

SUPPLEMENTARY INFORMATION

Reconstructing evolutionary trajectories of mutation signature
activities in cancer using TrackSig

Rubanova et al.

Supplementary Note 1: Computing activity to mutational signatures

We apply topic modeling⁷ to infer signature activities. Within the time point, we separate mutation into K mutation types. Mutation types relate to vocabulary in topic modeling. The types used in TrackSig are described in the Results section. Then we use mixture of discrete distributions to infer signature activities. We describe this model below.

We represent each mutation as a K -dimensional binary vector – "one-hot-encoding" of a mutation type. "One-hot-encoding" of a mutation of type k is a binary vector where k -th component is equal to 1, and other components are zeros. We will denote $\mathbf{x}^{(n)}$ to be the "one-hot-encoding of mutation n ". A sample containing N mutations is represented as a $N \times K$ binary matrix \mathbf{X} , where each column corresponds one mutation.

$$\mathbf{x}^{(n)} = \begin{bmatrix} 0 \\ 0 \\ \dots \\ 1 \\ \dots \\ 0 \end{bmatrix}; \quad x_k^{(n)} = \begin{cases} 1, & \text{mutation } n \text{ belongs to type } k \\ 0, & \text{otherwise} \end{cases} \quad (1)$$

A mutation process is represented as a distribution over mutation types, known as a mutation signature. We will denote signature multinomials as K -dimensional probability vectors \mathbf{s}_i , where $i = \{1..M\}$ is an index over signatures. Signatures are fixed and are not updated during the training.

We aim to estimate signature activities \mathbf{m} – the proportion of mutations generated by each signature.

We will use the following notation:

K – number of mutation types

M – number of signatures

N – number of mutations

$\mathbf{x}^{(n)}$ – K -dimensional binary vector of mutation n

$x_k^{(n)}$ – k -th component of vector $\mathbf{x}^{(n)}$

\mathbf{s}_i – i -th signature (K -dimensional vector)

s_{ik} – k -th component of vector \mathbf{s}_i

\mathbf{m} – signature activities (mixture coefficients, M -dimensional vector)

m_i – i -th component of \mathbf{m} (signature activity of signature i)

z_n – signature assignment for mutation n

We represent mutation matrix \mathbf{X} as a mixture of signature multinomials $\mathbf{s}_1, \dots, \mathbf{s}_M$ with mixture coefficients \mathbf{m} :

$$\mathbf{X} \sim \text{Multinomial}(N; \sum_{i=1}^M m_i \mathbf{s}_i) \quad (2)$$

We denote z_n to be the signature assignment of mutation n . The probabilities of mutation n to be assigned to i -th signature are equal to the mixing coefficients:

$$p(z_n = i | \mathbf{m}) = m_i; \quad i \in \{1..M\} \quad (3)$$

The probability of a mutation n to be generated by signature i is given by:

$$p(\mathbf{x}^{(n)} | z_n = i, \mathbf{m}, \mathbf{s}_1, \dots, \mathbf{s}_M) = \prod_{k=1}^K s_{ik}^{x_k^{(n)}}; \quad i \in \{1..M\}; \quad n \in \{1..N\} \quad (4)$$

Then log likelihood of the collection of mutations in a sample:

$$\log L(\mathbf{X}|\mathbf{m}, \mathbf{s}_1, \dots, \mathbf{s}_K) = \sum_{n=1}^N \log p(\mathbf{x}^{(n)}|\mathbf{m}, \mathbf{s}_1, \dots, \mathbf{s}_K) = \sum_{n=1}^N \log \sum_{i=1}^M p(\mathbf{x}^{(n)}|z_n = i, \mathbf{m}, \mathbf{s}_1, \dots, \mathbf{s}_K) p(z_n = i|\mathbf{m}) \quad (5)$$

To estimate the activities, we fit mixing coefficients \mathbf{m} in each bin using Expectation-Maximization (EM) algorithm². The EM algorithm iterates between updating a posterior distribution over z_n and updating an estimate of the mixing coefficients \mathbf{m}

We start with initializing EM algorithm with uniform mixing coefficients:

$$\pi_i^{(0)} = \frac{1}{M}; \quad i \in \{1..M\} \quad (6)$$

Then, we repeat the following E-step and M-step until the algorithm converges.

In E-step, at the t -th iteration, the posterior probabilities of mutation assignments to signatures are estimated as such:

$$p(z_n = i|\mathbf{x}^{(n)}, \mathbf{m}^{(t-1)}, \mathbf{s}_1, \dots, \mathbf{s}_K) = \pi_i^{(t-1)} \prod_{k=1}^K s_{ik}^{x_k^{(n)}}; \quad i \in \{1..M\}; \quad n \in \{1..N\} \quad (7)$$

In M-step we update the estimates of the mixing coefficients:

$$\pi_i^{(t)} = \frac{1}{N} \sum_{n=1}^N p(z_i|\mathbf{x}^{(n)}, \mathbf{m}^{(t-1)}, \mathbf{s}_1, \dots, \mathbf{s}_K); \quad i \in \{1..M\} \quad (8)$$

The algorithm has converged when the value of π is updated by less than 0.001 between iterations. The resulting mixture coefficients as the activities of the mutational signatures. We show the activities as percentage for the convenience of interpretation.

Supplementary Note 2: Pruned Exact Linear Time (PELT) Algorithm

We adapt Pruned Linear Exact Time (PELT)² algorithm to detect change points in activity trajectories given cost function (likelihood) and BIC penalty. PELT is based on dynamic programming and uses heuristics to prune the set potential changepoints, thus reducing the computational time.

In this section, we will use the following notation:

T – number of time points

P – number of changepoints

M – number of signatures

Locating change points

As described in the Methods section, we separate mutations into bins 100 mutations, each of which represents one time point. Our input is the set of mutation counts across 96 types for each time point: $y_{1:T} = (y_1, \dots, y_T)$. We aim to find P changepoints, or in other words, $P + 1$ segments. We denote $\tau_{1:P} = (\tau_1, \dots, \tau_P)$ to be the boundaries for our segments, meaning each segment will contain the data points $y_{\tau_{i-1}+1} \dots y_{\tau_i}$.

Given a set of changepoints we can compute the likelihood of the data the following way. We fit mutational signatures within each segment (treating all mutations within each segment as one bin) and compute the likelihood $\hat{L}(y_{\tau_{i-1}+1} \dots y_{\tau_i})$ as described in Supplementary Note 1. The total likelihood is the sum of likelihoods in each segment:

$$\hat{L} = \sum_{i=1}^{P+1} L(y_{(\tau_{i-1}+1):\tau_i})$$

We aim to minimize the Bayesian Information Criterion (BIC):

$$\text{BIC} = -2\ln\hat{L} + k \cdot \ln(T)$$

where k is the number of parameters in our model and T is the number of time points. In our case $k = (P + 1) \cdot (M - 1)$ as we fit $(M - 1)$ signature activities in $(P + 1)$ segments (recall that signature activities sum to 1).

We adapt PELT objective to minimize the BIC criterion. PELT aims to minimize sum of cost functions at each time point, while using a penalty β for each placed changepoint

$$\text{minimize} \sum_{i=1}^{P+1} C(y_{(\tau_{i-1}+1):\tau_i}) + \beta(P + 1)$$

Intuitively, we are trying to select changepoints which result in the lowest cost (or highest likelihood) while reducing the penalty associated with adding changepoints. We set the parameters as follows to make the PELT equivalent to BIC:

$$C = -2\hat{L}; \quad \beta = (M - 1)\ln(T)$$

TrackSig-PELT algorithm finds the changepoints as follows. The algorithm starts with finding a partial solution in a subset of the timeline and then increases the search space until changepoints are located over the whole timeline. The algorithm keeps track of the time points R_{τ^*} that satisfy the pruning condition and which will be considered as potential changepoints at further iterations. At each iteration τ^* , the algorithm considers adding a new changepoint out of the set of available time points R_{τ^*} . To score a potential new changepoint, the algorithm refits the activities in bins formed by a potential changepoint. It finds a time point τ' with the smallest likelihood and adds it to the list of changepoints cp. Then the list of available time points R_{τ^*} is updated: the potential changepoints are removed from further consideration if the increase in likelihood associated with this changepoint does not exceed the complexity penalty β .

Pruning

PELT provides an improvement in runtime by pruning certain changepoints from consideration. We prune time point t if for all $t < s < T$:

$$C(y_{(t+1):s}) + C(y_{(s+1):T}) + \beta \leq C(y_{(t+1):T}) \quad (9)$$

The cost of placing the last changepoint prior to T at t will always be higher than cost of placing the last changepoint prior to T at s . Given this result, we can eliminate t as a potential changepoint for all iterations of the dynamic programming algorithm as it will never be optimal going forwards.

Algorithm 1 TrackSig PELT Method²

Input: Mutation counts at each time step (y_1, y_2, \dots, y_T)

- 1: **Initialize:** Set $\beta = (M - 1)\ln(T)$; $F(0) = 0$; cp = {}; $R_1 = \{0\}$
 - 2: **for** $\tau^* = 1, \dots, T$ **do**
 - 3: Calculate $F(\tau^*) = \min_{\tau \in R_{\tau^*}} [F(\tau) + C(y_{(\tau+1):\tau^*}) + \beta]$, where $C(y_{(\tau+1):\tau^*}) = -2\hat{L}(y_{(\tau+1):\tau^*})$
 - 4: Let $\tau' = \arg \min_{\tau \in R_{\tau^*}} [F(\tau) + C(y_{(\tau+1):\tau^*}) + \beta]$
 - 5: Append τ' to cp
 - 6: Set $R_{\tau^*+1} = \{\tau \in R_{\tau^*} \cup \{\tau^*\} : F(\tau) + C(y_{\tau+1:\tau^*}) + \beta \leq F(\tau^*)\}$
 - 7: **end for**
 - 8: **return** cp – a set of changepoints
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Supplementary Note 3: Clonal evolution simulations

Choice of signatures We generate the simulations with four active signatures: S1, S5 and two randomly-sampled signatures, which we will call A1, A2. Two other signatures A1, A2 are sampled from uniformly from the set of PCAWG (excluding signatures S1, S5, S7 and "artifact signatures" S40-S60). We decided to exclude signature S7 (sum of signatures S7a, S7b, S7c, S7d) as it had a distribution similar to uniform and was easily confused with other signatures both by TrackSig and DeconstructSigs. We include signatures S1 and S5 in all simulations as they are present in all real samples in PCAWG.

