*1/*1, a low-metabolising genotype, needed dose reduction due to an increased risk of toxicity vs. *1F/*1F or 1F/1F (Median time to dose reduction: 2.33 months vs. not reached during study period; p < 0.006). Patients with CY2C9*1/*1, wild type genotype, had an increased risk of dose reductions due to toxicity versus other genotypes (Median time to dose reduction: 2.8 months vs. 9.73 months; P < 0.021). No statistically significant associations were observed among drug metabolising genes and PFS or OS.

Val(158)Met Catechol-O-methyltransferase (COMT) gene polymorphisms have been associated with PFS and OS. We found that Met/Met carriers, low metabolising allele, had longer PFS and OS compared to those with Met/Val (PFS not reached vs. 15 months; OS not reached vs 17.2 months) and Val/Val (PFS = 3.3 months; OS = 4.4 months) phenotypes (P = 0.005 for PFS and P = 0.005 for OS).