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SUPPLEMENTARY INFORMATION

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Pan-cancer analysis of whole genomes identifies driver rearrangements promoted by LINE-1 retrotransposition

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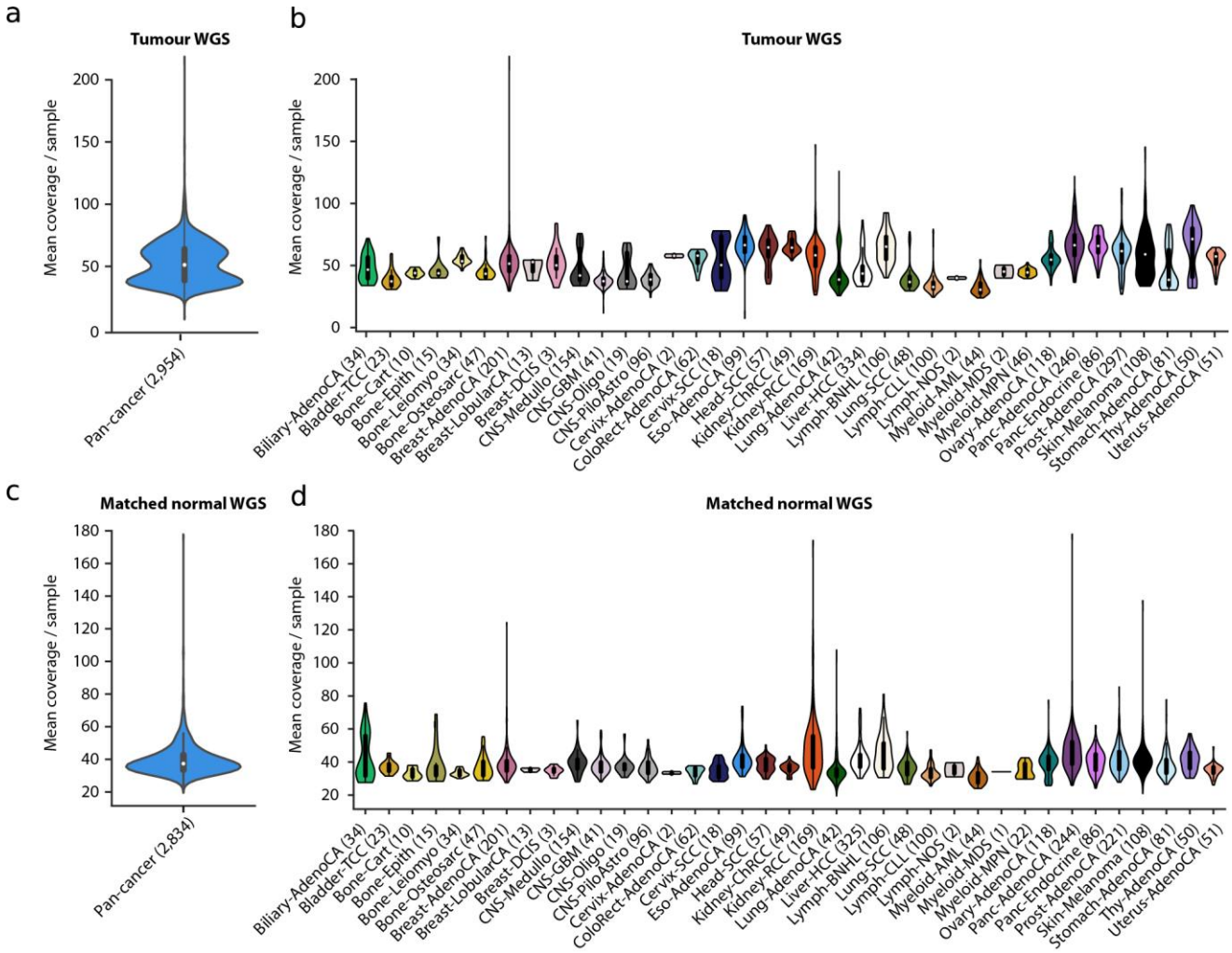
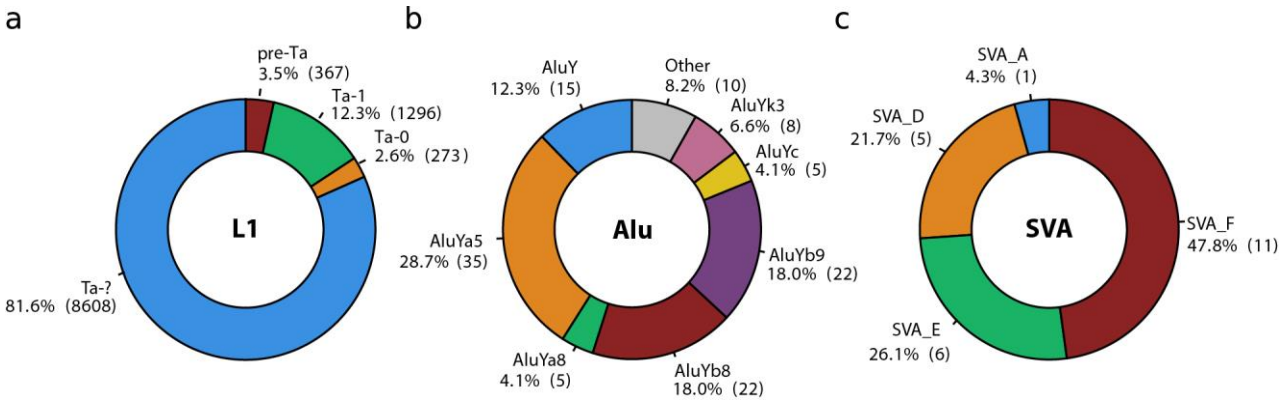


Supplementary Figure 1

Whole-genome sequencing coverage in tumours and matched-normal genomes in the PCAWG cohort.

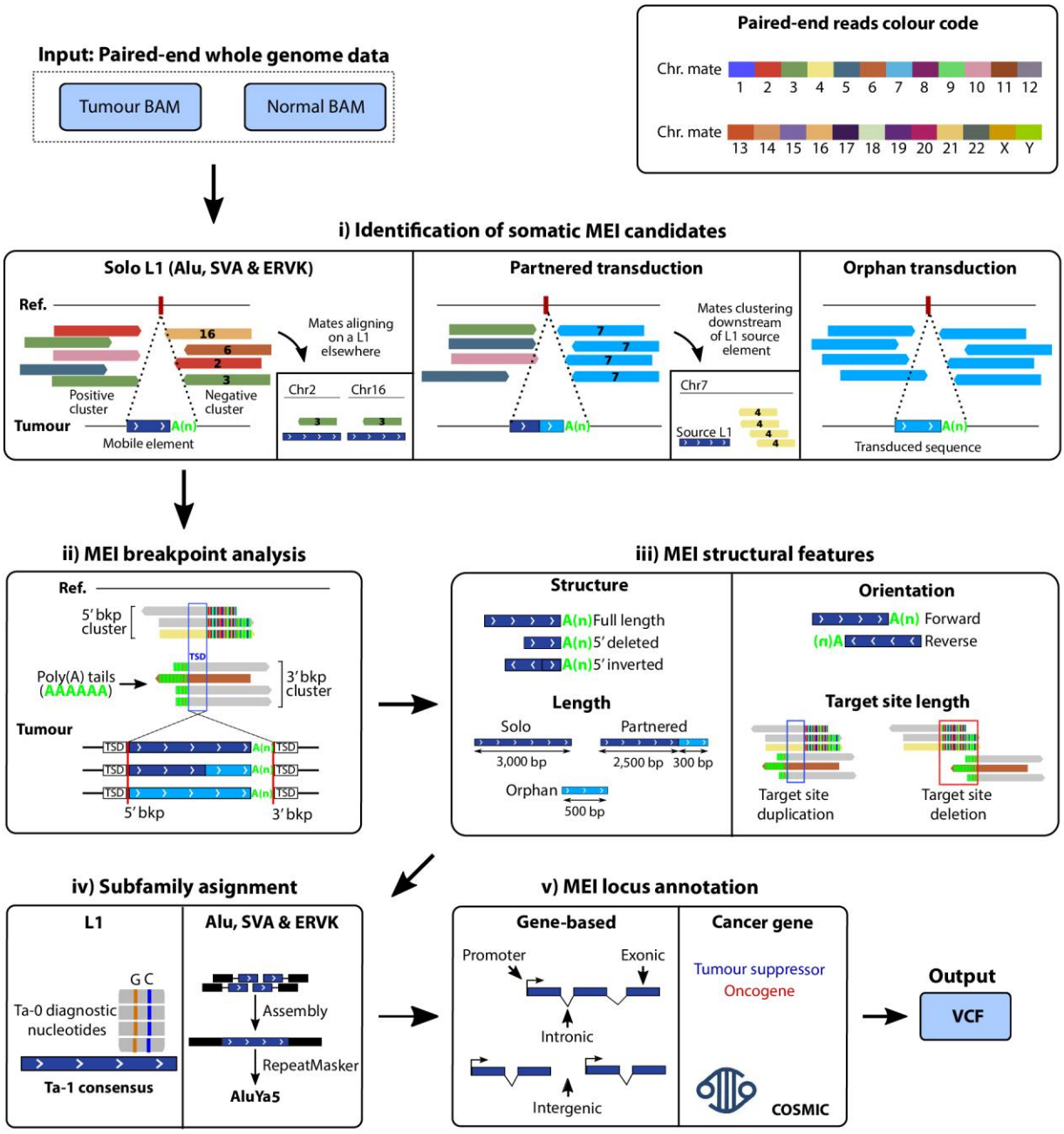
(a) Violin plot for the distribution of the mean coverage from all tumours (N=2,954) analyzed shows a bimodal distribution with maxima at 38 and 60 reads per base-pair. White point, median; box, 25th to 75th percentile (interquartile range, IQR); whiskers, data within 1.5 times the IQR. (b) Tumour samples mean depth of coverage distribution by cancer type. The number of samples per tumour type is shown in parenthesis. Violin plot features are as in 'a'. (c) Violin plot for the distribution of the mean coverage from all PCAWG matched-normal samples (N=2,834) analyzed shows a mean coverage of 30 reads per genome. Violin plot features are as in 'a'. (d) Matched-normal samples mean depth of coverage distribution by cancer type. Number of samples per tumour type is shown in parenthesis. Violin plot features are as in 'a'.



Supplementary Figure 2

Distribution of somatic retrotranspositions according to subfamily.

(a) L1 subfamilies. Ta-1 and Ta-0 elements – the youngest subfamilies of L1 retrotransposons – represent 97.5% of all L1 somatic mobilizations that were characterized to subfamily level, although we also find 367 L1 events bearing the diagnostic hallmarks of pre-Ta elements, which have been shown to retain retrotransposition activity in modern humans[5]. The category “Ta-?” contains those L1-Ta events for which it was not possible to detect the Ta-0 or Ta-1 diagnostic nucleotides. (b) Alu subfamilies. (c) SVA subfamilies.