We sample activities separately for each cluster. We sample the activity of S1 from [0.03, 0.1] interval, S5 from [0.05, 0.15] interval, A1 from [0.4, 0.7] interval. The remaining activity is assigned to signature A2 (all signature activities have to sum to 1).

Sampling mutation types To sample mutation types from a signature, we treat it as a multinomial distribution and sample from it. The number of mutations sampled from each signature is equal to the activity of this signature multiplied by the total number of mutations.

Sampling number of ref and alt alleles Here we describe sampling number of ref and alt alleles for each mutation of the cluster, given the cluster CCF, number of mutation in the cluster and desired mean mutation depth. We tested mean mutation depths of 10, 30 and 100.

For each mutation, we sample read depth d from Poisson distribution with specified mean depth. Then we compute the probability of alt allele as $p = ccf * \frac{\text{mutantCN}}{\text{totalCN}}$, where ccf is CCF of the current cluster. Finally, we sample number of alt alleles a from a Binomial(d, p) and set the number of ref alleles to be the difference between depth and alt alleles.

In simulations with one and two clusters we use normal copy number of 2, mutant copy number of 1 and purity 1. Each simulation has 5000 mutations in total. We generate 100 simulations of each of five simulation types (one-cluster, two-cluster, branching, cna gain and infinite site assumption) and for each read depth that we tested.

Basic simulations

First, we create simple one- and two-cluster simulations.

One-cluster simulations We create one cluster with the average cluster CCF=1. Number of ref and alt alleles for each mutation is sampled as described in the previous section. We sample activity of the first active signature A1 from the interval Uniform([0.4,0.7]), activity of time-related signature S1 from Uniform([0.03,0.1]), and time-related signature S5 from Uniform([0.05,0.15]). The remaining activity is assigned to the signature A2. Finally, we sample mutation types from each of active signatures. Number of mutation types sampled from each signature is proportional to their activities.

Two-cluster simulations We create the first cluster with CCF=1 as described above. For the second cluster we sample ccf from Uniform([0.2,0.6]) distribution. To sample signature activities, we follow the procedure similar to one-cluster simulations. We sample activity of the first active signature A1 from Uniform([0.4,0.7]) for the clonal cluster, and Uniform([0.2,0.4]) for the second cluster to ensure the signature activity change between the two clusters. Full procedure is shown in Supplementary Note 6.

Branching

To test violation of TrackSig assumptions, we create simulations with branching, CNA gain or violation of infinite site assumption.

To simulate branching, we create three clusters. The clonal cluster is always assigned CCF=1. The CCF for the last cluster (with the smallest CCF) is sampled uniformly from [0.2, 0.35]. The middle cluster CCF is sampled such that it has at least 0.15 gap on CCF scale with other clusters. Additionally, we ensure that sum of CCFs of the second and third clusters does not exceed 1 (otherwise the clusters cannot be branched).

In branching simulations, we expect to see the signature activity for A1 signature decreases at the transition to the second cluster and increases again at the transition to the third subclone. If such step-like behavior of is observed in real data, we consider this a sign of branching. Note that if we reversed the order of the branched clusters and assigned the same signature activities to the first and second clusters, it wouldn't be possible to distinguish between these two clusters since TrackSig can only find changepoints based on signature change.

To show the effect of branching on signature trajectories, we assign similar activities to the first (clonal) and third cluster (with the smallest CCF), but introduce a signature change in the second (middle) cluster. To do this, we sample signature activity for A1 from Uniform([0.4,0.7]), calculate the exposures for other signatures and assign the same activities to the first and last cluster. For the middle cluster, we sample activity for A1 signature from Uniform([0.2, 0.4]). As before, we sample activity of time-related signature S1 from Uniform([0.03,0.1]), and time-related signature S5 from Uniform([0.05,0.15]) and assign the remaining activity to A2.

CNA gain

CNA gain simulations are based on the branching simulations described above and has three clusters: clonal and two subclones.

We introduce a CNA gain for 10% of mutations in the clonal cluster: 5% of mutations have CNA gain on the mutant allele and 5% have CNA gain on reference allele. Thus, 10% of mutations get total copy number 3 and mutant copy number of 2 and 1 respectively. We assume that these copy number changes are inherited by both subclones. To simulate the CNA change, we adjust the mutantCN and totalCN parameter in Supplementary Note 7 for 10% of mutations in each cluster. We provide total copy number a input to both TrackSig and SciClone.

Violation of infinite site assumption

To simulate the violation of infinite site assumption (ISA), we create four clusters. The first three clusters are created the same way as in the branching simulation. The forth cluster simulates mutations that occurred in both clusters independently, thus violating ISA. The CCF of the forth cluster is the sum of CCFs of the two subclonal clusters. We assign 3% of all mutation to the forth cluster. As expected, the presence of mutations that violate ISA don't affect signature activity trajectories.

Neutral Evolution Mutations

To make our simulations more realistic, we add mutations which emerged due to neutral evolution. We follow Williams et al.² for generating mutations from neutral evolution. First, we establish the number of neutral mutations to be generated. Then we sample those mutations according to the power-law distribution $\frac{1}{f^2}$, where f is variant allele frequency. Both steps are described in more detail below.

The number of neutral mutations is computed as follows:

$$M(f_c) = s_e \left(\frac{1}{f_{\min}} - \frac{1}{f_c} \right) \quad (10)$$

where f_c is the variant allele frequency (VAF) of the cluster, f_{\min} is a minimal VAF in consideration and s_e is effective mutation rate. For clonal cluster, $f_c = 0.5$. We only consider mutations with 3 or more mutant reads. Therefore, we set $f_{\min} = \frac{3}{d}$, where d is the mean depth of the simulation. We use $s_e = 16^2$.

Next, we sample $M(f_c)$ mutations according to the power-law distribution on interval $[f_{\min}; f_c]$. Cumulative distribution function (CDF) of power-law distribution on the interval $[f_{\min}; f_c]$ is the following:

$$\text{CDF}(f) = \frac{\frac{1}{f_c} - \frac{1}{f}}{\frac{1}{f_c} - \frac{1}{f_{\min}}} \quad (11)$$

To sample from this distribution, we take samples from uniform distribution and then use inverse cumulative distribution function (I-CDF) to transform them into samples from power-law distribution. Inverse CDF function takes the following form:

$$f = \frac{1}{\frac{1}{f_c} - u\left(\frac{1}{f_c} - \frac{1}{f_{\min}}\right)} \quad (12)$$

where f is our target allele frequency (i.e. sample from the power-law) and u is a sample from uniform distribution.

Note that the approach we used to sample neutrally-evolving mutations may not reflect the true, complex clonal dynamics that would be better represented with a branching process. Although our one cluster case precisely matches a standard neutral model², using the same model for the two cluster simulations ignores the effect that the introduction of subclone has on the number and VAF distribution of neutrally-evolving mutations.

It does, however, establish a lower bound on performance. The introduction of a subclone is likely to reduce the number of neutral mutations, though their VAF distributions would not drastically differ, and the “neutral mode” near the detection limit would be composed of mutations from both clones rather than from just one clone. These differences would make the reconstruction problem easier for TrackSig. As such, although the neutral model is not correct in the two cluster case, the one we used provides lower bound on TrackSig’s performance.

Our results are shown in additional bar in Figure 3b and Supplementary Figure 2. At depth 10 and 30 TrackSig’s ability to detect subclones is not impacted by neutral mutations. At depth 100, both TrackSig and SciClone detect an extra cluster, which is consistent findings of Williams et al.²: neutral evolution can be detected at a minimal depth of 100. Figure 4b shows the example of generated simulation at depth 10.

Supplementary Note 4: SciClone+DeconstructSigs baseline

To showcase the potential of our method, we compared TrackSig to SciClone+DeconstructSigs pipeline which is commonly used to infer signature activities.

SciClone + DeconstructSigs

First, we clustered SNVs using SciClone (v1.1)². SciClone uses variational Bayesian mixture model to cluster SNVs based on their CCF. We provided CNA calls as a part of input for SciClone, same as we do in TrackSig. Since we needed to test clustering at low depth, we used minimum read depth of 1. We report the results with two clustering methods in SciClone: Beta mixture model (BMM, default) and Beta-binomial mixture model (Binomial BMM).

Finally, we took the mutation clustering performed by SciClone and computed activities of mutational signatures withing each cluster using DeconstructSigs (v1.8.0)². We used the same set of PCAWG signatures as we used in TrackSig. We fit the same set of active signatures with DeconstructSigs as we do in TrackSig.

PyClone

We attempted to use PyClone (v0.13.1)² instead of SciClone. However, PyClone uses a Markov Chain Monte Carlo (MCMC) approach, and has a time complexity of $O(n^2)$. This is feasible for the number of mutations validated on in the paper PyClone is described, but quickly becomes intractable for whole genome sequencing containing thousands of mutations. We didn’t manage to run the on samples containing more than 1000 SNVs.

Supplementary Note 5: Analysis of multi-region cases

To compare mutational signatures across multiple samples, we run TrackSig separately on each sample. Samples from the same tumour can have different active signatures. Therefore, for each tumour, we split the samples into groups that have the same set of active signatures and compare samples only within the group. To compare signatures of the clonal cluster, we compute KL divergence and mean activity difference between the first time points of the samples with the same active signatures. Within each group of samples with the same set of active signatures, we compute the pair-wise mean activity difference and KL difference between all pairs of samples within the group. We report the mean metrics of all pairs within the signature group.

