SUPPLEMENTAL MATERIALS

SUPPLEMENTAL EXPERIMENTAL PROCEDURES

Cell Culture Procedures and Plasmids

RPE1-hTERT cells were obtained from the American Type Culture Collection (ATCC) and cultured in a 1:1 mixture of Dulbecco's Modified Eagle Medium (DMEM) and Ham's F-12 medium (Gibco) (DMEM/F12). HTC75 and Phoenix virus packaging cells were grown in DMEM. Media was supplemented with 10% fetal bovine serum (Gibco), 100 U/ml penicillin/streptomycin (Life Technologies), and 2.5 mM L-glutamine (Life Technologies). Doxycycline was used at 1 μg/ml, blebbistatin (Tocris Bioscience) at 30 μM, monastrol (Tocris Bioscience) at 100 μM, nocodazole (Sigma) at 100 ng/ml, and reversine (Cayman Chemical) at 1 μM. Unless indicated otherwise, analysis was performed 48 h after TRF2-DN transgene induction.

For retroviral transduction, open reading frames were cloned into pQCXIN, pQCXIP, pQCXIB (Clontech) or pQCXIZ, which confer resistance to G418, puromycin, blasticidin, and zeocin, respectively. Constructs were transfected into Phoenix amphotropic packaging cells using calcium phosphate precipitation. Retroviral supernatants were filtered, mixed 1:1 with target cell media and supplemented with 4 μg/ml polybrene. Target cells were infected with retroviral supernatants four times at 12 h intervals. Successfully targeted cells were selected using G418 (0.4 mg/ml), puromycin (5 μg/ml), blasticidin (5 μg/ml), or zeocin (Life Technologies) (400 μg/ml). Clones were isolated by limiting dilution. If transgenes incorporated a fluorescent protein, transduced cells were isolated by FACS. Either pooled cells were analyzed or clones were isolated by subcloning into 96-well plates.

Target sequence for CRISPR/Cas9 mediated gene knockouts identified by ZiFit (http://zifit.partners.org): sgTREX1-2, 5'-GAGCCCCCCCACCTCTC-(PAM)-3'. Target sequences were incorporated into an AflII-linearized gRNA-cloning vector (Addgene) by Gibson Assembly (New England BioLabs). gRNA plasmids were co-transfected into target cells with an hCas9 expression plasmid (Addgene) by nucleofection (Lonza apparatus). 700,000 cells were mixed with electroporation buffer (freshly mixed 125 mM Na_2HPO_4, 12.5 mM KCl, 55 mM MgCl2 pH 7.75), 5 µg Cas9 plasmid, and 5 µg gRNA plasmid, transferred to an electroporation cuvette (BTX), and electroporated with program T23 for RPE-1 cells or program L005 for HTC75 cells. Cells were then allowed to recover for 48 h before a second round of electroporation. Successful CRISPR/Cas9 editing was confirmed at the polyclonal stage by mutation detection with the SURVEYOR nuclease assay (Transgenomic). Briefly, the region surrounding the predicted Cas9 cut site was PCR amplified (Primer 1: CCTCACCCTCTCCAACTTCC, Primer 2: GCAGCGGGGGTCTTTATTCG), melted, and reannealed. Reannealed PCR products were then incubated with the SURVEYOR nuclease for one hour at 42ºC, and analyzed on a 2% agarose gel with ethidium bromide. Clones were isolated by limiting dilution and screened for TREX1 deletion by Western blotting. Biallelic targeting was verified by sequencing of TOPO-cloned PCR products.

Immunoblotting and immunofluorescence

For immunoblotting, cells were harvested by trypsinization and lysed in 1x Laemmli buffer (50 mM Tris, 10% glycerol, 2% SDS, 0.01% bromophenol blue, 2.5% β-mercaptoethanol) at 10^7 cells/ml. Lysates were denatured at 100ºC and DNA was sheared with a 28 1/2 gauge insulin needle. Lysate equivalent to 10^5 cells was resolved on 8% or 10% SDS/PAGE (Life Technologies) and transferred to nitrocellulose membranes. Membranes were blocked in 5% milk in TBS with 0.1% Tween-20 (TBS-T)

and incubated with primary antibody overnight at 4ºC, washed 3 times in TBS-T, and incubated for 1 h at room temperature with horseradish-peroxidase-conjugated sheep anti-mouse or donkey anti-rabbit secondary antibodies. After three washes in TBS-T, membranes were rinsed in TBS and proteins were developed using enhanced chemiluminescence (Amersham).