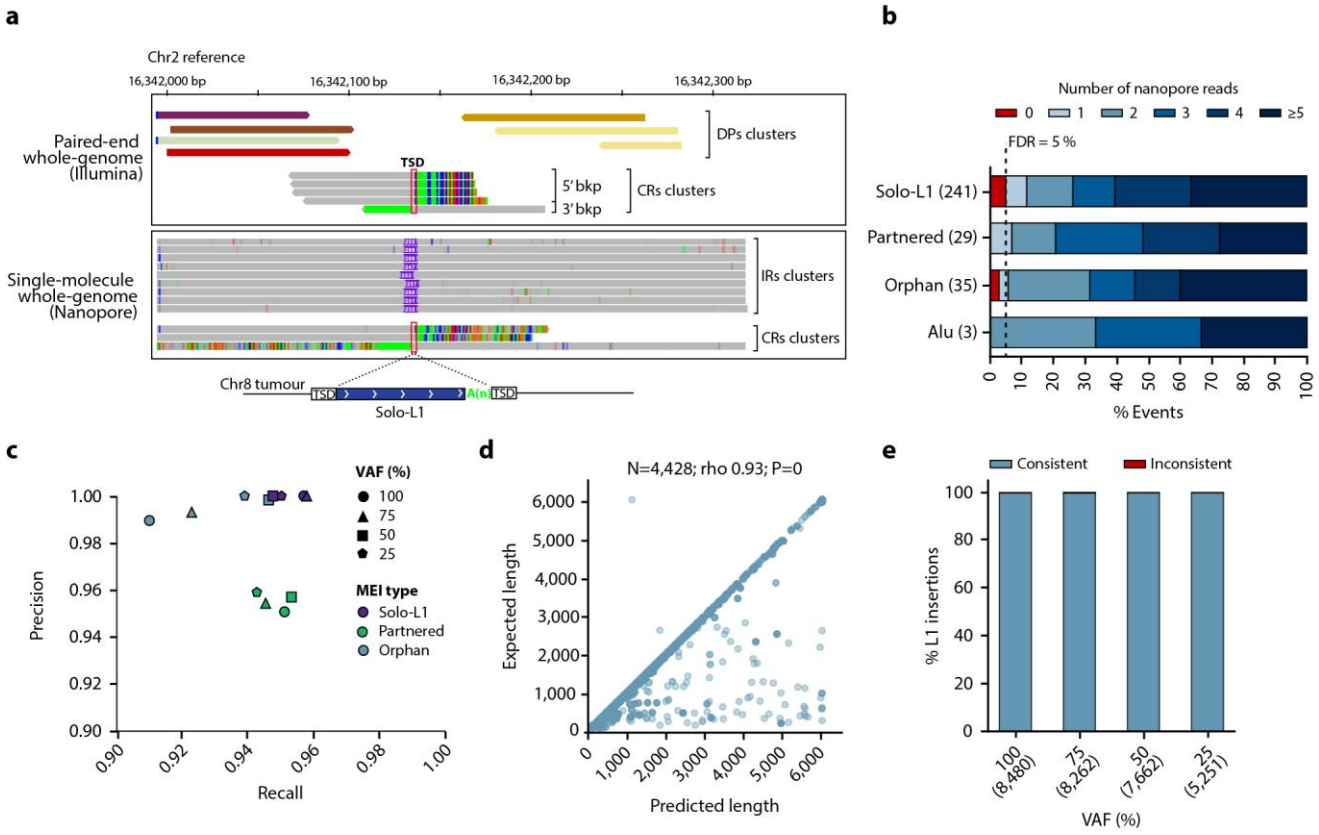


Supplementary Figure 3

Overview of TraFiC-mem.

TraFiC-mem analyzes Illumina paired-end mapping data (see **Supplementary Note**). i) Identification of candidate somatic mobile element insertions (MEIs) by TraFiC-mem. Solo-retrotransposon insertions are detected by the identification of two reciprocal clusters (positive and negative, or head-to-head) of interchromosomal reads whose mates map onto retrotransposons of the same type located elsewhere in the genome. Partnered transductions are detected by the identification of one cluster of interchromosomal reads whose mates map onto L1 retrotransposons of the same family elsewhere in the genome, and one single reciprocal cluster of reads whose mates are clusterered at a unique region adjacent to a donor source L1 element (the example illustrates a transduction from chromosome 7). Orphan transductions are detected by the identification of two reciprocal clusters whose mates map downstream to a source element as described for partnered transductions. ii) MEI breakpoint analysis. TraFiC-mem seeks for two additional clusters (5'

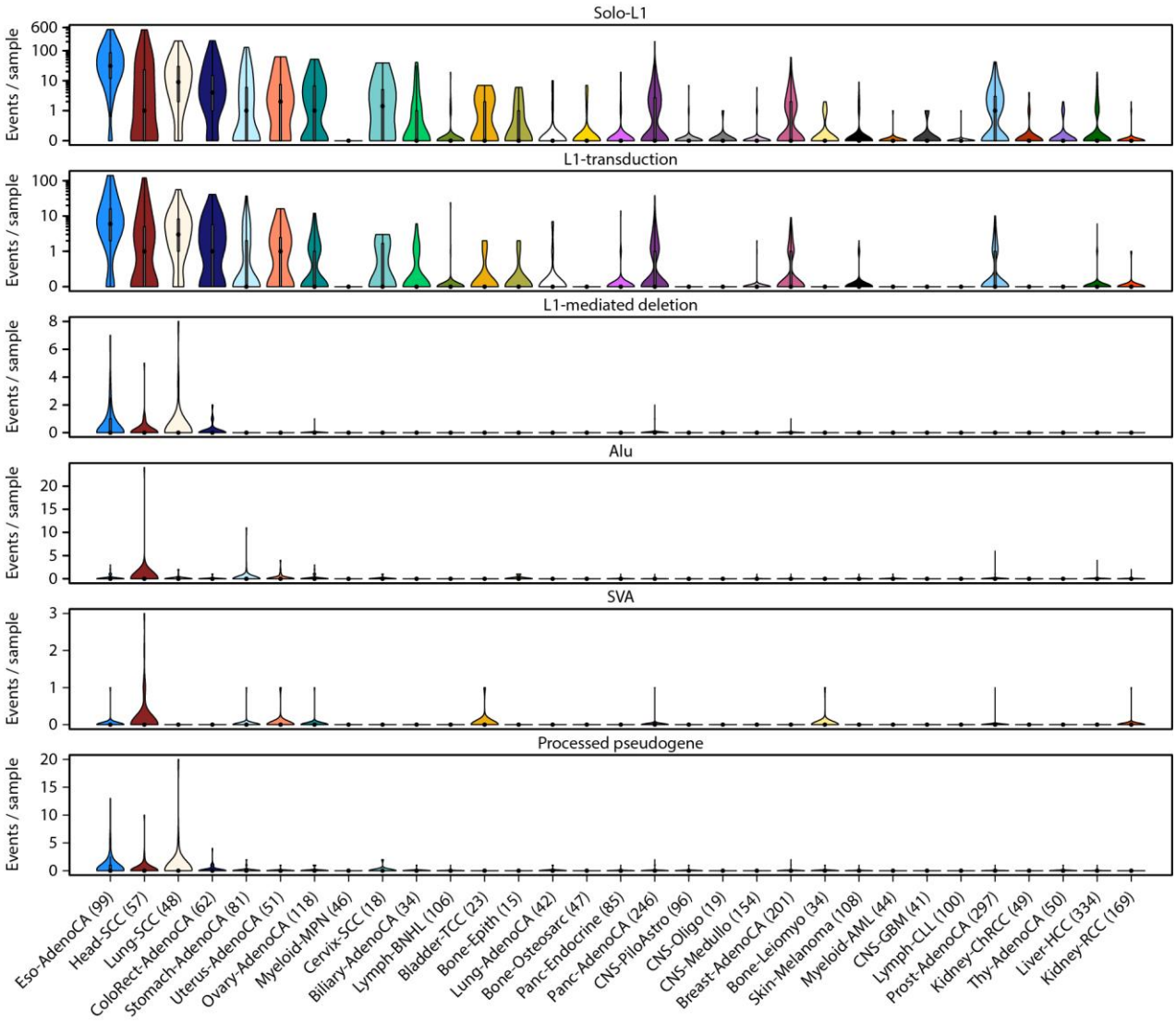
brakpoint cluster and 3’ breakpoint cluster) of clipped reads in the candidate insertion region, in order to reveal the 5’ and 3’ insertion breakpoint coordinates to base-pair resolution. iii) MEI structural features annotation. iv) Subfamily assignment. Subfamily specific diagnostic nucleotides are used to determine the subfamily for L1 events. v) MEI locus annotation: The target genomic region is annotated and MEIs inserted within cancer genes, according to the COSMIC database, are flagged. Output is a VCF file.



Supplementary Figure 4

Validation and evaluation of TraFiC-mem.

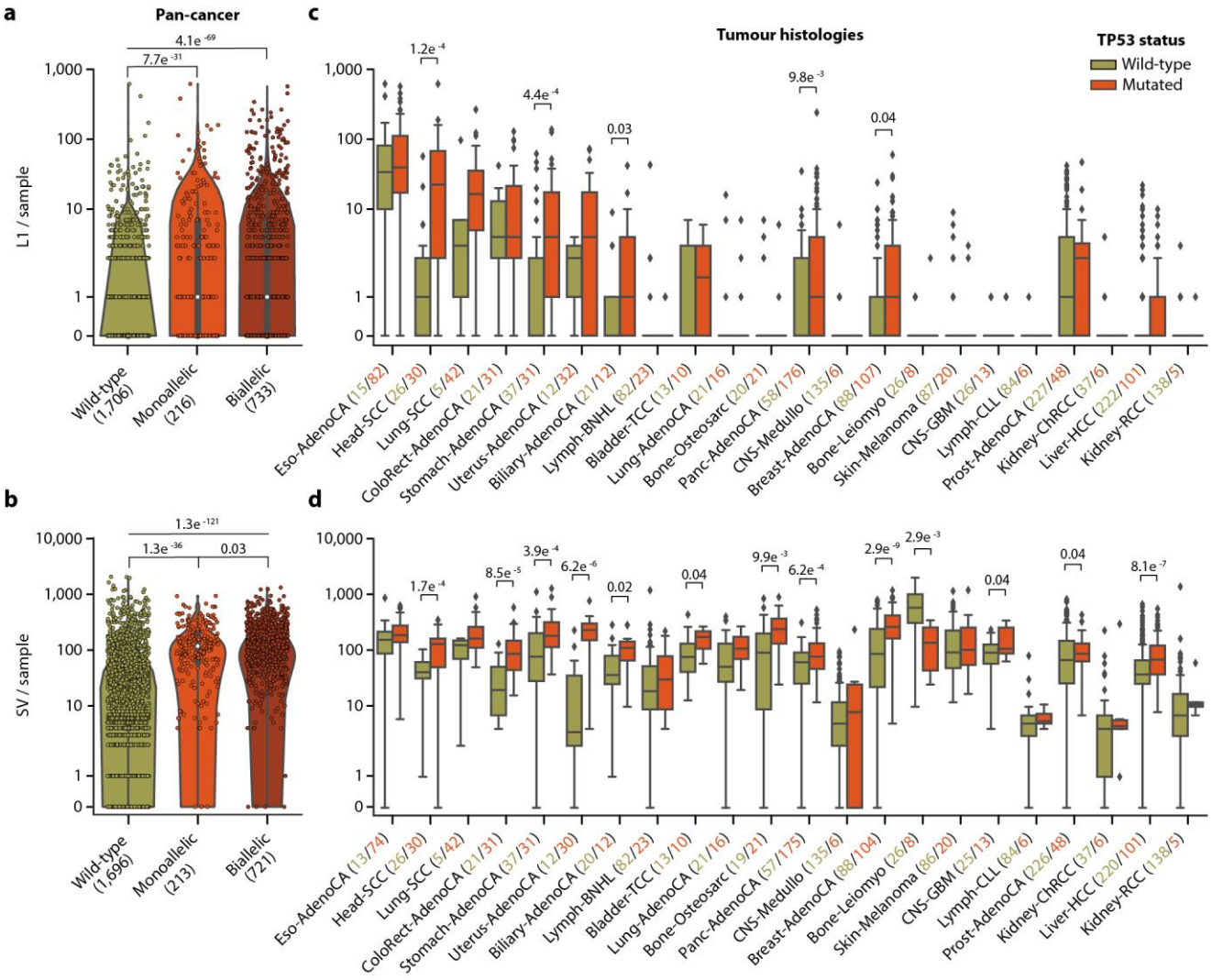
(a) Retrotransposition breakpoint validation approach using long-reads with Oxford Nanopore Technologies (ONT). Illustrative example of a Solo-L1 insertion in cancer cell-line NCI-H2087 detected with short and long-reads. Top, TraFiC-mem relies on the identification of discordant read-pairs (DPs) and clipped reads (CRs) to detect a Solo-L1 insertion using Illumina paired-end data. Bottom, indel reads (IRs) and clipped reads confirmation using ONT. (b) For each type of insertion (solo-L1, partnered transductions, orphan transductions, Alu), the proportion of events that are supported by different counts of long-reads is represented (from zero to more than 5 reads). Events supported by at least one long-read and absent in the matched-normal sample were considered true positive (i.e., somatic), while those not supported by ONT and/or present in the matched-normal sample were considered false positive. The total number of events assessed for each retrotransposition category is shown in parenthesis. (c) Precision and recall of TraFiC-mem after in-silico simulation of 10,000 L1 insertions (Solo-L1, partnered and orphan transductions) in tumors of different clonalities at 25%, 50%, 75% and 100%. (d) Plot showing the correlation between the observed and expected lengths for 8,025 Solo-L1 insertions simulated in-silico. Sample size (N), Spearman's rho and *P-value* are displayed above the panel. (e) Fraction of true positive Solo-L1 events with a predicted orientation consistent (green), and inconsistent (red), with the expected. Orientation consistency was assessed for four clonality levels (25%, 50%, 75%, 100%).