Supplementary Note 6 Simulation algorithm for two clusters

Input: mean mutation depth d , number of mutations N

```
1: mutantCN = 1
2: totalCN = 2
3: Sample CCFs for each cluster:
4:  $ccf_1 = 1$ . ▷ CCF of the clonal cluster is set to 1.
5:  $ccf_2 \sim \text{Uniform}(0.2, 0.6)$ 
6: Sample number of mutations per cluster:
7:  $N_{c2} = \lfloor ccf_2 * N \rfloor$ 
8:  $N_{c1} = N - N_{c2}$ 
9: Sample two active signatures A1, A2 for the current tumour sample, excluding S1 and S5
10: Set active signatures to (S1, S5, A1, A2)
11: for each cluster  $i$  in 1..2 do
12:   Sample signature activities:
13:      $e_{S1} \sim \text{Uniform}(0.03, 0.1)$ 
14:      $e_{S5} \sim \text{Uniform}(0.05, 0.15)$ 
15:      $e_{A1} \sim \text{Uniform}(0.45, 0.7)$  for the first cluster and  $e_{A1} \sim \text{Uniform}(0.2, 0.4)$  for the second cluster
16:      $e_{A2} = 1 - \text{sum}(e_{S1}, e_{S5}, e_{A1})$ 
17:   Generate number of alt and ref alleles for  $N_{c_i}$  mutations:
18:   for each mutation  $j$  do
19:      $\text{depth}_j = \text{Poisson}(d)$  ▷ Sample depth for each mutation
20:      $\text{prob}_j = ccf_i * \frac{\text{mutantCN}}{\text{totalCN}}$  ▷ Sample probability of mutant allele for mutation  $j$ 
21:      $\text{alt}_j \sim \text{Binomial}(\text{depth}_j, \text{prob}_j)$  ▷ Sample number of variant alleles from a Binomial)
22:      $\text{ref}_j = \text{depth}_j - \text{alt}_j$ 
23:   end for
24:   Sample types of mutations according to signature activities
25:   for each signature  $s$  in active signatures (S1, S5, A1, A2) do
26:     Notation:
27:      $e_s$  ▷ Signature activity
28:      $n_s$  ▷ Mutations per signature
29:     def $_s$  ▷ Signature definition
30:
31:      $n_s = N_{c_i} * e_s$ 
32:     Create vector of trinucleotide counts across  $n_s$  mutations from signature  $s$ :
33:      $c_s \sim \text{Multinom}(n_s, \text{def}_s)$ 
34:   end for
35:   Sum counts  $c_s$  over all signatures
36:   Convert trinucleotide counts into a vector of mutation types of length  $N_{c_i}$ 
```

Supplementary Note 7 Simulation algorithm for branching with three clusters

Input: mean mutation depth d , number of mutations N

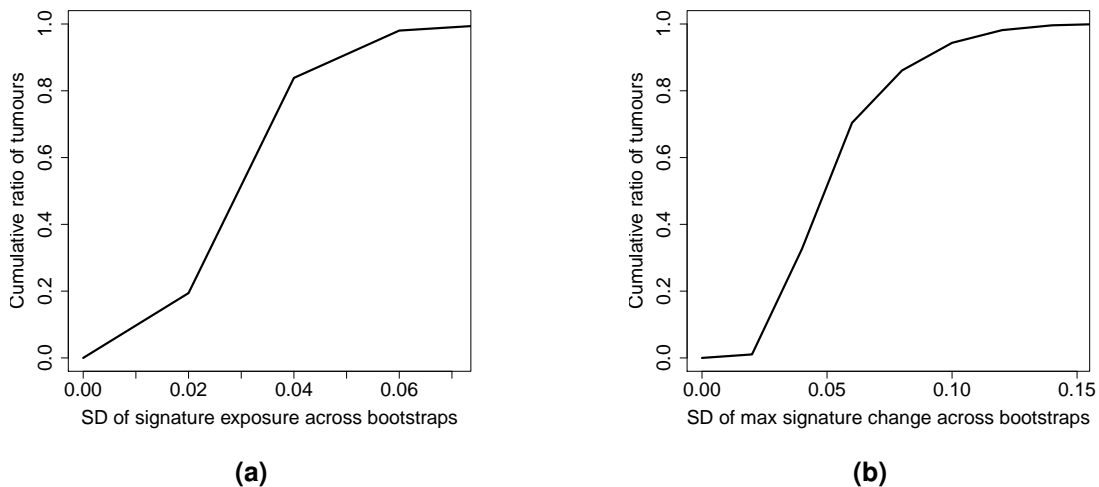
```
1: mutantCN = 1
2: totalCN = 2
3: Sample CCFs for each cluster:
4:  $ccf_1 = 1$ . ▷ CCF of the clonal cluster is set to 1.
5:  $ccf_3 \sim \text{Uniform}(0.2, 0.35)$ 
6:  $ccf_2 \sim \text{Uniform}(ccf_2 + 0.15; 1 - ccf_2 - 0.15)$  ▷ CCF of the middle cluster is set to be at least 0.15 CCF
   apart from third cluster. Sum of CCFs from second and third clusters should not exceed 1 because they
   are branched.
7: Sample number of mutations per cluster:
8:  $N_{c3} = \lfloor ccf_3 * N \rfloor$ 
9:  $N_{c2} = \lfloor ccf_2 * N \rfloor$ 
10:  $N_{c1} = N - N_{c2} - N_{c3}$ 
11: Sample two active signatures A1, A2 for the current tumour sample, excluding S1 and S5
12: Set active signatures to (S1, S5, A1, A2)
13: for each cluster  $i$  in 1..3 do
14:   Sample signature activities:
15:      $e_{S1} \sim \text{Uniform}(0.03, 0.1)$ 
16:      $e_{S5} \sim \text{Uniform}(0.05, 0.15)$ 
17:      $e_{A1} \sim \text{Uniform}(0.45, 0.7)$  if  $i \in \{1, 3\}$  and  $e_{A1} \sim \text{Uniform}(0.2, 0.4)$  if  $i == 2$ 
18:      $e_{A2} = 1 - \text{sum}(e_{S1}, e_{S5}, e_{A1})$ 
19:   Generate number of alt and ref alleles for  $N_{c_i}$  mutations:
20:   for each mutation  $j$  do
21:      $\text{depth}_j = \text{Poisson}(d)$  ▷ Sample depth for each mutation
22:      $\text{prob}_j = ccf_i * \frac{\text{mutantCN}}{\text{totalCN}}$  ▷ Sample probability of mutant allele for mutation  $j$ 
23:      $\text{alt}_j \sim \text{Binomial}(\text{depth}_j, \text{prob}_j)$  ▷ Sample number of variant alleles from a Binomial)
24:      $\text{ref}_j = \text{depth}_j - \text{alt}_j$ 
25:   end for
26:   Sample types of mutations according to signature activities
27:   for each signature  $s$  in active signatures (S1, S5, A1, A2) do
28:     Notation:
29:      $e_s$  ▷ Signature activity
30:      $n_s$  ▷ Mutations per signature
31:     def $_s$  ▷ Signature definition
32:
33:      $n_s = N_{c_i} * e_s$ 
34:     Create vector of trinucleotide counts across  $n_s$  mutations from signature  $s$ :
35:      $c_s \sim \text{Multinom}(n_s, \text{def}_s)$ 
36:   end for
37:   Sum counts  $c_s$  over all signatures
38:   Convert trinucleotide counts into a a vector of mutation types of length  $N_{c_i}$ 
```

Supplementary Tables

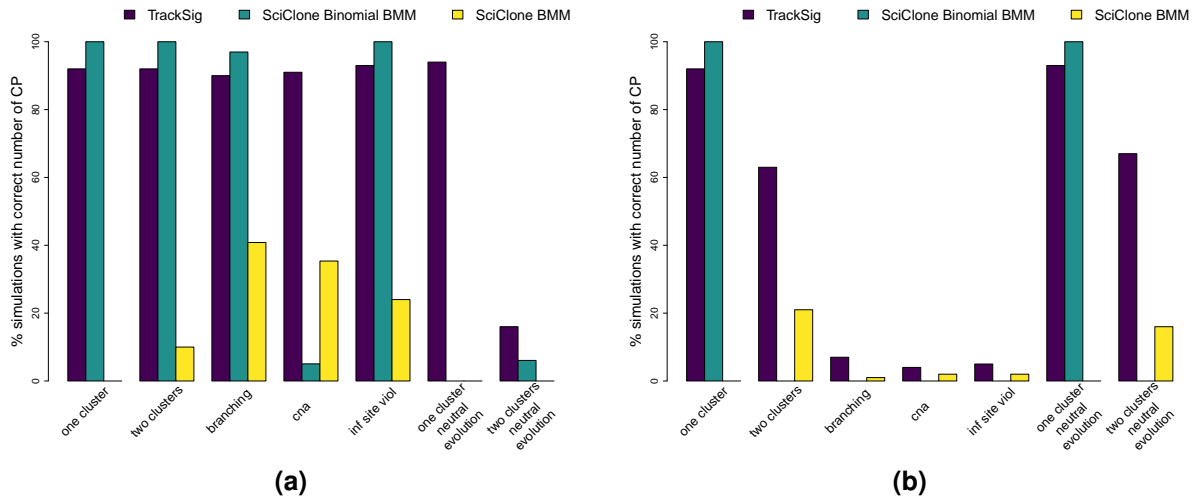
		# true changepoints			
		0	1	2	3
# predicted changepoints	0	0.91	0.004	0	0
	1	0.061	0.9	0.019	0.001
	2	0.024	0.078	0.898	0.037
	3	0.006	0.02	0.075	0.861
	4	0.002	0.001	0.009	0.091
	5	0	0.001	0.001	0.002

Supplementary Table 1. TrackSig change-point prediction performance in non-parametric simulations. Each cell shows the percentage of simulations which have certain number of predicted change-points (normalized within a column). Note that there might be several predicted change-points that correspond to the same change-point in the ground truth. We consider predicted change-points to match the ground-truth change-point if it is located no more than 3 time points away.

