

Supplementary Material: Appendix 2 – Results: Local histopathological and mutational analyses

The results of the local histopathological and mutational analyses are shown in Supplementary Table S2; a total of 300 samples (100%) were available for analysis.

Supplementary Table S2 Summary of findings from local histopathological and mutational analyses

Local histopathological analysis, <i>n</i> (%)		<i>n</i> = 300
Samples available and analysed		300 (100)
c-KIT positive (GIST confirmed; immunohistochemical analysis)		245 (82)
Analysis missing		31 (10)
<i>c-KIT</i> mutational status	Exon 9	8 (3)
	Exon 11	74 (30)
	Exon 13	0 (0)
	Exon 17	5 (2)
	Wild type	2 (1)
	Unknown	139 (57)
c-KIT positivity missing data		2 (1)
c-KIT negative		24 (8)
<i>PDGFRAα</i> mutational status	Exon 12	2 (1)
	Exon 18	12 (4)
Multiple loci mutations		15 (5)

Percentages may not add up to 100 due to rounding

Discussion and conclusions: Local histopathological and mutational analyses

Overall, local histopathological and mutation analyses performed in this study showed that the majority of the GIST samples (82%) were c-KIT positive; 8% of the GIST samples were c-KIT negative. The remaining 10% of results were not reported. These findings are in line with published reports of KIT mutations being present in most (>70%) of GISTs [1-3].

With regard to the frequency of specific KIT mutations in GIST samples in the present study, findings from local analysis tend to differ from published reports. The finding of 3% exon 9 mutations, for example, is slightly lower than the published range of 4–17%, while the finding of 30%

exon 11 mutations is much lower than the published range of 57–77% [3]. However, the frequency of mutations in exons 13 and 17 (0–2%) is in line with published reports of these mutations being rare (<2%) [2]. Also, similar to published reports, a small percentage of KIT-negative GISTs with PDGFRA α mutations were found (1–4%) [2].

References

1. Tosoni A, Nicolardi L, Brandes AA (2004) Current clinical management of gastrointestinal stromal tumors. *Expert Rev Anticancer Ther* 4:595–605
2. Corless CL, Fletcher JA, Heinrich M (2004) Biology of gastrointestinal stromal tumors. *J Clin Oncol* 22:3813–3825
3. Heinrich MC, Blanke CD, Druker BJ, Corless CL (2002) Inhibition of KIT tyrosine kinase activity: a novel molecular approach to the treatment of KIT-positive malignancies. *J Clin Oncol* 20:1692–1703