Supplementary Figure 5

Rates of somatic retrotransposition across PCAWG tumor types.

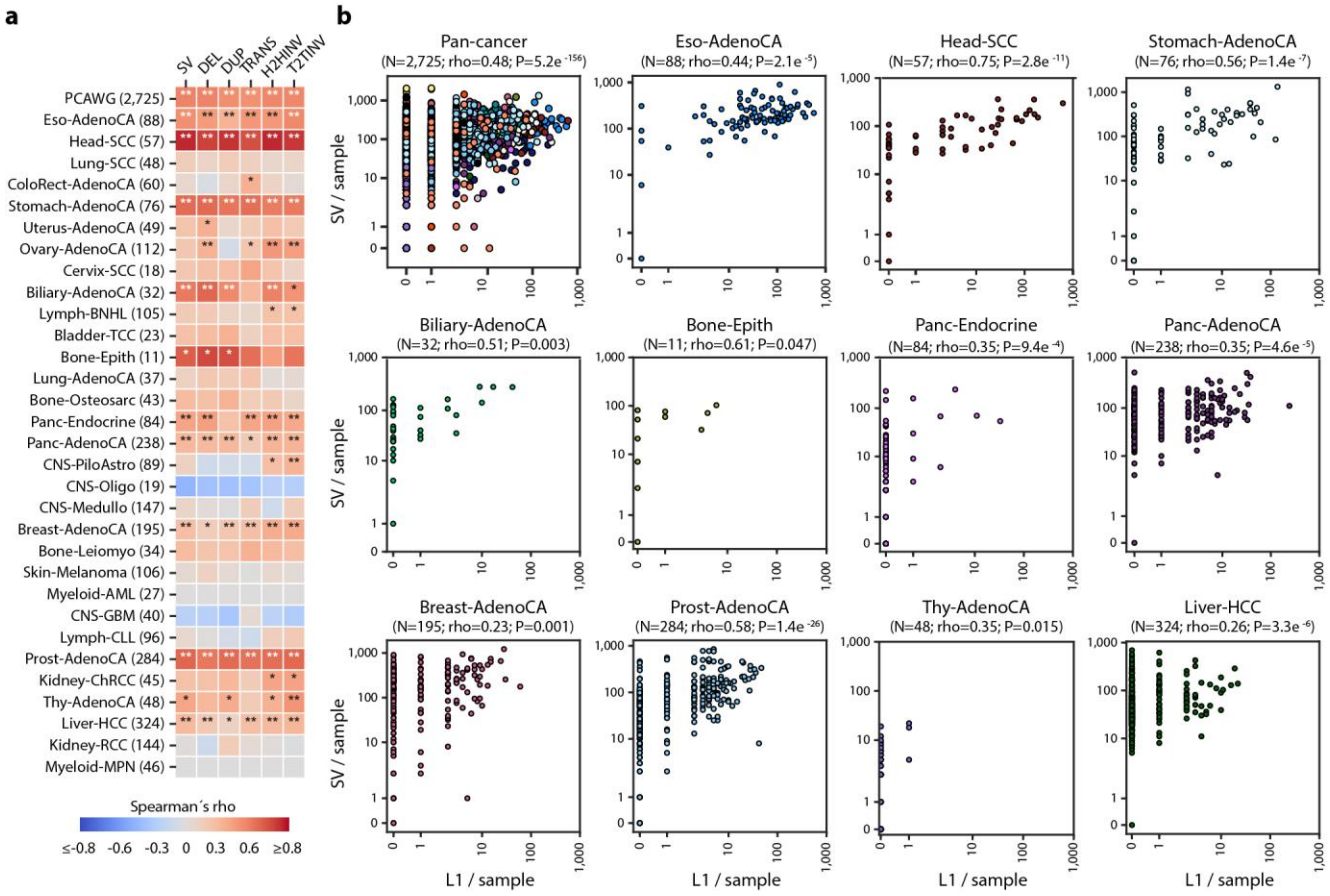
Violin plots showing the distribution of the number of retrotranspositions per sample across cancer types, for the six different categories of retrotranspositions that were analyzed (Solo-L1, L1-transductions, L1-mediated deletions, Alu, SVA and Processed pseudogenes). The number of samples per tumor type is shown in parenthesis. Y-axis is represented in a logarithmic scale. Black points, median; boxes, 25th to 75th percentile (interquartile range, IQR); whiskers, data within 1.5 times the IQR.



Supplementary Figure 6

TP53 mutation is associated with high rates of L1 retrotransposition and structural variants.

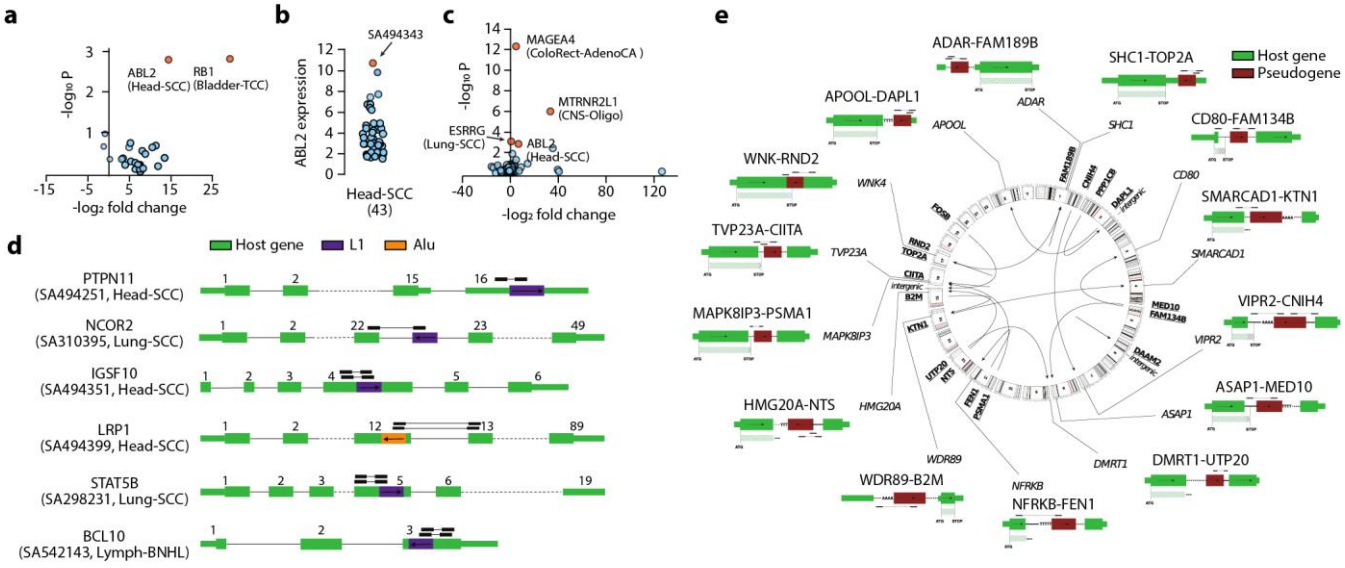
(a) Distribution of L1 counts of three sample groups according to their *TP53* mutational status: wild-type, monoallelic and biallelic driver mutation. The number of samples per group is shown within parenthesis. Point, median; box, 25th to 75th percentile (interquartile range, IQR); whiskers, data within 1.5 times the IQR. *P-value*s indicate significance from a two-tailed Mann–Whitney *U*-test. Y-axis is presented in a logarithmic scale. (b) The same for structural variant (SV) counts. (c) Distribution of L1 counts across tumor types from samples grouped in two categories: *TP53* wild-type and *TP53*-mutated (monoallelic or biallelic). The number of samples per tumor type and TP53 status (green: wild-type; orange: mutated) is shown within parenthesis. Violin plot features are as in 'a'. Outlier values outside 1.5 times the IQR are represented as diamonds. *P-value*s indicate significance from a two-tailed Mann–Whitney *U*-test. Y-axis is presented in a logarithmic scale. (d) The same for structural variant (SV) counts.



Supplementary Figure 7

Correlation between L1 retrotransposition and structural variation burden.

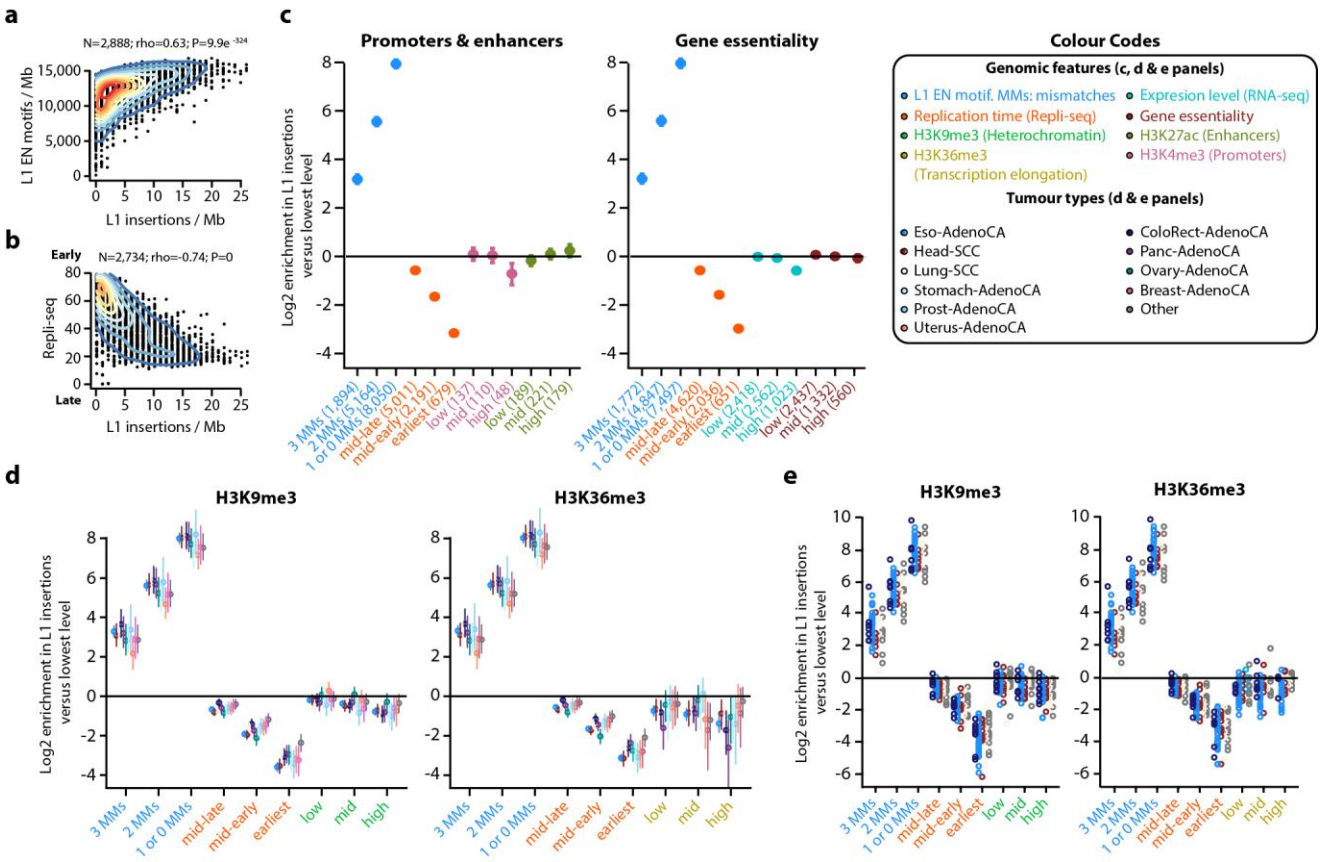
(a) Heatmap showing the correlation between the number of L1 events, the total number of structural variants (SVs) and the number of 5 different types of SVs per sample: deletions (DEL), duplications (DUP), translocations (TRANS), head-2-head inversions (H2HINV) and tail-2-tail inversions (T2TINV). Correlation was assessed both at Pan-Cancer and tumor type levels. Spearman´s correlation coefficients are shown in a blue (negative) to a red (positive) colored gradient. *P-value*s lower than 0.05 and 0.01 are represented as single and double asterisks, respectively. (b) Scatter plots showing correlations between the number of L1 events and the total number of SVs per sample at both Pan-Cancer and tumour type levels, for those comparisons that were significant in panel 'a'. The sample size (N) together with Spearman's rho and *P-value* are displayed above each chart. Both axes are displayed on a symlog scale.