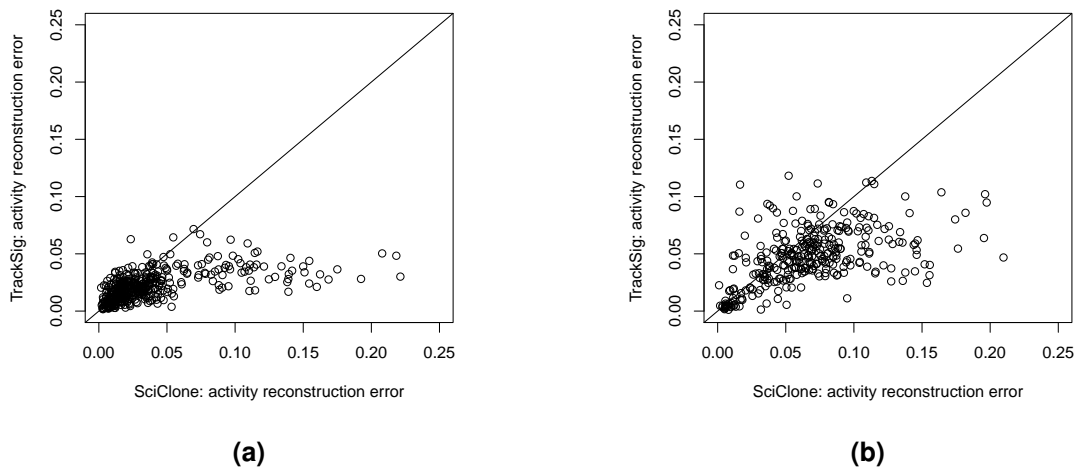
Supplementary Figures



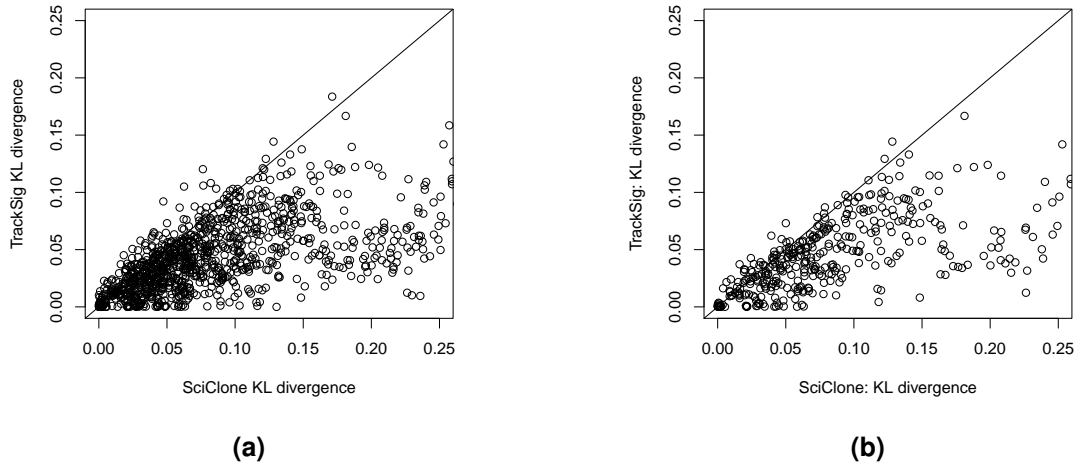
Supplementary Figure 1. Discrepancies in signature activities on bootstrap data. (a) Standard deviations of signature activities at each time point for each signature across bootstraps. (b) Standard deviations of change in signature activity at each time point for each signature across bootstraps.



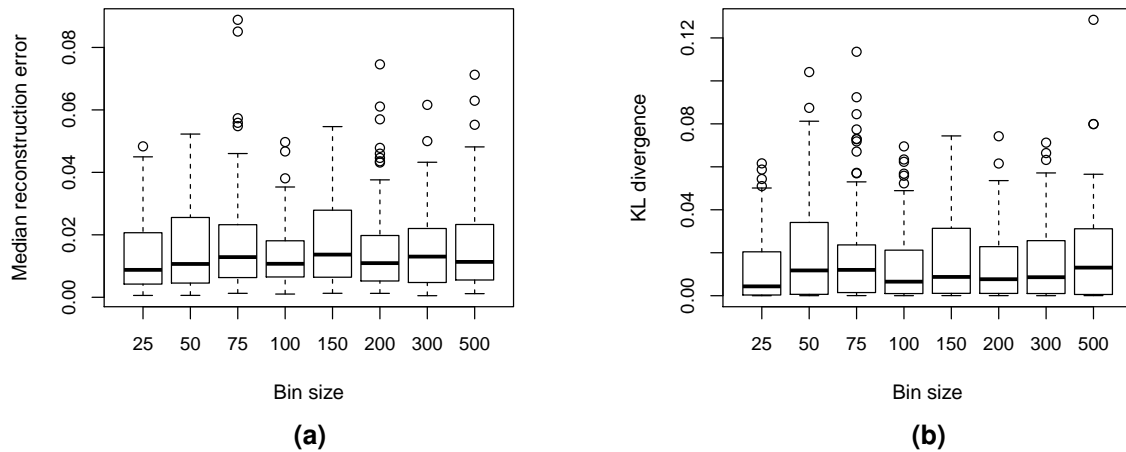
Supplementary Figure 2. Subclone detection accuracy in clonal evolution simulations. Each method was evaluated on all simulation scenarios in Supplementary Note 3, shown in X-axis. Y-axis shows the percentage of simulations where the method predicted the correct number of change points. Comparison was performed on simulated data with read depth (a) 100 and (b) 10.



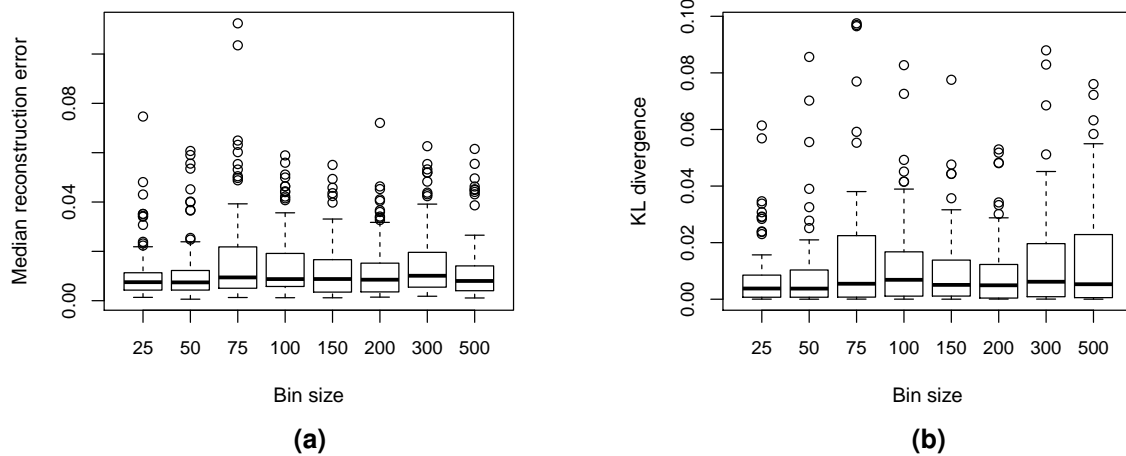
Supplementary Figure 3. Absolute activity errors in clonal evolution simulations. Scatterplots show median per-mutation activity reconstruction error (absolute activity difference) between the method (TrackSig and SciClone) and the true activities on clonal evolution simulations. (a) Depth 100. Mean activity error: TrackSig 0.022, SciClone 0.039. (b) Depth 10. Mean activity error: TrackSig 0.048, SciClone 0.068.



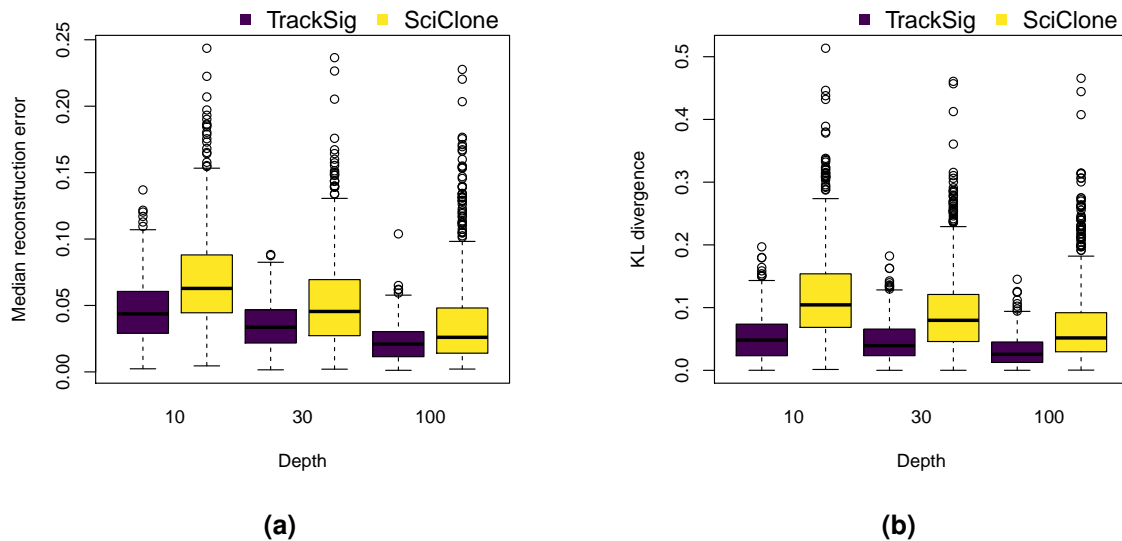
Supplementary Figure 4. KL divergences in clonal evolution simulations. Scatterplots show mean per-mutation KL divergence between predicted and true exposures on clonal evolution simulations. **(a)** All simulations. Mean per-mutation KL divergence: TrackSig 0.044, SciClone 0.091. **(b)** Depth 30. Mean KL divergence: TrackSig 0.047, SciClone 0.095.



Supplementary Figure 5. Dependence of activity errors on TrackSig bin sizes at depth 30. **(a)** Median absolute, per-mutation difference between true activities and activities estimated by TrackSig for different bin sizes at depth 30. **(b)** Mean per-mutation KL divergence between estimated and true activities for different bin sizes at depth 30. In all subfigures, error bars extend to 1.5 times the inter-quartile range, or the point the furthest from the mean, whichever is less.