For IF, cells were grown on coverslips for 48 h prior to fixation in 2% paraformaldehyde in PBS for 10 min or in ice-cold methanol for 10 min. Coverslips were incubated in blocking buffer (1 mg/ml BSA, 3% goat serum, 0.1% Triton X-100, 1 mM EDTA in PBS) for 30 min, followed by incubation with primary antibodies diluted in blocking buffer for 2 h, washed 3 times with PBS with 0.1% Triton, incubated with secondary antibodies diluted in blocking buffer, and finally DNA was stained with either DAPI or YOYO-1 Iodide (Life Technologies). Coverslips were then mounted in ProLong Gold Antifade Mountant (Life Technologies). Digital images were acquired on a Zeiss Axioplan II microscope equipped with a Hamamatsu C4742-95 camera using Volocity software or with an image restoration system (DeltaVision; Applied Precision) equipped with a cooled charge-coupled device camera (CoolSnap QE; Photometrics), a PlanApo 60x 1.40 NA objective (Olympus America, Inc.), and SoftWoRx software.

For EdU labeling, cells were incubated with EdU for 30 minutes prior to PFA fixation. Incorporated EdU was detected using a Click-iT EdU Alexa Fluor 647 imaging kit (Life Technologies).

The following primary antibodies were used: anti-hTRF2 (rabbit polyclonal, de Lange lab, #647) Zhu et al, 2000; anti-γ-tubulin (mouse monoclonal, Abcam, ab11316); anti-myc (mouse monoclonal, MSKCC Hybridoma Core Facility, #9E10); anti-TREX1 (rabbit monoclonal, Abcam, ab185228); anti-Lamin A/C (mouse monoclonal, Santa Cruz, sc-7292); anti-γH2A.X (mouse monoclonal, Millipore, 05-636); anti-53BP1 (rabbit polyclonal, Novus, 100-304A); anti-FLAG (mouse monoclonal, Sigma, F3165); anti-

RPA32 (mouse monoclonal, Abcam, ab2175); anti-Tpr (mouse monoclonal, Santa Cruz, sc-271565); anti-Histone H2A (rabbit polyclonal, Abcam, ab18255), anti-Histone H2B (rabbit polyclonal, Abcam, ab1790); anti-Histone H4 (rabbit polyclonal, Abcam, ab10158); anti-Lamin B1 (rabbit polyclonal, Abcam, ab16048); anti-BAF1 (rabbit monoclonal, Abcam, ab129184); anti-SUN1 (rabbit polyclonal, Abcam, ab74758); anti-SUN2 (rabbit polyclonal, Abcam, ab87036); anti-Mre11 (rabbit polyclonal, Bethyl) ; anti-Mad1 (rabbit polyclonal, Santa Cruz, sc-67338); anti-LAP2 (rabbit polyclonal, Bethyl); anti-mAb414 (mouse monoclonal, Abcam, ab24609).

Live-cell Imaging

Cells were plated onto 35 mm glass bottom dishes (MatTek) 48 h before imaging. One h before imaging cell culture media was replaced with phenol red-free DMEM/F12 medium. Live cell imaging was performed using a CellVoyager CV1000 spinning disk confocal system (Yokogawa, Olympus) equipped with 445, 488, and 561 nm lasers, a Hamamatsu 512 x 512 EMCCD camera. Pinhole size was 50 µm. Fluorescence images were acquired at the indicated intervals using a UPlanSApo 60x/1.3 silicon oil objective with the correction collar set to 0.17. The pixel size in the image was 0.27 µm. The following emission filters were used for image acquisition: 480/40 or 470/24 for mTurquoise2-tagged proteins, and 617/73 for mCherry-tagged proteins. 17 µm z-stacks were collected at 0.5 µm steps or 16 µm z-stacks were collected at 2.0 µm steps. Temperature was maintained at 37ºC in a temperature-controlled enclosure with CO_2 support. Maximum intensity projection of z-stacks and adjustment of brightness and contrast were performed using Fiji software. Image stitching was done with the Fiji plugin Grid/Collection stitching (Preibisch et al., 2009) with 20% tile overlap, linear blending, a 0.30 regression threshold, a 2.50 max/avg. displacement threshold, and a 3.50 absolute

displacement threshold. Images were cropped and assembled into figures using Photoshop CS5.1 (Adobe).