Supplementary Figure 8

Some gene expression effects associated with somatic retrotransposition in PCAWG.

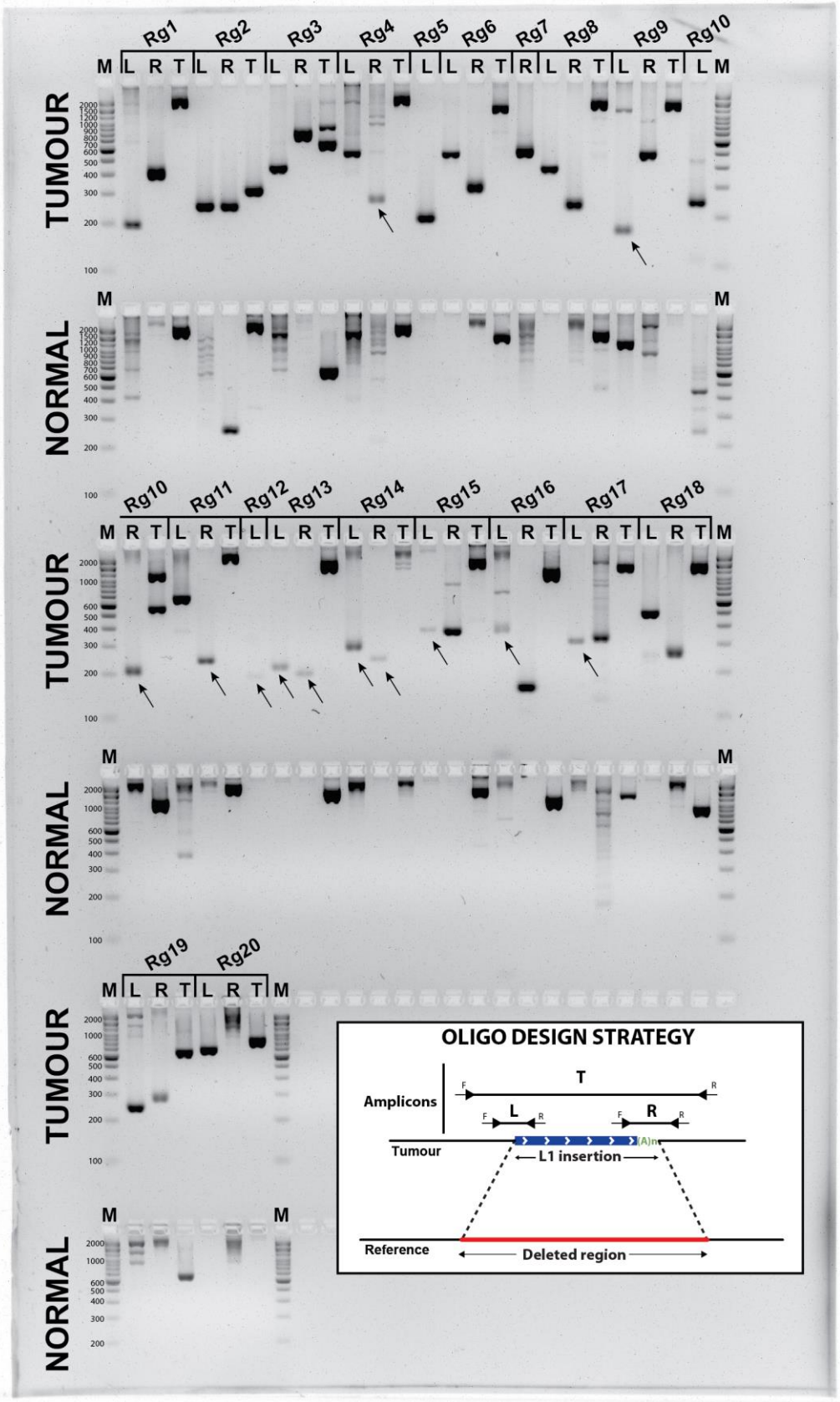
(a) Volcano plot showing the impact of L1 integration in the expression of cancer genes. Gene expression fold-change (x axis) is represented versus inverted significance (y axis). Red dots indicate significant associations under Benjamini–Hochberg adjusted *p-value*s < 0.1 from two-tailed Student's *t*-test. Adjusted *p-value*s for *ABL2* and *RB1* are 0.0017 and 0.0014, respectively. (b) Up-regulation of the *ABL2* oncogene in tumour SA494343, a head-and-neck squamous carcinoma (Head-SCC), relative to the expression of the same oncogene in other Head-SCC samples (blue) from PCAWG. Expression levels measured as Fragments Per Kilobase Million (FPKM). (c) Gene expression differences for genes with L1-retrotranspositions in promoter regions reveals significant upregulation in four genes. Volcano plot features are as in 'a'. Adjusted *p-value*s for *MTRNR2L1*, *ESRRG*, *ABL2* and *MAGEA4* are 1.1294^{e-06}, 0.0009, 0.0017 and 6.0019^{e-13}, respectively. (d) Exonization of somatic retrotranspositions, including cancer genes *PTPN11* and *NCOR2*. Green boxes are exons, thinner green blocks UTRs, lines introns and dotted lines means that a piece of the gene model is not shown for visualization purposes. Purple and orange boxes correspond to L1 and Alu integrations, respectively. Discordant read-pairs supporting fusion transcript expression are shown above the predicted transcript. (e) Host gene and processed pseudogene fusion transcripts. Arcs with arrows within the circos indicate the processed pseudogene events, connecting the source gene (underlined and bold) with the corresponding integration site. Predicted fusion transcript structures are shown in the outermost layer of the figure. Coding potential is shown underneath the fusion transcript representation. Start codon is denoted as ATG, termination codon as STOP, and uncertain termination is represented using dots.



Supplementary Figure 9

L1 integration and genomic features.

(a) Correlation between the number of L1 events and L1 endonuclease (EN) motif instances per 1 Mb-windows. The sample size (N) together with Spearman's rho and *P-value* is displayed above the plot. 2D Kernel density estimate (KDE) is displayed over the data points in a blue to red gradient. (b) Correlation between the number of somatic L1 insertions per Mb and replication timing, which is measured through Repli-seq wavelet-smoothed signal (late to early replication) and averaged per Mb. Plot features are as in 'a'. (c-e) In each panel, enrichment scores are shown, adjusted for multiple covariates and comparing the L1 insertion rate in bins 1-3 for a particular genomic feature versus bin 0 of the same feature, which therefore always has log enrichment=0 by definition and is not shown. The error bars represent 95% confidence intervals. The number of observations per bin is provided between parenthesis whenever possible. For replication time, bin 0 is the latest-replicating quarter of the genome. For essentiality, bin 0 is the non-essential genes. For the L1 motif, bin 0 denotes a non-match (4 or more mismatches). MMs stands for the number of mismatches relative to the consensus L1 EN motif. Additional information in **Supplementary Note**. (d) Association between L1 insertion rate and multiple genomic features for those tumour types with at least 100 L1 events. Data colored according to tumor type. (e) Association between L1 insertion rate and multiple genomic features in samples with at least 100 L1 events. Each data point is colored according tumour type.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TUMOUR
NORMAL
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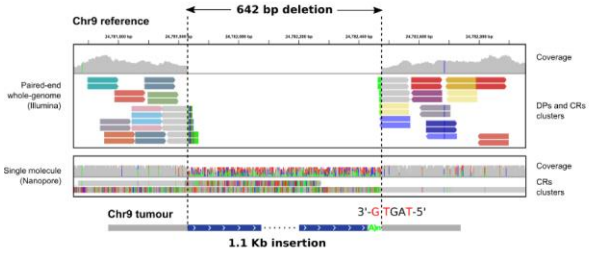
R
F
R
Tumour
(A)n
L1 insertion
Reference
Deleted region

Supplementary Figure 10

PCR validation of somatic L1-mediated rearrangement calls.

Gel showing PCR results on cancer cell-lines (NCI-H2009 and NCI-H2087) and their matched-normal cell-lines (NCI-BL2009 and NCI-BL2087). We performed validation of 20 L1-mediated rearrangements (for details, see Supplementary Table 7): 16 L1-mediated deletions (Rg1, Rg2, Rg3, Rg4, Rg6, Rg8, Rg9, Rg10, Rg11, Rg13, Rg14, Rg15, Rg16, Rg17, Rg18, Rg19), 1 L1-mediated translocation (Rg20) and 3 independent L1 breakpoints associated with a copy number change from an unknown rearrangement type (Rg5, Rg7, Rg12). For each rearrangement, except those where only one breakpoint is known, at least three regions were amplified in the tumours (see Online Methods): left breakpoint (L), right breakpoint (R), and the target site (T). Arrows are used to highlight the position of some somatic amplicons. Note that the target site could also amplify in the matched-normal sample if the deletion is not too long. “M” denotes the size marker. For illustrative purposes, the oligo design strategy is shown in a panel at the bottom of the figure: amplicons (L, R and T) and oligos – forward (F) and reverese (R) – are represented. This experiment was repeated 3 times with the same result, and results were further confirmed by single-molecule sequencing of the amplicons with ONT (see Online Methods).

a **Rg18 (NCI-H2087)**

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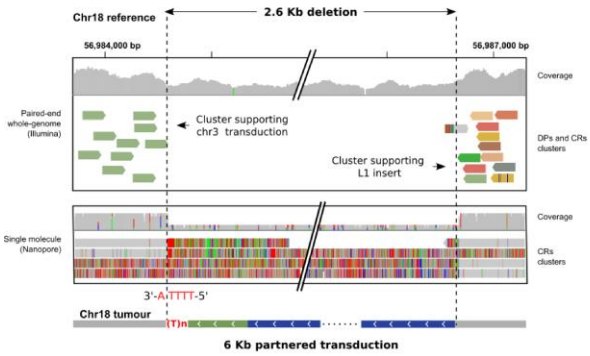
b **Rg11 (NCI-H2009)**

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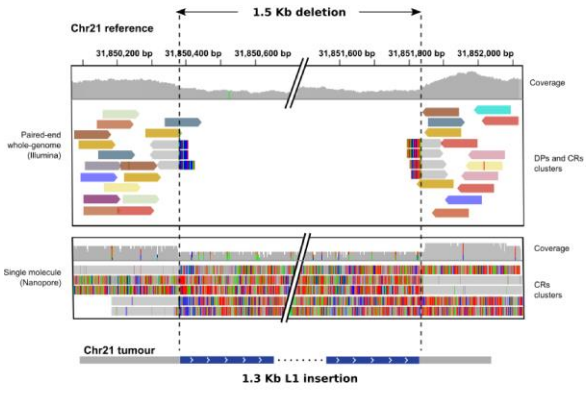


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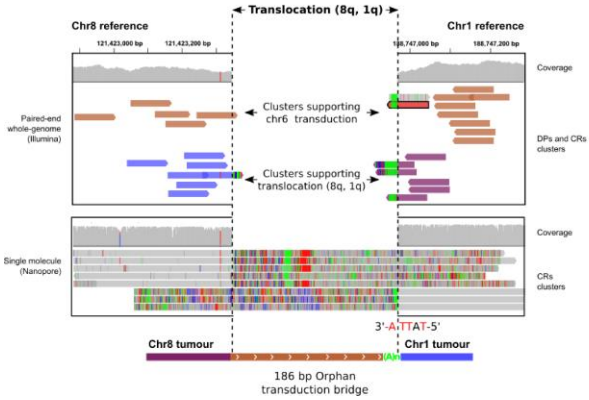
d **Rg20 (NCI-H2087)**