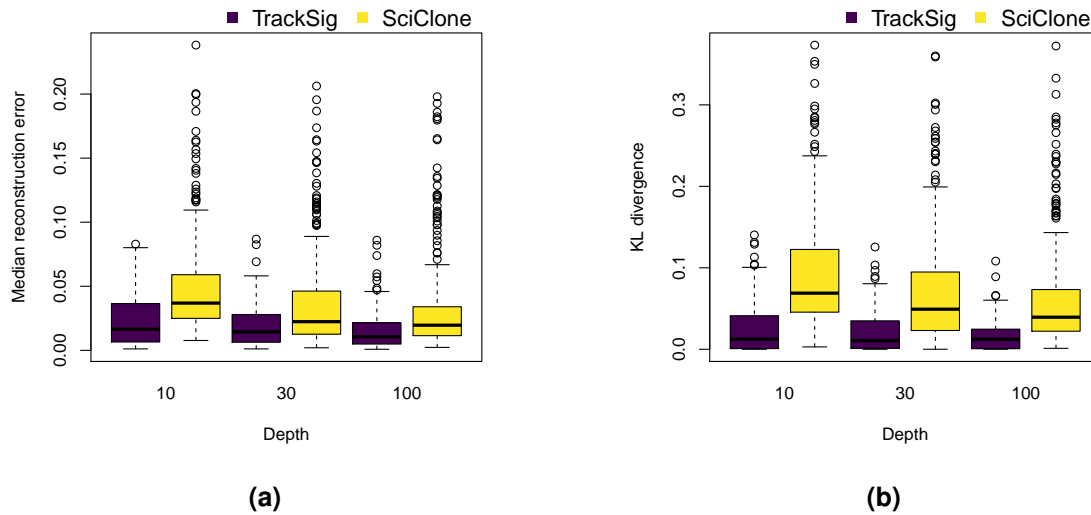


Supplementary Figure 6. Dependence of activity errors on TrackSig bin sizes at depth 100. (a) Median absolute, per-mutation difference between true activities and activities estimated by TrackSig for different bin sizes at depth 100. (b) Mean per-mutation KL divergence between estimated and true activities for different bin sizes at depth 100. In all subfigures, error bars extend to 1.5 times the inter-quartile range, or the point the furthest from the mean, whichever is less.



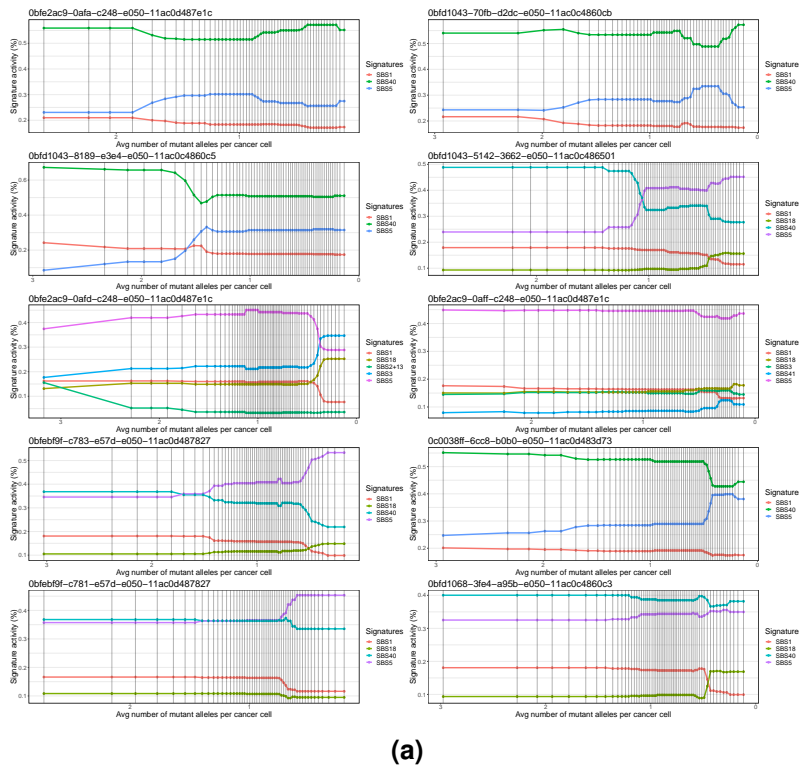
Supplementary Figure 7. Activity errors by depth in non-neutral simulations. Comparison of TrackSig and SciClone (BMM noise model, default) on all simulation scenarios described in Supplementary Note 3, except neutral evolution. Performance was evaluated across different simulated read depths, shown in X-axis.

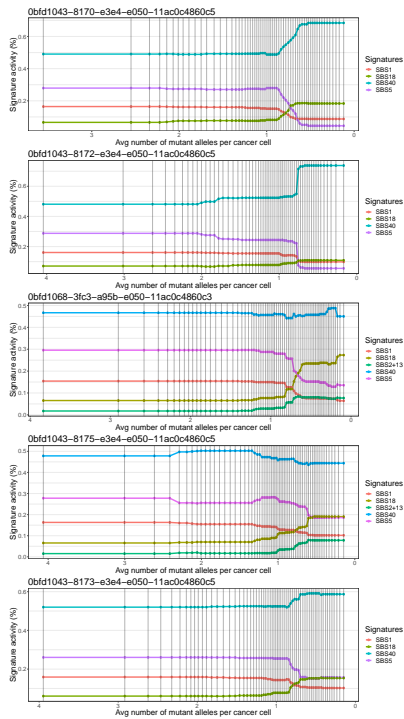
In all subfigures, error bars extend to 1.5 times the inter-quartile range, or the point the furthest from the mean, whichever is less. (a) Median absolute, per-mutation difference between true activities and activities estimated by each method. (b) Mean per-mutation KL divergence between true activities and activities estimated by each method.



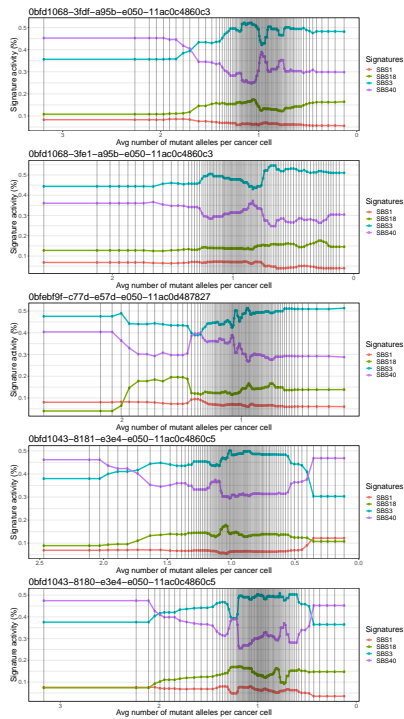
Supplementary Figure 8. Activity errors by depth in simulations with neutral mutations.

Comparison of TrackSig and SciClone (BMM noise model, default) on one and two cluster simulations with the inclusion of neutral mutations, as described in Supplementary Note 3. Performance was evaluated across different simulated read depths, shown in X-axis. In all subfigures, error bars extend to 1.5 times the inter-quartile range, or the point the furthest from the mean, whichever is less. **(a)** Median absolute, per-mutation difference between true activities and activities estimated by each method. **(b)** Mean, per-mutation, KL divergence between true activities and activities estimated by each method.

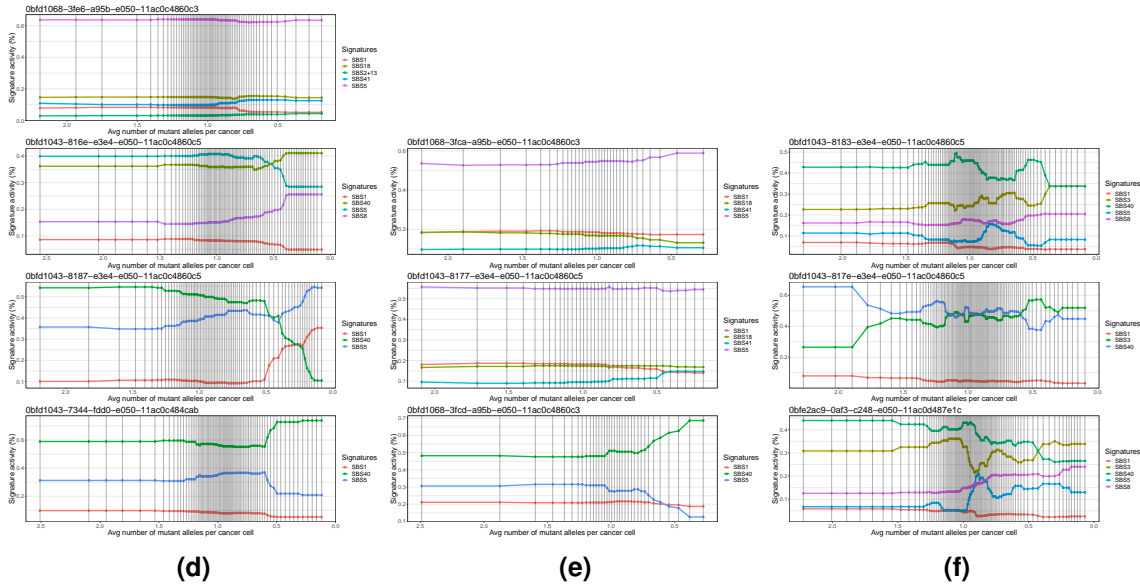




(b)



(c)



Supplementary Figure 9. Evolutionary trajectories for multiple samples from same tumour. Each subplot shows signature trajectories for different samples from the same tumour. Signature trajectories shown are the mean of 30 bootstrap trajectories and therefore are not piece-wise constant. We report mean activity difference and KL divergence between the activities in the clonal cluster only. We compare clonal activities across the groups of samples with the same set of active signatures.

Tumour DO51954: Group 1 with active signatures "SBS1 SBS5 SBS40": mean activity diff 0.0573, KL divergence 0.05. Group 2 with signatures "SBS1 SBS5 SBS18 SBS40": mean activity diff 0.036, KL divergence 0.018

Tumour DO51958: Group 1 with active signatures "SBS1 SBS5 SBS18 SBS40": mean activity diff 0.014, KL divergence 0.002. Group 2 with signatures "SBS1 SBS5 SBS18 SBS40 SBS2+13": mean activity diff 0.008, KL divergence 0.001.

Tumour DO51965: Group with active signatures "SBS1 SBS3 SBS18 SBS40": mean activity diff 0.042, KL divergence 0.028.

Tumour DO51953: Group with active signatures "SBS1 SBS5 SBS40": mean activity diff 0.031, KL divergence 0.005.

Tumour DO51959: Group with active signatures "SBS1 SBS5 SBS18 SBS41": mean activity diff 0.011 , KL divergence 0.0014.

Tumour DO51962: Group with signatures "SBS1 SBS3 SBS5 SBS8 SBS40": mean activity diff 0.037, KL divergence 0.031.