Telomere fusion PCR

Fusion PCR to detect telomeric fusions was performed essentially as described (Letsolo et al., 2010; Capper et al., 2007). Genomic DNA was extracted using phenol/chloroform, solubilized by digestion with EcoRI, and quantitated by Hoechst 33258 fluorometry (Hoefer). Subtelomeric primers used for amplification of telomeric fusions were XpYpM: ACCAGGTTTTCCAGTGTGTT, 17p6: GGCTGAACTATAGCCTCTGC, 21q4: GGGACATATTTTGGGGTTGC. XpYpc2tr: GCTATGGCTTCTTGGGGC, was included for control amplification of XpYp subtelomeric DNA. PCR conditions were as follows: 50 ng of input genomic DNA, supplemented with Fail Safe PCR buffer H and Fail Safe Enzyme Mix (Epicentre). Cycling conditions were 1 cycle of 95ºC for 15 seconds, followed by 26 cycles of 95ºC for 15 seconds, 58º C for 20 seconds, 68ºC for 10 minutes, and finally 1 cycle of 68ºC for 10 min.

Products were resolved by 0.7% 1x TAE agarose gel electrophoresis, transferred to a Hybond membrane, and detected with a random-primed α-^{32}P-labeled (GE Healthcare) probe specific for the TelBam11 family of subtelomeres (Brown et al., 1990; Riethman et al., 2004). Primers used for TelBam11 probe generation were 21q4 and 21q-seq-rev2: ACACAGAAGGTTGATATACACAG. Control amplification of the XpYp subtelomere was detected with an XpYp subtelomere specific probe generated by PCR with the following primers: XpYpO: CCTGTAACGCTGTTAGGTAC, XpYpG: AATTCCAGACACACTAGGACCCTGA. Hybridized signal was detected by exposing the membrane to a PhosphorImager screen overnight and imaging with a Storm 820 Molecular Imager (Molecular Dynamics).

Analysis of telomere fusions and karyotypes

Cells were incubated with 0.1 μg/ml colcemid (Roche) for 30 min, harvested by trypsinization, resuspended in 0.075 M KCl, incubated at 37ºC for 10 min, and fixed overnight in methanol and acetic acid (3:1) at 4ºC. Fixed cells were dropped onto glass slides and the slides were dried overnight. Cells were rehydrated in PBS for 10 min, dehydrated with 75%, 95%, and 100% ethanol and air dried before hybridization in hybridization solution (70% formamide, 1 mg/ml blocking reagent (Roche), 10 mM Tris-HCl (pH 7.2), TAMRAOO-$(TTAGGG)_3$ PNA probe (Applied Biosystems). Slides were then denatured at 80ºC for 5 min and hybridized for 2 h at room temperature. Slides were washed 2 times for 15 min in wash solution #1 (70% formamide, 10 mM Tris-HCl, (pH 7.2)), and 3 times for 5 min in wash solution #2 (0.1M Tris-HCl (pH 7.2), 0.15M NaCl, 0.08% Tween-20). DNA was stained with DAPI. Slides were mounted in ProLong Gold Antifade Mountant (Life Technologies).

Karyotypic analysis was done on DAPI stained metaphases, G-banded metaphases, or by spectral karyotyping. For spectral karyotyping, cell lines were cultured with colcemid (0.005 μg/mL) at 37°C for 45 minutes, resuspended in 0.075 M KCl, incubated at 37°C for 10 minutes, and then fixed in methanol-acetic acid (3:1). Metaphase spreads from the fixed cell suspension were hybridized with SKY painting probe according to the manufacturer's protocol (Applied Spectral Imaging, Carlsbad, CA). SKY images were acquired with an SD300 Spectracube (Applied Spectral Imaging, Carlsbad, CA) mounted on a Nikon Eclipse E800 microscope using a custom-designed optical filter (SKY-1) (Chroma Technology, Brattleboro, VT). For each cell line, a minimum of 20 metaphases were captured and fully karyotyped. The breakpoints on the SKY-painted chromosomes were determined by comparison with corresponding DAPI karyotype and chromosomal abnormalities described according to the International

System of Human Cytogenetic Nomenclature (ISCN) 2013. CMA-chromosome banding was performed using standard methods.