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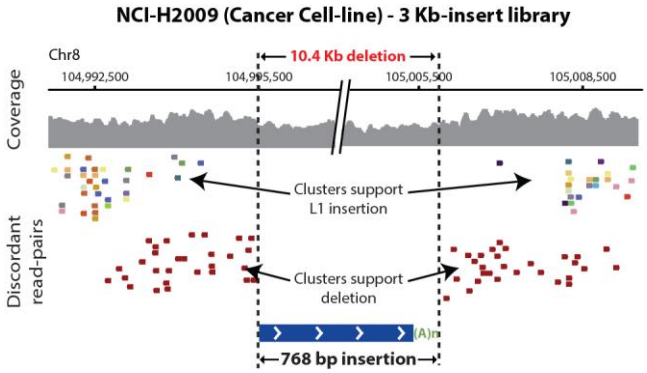
ttaatcactg gctcttaatt catatatgtt tatccaaaat tgaatctcaa  6100
aaatattgga gtattaggtg gggatttgct tttattacat tatcaaactg  6150
gattttacac ttacagttaa aattgtgtgc tatatctagt tgtatttcag  6200
```

Supplementary Figure 11

Single-molecule sequencing validation of somatic L1-mediated rearrangement calls.

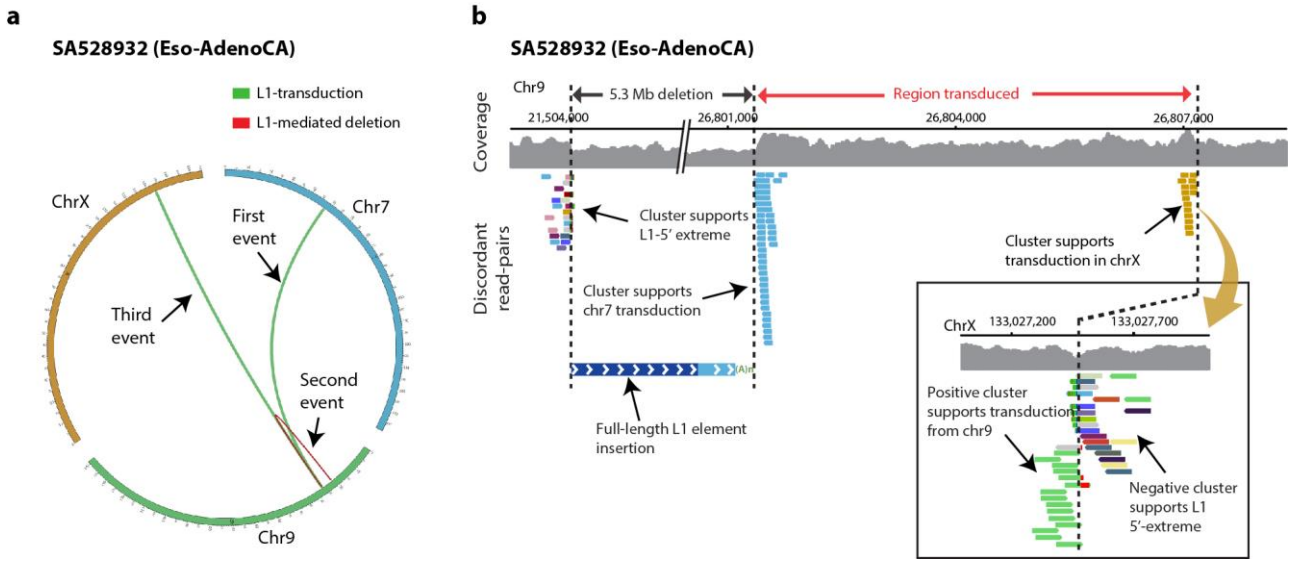
We sequenced to high-coverage (>1,000x) the PCR amplicons shown in Supplementary Fig. 10 using single-molecule sequencing with a MinION sequencer (Oxford Nanopore Technologies). We also carried out whole-genome single-molecule sequencing to low coverage of the same two tumor cell-lines (NCI-H2009 and NCI-H2087) subjected to PCR validation. For illustrative purposes, this figure only shows the validation of four representative rearrangements (Rg18, Rg11, Rg13, Rg20). The sequences of the remaining PCR amplicons can be found in Supplementary Table 7. On the left side of each panel, paired-end and Oxford Nanopore reads supporting a given rearrangement are displayed over a virtual reconstruction of the rearrangement breakpoints. On the right side of each panel, nucleotide sequence obtained by single-molecule sequencing validating each event shown in left. Nucleotide colors match those in the virtual reconstruction of the rearrangement (blue for L1, bright-green for poly-A, grey for target region, light-green for transduction). (a) Solo-L1 insertion mediating a 642 bp deletion. (b) Partnered transduction promoting a 2.6 Kb long deletion. (c) A 1.5 Kb deletion generated through an endonuclease independent L1 integration. Long reads confirm the truncation of the L1 element at its 5′ and 3′ ends. (d) Translocation between 1q31.1 and 8q24.12 mediated by an orphan transduction (same rearrangement as in **Fig. 6b**). Nanopore reads validate the orphan transduction bridge between both chromosomes.



Supplementary Figure 12

Validation of L1-mediated rearrangements in cancer cell lines by mate-pairs sequencing.

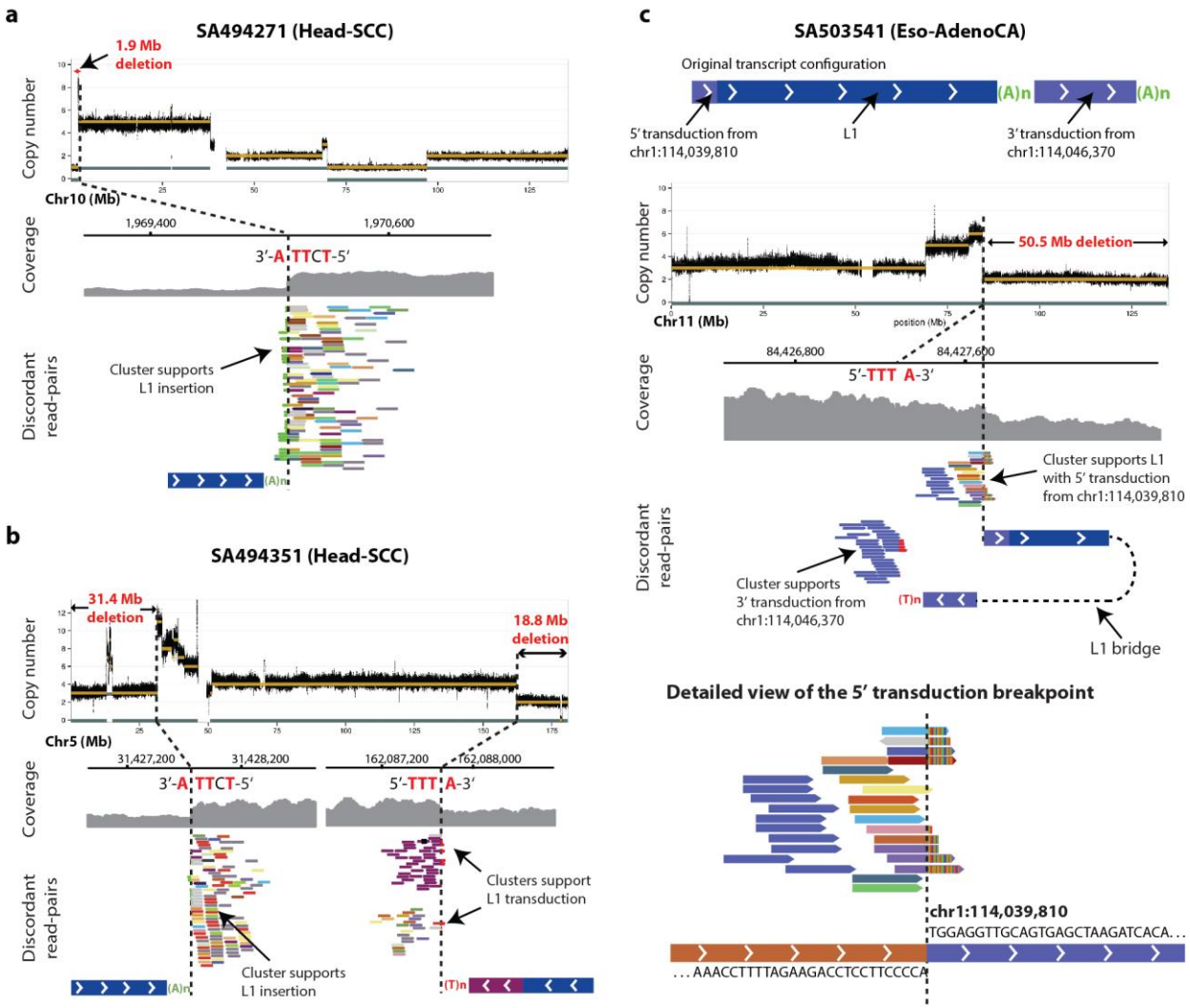
In order to further validate L1-mediated deletions, we performed mate-pair sequencing of long-inserts libraries (3 kb and 10 kb) on two cancer cell-lines with high-retrotransposition rates. In these samples, our algorithms confirmed 16 events with the hallmarks of L1-mediated deletions, in which the mate-pair data confirmed a single L1-derived (i.e., solo-L1 or L1-transduction) retrotransposition as the cause of the copy number loss, and identified the sizes of the deletion and the associated insertion. For illustrative purposes, here it is shown the validation of a 10.4 kb long deletion promoted by integration of a 768 bp L1 insertion in the cancer cell-line NCI-H2009. The L1 element inserted within the deletion breakpoints is too long to be characterized using standard paired-end sequencing libraries, but the mate-pairs successfully span the breakpoints of the deletion and confirm a single L1 insertion associated with the rearrangement.



Supplementary Figure 13

Some L1-mediated deletions are transduction-competent.

(a) Circos plot summarizing the three concatenated retrotransposition events shown in the panel b. First event, an L1 transduction mobilized from chromosome 7 is integrated into chromosome 9. Second event, this insertion concomitantly causes a 5.3 Mb deletion in the acceptor chromosome 9. Third event, the L1 element causing the deletion is subsequently able to promote a transduction that integrates into chromosome X. (b) Discordant read-pairs in chromosome 9 supports a 5.3 Mb deletion generated by the integration of a transduction from chromosome 7, and reveals an L1-event with full-length structure. Five kilobases downstream, a positive cluster of reads supports a transduction from this L1-retrotransposition event into chromosome X.



Supplementary Figure 14

Somatic integration of L1 and telomere loss.

The PCAWG-11 consensus total copy number and the copy number of the minor allele are plotted as gold and gray bands, respectively. (a) In a head-and-neck tumor, SA494271, deletion of 1.9 Mb at the short arm of chromosome 10, which involves the telomeric region, is associated with the somatic integration of an L1 retrotransposon. (b) In another head-and-neck tumor, SA494351, two independent L1 events promote deletion of both ends of chromosome 5. (c) In a Lung squamous carcinoma, SA503541, the aberrant integration of an L1 event bearing 5' and 3' transductions causes a complex rearrangement with loss of 50.5 Mb from the long arm of chromosome 11 that includes the telomere. Only the two clusters supporting both extremes of a putative L1-mediated fold-back inversion are shown. Below, a detailed view of the 5'-transduction breakpoint.

SUPPLEMENTARY NOTES

Pan-cancer analysis of whole genomes identifies driver rearrangements promoted by LINE-1 retrotransposition

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TABLE OF CONTENTS

1. ANALYSIS OF SOMATIC RETROTRANSPOSITION

1.1. Detection of mobile element insertions using TraFiC-mem

BAM files from tumour and matched-normal pairs were processed with TraFiC-mem v1.1.0 (https://gitlab.com/mobilegenomes/TraFiC) to identify somatic mobile element insertions (MEIs) including solo-L1, L1-mediated transductions, Alu, SVA and ERV-K, using Illumina paired-end mapping data. In donors where multiple samples of primary, metastatic and/or recurrent tumours were available, each sample was independently processed and a list of non-redundant MEI calls for each donor was generated by merging the MEI calls from multiple samples as follows: those MEIs within a breakpoint offset of ±15 bp were clustered together, and the call supported by the highest number of discordant read-pairs in each cluster was selected as representative in the merging process.

TraFiC-mem starts by identifying candidate somatic MEIs via the analysis of discordant read-pairs. Contrary to previous version of the algorithm [1] , the new pipeline uses BWA-mem instead of RepeatMasker as search engine for the identification of retrotransposon-like sequences in the sequencing reads. Calls obtained at this step are preliminary, in which MEI features are outlined and insertion coordinates represent ranges surrounding the breakpoints. Then, a new module of TraFiC-mem, called MEIBA (from Mobile Element Insertion Breakpoint Analyzer) (https://github.com/brguez/MEIBA/tree/master/src/python), is used to identify the integration breakpoints to base-pair resolution, and to perform a detailed characterization of MEI features, including structure, subfamily assignment, and insertion site annotation. TraFiC-mem is illustrated in **Supplementary Fig. 3**.

1.1.1. Identification of MEI candidates via discordant reads analysis

- Identification of Solo-element events: TraFic-mem identifies reads from BWA-mem mapping that are likely to provide information pertaining to mobile elements site inclusion. Two different read-pair types are considered for the identification of insertions, named INTER_CHROM (each end of the pair are mapped to different chromosomes, where the end with the highest mapping quality (MAPQ) is considered the anchor in the reference genome), and ABERRANT (both ends of the pair are impropertly mapped to the same chromosome, where the end with the highest

MAPQ is considered the anchor). In all cases the anchor end must have a MAPQ higher than zero. The pair is excluded if any of the reads is not a primary alignment, fails platform/vendor quality checks, or is PCR or optical duplicate. Then, all non-anchor reads with an anchored mate MAPQ > 0 are interrogated for the existence of mobile element-like sequences. At this step, non-anchor reads are realigned with BWA-mem v0.7.17[1] into a database containing a set of human full-length mobile element consensus sequences, including L1, Alu, SVA and ERV-K. After BWA-mem search, all anchored reads with mates containing sequences from the same mobile element type are clustered together if (a) they have the same orientation – positive or negative – and (b) the distance relative to the nearest mapped read of the same cluster is equal or less than the average read size. Two main cluster categories are considered, namely positive and negative clusters (i.e., anchor reads mapped onto the reference genome with positive and negative orientation, respectively). Initially, a range of genome coordinates is associated with each single cluster (breakpoint coordinates are refined in a later step – see "Reconstruction of MEI breakpoints via clipped-reads analysis"). Ranges are conformed by a lower coordinate (P_L_POS and N_L_POS, respectively for positive and negative clusters) and an upper coordinate (P_R_POS and N_R_POS, for positive and negative). One positive and one negative cluster are reciprocal if P_R_POS ≥ N_L_POS and abs(N_L_POS – P_R_POS) ≤ 2(average read size). Only positive and negative clusters conformed of 4 or more reads are employed in the assignment of reciprocal clusters. Each reciprocal cluster identifies a candidate mobile element insertion (**Supplementary Fig. 3a**).

- Identification of L1 transductions: Two types of L1-mediated transductions were identified [1], namely partnered transductions, in which an L1 and downstream nonrepetitive sequence are retrotransposed together, and orphan transductions, in which only the unique sequence downstream of an active L1 is retrotransposed without the cognate L1. As above, two different read-pair types, INTERCHROM and ABERRANT are considered. In all cases the anchor read and the corresponding mate must have a MAPQ ≥ 37. Then, anchored reads are clustered together if (a) they share the same orientation, (b) the distance relative to the nearest mapped read of the cluster is equal or less than the average read size, and (c) their mates are also clustered together. Two main cluster categories are considered, namely positive and negative clusters, as above. The integration breakpoint of a potential partnered transduction is defined by reciprocal

clusters conformed of one-single cluster of (a) INTER_CHROM and/or ABERRANT reads that support an L1 and (b) one-single cluster of INTER_CHROM and/or ABERRANT reads that supports the integration of a unique DNA region from elsewhere in the genome that is located downstream to an L1 source element locus. One L1 cluster and one INTER_CHROM and/or ABERRANT cluster are reciprocal if they are in opposite orientation and P_R_POS ≥ N_L_POS and abs(N_L_POS – P_R_POS) ≤ 2(average read size). Only positive and negative clusters conformed of 4 or more reads are employed in the assignment of reciprocal clusters. Reciprocal clusters represent preliminary transduction calls that must pass the filters described below to be finally selected. The integration breakpoint of a potential orphan transduction is defined by two reciprocal clusters conformed of INTER_CHROM and/or ABERRANT reads. In this case, two clusters of INTER_CHROM and/or ABERRANT reads are reciprocal if (a) they are in opposite orientation, (b) P_R_POS ≥ N_L_POS and abs(N_L_POS – P_R_POS) ≤ 2(average read size), and (c) the two single clusters that constitute their mates are mapped within a distance of 10 kb to each other (**Supplementary Fig. 3a**).