Supplementary Note 8

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Patterns of structural variations, signatures, genomic correlations, retrotransposons, mobile elements

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Mutation signatures and processes

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Germline cancer genome

Ludmil B Alexandrov^{2,317}, Eva G Alvarez^{287,288,289}, Adrian Baez-Ortega²⁹⁰, Matthew H Bailey^{138,139}, Matia Bosio^{46,136,147}, G Steven Bova³³⁰, Alvis Brazma⁷, Alicia L Bruzos^{287,288,289}, Ivo Buchhalter^{52,53,54}, Carlos D Bustamante^{60,61}, Atul J Butte³³¹, Andy Cafferkey⁷, Claudia Calabrese^{7,8}, Peter J Campbell^{1,2}, Stephen J Chanock³³², Nilanjan Chatterjee^{333,334}, Jieming Chen^{123,335}, Francisco M De La Vega^{59,59,60,61,62}, Olivier Delaneau^{336,337,338}, German M Demidov^{136,147,339}, Anthony DiBiase³⁴⁰, Li Ding^{138,139,146}, Oliver Drechsel^{136,147}, Lewis Jonathan Dursi^{9,65}, Douglas F Easton^{341,342}, Serap Erkek⁸, Georgia Escaramis^{147,343,344}, **Xavier Estivill**²⁹⁶, Erik Garrison², Mark Gerstein^{120,121,122,123}, Gad Getz^{3,4,5,6}, Dmitry A Gordenin²⁹⁷, Nina Habermann⁸, Olivier Harismendy⁷³, Eoghan Harrington³⁴⁵, Shuto Hayashi⁸⁷, José María Heredia-Genestar³⁴⁶, Aliaksei Z Holik¹⁴⁷, Xing Hua³³², Kuan-lin Huang^{139,347}, Seiya Imoto^{86,87}, Sissel Juul³⁴⁵, Ekta Khurana^{117,118,130,131}, Hyung-Lae Kim²⁷, Youngwook Kim^{96,97}, Leszek J Klimczak³⁰⁶, **Jan O Korbel**^{7,8}, Roelof Koster³⁴⁸, Sushant Kumar^{122,123}, Ivica Letunic¹⁵⁵, Yilong Li², Tomas Marques-Bonet^{111,135,346,349}, R Jay Mashl^{139,156}, Simon Mayes³⁵⁰, Michael D McLellan^{138,139,146}, Lisa Mirabello³³², Francesc Muias^{136,147,339}, Hidewaki Nakagawa⁴⁸, Arcadi Navarro^{111,135,346}, Steven J Newhouse⁷, Stephan Ossowski^{136,147,157}, Esa Pitkänen⁸, Aparna Prasad¹³⁶, Raquel Rabionet^{136,147,351}, Benjamin Raeder⁸, Tobias Rausch⁸, Steven A Roberts³¹³, Bernardo Rodriguez-Martin^{287,288,289}, Gunnar Rättsch^{90,93,94,107,108,109}, Natalie Saini²⁹⁷, Matthias

Schlesner^{52,110}, Roland F Schwarz^{7,84,250,260}, Ayellet V Segre^{3,3,352}, Tal Shmaya⁵⁹, Suyash S Shringarpure⁶¹, Nikos Sidiropoulos¹¹³, Reiner Siebert^{262,761}, Jared T Simpson^{9,160}, Lei Song³³², Oliver Stegle^{7,8,265}, Hana Susak^{136,147}, Tomas J Tanskanen³⁵³, Grace Tiao³, Marta Tojo²⁸⁹, Jose MC Tubio^{287,288,289}, Daniel J Turner³⁵⁰, Lara Urban^{7,8}, Sebastian M Waszak⁸, David C Wedge^{2,354,355}, Joachim Weischenfeldt^{8,113,114}, David A Wheeler^{164,165}, Mark H Wright⁶¹, Dai-Ying Wu⁵⁹, Tian Xia³⁵⁶, Sergei Yakneen⁸, Kai Ye^{161,166}, Venkata D Yellapantula^{167,168}, Jorge Zamora^{2,287,288,289} and Bin Zhu³³²

Tumor subtypes and clinical translation

Fatima Al-Shahrour³⁵⁷, Gurnit Atwal^{9,10,358}, Peter J Bailey²³⁶, **Andrew V Biankin**^{359,360,361,362}, Paul C Boutros^{9,133,142,143}, Peter J Campbell^{1,2}, David K Chang^{360,362}, Susanna L Cooke³⁶², Vikram Deshpande¹⁰⁶, Bishoy M Faltas¹⁰⁹, William C Faquin¹⁰⁶, **Levi Garraway**⁴⁹, Gad Getz^{3,4,5,6}, **Sean M Grimmond**³⁶³, Syed Haider⁹, **Katherine A Hoadley**^{247,248}, Wei Jiao⁹, Vera B Kaiser³⁶⁴, Rosa Karlič³⁶⁵, Mamoru Kato³⁶⁶, Kirsten Kübler^{3,6,106}, Alexander J Lazar³⁶⁷, Constance H Li^{9,133}, David N Louis¹⁰⁶, Adam Margolin³⁶⁸, Sancha Martin^{2,369}, Hardeep K Nahal-Bose⁴⁵, G Petur Nielsen¹⁰⁶, Serena Nik-Zainal^{2,324,325,326}, Larsson Omberg¹⁰⁰, Christine P'ng⁹, Marc D Perry^{45,103}, Paz Polak^{3,4,6}, Esther Rheinbay^{3,6,106}, Mark A Rubin^{131,194,210,211,212}, Colin A Semple³⁶⁴, Dennis C Sgroi¹⁰⁶, Tatsuhiro Shibata^{33,34}, Reiner Siebert^{262,761}, Jaclyn Smith³⁶⁸, **Lincoln D Stein**^{9,10}, Miranda D Stobbe^{135,136}, Ren X Sun⁹, Kevin Thai⁴⁵, Derek W Wright^{370,371}, Chin-Lee Wu¹⁰⁶, Ke Yuan^{294,369,372} and Junjun Zhang⁴⁵

Evolution and heterogeneity

David J Adams², Pavana Anur³⁷³, Rameen Beroukhim^{3,6,172}, Paul C Boutros^{9,133,142,143}, David D L Bowtell^{187,291}, Peter J Campbell^{1,2}, Shaolong Cao¹⁴⁸, Elizabeth L Christie¹⁸⁷, Marek Cmero^{374,375,376}, Yupeng Cun³⁷⁷, Kevin J Dawson², Jonas Demeulemeester^{63,64}, Stefan C Dentre^{2,64,354}, Amit G Deshwar³⁷⁸, Nilgun Donmez^{151,159}, Ruben M Drews²⁹⁴, Roland Eils^{52,54,66,67}, Yu Fan¹⁴⁸, Matthew W Fittall⁶⁴, Dale W Garsed^{187,188}, Moritz Gerstung^{7,8}, Gad Getz^{3,4,5,6}, Santiago Gonzalez^{7,8}, Gavin Ha³, Kerstin Haase⁶⁴, Marcin Imielinski^{299,300}, Lara Jerman^{8,379}, Yuan Ji^{380,381}, Clemency Jolly⁶⁴, Kortine Kleinheinz^{52,54}, Juhee Lee³⁸², Henry Lee-Six², Ignaty Leshchiner³, Dimitri Livitz³, Geoff Macintyre²⁹⁴, Salem Malikic^{151,159}, Florian Markowetz^{294,295}, Iñigo Martincorena², Thomas J Mitchell^{2,295,383}, Quaid D Morris^{358,384}, Ville Mustonen^{320,321,322}, Layla Oesper³⁸⁵, Martin Peifer³⁷⁷, Myron Peto³⁸⁶, Benjamin J Raphael¹²⁰, Daniel Rosebrock³, Yulia Rubanova^{160,358}, S Cenk Sahinalp^{151,158,159}, Adriana Salcedo⁹, Matthias Schlesner^{52,110}, Steven E Schumacher^{3,217}, Subhajit Sengupta³⁸⁷, Ruian Shi³⁸⁴, Seung Jun Shin²⁶⁴, **Paul T Spellman**³⁸⁸, Oliver Spiro³, Lincoln D Stein^{9,10}, Maxime Tarabichi^{2,64}, **Peter Van Loo**^{63,64}, Shankar Vembu^{384,389}, Ignacio Vázquez-García^{2,167,327,328}, Wenyi Wang¹⁴⁸, **David C Wedge**^{2,354,355}, David A Wheeler^{164,165}, Jeffrey A Wintersinger^{192,358,390}, Tsun-Po Yang³⁷⁷, Xiaotong Yao^{299,316}, Kaixian Yu³⁹¹, Ke Yuan^{294,369,372} and Hongtu Zhu^{392,393}

Exploratory: portals, visualization and software infrastructure

Fatima Al-Shahrour³⁵⁷, Elisabet Barrera⁷, Wojciech Bazant⁷, Alvis Brazma⁷, Isidro Cortés-Ciriano^{237,238,239}, Brian Craft²⁴⁰, David Craft³, Vincent Ferretti^{45,69}, Nuno A Fonseca^{7,70}, Anja Füllgrabe⁷, Mary J Goldman²⁴⁰, **David Haussler**^{240,394}, Wolfgang Huber⁸, Maria Keays⁷, Alfonso Muñoz⁷, Brian D O'Connor^{45,50}, Irene Papatheodorou⁷, Robert Petryszak⁷, Elena Piñeiro-Yáñez³⁵⁷, Alfonso Valencia^{105,111}, **Miguel Vazquez**^{105,112}, John N Weinstein^{395,396}, Qian Xiang¹¹⁶, Junjun Zhang⁴⁵ and **Jingchun Zhu**²⁴⁰

Exploratory: mitochondrial variants and HLA/immunogenicity

Peter J Campbell^{1,2}, Yiwen Chen¹⁴⁸, Chad J Creighton²⁴¹, Li Ding^{138,139,146}, Akihiro Fujimoto⁴⁸, Masashi Fujita⁴⁸, Gad Getz^{3,4,5,6}, Leng Han²³¹, Takatori Hasegawa⁸⁷, Shuto Hayashi⁸⁷, Seiya Imoto^{86,87}, Young Seok Ju^{2,181}, Hyung-Lae Kim²⁷, Youngwook Kim^{96,97}, Youngil Koh^{307,308}, Mitsuhiro Komura⁸⁷, Jun Li¹⁴⁸, **Han**