Image quantitation

Chromatin bridge resolution was determined by manually tracking pairs of daughter cells. Bridge resolution was inferred to take place when the base of the bridge became slack and/or recoiled. The mean fluorescence intensity of mTurquoise2-tubulin at the midbody was measured every 10 minutes using Fiji software. Values were corrected for background and the initial signal intensity of each cell was set to 1. S phase timing was marked by the appearance of stable RPA foci in the primary nucleus.

Dynamics of NERDI events were calculated as described (Hatch et al., 2013). Briefly, the mean fluorescence intensity of NLS-3xmTurquoise2 in a region of interest in the nucleus was measured every 30 seconds over the course of a rupturing event using Fiji software. The peak fluorescence intensity was set to 1, and the lowest fluorescence intensity was set to 0. Rupturing curves were fit to a plateau followed by a one-phase decay curve where the plateau value was set to 1. Recovery curves were fit to one-phase associations. Curve fitting was performed using Prism 6 (GraphPad).

RPA signals on chromatin bridges were quantified by measuring the mean RPA intensity of 10 relevant regions on each chromatin bridge in fixed samples using Fiji software. Background intensity was subtracted and values were averaged within each experiment. Independent experiments were averaged to produce the graph in Figure 4H. Since RPA accumulation on chromatin bridges is usually a late event only bridges greater than 50 μm in length were included in this analysis. For RPA signals quantified in live cell samples (Fig. S4C,F) measurements were taken using Fiji software immediately before bridge rupture or within 16 hours of anaphase.

X-ten sequencing

DNA from the post-crisis samples and parental lines was sequenced on the Illumina X10 platform to a target coverage of 30x whole human genome. Sequencing libraries were synthesized from genomic DNA on robots according to manufacturer's protocols. Cluster generation and sequencing were performed using the manufacturer pipelines. Average sequence coverage achieved across the samples was 33.3x (range, 27.4-35.9x).

Mapping and analysis

Mapping to the human genome was performed using the BWA algorithm (Li and Durbin, 2010), using the BWA mem version 0.7.8. The exact genome build used was hs37d5.

Copy number analysis

The reference genome was divided into windows expected to generate an equivalent number of reads as done previously (Campbell et al., 2008; Li et al., 2009) was used to extract mapped reads with a mapping quality of at least 35 and with the following flags:

- Properly paired
- Non-secondary
- QC-pass
- Non-duplicate
- Non-supplementary

The number of reads overlapping with each genomic window was counted using BEDTools (Quinlan, 2014). Copy number was inferred from read depth data as described previously (Li et al., 2014).

Rearrangement calling and chromothripsis

The sequence data from all sequenced samples were merged. Clusters of abnormally paired read pairs were identified from the merged sequence data using an in-house algorithm 'Brass'. Raw rearrangement calls supported by clusters of abnormally mapped read pairs were called if the clusters were formed of at least four read pairs all from the same sample. An exception was made with X-37, whose rearrangement data was noisier in other samples; for rearrangement calls to be made in X-37 at least six read pairs were required. The raw rearrangements were filtered, as has been described previously (Li et al., 2014). Statistical testing for segment orders was performed using Spearman correlation and for other criteria done as in Li et al., 2014).

Published chromothripsis criteria (Korbel and Campbell, 2013) were used to assess regions with complex clusters of chromothripses. 1. Clustering of breakpoints: Applicable. Five out of nine regions with high density of rearrangements show strong clustering of rearrangement breakpoints. 2. Regularity of oscillating copy-number stages: Applicable. As opposed to "conventional chromothripsis," copy numbers oscillate over multiple copy number states since chromothripsis takes place on chromosomes amplified through BFBs. 3. Interspersed loss and retention of heterozygosity: Applicable. Interspersed loss and retention of heterozygosity was present in multiple chromosomes with chromothripsis. 4. Prevalence of rearrangements affecting a specific haplotype: Applicable. Karyotyping analysis confirms prevalence of rearrangements on a specific haplotype. 5. Randomness of DNA segment order and fragment joins: Applicable. Rearrangements join orientations in chromothripsis regions are consistent with random draws from a uniform multinomial distribution. However, the chromothripsis events involve fairly low numbers of intra-chromosomal rearrangements, which would decrease power in finding statistical departures from a uniform multinomial distribution. 6. Ability to

walk the derivative chromosome: Not applicable, as chromothripsis takes place on chromosomes with preceding duplication through BFBs. In this scenario, it would be impossible to walk the derivative chromosome (Li et al., 2014).