TraFiC-mem performs the actions described above in both, the tumour and the matched normal genomes. In order to remove potential germline calls, MEI candidates are filtered out from the tumour sample if: (a) they are located within 200 bp of a cluster from the same retrotransposon family in the matched normal sample that is supported by at least 3 reads; and/or (b) there is a polymorphic insertion from the same retrotransposon family within a range of 200 bp that is present in 'TraFiC-ip db' [1], dbRIP [2], the 1,000 Genomes Project Phase 3 callset [3,4] or the dataset of germline events identified by our group across PCAWG[5]. Finally, we noticed the existence of mapping artefacts leading to quite frequent false positive insertion calls (particularly in Alu and SVA calls), located within or between repeats of the same family in the reference genome. So, an additional filter is applied to remove those insertions located within a range of ±150 bp of an element from the same family that shows ≥85% of nucleotide identity relative to the consensus sequence of the family.

1.1.2. Reconstruction of MEI breakpoints via clipped-reads analysis

A new module of TraFiC-mem, called MEIBA, is used to identify the breakpoints of an insertion to base-pair resolution. The algorithm uses two classes of reads mapped

with BWA-mem, namely soft and hard clipped reads, which overlap a putative insertion breakpoint. These reads consist of two segments, one that aligns onto the insertion target region and a second that aligns onto a mobile element elsewhere in the reference genome. Thus, once MEI candidates have been identified, TraFic-mem seeks for two additional clusters of clipped reads (CRs) that would indicate the exact insertion breakpoint coordinates (each individual insertion has two breakpoints, namely 5' and 3' breakpoints). Soft and hard CRs are extracted within a range of ±50 bp to the positive cluster P_R_POS cordinate identified in step "i" of the pipeline via discordant read-pairs. Reads marked as duplicates and reads clipped both at the beginning and ending extremes are filtered out, as they usually constitute mapping artefacts. The same approach is applied to the negative cluster N_L_POS coordinate of the same putative MEI, and both sets of reads, belonging to positive and negative clusters, are merged into a non-redundant dataset as follows: CRs are organized into clusters supporting the same breakpoint position using a maximum breakpoint offset of 3 bp. Those clusters in the tumour that are also detected in the matched-normal genome, and/or those clusters with an overrepresentation of CRs overlapping the breakpoint (we used a cut-off of more than 500 CRs), are excluded. Then, for each breakpoint cluster, supporting CRs are submitted to multiple sequence alignment using MUSCLE v3.8.31 [6], and a consensus sequence spanning the insertion breakpoints is constructed with "Cons" from the EMBOSS suit v6.6.0 [7]. The consensus sequences obtained are processed to assess if they span the target genome region and mobile element breakpoint junction (5' breakpoint), or the target region and poly(A) tail junction (3' breakpoint). If more than one 5' and/or 3' breakpoints are generated, the one supported by the highest number of CRs is selected. Finally, insertion breakpoints are required to be consistetly supported by at least two CRs, and candidate MEIs are filtered out if they do not have at least one of the two insertion breakpoints characterized to base pair resolution.

1.1.3. MEI structural features annotation

MEI structural features including insertion length, structure condition (full-length, partial, inverted), orientation, and size of target site duplication (TSD) or target site deletion, are determined for the insertions with both breakpoints successfully reconstructed. In order to compute the insertion size, the consensus sequence spanning the 5' breakpoint is realigned to the corresponding L1, Alu, SVA or ERV-K reference

sequence using Blat v34.0 [8]. Next, as retrotransposons usually only get truncated at their 5'-extreme, the insertion length is computed as the distance between the beginning of the alignment and the end of the reference sequence. Insertions spanning less than 98% of the consensus sequence for each family of retrotransposons are considered 5'-truncated, and/or if the sequence aligns in opposite orientation than the insertion DNA strand are considered 5'-inverted; otherwise, insertions are catalogued as full-length. MEIs with positive orientation are supported by clusters at the 5' and 3' breakpoints whose CRs are clipped at their ending (end-clipped) and their beginning (beg-clipped), respectively; while MEIs with negative orientation show the opposite clipping pattern. TSD and target-site deletion sizes are estimated as the distance between the two insertion breakpoints. Insertions with TSD show a breakpoint coordinate supported by the end-clipped cluster that is higher than the breakpoint coordinate supported by the beg-clipped cluster. Target site deletions show the opposite pattern.

1.1.4. MEI subfamily assignment

Two different strategies are applied to infer the subfamily of the inserted L1, Alu, SVA and ERV-K element. For L1 insertions, discordant read-pairs from supporting reciprocal clusters are realigned onto an L1 consensus sequence (GenBank identifier: L19088.1) using BWA-mem v0.7.17. The resulting SAM is converted into a binary sorted BAM file using samtools v1.7 [9]. Then, genotype likelihoods at each genomic position are computed with samtools mpileup, reference and variant sites are called with bcftools v1.7 consensus caller [10] and filtered requesting a quality score higher than 20 and a minimum read depth of 2. Finally, subfamily inference is done based on the identification of subfamily diagnostic nucleotide positions [11]: L1 integrations bearing the diagnostic "ACG" or "ACA" triplet at 5,929-5,931 position are classified as "pre-Ta" and "Ta", respectively. Ta elements are subclassified into "Ta-0" or "Ta-1" according to diagnostic bases at 5,535 and 5,538 positions (Ta-0: G and C; Ta-1: T and G). When sequencing reads do not cover the diagnostic nucleotides, subfamily cannot be inferred. For Alu, SVA and ERV-K, discordant read-pairs from reciprocal clusters supporting the insertions are assembled with velvet v1.2.10 [12], using a k-mer length of 21 bp, and the resulting contig is processed with RepeatMasker v4.0.7 to determine the subfamily. If multiple RepeatMasker hits are obtained, the one with the highest Smith-Waterman score is selected as representative. MEIs will be discarded if preliminary family assigment in "i" and subfamily assigment are not consistent.

1.1.5. MEI locus annotation

The target genomic region is annotated using the software ANNOVAR v2016-02-01 [13] and GENCODE v19 basic annotation [14]. MEIs inserted within cancer genes, according to the Cancer Gene Census COSMIC database v77 [15], are flagged.

1.1.6. TraFiC-mem output

The primary TraFiC-mem output is a standard Variant Call Format (VCF) v4.2 file containing all somatic MEI calls coordiantes with annotation features, including family, subfamily, insertion length, structural condition, orientation, size of TSD or deletion, gene annotation, number of supporting reads, and consensus sequences spanning the breakpoint junctions. Additional information is provided for L1-mediated transductions, which includes the transduced sequence length, the genomic position of the source element, and source element. MEI candidates that were filtered out are also reported together with filtering reasons.

1.1.7. TraFiC-mem availability and distribution

TraFiC-mem is implemented using Snakemake [16], a flexible Python-based workflow language, that allows to execute the pipeline from single-core workstations to computing clusters, without the need to modify the workflow. In order to enhance reproducible research, TraFiC-mem and its third party dependencies are also distributed as a Docker image (https://hub.docker.com/r/mobilegenomes/trafic). TraFiC-mem is distributed together with complete documentation and tutorial (https://gitlab.com/mobilegenomes/TraFiC).

1.2. Identification of L1-mediated deletions

Independent read clusters, identified with TraFic-mem, supporting an L1 event (i.e., clusters of discordant read-pairs with no apparent reciprocal cluster within the proximal 500 bp, and whose mates support a somatic L1 retrotransposition event) were interrogated for the presence of an associated copy number change in its proximity. Briefly, we looked for copy number loss calls from PCAWG-11 (see “Copy number dataset” above) for which the following conditions were fulfilled: (i) the upstream breakpoint matches an independent L1 cluster in positive orientation, (ii) the

corresponding downstream breakpoint, if any, from the same copy number change matches an independent L1 cluster in negative orientation, and (iii) the reconstruction of the structure of the putative insertion causing the deletion is compatible with one-single retrotransposition event. We used MEIBA – described above – to reconstruct the insertion breakpoint junctions in order to confirm the ends of the events and identify hallmarks of retrotransposition, including the poly(A) tract and duplication of the target site.

Further to the strategy described above, an additional strategy was adopted to identify L1-mediated deletions shorter than 100 kb, as follows. Coverage drops in the proximity of each independent cluster were detected by, first, normalizing read depth on each side of the cluster using the matched normal sample as a reference. Then, the ratio between the normalized read depth on both sides of the cluster was computed. This calculation was performed for window sizes ranging between 200 and 5,000 bp, with the windows on both sides of the cluster always having the same length. An immediately adjacent 'buffer' region of 300 bp was defined on each side of the cluster, and reads within these regions were omitted in read depth calculations, in order to prevent false positives due to sequence repeats at the cluster location. Subsequently, pairs of independent reciprocal (positive–negative) clusters were selected for which (i) the two clusters were located less than 100 kb apart, (ii) a potential drop in read depth ratio was identified, extending from the positive cluster to the negative cluster (statistical significance of read depth ratios was estimated non-parametrically, as described below), and (iii) the reconstruction of the structure of the putative insertion causing the deletion was compatible with a single L1 event. For each selected cluster pair, the continuity and reliability of the copy number drop was assessed by measuring the normalized read depth ratio between non-overlapping 500 bp windows spanning the region between the positive and negative clusters (i.e. within the putative deletion) and windows located upstream and downstream of the positive and negative cluster (i.e. outside the putative deletion), respectively. The significance of each drop in read depth ratio was estimated non-parametrically using a null distribution of normalized read depth ratios. This distribution was obtained for each tumour sample by randomly sampling 100,000 genomic locations, drawn from the copy number segments with the predominant copy number in that particular sample (If the predominant copy number was 1, then segments with a copy number of 2 were used instead to avoid extreme read depth ratios that could