Liang³⁹⁷, Iñigo Martincorena², Satoru Miyano⁸⁷, Shinichi Mizuno³⁹⁸, **Hidewaki Nakagawa**⁴⁸, Keunchil Park^{206,207}, Eigo Shimizu⁸⁷, Yumeng Wang^{148,399}, John N Weinstein^{395,396}, Yanxun Xu⁴⁰⁰, Rui Yamaguchi⁸⁷, Fan Yang³⁸⁴, Yang Yang²³¹, Christopher J Yoon¹⁸¹, Sung-Soo Yoon³⁰⁸, Yuan Yuan¹⁴⁸, Fan Zhang²⁴⁶ and Zemin Zhang^{246,271}

Exploratory: pathogens

Malik Alawi^{401,402}, Ivan Borozan⁹, Daniel S Brewer^{403,404}, Colin S Cooper^{404,405,406}, Nikita Desai⁴⁵, **Roland Eils**^{52,54,66,67}, Vincent Ferretti^{45,69}, Adam Grundhoff^{401,407}, Murat Iskar⁴⁰⁸, Kortine Kleinheinz^{52,54}, Peter Lichter⁴⁰⁸, **Hidewaki Nakagawa**⁴⁸, Akinyemi I Ojesina^{255,256,257}, Chandra Sekhar Pedamallu^{3,6,172}, Matthias Schlesner^{52,110}, Xiaoping Su¹⁴⁴ and Marc Zapatka⁴⁰⁸

Tumor Specific Providers – Australia (Ovarian cancer)

Kathryn Alsop^{409,410}, Australian Ovarian Cancer Study Group^{187,311,411}, **David D L Bowtell**^{187,291}, Timothy JC Bruxner¹⁸⁵, Angelika N Christ¹⁸⁵, Elizabeth L Christie¹⁸⁷, Stephen M Cordner⁴¹², Prue A Cowin¹⁸⁷, Ronny Drapkin⁴¹³, Dariush Etemadmoghadam^{187,188}, Sian Fereday⁴¹⁴, Dale W Garsed^{187,188}, Joshy George¹⁷⁰, Sean M Grimmond³⁶³, Anne Hamilton¹⁸⁷, Oliver Holmes^{311,312}, Jillian A Hung^{415,416}, Karin S Kassahn^{185,417}, Stephen H Kazakoff^{311,312}, Catherine J Kennedy^{418,419}, Conrad R Leonard^{311,312}, Linda Mileschkin¹⁸⁷, David K Miller^{185,360,420}, Gisela Mir Arnau¹⁸⁷, Chris Mitchell¹⁸⁷, Felicity Newell^{311,312}, Katia Nones^{311,312}, Ann-Marie Patch^{311,312}, John V Pearson^{311,312}, Michael C Quinn^{311,312}, Mark Shackleton^{188,218}, Darrin F Taylor¹⁸⁵, Heather Thorne¹⁸⁷, Nadia Traficante¹⁸⁷, Ravikiran Veduru¹⁸⁷, Nick M Waddell³¹², Nicola Waddell^{311,312}, Paul M Waring²⁵³, Scott Wood^{311,312}, Qinying Xu^{311,312} and Anna deFazio^{421,422,423}

Tumor Specific Providers – Australia (Pancreatic cancer)

Matthew J Anderson¹⁸⁵, Davide Antonello⁴²⁴, Andrew P Barbour^{425,426}, Claudio Bassi⁴²⁴, Samantha Bersani⁴²⁷, **Andrew V Biankin**^{359,360,361,362}, Timothy JC Bruxner¹⁸⁵, Ivana Cataldo^{427,428}, David K Chang^{360,362}, Lorraine A Chantrill³⁶⁰, Yoke-Eng Chiew⁴²¹, Angela Chou^{360,429}, Angelika N Christ¹⁸⁵, Sara Cingarlini³⁷, Nicole Cloonan⁴³⁰, Vincenzo Corbo^{428,431, 432}, Fraser R Duthie^{433,434}, J Lynn Fink^{105,185}, Anthony J Gill^{360,435}, Janet S Graham^{362,436}, **Sean M Grimmond**³⁶³, Ivon Harliwong¹⁸⁵, Oliver Holmes^{311,312}, Nigel B Jamieson^{361,362,437}, Amber L Johns^{360,420}, Karin S Kassahn^{185,417}, Stephen H Kazakoff^{311,312}, James G Kench^{360,435,438}, Luca Landoni⁴²⁴, Rita T Lawlor⁴²⁸, Conrad R Leonard^{311,312}, Andrea Mafficini⁴²⁸, Neil D Merrett^{424,439}, David K Miller^{185,360,420}, Marco Miotto⁴²⁴, Elizabeth A Musgrove³⁶², Adnan M Nagrial³⁶⁰, Felicity Newell^{311,312}, Katia Nones^{311,312}, Karin A Oien^{253,440}, Marina Pajic³⁶⁰, Ann-Marie Patch^{311,312}, John V Pearson^{311,312}, Mark Pinese³⁶⁰, Andreia V Pinho³⁶⁰, Michael C Quinn^{311,312}, Alan J Robertson¹⁸⁵, Ilse Rومان³⁶⁰, Borislav C Rusev⁴²⁸, Jaswinder S Samra^{424,435}, Maria Scardoni⁴²⁷, Christopher J Scarlett^{360,441}, Aldo Scarpa⁴²⁸, Elisabetta Sereni⁴²⁴, Katarzyna O Sikora⁴²⁸, Michele Simbolo⁴³¹, Morgan L Taschuk⁴⁵, Christopher W Toon³⁶⁰, Giampaolo Tortora^{37,38}, Caterina Vicentini⁴²⁸, Nick M Waddell³¹², Nicola Waddell^{311,312}, Scott Wood^{311,312}, Jianmin Wu³⁶⁰, Qinying Xu^{311,312} and Nikolajs Zeps⁴⁴²

Tumor Specific Providers – Australia (Skin cancer)

Lauri A Aaltonen⁴⁴³, Andreas Behren⁴⁴⁴, Hazel Burke⁴⁴⁵, Jonathan Cebon⁴⁴⁴, Rebecca A Dagg⁴⁴⁶, Riccardo De Paoli-Iseppi⁴⁴⁷, Ken Dutton-Regester³¹¹, Matthew A Field⁴⁴⁸, Anna Fitzgerald⁴⁴⁹, Sean M Grimmond³⁶³, **Nicholas K Hayward**^{311,445}, Peter Hersey⁴⁴⁵, Oliver Holmes^{311,312}, Valerie Jakrot⁴⁴⁵, Peter A Johansson³¹¹, Hojabr Kakavand⁴⁴⁷, Stephen H Kazakoff^{311,312}, Richard F Kefford⁴⁵⁰, Loretta MS Lau⁴⁵¹, Conrad R Leonard^{311,312}, Georgina V Long⁴⁵², **Graham J Mann**^{453,454}, Felicity Newell^{311,312}, Katia Nones^{311,312}, Ann-Marie Patch^{311,312}, John V Pearson^{311,312}, Hilda A Pickett⁴⁵¹, Antonia L Pritchard³¹¹, Gulietta M Pupo⁴⁵⁵, Robyn PM Saw⁴⁵², Sarah-Jane Schramm⁴⁵⁶, **Richard A Scolyer**^{422,452,457,458}, Mark

Shackleton^{188,218}, Catherine A Shang⁴⁵⁹, Ping Shang⁴⁵², Andrew J Spillane⁴⁵², Jonathan R Stretch⁴⁵², Varsha Tembe⁴⁵⁶, John F Thompson⁴⁵², Ricardo E Vilain⁴⁵⁷, Nick M Waddell³¹², Nicola Waddell^{311,312}, James S Wilmott⁴⁵², Scott Wood^{311,312}, Qinying Xu^{311,312} and Jean Y Yang⁴⁶⁰

Tumor Specific Providers – Canada (Pancreatic cancer)

John Bartlett^{461,462}, Prashant Bavi⁴⁶³, Ivan Borozan⁹, Dianne E Chadwick⁴⁶⁴, Michelle Chan-Seng-Yue⁴⁶³, Sean Cleary^{463,465}, Ashton A Connor^{466,467}, Karolina Czajka⁴⁶⁸, Robert E Denroche⁴⁶³, Neesha C Dhani⁴⁶⁹, Jenna Eagles⁷⁹, Vincent Ferretti^{45,69}, Steven Gallinger^{463,466,467}, Robert C Grant^{463,470}, David Hedley⁴⁶⁹, Michael A Hollingsworth⁴⁷¹, **Thomas J Hudson**^{78,79}, Gun Ho Jang⁴⁶³, Jeremy Johns⁷⁹, Sangeetha Kalimuthu⁴⁶³, Sheng-Ben Liang⁴⁷², Ilinca Lungu^{463,473}, Xuemei Luo⁹, Faridah Mbabaali⁷⁹, **John D McPherson**^{79,463,474}, Treasa A McPherson⁴⁷⁰, Jessica K Miller⁷⁹, Malcolm J Moore⁴⁶⁹, Faiyaz Notta^{463,475}, Danielle Pasternack⁷⁹, Gloria M Petersen⁴⁷⁶, Michael H A Roehrl^{133,463,477,478,479}, Michelle Sam⁷⁹, Iris Selander⁴⁷⁰, Stefano Serra²⁵³, Sagedeh Shahabi⁴⁷², **Lincoln D Stein**^{9,10}, Morgan L Taschuk⁴⁵, Sarah P Thayer¹⁰⁶, Lee E Timms⁷⁹, Gavin W Wilson^{9,463}, Julie M Wilson⁴⁶³ and Bradly G Wouters⁴⁸⁰

Tumor Specific Providers – Canada (Prostate cancer)

Timothy A Beck⁴⁵, Vinayak Bhandari⁹, Paul C Boutros^{9,133,142,143}, **Robert G Bristow**^{133,481,482,483,484}, Colin C Collins¹⁵¹, Shadrielle MG Espiritu⁹, Neil E Fleshner⁴⁸⁵, Natalie S Fox⁹, Michael Fraser⁹, Syed Haider⁹, Lawrence E Heisler⁴⁸⁶, Vincent Huang⁹, Emilie Lalonde⁹, Julie Livingstone⁹, John D McPherson^{79,463,474}, Alice Meng⁴⁸⁷, Veronica Y Sabelnykova⁹, Adriana Salcedo⁹, Yu-Jia Shiah⁹, Theodorus Van der Kwast⁴⁸⁸ and Takafumi N Yamaguchi⁹

