Supplemental Table and Figure Legends

Table S1. Gut adenocarcinoma sample cohort and methods of data generation.

Table S2. Significantly amplified focal SCNAs in a) colorectal, b) gastric, and c) esophageal adenocarcinomas (q-value <0.01).

Table S3. Focal amplifications in gut adenocarcinomas shared with all cancers. Significantly amplified (q-value < 0.01) genes and peaks from the gut adenocarcinoma cohort was compared to 2,311 other cancers not including these tumor types (12).

Table S4. Significantly deleted focal SCNAs in a) colorectal, b) esophageal, and c) gastric adenocarcinomas (q-value <0.01).

Table S5. Focal deletions in gut adenocarcinomas shared with all cancers. Significantly amplified (q-value < 0.01) genes and peaks from the gut adenocarcinoma cohort was compared to 2,311 other cancers not including these tumor types (12).

Table S6. Significantly a) amplified and b) deleted focal SCNAs in a cohort of esophageal, gastric, and colorectal adenocarcinomas from only SNP6.0 arrays (q-value <0.01).

Figure S1. Average levels of focal amplification and deletion along the chromosome arm. Mean GISTIC G-scores were calculated for all chromosome arms scaled to a common length across the samples for each disease-type, as well as the composite dataset. The dashed lines indicate the score corresponding to the False Discovery Rate threshold of 0.01. Lower rates of alteration are seen in interstial regions, but neither telomeric nor centromeric regions were significantly altered on average.

Figure S2. Patterns of chromosomal instability differ across adenocarcinomas of the gastrointestinal tract after removing samples without arm-level alterations. Numbers of a) focal and b) arm-level SCNAs following removal of 11 EA, 14 GC, and 37 CRC samples without arm-level changes. Solid bar represents median number of events per sample. Error bars represent S.E.M. ***, $P < 0.0005$; **, $P < 0.005$; *, $P < 0.05$; n.s., not significant.

Figure S3. Numbers of high-level amplifications and deletions using arm-level based thresholds. Thresholds were determined by using the most extreme arm-level event for either amplifications or deletions. Focal SCNAs above or below this sample-specific threshold were called as high-level. ***, $P < 0.0005$; **, $P < 0.005$; *, $P < 0.05$; n.s., not significant.

Figure S4. Identification of arm-level alterations in esophageal, gastric, and colorectal adenocarcinomas. Arm-level q-values (x-axis) for a) amplifications and b) deletions are plotted across the genome (y-axis).

Figure S5. Comparison of SNP6.0 and 250K adenocarcinoma data to Progenetix database. Frequency of copy-number alteration at the cytoband level was compared between a) 190 colorectal cancer copy-number profiles from SNP6.0 and 250K arrays with 998 aCGH and cCGH Progenetix colorectal samples, b) 110 gastric adenocarcinoma copy-number profiles from SNP6.0 and 250K arrays with 741 aCGH and cCGH Progenetix gastric profiles, and c) 186 esophageal adenocarcinoma copy-number profiles from SNP6.0 and 250K arrays with 71 aCGH and cCGH esophageal adenocarcinoma Progenetix profiles from (http://www.progenetix.net).

Figure S6. Significance of focal amplifications in esophageal, gastric, and colorectal adenocarcinomas. GISTIC q-values (x-axis) for amplifications are plotted across the genome (y-axis).

Figure S7. Variable levels of focal alterations across gut adenocarcinomas. Plots of copy-number data for amplification of a) *ERBB2* (17q12), b) *GATA6* (18q11.2), c) *GATA4* (8p23.1), and d) deletion of *CDKN2A* (9p21.1). *P*-values shown in italics represent Fisher's exact test results comparing numbers of samples with and without each alteration in each tumor type.

Figure S8. *KRAS* alterations differ across gut adenocarcinomas. a) Plot of copy-number data for amplification of the *KRAS* locus (12p12.1) in esophageal, gastric, and colorectal tumors. *P*-values shown in italics represent Fisher's exact test results

comparing numbers of samples with and without *KRAS* alterations in each tumor type. b) Percentages of *KRAS* amplification and mutation (MacConaill LE, et al. 2009) in GI adenocarcinomas compared to colorectal adenocarcinomas.

Figure S9. Quantitative real-time PCR confirms *FGFR2* amplification in EA tumors.

Figure S10. Focal alterations in gut adenocarcinomas shared with all cancers. Significantly amplified or deleted (q-value < 0.01) genes and peaks from the gut adenocarcinoma cohort was compared to 2,311 other cancers excluding these tumor types (Beroukhim, et al. 2010).

Figure S11. Significance of focal deletions in esophageal, gastric, and colorectal adenocarcinomas. GISTIC q-values (x-axis) for deletions are plotted across the genome (y-axis).

Figure S12. Length distribution of SCNAs in gut adenocarcinomas. Data are presented by cancer type and experimental platform.

Figure S13. Observed distribution of copy number event amplitudes in the data set. Amplifications are shown as positive amplitudes and deletions as negative amplitudes on the histogram. SCNAs of magnitude < 0.2 copies that were excluded from the analysis as likely artifacts fall within the uncolored bars.

Figure S14. Hierarchical clustering of copy-number profiles. Data were clustered using a correlation distance metric and copy-numbers at peak regions in the combined GISTIC analysis across all tumors.