arise from potentially undetected deletions). Specifically, read depth ratios were calculated from this sample of locations by comparing the normalized read depth between two 2,500-bp windows located immediately upstream and downstream of each location. Non-parametric *p*-values were calculated by comparing the observed read depth ratios with the ones in this null distribution, and adjusted via Benjamini–Hochberg (BH) multiple-testing correction. Three groups of output clusters were produced, corresponding to decreasing significance of the candidate deletions: first, pairs of reciprocal clusters where both clusters present an adjusted p-value below 0.1 ('Tier 1' candidates); second, pairs of reciprocal clusters where only one cluster presents an adjusted p-value below 0.1 ('Tier 2' candidates); and third, any individual clusters presenting an adjusted p-value below 0.1 ('Tier 3' candidates). The resulting L1-mediated deletion candidates (Tiers 1 and 2) were subsequently confirmed via visual inspection using the Integrative Genomics Viewer (igv) [17].

1.3. Analysis of the association between L1 insertion rate and genomic features

L1 insertion rate was calculated as the total number of somatic L1 insertions, identified across the complete PCAWG cohort, per 1-Mb window. L1 endonuclease motif density was computed as the number of canonical endonuclease motifs, here defined as TTTT|R (where R is A or G) or Y|AAAA (where Y is C or T), per 1-Mb. Bivariate correlations between L1 insertion rate, endonuclease motif density and replication timing were assessed using Spearman's rank.

To study the association of L1 insertion rate with multiple predictor variables at single-nucleotide resolution we used a statistical framework based on negative binomial regression, as described in detail previously [18]. This method was adapted herein such that originally the regression adjusted for content of trinucleotides in each genomic bin, while in this case we instead adjusted for the content of the L1 endonuclease motif. More specifically, we stratified the genome into four bins (0-3) by the closeness of match to the canonical L1 motif, here defined as TTTT|R (where R is A or G). The bin 0 contains dissimilar DNA motifs, which have 4 or more (out of 5) mismatches (MMs), encompassing 1149.7 Mb of the genome. Bin 1, 2 and 3 contain genome segments with exactly 3, exactly 2 and at most 1 MM, encompassing 749.4 Mb, 380.2 Mb and 114.1 Mb of the GRCh37 assembly, respectively. The closest match of either of the two DNA strands was considered.

Histone mark data (ChIP-Seq for H3K9me3, H3K4me3, H3K36me3, H3K27ac) and DNase hypersensitivity (DHS) data for the regional analyses was collected from Roadmap Epigenomics Consortium by averaging fold-enrichment signal over 8 cell types (E017, E114, E117, E118, E119, E122, E125 and E127) and processed by stratifying into four genomic bins, as described previously [18]. For histone marks and DHS, bin 0 are the areas of the genome with below-baseline signal (Roadmap fold-enrichment compared to input < 1), while bins 1-3 are approximately equal-sized bins covering the remaining parts of the genome with above-average fold-enrichment score. In particular, DHS bins 1-3 encompass 122.8-123.0 Mb each; for H3K36me3 129.1-136.0 Mb each; for H3K4me3 43.2-43.7 Mb each; for H3K27ac 73.6-75.1 Mb each. RNA-Seq data was also collected from Roadmap and processed as previously [18] by averaging over 8 cell types (E071, E096, E114, E117, E118, E119, E122, E127): bin 0 consisted of non-expressed genes (FPKM=0) and intergenic DNA that was not explicitly listed as expressed (total 1076.6 Mb), while bin 1 (up to 0.59 FPKM), 2 (up to 5.68 FPKM) and 3 (above 5.68 FPKM) spanned 389.9, 462.1 and 473.8 Mb of the genome, respectively. Replication time (RT) data was processed similarly as histone marks, but collected from ENCODE and processed by averaging the wavelet-smoothed signal over 8 cell types (HeLa S3, HEP G2, HUVEC, NHEK, BJ, IMR-90, MCF-7 and SK-N-SH) and then dividing into four equal-sized genomic bins (quartiles), where bin 0 is the latest-replicating and bin 3 is the earliest replicating. Essential genes were determined by CERES score based on CRISPR essentiality screens, ordering by median score across all 342 cell lines tested [19] and then stratifying genes into equal-frequency bins, from less negative to more negative median CERES score (implying commonly essential genes). For the purposes of finding L1 rates in CERES essential genes an additional 1 kb flanking the transcript was also considered together with the gene. All enrichment scores shown in plots compare bins 1-3 for a particular feature (RT, histone marks, gene expression, L1 motif) versus bin 0 of the same feature, which therefore always has log enrichment=0 by definition and is not shown on enrichment plots. The regional analyses are restricted to parts of the genome with perfect mappability scores, according to the CRG Alignability 75 track of the UCSC browser.

Further to what is reported in the main text, our analyses confirm fewer L1 events at active promoters (1.63-fold), here detected by the H3K4me3 histone mark [20], yet there

is no decrease at active enhancers, marked by H3K27ac; and we detect no significant association between gene essentiality and L1 rates (1.03-fold decrease in essential genes), suggesting that only a minor fraction of the somatic L1 events may be under negative selection (**Supplementary Fig. 9c**). Different cancer types and different samples appear remarkably consistent in the biases of L1 events towards later-replicating DNA and towards other epigenomic features examined (**Supplementary Fig. 9d-e**)

1.4. Validation of TraFiC-mem calls using single-molecule sequencing

Due to the unavailability of Pan-Cancer DNA specimens, in order to evaluate our algorithm for the identification of retrotransposon integrations, we performed validation of 308 putative somatic retrotranspositions identified with TraFiC-mem in one cancer cell-line (NCI-H2087) with high retrotransposition rate, and absent in its matched normal cell-line (NCI-BL2087) derived from blood, by single-molecule sequencing using Oxford Nanopore technology. Genomic DNA was sheared to 10 kb fragments using Covaris g-TUBEs (Covaris), cleaned with 0.4x Ampure XP Beads (Beckman Coulter Inc). After end-repairing and dA-tailing using the NEBNext End Repair/dA-tailing module (NEB), whole-genome libraries were constructed with the Oxford Nanopore Sequencing 1D ligation library prep kit (SQK-LSK108, Oxford Nanopore Technologies Ltd). We obtained four and five libraries for NCI-H2087 and NCI-BL2087, respectively. Genomic libraries were loaded on MinION R9.4 flowcells (FLO-MIN106, Oxford Nanopore Technologies Ltd), and sequencing runs were controlled using the Oxford Nanopore MinKNOW software v18.01.6. We used the Oxford Nanopore basecaller Albacore v2.0.1 to identify DNA sequences directly from raw data and generate fatsq files. Files with quality score values below 7 were excluded at this point. Minion adapter sequences were trimmed using Porechop v0.2.3 (https://github.com/rrwick/Porechop). Then, we used minimap2 v2.10-r764-dirty[21] to map sequencing reads onto the hs37d5 human reference genome, and the SAM files were converted to BAM format, sorted and indexed with Samtools v1.7 for each one of sequencing runs. BAM files were merged, sorted and indexed. After this process, sequencing coverage were 8.2x (NCI-BL2087) and 9.17X (NCI-H2087), and average read size of mapped reads were ~4.5 kb (NCI-BL2087) and ~11 kb (NCI-H2087).

Once having the whole-genome BAM files, for each one of the 308 putative somatic retrotransposition call identified with TraFic-mem, we interrogated the long-read tumour BAM file to seek for reads validating the event. Two types of MEI supporting clusters of sequencing reads were catalogued (**Supplementary Fig. 4a**), namely (i) "indel-read clusters", composed of Nanopore-reads completely spanning the insertion, so they can be identified as a standard insertion on the reference, and (ii) "clipped-read clusters", composed of Nanopore-reads spanning only one of the inserted element extremes, so they get clipped during the alignment in the reference. We observe that short MEI insertions are predominantly supported by indel-read clusters, while longer MEI insertions are mainly supported by clipped-read clusters. MEIs supported by at least one Nanopore-read in the tumour and absent in the matched-normal sample were considered true positive (TP) somatic events, while MEIs not supported by long-reads in the tumour and/or present in the matched-normal were considered false positive (FP) calls. Overall, we find 4.22% (13/308) false positive events, which showed to be particularly frequent in regions with low sequencing coverage. However, we cannot not rule out the possibility that these are true positive events, as they were not found in the matched-normal sample. False discovery rate (FDR) was estimated as follows: FDR = FP / (TP + FP).

1.5. Validation of L1-mediated rearrangements with PCR and single-molecule sequencing

Due to the unavailability of Pan-Cancer DNA specimens, we performed validation of 20 somatic L1-mediated rearrangements, mostly deletions, identified in two cancer cell-lines with high retrotransposition rates (NCI-H2009 and NCI-H2087). We carried out PCR followed by single-molecule sequencing of amplicons from the two tumour cell-lines and their matched normal samples (NCI-BL2009 and NCI-BL2087), using a Minion sequencer from Oxford Nanopore. PCR primers were designed with Primer3 v0.4.0 [22] , to amplify three regions from each event (namely, 5'-extreme, 3'-extreme and target site) as follows. For the amplification of the 3'-extreme of the event (the one that contains the poly(A) tract), we designed one forward oligo to hybridize the 3' extreme of the MEI, and a reverse oligo that hybridizes the DNA downstream. In the case of Solo-L1s, we used an L1Hs specific forward oligo matching the 3'-UTR: 5'-GGGAGATATACCTAATGCTAGATGACAC-3' [23], or an alternative forward oligo that matches other region at the 3'-extreme of the element. For the amplification of the

target site in the tumour and matched normal, we designed primers to amplify the DNA sequence between both breakpoints (5' and 3') of the rearrangement. For the amplification of the 5'-extreme of a MEI in a tumour, we designed one forward oligo to match the non-repetitive region immediately adjacent to the 5'-extreme of the element, and a reverse oligo that hybridizes the 5' extreme of the MEI.

Each PCR mixture contains 10ng of DNA, 5pmol of each primer, 5U Taq DNA polymerase (Sigma-Aldrich, catalog number D1806) with 1x Buffer containing $MgCl_2$, 0.2mM of each dNTPs, and water to a final volume of 25µl. PCR conditions were as follows: initial denaturation at 95ºC for 7 minutes; then 30-35 cycles of 95ºC for 30 seconds, 60ºC for 30 seconds, 72ºC for 45 seconds; and a final extension of 72ºC for 7 minutes. In some cases, when amplification was tricky, we used Platinum Taq High-fidelity, with a 94ºC denaturation and a 68ºC extension.