Tumor Specific Providers – China (Gastric cancer)

Shuai Ding⁴⁸⁹, Daiming Fan⁴⁹⁰, Yong Hou^{39,249}, Yi Huang^{153,154}, Lin Li³⁹, Siliang Li^{39,249}, Dongbing Liu^{39,249}, Xingmin Liu^{39,249}, **Yuyong Lu**^{28,29,30}, Yongzhan Nie^{490,491}, Hong Su^{39,249}, Jian Wang³⁹, Kui Wu^{39,249}, Xiao Xiao¹⁵⁴, Rui Xing^{29,492}, **Huanming Yang**³⁹, Shanlin Yang⁴⁸⁹, Yingyan Yu⁴⁹³,²³⁰, Xiuqing Zhang³⁹, Yong Zhou³⁹ and Shida Zhu^{39,249}

Tumor Specific Providers – EU: France (Renal cancer)

Rosamonde E Banks⁴⁹⁴, Guillaume Bourque^{495,496}, Alvis Brazma⁷, Paul Brennan⁴⁹⁷, **Mark Lathrop**⁴⁹⁶, Louis Letourneau⁴⁹⁸, Yasser Riazalhosseini⁴⁹⁶, Ghislaine Scelo⁴⁹⁷, **Jörg Tost**⁴⁹⁹, Naveen Vasudev⁵⁰⁰ and Juris Viksna⁵⁰¹

Tumor Specific Providers – EU: United Kingdom (Breast cancer)

Sung-Min Ahn⁵⁰², Ludmil B Alexandrov^{2,317}, Samuel Aparicio⁵⁰³, Laurent Arnould⁵⁰⁴, MR Aure⁵⁰⁵, Shri-ram G Bhosle², E Birney⁷, Ake Borg⁵⁰⁶, S Boyault⁵⁰⁷, AB Brinkman⁵⁰⁸, JE Brock⁵⁰⁹, A Broeks⁵¹⁰, Adam P Butler², AL Børresen-Dale⁵⁰⁵, C Caldas^{511,512}, Peter J Campbell^{1,2}, Suet-Feung Chin^{511,512}, Helen Davies², C Desmedt⁵¹³, L Dirix⁵¹⁴, S Dronov², Anna Ehinger⁵¹⁵, JE Eyfjord⁵¹⁶, GG Van den Eynden⁵¹⁷, A Fatima²¹⁷, Jorge Reis Filho⁵¹⁸, JA Foekens⁵¹⁹, PA Futreal⁵²⁰, Øystein Garred^{521,522}, Moritz Gerstung^{7,8}, Dilip D Giri⁵¹⁸, D Glodzik², Dorthe Grabau⁵²³, Holmfridur Hilmarsdottir⁵¹⁶, GK Hooijer⁵²⁴, Jocelyne Jacquemier⁵²⁵, SJ Jang⁵²⁶, Jon G Jonasson⁵¹⁶, Jos Jonkers⁵²⁷, HY Kim⁵²⁵, Tari A King^{528,529}, Stian Knappskog², G Kong⁵²⁵, S Krishnamurthy⁵³⁰, S Van Laere⁵¹⁴, SR Lakhani⁵³¹, A Langerød⁵⁰⁵, Denis Larsimont⁵³², HJ Lee⁵²⁶, JY Lee⁵³³, Ming Ta Michael Lee⁵²⁰, Yilong Li², Ole Christian Lingjærde⁵³⁴, Gaetan MacGrogan⁵³⁵, JW Martens⁵³⁶, Sancha Martin^{2,369}, Iñigo Martincorena², Andrew Menzies², Sandro Morganella², Ville Mustonen^{320,321,322}, Serena Nik-Zainal^{2,324,325,326}, Sarah O'Meara², I Pauporté¹⁸, Sarah Pinder⁵³⁷, X Pivot⁵³⁸, Elena Provenzano⁵³⁹,

CA Purdie⁵⁴⁰, Keiran M Raine², M Ramakrishna², K Ramakrishnan², AL Richardson²¹⁷, M Ringnér⁵⁰⁶, Javier Bartolomé Rodríguez¹⁰⁵, FG Rodríguez-González¹⁷⁵, G Romieu⁵⁴¹, Roberto Salgado²⁵³, Torill Sauer⁵³⁴, R Shepherd², AM Sieuwerts¹⁷⁷, PT Simpson⁵³¹, M Smid⁵⁴², C Sotiriou⁵⁵, PN Span⁵⁴³, J Staaf⁵⁰⁶, Lucy Stebbings², Ólafur Andri Stefánsson⁵⁴⁴, Alasdair Stenhouse⁵⁴⁵, **Michael Rudolf Stratton**², HG Stunnenberg^{249,546}, Fred Sweep⁵⁴⁷, BK Tan⁵⁴⁸, Jon W Teague², Gilles Thomas⁵⁴⁹, AM Thompson⁵⁴⁵, S Tommasi⁵⁵⁰, I Treilleux^{551,552}, Andrew Tutt²¹⁷, NT Ueno³⁹³, Peter Van Loo^{63,64}, P Vermeulen⁵¹⁴, Alain Viari⁴²⁸, MJ van de Vijver²⁵³, A Vincent-Salomon⁵⁴⁶, David C Wedge^{2,354,355}, Bernice Huimin Wong⁵⁵³, Lucy Yates², X Zou², CHM van Deurzen⁵³⁶ and L van't Veer^{554,555}

Tumor Specific Providers – Germany (Malignant lymphoma)

Ole Ammerpohl^{556,557}, Sietse Aukema^{558,559}, Anke K Bergmann⁵⁶⁰, Stephan H Bernhart^{276,277,281}, Hans Binder^{276,277}, Arndt Borkhardt⁵⁶¹, Christoph Borst⁵⁶², Benedikt Brors^{82,119,278}, Birgit Burkhardt⁵⁶³, Alexander Claviez⁵⁶⁴, Roland Eils^{52,54,66,67}, Maria Elisabeth Goebler⁵⁶⁵, Andrea Haake⁵⁵⁶, Siegfried Haas⁵⁶², Martin Hansmann⁵⁶⁶, Jessica I Hoell⁵⁶¹, Steve Hoffmann^{277,279,280,281}, Michael Hummel⁵⁶⁷, Daniel Hübschmann^{54,66,83,84,85}, Dennis Karsch⁵⁶⁸, Wolfram Klapper⁵⁵⁹, Kortine Kleinheinz^{52,54}, Michael Kneba⁵⁶⁸, Jan O Korbel^{7,8}, Helene Kretzmer^{277,281}, Markus Kreuz⁵⁶⁹, Dieter Kube⁵⁷⁰, Ralf Küppers⁵⁷¹, Chris Lawerenz⁶⁷, Dido Lenze⁵⁶⁷, Peter Lichter⁴⁰⁸, Markus Loeffler⁵⁶⁹, Cristina López^{262,556}, Luisa Mantovani-Löffler⁵⁷², Peter Möller⁵⁷³, German Ott⁵⁷⁴, Bernhard Radlwimmer⁴⁰⁸, Julia Richter^{556,559}, Marius Rohde⁵⁷⁵, Philip C Rosenstiel⁵⁷⁶, Andreas Rosenwald⁵⁷⁷, Markus B Schilhabel⁵⁷⁶, Matthias Schlesner^{52,110}, Stefan Schreiber⁵⁷⁸, **Reiner Siebert**^{262,761}, Peter F Stadler^{276,277,281}, Peter Staib⁵⁷⁹, Stephan Stilgenbauer⁵⁸⁰, Stephanie Sungalee⁸, Monika Szczepanowski⁵⁵⁹, Umut H Toprak^{54,581}, Lorenz HP Trümper⁵⁷⁰, Rabea Wagener^{262,556} and Thorsten Zenz⁸²

Tumor Specific Providers – Germany (Pediatric Brain cancer)

Ivo Buchhalter^{52,53,54}, Juergen Eils^{66,67}, Roland Eils^{52,54,66,67}, Volker Hovestadt⁴⁰⁸, Barbara Hutter^{80,81,82}, David TW Jones^{301,302}, Natalie Jäger⁵², Christof von Kalle⁸⁴, Marcel Kool^{98,301}, Jan O Korbel^{7,8}, Andrey Korshunov⁹⁸, Pablo Landgraf⁵⁸², Chris Lawerenz⁶⁷, Hans Lehrach⁵⁸³, **Peter Lichter**⁴⁰⁸, Paul A Northcott⁵⁸⁴, Stefan M Pfister^{98,301,585}, Bernhard Radlwimmer⁴⁰⁸, Guido Reifenberger⁵⁸², Matthias Schlesner^{52,110}, Hans-Jörg Warnatz⁵⁸³, Joachim Weischenfeldt^{8,113,114}, Stephan Wolf⁵⁸⁶, Marie-Laure Yaspo⁵⁸³ and Marc Zapatka⁴⁰⁸

Tumor Specific Providers – Germany (Prostate cancer)

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Tumor Specific Providers – India (Oral cancer)

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Tumor Specific Providers – Italy (Pancreatic cancer)

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Tumor Specific Providers – Japan (Biliary tract cancer)

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Tumor Specific Providers – Japan (Gastric cancer)

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Tumor Specific Providers – Japan (Liver cancer)

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Tumor Specific Providers – Singapore (Biliary tract cancer)

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Tumor Specific Providers – South Korea (Blood cancer)

Keun Soo Ahn⁶¹⁷, Hyung-Lae Kim²⁷, Youngil Koh^{307,308} and **Sung-Soo Yoon**³⁰⁸

Tumor Specific Providers – Spain (Chronic Lymphocytic Leukemia)

Marta Aymerich⁶¹⁸, **Elias Campo**^{619,620}, Josep Ll Gelpi^{46,71}, Ivo G Gut^{135,136}, Marta Gut^{135,136}, Armando Lopez-Guillermo⁶²¹, Carlos López-Otín⁶²², Xose S Puente⁶²³, Romina Royo¹⁰⁵ and David Torrents^{105,111}

Tumor Specific Providers – United Kingdom (Bone cancer)

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Tumor Specific Providers – United Kingdom (Chronic myeloid disorders)

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Tumor Specific Providers – United Kingdom (Esophageal cancer)

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Tumor Specific Providers – United Kingdom (Prostate cancer)

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Tumor Specific Providers – United States (TCGA)

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