PCR amplicons were sequenced with single-molecule sequencing using a MinION from Oxford Nanopore. Amplicons were pooled and total DNA was cleaned with 0.4x AMPure XP Beads (Beckman Coulter Inc). After end-repairing and dA-tailing using the NEBNext End Repair/dA-tailing module (NEB), the sequencing library was constructed with the Oxford Nanopore Sequencing 1D ligation library prep kit (SQK-LSK108, Oxford Nanopore Technologies Ltd) and loaded on a MinION R9.4 flowcell (FLO-MIN106, Oxford Nanopore Technologies Ltd). Mapping to human reference genome was performed as described above, with minor modifications.

2. INTERPRETATION OF THE PAIRED-END MAPPING DATA FROM FIGURES

TraFiC-mem analyzes Illumina paired-end whole-genome sequencing data, aligned with BWA-mem, from a pair of tumour and matched-normal samples, to identify somatically acquired mobile element insertions (MEIs). All figures in this paper use default read colours by Integrative Genomics Viewer (igv)[17], where paired-end reads are coloured by the chromosome on which their mates can be found. Thus, retrotransposon-specific clusters (i.e., clusters of reads supporting the integration of a retrotransposon) are conformed of multicoloured reads, because mates can map

ambiguously elsewhere in the reference genome where a retrotransposon of the same family is present.

Supplementary Fig. 3 illustrates how TraFiC-mem identifies different types of candidate somatic MEIs. Briefly, the integration of a Solo-retrotransposon is detected by the identification of two reciprocal clusters (positive and negative) of interchromosomal (multicoloured) reads whose mates map onto retrotransposon of the same type located elsewhere in the genome. The integration of a partnered transduction from chromosome 7 is detected by the identification of two different types of reciprocal clusters: one cluster of multicoloured interchromosomal reads whose mates map onto L1 retrotransposons of the same family elsewhere in the genome, and one single-coloured cluster of reads whose mates are clustered at a unique region adjacent to a donor source L1 element – for illustrative purposes, this last cluster identifies a transduction from chromosome 7, and reads from this cluster are homogeneously coloured in light-blue because that is the default colour in igv for chromosome 7 –. The integration of an orphan transduction from chromosome 7 is detected by the identification of two reciprocal clusters of the same type, but this time both clusters are single-coloured because mates are clustered on the same chromosome.

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Mering[120,126]

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Macintyre[278], Florian Markowetz[278,279], Iñigo Martincorena[1], Alexander Martinez-Fundichely[110,111,123], Satoru Miyano[83], Hidewaki Nakagawa[47], Fabio CP Navarro[114], Stephan Ossowski[128,139,148], Peter J Park[223,225], John V Pearson[295,296], Montserrat Puiggròs[45], Karsten Rippe[81], Nicola D Roberts[1], Steven A Roberts[297], Bernardo Rodriguez-Martin[270,271,272], Steven E Schumacher[3,204], Ralph Scully[298], Mark Shackleton[178], Nikos Sidiropoulos[108], Lina Sieverling[112,171], Chip Stewart[3], David Torrents[45,105], Jose MC Tubio[270,271,272], Izar Villasante[45], Nicola Waddell[295,296], Jeremiah A Wala[3,6,48], Joachim Weischenfeldt[8,107,108], Lixing Yang[299], Xiaotong Yao[284,300], Sung-Soo Yoon[292], Jorge Zamora[1,270,271,272] and Cheng-Zhong Zhang[3,301]

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Tubio[270,271,272], Daniel J Turner[336], Lara Urban[7,8], Sebastian M Waszak[8], David C Wedge[1,340,341], Joachim Weischenfeldt[8,107,108], David A Wheeler[154,155], Mark H Wright[59], Dai-Ying Wu[57], Tian Xia[342], Sergei Yakneen[8], Kai Ye[152,156], Venkata D Yellapantula[138,157], Jorge Zamora[1,270,271,272] and Bin Zhu[318]

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PCAWG Evolution & Heterogeneity Working Group

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Anja Füllgrabe[7], Mary J Goldman[12], **David Haussler#**[12,374], Wolfgang Huber[8], Maria Keays[7], Alfonso Muñoz[7], Brian D O'Connor[44,95], Irene Papatheodorou[7], Robert Petryszak[7], Elena Piñeiro-Yáñez[343], Alfonso Valencia[45,105], **Miguel Vazquez#**[45,106], John N Weinstein[375,376], Qian Xiang[44], Junjun Zhang[44] and **Jingchun Zhu#**[12]

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PCAWG Tumour-specific providers (pancreatic cancer) in Canada

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PCAWG Tumour-specific providers (prostate cancer) in Canada

Timothy A Beck[44,442], Vinayak Bhandari[9], **Paul C Boutros#**[9,125,134,135], **Robert G Bristow#**[125,443,444,445,446], Colin C Collins[142], Shadrielle MG Espiritu[9], Neil E Fleshner[447], Natalie S Fox[9], Michael Fraser[9], Syed Haider[9], Lawrence E Heisler[44], Vincent Huang[9], Emilie Lalonde[9], Julie Livingstone[9], John D McPherson[76,429,436], Alice Meng[448], Veronica Y Sabelnykova[9], Adriana Salcedo[9], Yu-Jia Shiah[9], Theodorus Van der Kwast[441] and Takafumi N Yamaguchi[9]

PCAWG Tumour-specific providers (gastric cancer) in China

Shuai Ding[449], Daiming Fan[450], Yong Hou[38,235], Yi Huang[144,145], Lin Li[38], Siliang Li[38,235], Dongbing Liu[38,235], Xingmin Liu[38,235], **Youyong Lu#**[28,29,30], Yongzhan Nie[450,451], Hong Su[38,235], Jian Wang[38], Kui Wu[38,235], Xiao Xiao[145], Rui Xing[30], **Huanming Yang#**[38], Shanlin Yang[449], Yingyan Yu[452], Xiuqing Zhang[38], Yong Zhou[38] and Shida Zhu[38,235]

PCAWG Tumour-specific providers (renal cancer) in the EU & France

Rosamonde E Banks[453], Guillaume Bourque[454,455], Alvis Brazma[7], Paul Brennan[456], **Mark Lathrop#**[455], Louis Letourneau[457], Yasser Riazalhosseini[455], Ghislaine Scelo[456], **Jörg Tost#**[458], Naveen Vasudev[459] and Juris Viksna[460]

PCAWG Tumour-specific providers (breast cancer) in the EU & United Kingdom

Sung-Min Ahn[461], Ludmil B Alexandrov[1,70,302], Samuel Aparicio[462], Laurent Arnould[463], MR Aure[464], Shriram G Bhosle[1], Ewan Birney[7], Ake Borg[465], Sandrine Boyault[466], Arie B Brinkman[467], Jane E Brock[468], Annegien Broeks[469], Adam P Butler[1], Anne-Lise Børresen-Dale[464,470], Carlos Caldas[278,471], Peter J Campbell[1,2], Suet-Feung Chin[278,471], Helen Davies[1,309,310], Christine Desmedt[472,473], Luc Dirix[474], Serge Serge[1], Anna Ehinger[475], Jorunn E Eyfjord[476], Aquila Fatima[204], John A Foekens[477], P Andrew Futreal[478], Øystein Garred[479,480], Moritz Gerstung[7,8], Dilip D Giri[481], Dominik Glodzik[1], Dorthe Grabau[482], Holmfridur Hilmarsdottir[476], Gerrit K Hooijer[483], Jocelyne Jacquemier[484], Se Jin Jang[485], Jon G Jonasson[476], Jos Jonkers[486], Hyung-Yong Kim[484], Tari A King[487,488,489], Stian Knappskog[1,490], Gu Kong[484], Savitri Krishnamurthy[352,491], Sunil R Lakhani[492], Anita Langerød[464], Denis Larsimont[493], Hee Jin Lee[485], Jeong-Yeon Lee[494], Ming Ta Michael Lee[478], Yilong Li[1], Ole Christian Lingjærde[495], Gaetan MacGrogan[496], John WM Martens[477], Sancha Martin[1,353], Iñigo Martincorena[1], Andrew Menzies[1], Sandro Morganella[1], Ville Mustonen[305,306,307], Serena Nik-Zainal[1,309,310,311], Sarah O'Meara[1], Iris Pauporté[20], Sarah Pinder[497], Xavier Pivot[498], Elena Provenzano[499], Colin A Purdie[500], Keiran M Raine[1], Manasa Ramakrishna[1], Kamna Ramakrishnan[1], Jorge Reis-Filho[481], Andrea L Richardson[204], Markus Ringnér[501], Javier Bartolomé

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PCAWG Tumour-specific providers (malignant lymphoma) in Germany

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PCAWG Tumour-specific providers (paediatric brain cancer) in Germany

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PCAWG Tumour-specific providers (prostate cancer) in Germany

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PCAWG Tumour-specific providers (oral cancer) in India

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PCAWG Tumour-specific providers (pancreatic cancer) in Italy

Davide Antonello[396], Stefano Barbi[403], Claudio Bassi[396], Samantha Bersani[399], Giada Bonizzato[400], Cinzia Cantù[400], Ivana Cataldo[399,400], Sara Cingarlini[36], Vincenzo Corbo[400,403], Maria Vittoria Davi[404], Angelo P Dei Tos[557], Matteo Fassan[558], Sonia Grimaldi[400], Luca Landoni[396], Rita T Lawlor[400], Claudio Luchini[399], Andrea Mafficini[400], Giuseppe Malleo[396], Giovanni Marchegiani[396], Michele Milella[36], Marco Miotto[396], Salvatore Paiella[396], Antonio Pea[396], Paolo Pederzoli[396], Borislav C Rusev[400], Andrea Ruzzenente[396], Roberto Salvia[396], Maria Scardoni[399], **Aldo Scarpa#**[400], Elisabetta Sereni[396], Michele Simbolo[403], Nicola Sperandio[400], Giampaolo Tortora[36,37] and Caterina Vicentini[400]

PCAWG Tumour-specific providers (biliary tract cancer) in Japan

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PCAWG Tumour-specific providers (gastric cancer) in Japan

Hiroyuki Aburatani#[256], Yasuhito Arai[33], Masashi Fukayama[562], Natsuko Hama[33], Fumie Hosoda[33], Shumpei Ishikawa[563], Hitoshi Katai[564], Mamoru Kato[351], Hiroto Katoh[563], Daisuke Komura[563], Genta Nagae[256,267], Hiromi Nakamura[33], Hirofumi Rokutan[351], Mihoko Saito-Adachi[351], **Tatsuhiro Shibata#**[33,34], Akihiro Suzuki[256,565], Hirokazu Taniguchi[34], Kenji Tatsuno[256], Yasushi Totoki[33], Tetsuo Ushiku[562], Shinichi Yachida[33,566] and Shogo Yamamoto[256]

PCAWG Tumour-specific providers (liver cancer) in Japan

Hiroyuki Aburatani[256], Hiroshi Aikata[567], Koji Arihiro[567], Shun-ichi Ariizumi[568], Keith A Boroevich[46,47], Kazuaki Chayama[567], Akihiro Fujimoto[47], Masashi Fujita[47], Mayuko Furuta[47], Kunihito Gotoh[569], Natsuko Hama[33], Takanori Hasegawa[83], Shinya Hayami[570], Shuto Hayashi[83], Satoshi Hirano[571], Seiya Imoto[83], Mamoru Kato[351], Yoshiiku Kawakami[567], Kazuhiro Maejima[47],

Satoru Miyano[83], Genta Nagae[256,267], **Hidewaki Nakagawa#**[47], Hiromi Nakamura[33], Toru Nakamura[571], Kaoru Nakano[47], Hideki Ohdan[567], Yasushi Rino[572], Aya Sasaki-Oku[47], **Tatsuhiro Shibata#**[33,34], Yuichi Shiraishi[83], Hiroko Tanaka[83], Yasushi Totoki[33], Tatsuhiko Tsunoda[46,207,208,209], Masaki Ueno[570], Rui Yamaguchi[83], Masakazu Yamamoto[568] and Hiroki Yamaue[570]

PCAWG Tumour-specific providers (biliary tract cancer) in Singapore

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PCAWG Tumour-specific providers (blood cancer) in South Korea

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PCAWG Tumour-specific providers (chronic lymphocytic leukaemia) in Spain

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PCAWG Tumour-specific providers (bone cancer) in the United Kingdom

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PCAWG Tumour-specific providers (chronic myeloid disorders) in the United Kingdom

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PCAWG Tumour-specific providers (oesophageal cancer) in the

United Kingdom

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PCAWG Tumour-specific providers (prostate cancer) in the United Kingdom

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PCAWG Tumour-specific providers (TCGA) in the United States

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