Computation of variant allele frequency

Samtools (v1.2) and bcftools (v1.2) were used to generate a list of variants in the parental clone (Phi) with default parameters. Of the output variants, only heterozygous biallelic SNPs with the highest variant quality (222) were included, yielding 1,669,777 heterozygous loci in the clone. Samtools and bcftools were then used to compute the variant allele frequency at these reference heterozygous loci.

Mutation calling and kataegis

Point mutations were called using an in-house algorithm 'Caveman' as before (Nik-Zainal et al., 2012a). The RPE-1/Rbsh/p21sh/rtTA clone from which T2p1 and T2cl.24 were derived (see Table S1) was used as the reference sample. Raw mutations were filtered as follows:

- Homopolymer filter. Mutations which had a homopolymer repeat of at least six bases on either side of the mutation and where the mutated base was same as the base of the homopolymer repeat(s) were removed.
- Soft-clip filter. Mutations where more than half of the supporting reads were soft-clipped were removed.
- Remapping filter. For each raw mutation, all the mutation-supporting reads were extracted. The reads were mapped, using BWA mem, to a database of 'decoy-sequences', which included all the sequences other than autosomes, chrX, chrY and chrM in GRCh38 and hs38d1. In addition, the HLA region alternative haplotypes were obtained from

http://hgdownload.soe.ucsc.edu/goldenPath/hg19/bigZips/chromFa.tar.gz and added to the decoy sequence database. The remapping difference score was obtained for each read by subtracting the original BWA mem mapping score with the mapping score from remapping the read to the decoy sequence database. Mutations that had an average remapping score over supporting reads < 40 were removed.

Kataegis mutation clusters were detected using visual inspection based on the criteria of short inter-mutation distance (generally <2kb) between cytosine mutations that were processive and enriched with TpC context.

Association of mutation clusters and rearrangements

The distances from the center (middle point between the first and the last mutation) of kataegis events to rearrangement breakpoints were computed manually. To associate a rearrangement breakpoint with a kataegis cluster, the rearrangement breakpoint had to have the right orientation with respect to its relative position to the kataegis cluster. Thus, rearrangements before and after a kataegis cluster in reference genome coordinates were required to be of - and + orientation, respectively.

In some cases a kataegis event was clearly adjacent to a rearrangement breakpoint based on copy number data, but the associated rearrangement call was missed (e.g. X-25 chromosome 13, Figure 5B). In these cases the distance from the kataegis event to the copy number segmentation breakpoint was used instead. Analogous to the rearrangement orientation requirement, copy number segmentation breakpoints before or after a kataegis cluster were required to have a copy number increase or decrease, respectively.

SUPPLEMENTAL TABLE S1. Derivation of cell lines used in this study. Related to Figure 1-4.

Derivation of vp1, T2p1, and T2cl24				
	RPE-1	hTERT/hygro		
	RPE-1	hTERT/hygro	Rbsh+p21sh (GFP/puro)	
	RPE-1	hTERT/hygro	Rbsh+p21sh (GFP/puro)	rtTA (blast)
clone	RPE-1/Rbsh/p21sh/rtTA			
vp1 pool	RPE-1/Rbsh/p21sh/rtTA		dox vector (neo)	
T2p1 pool	RPE-1/Rbsh/p21sh/rtTA		dox mycTRF2-DN(IRESneo)	
T2cl24	RPE-1/Rbsh/p21sh/rtTA		dox mycTRF2-DN(neo)	
RPE-1 derivatives				
RPE-1	NLS-3xmTurquoise2 (FACS, clone)			
vp1 derivatives				
vp1	H2B-mCherry (FACS, pool)			
vp1	NLS-3xmTurquoise2 (FACS, clone)			
T2cl24 derivatives				
T2cl24	H2B-mCherry (FACS, pool)			
T2p1 derivatives				
T2p1	NLS-3xmTurquoise2 (FACS, clone)			
T2p1	H2B-mCherry (FACS, clone)			
T2p1 NLS-3xmTurquoise2 derivatives				
T2p1	mCherry-LaminB1 (FACS, pool)			
T2p1	mCherry-LAP2β (FACS, pool)			
T2p1 H2B-mCherry derivatives				
T21	H2B-mCherry (FACS, clone) myrPALM-mTurquoise2 (FACS, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-TREX1-D18N (FACS, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-tubulin (FACS, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-BP1-2 (FACS, pool)			
T2p1 H2B-mCherry mTurquoise2-RPA70 derivatives				
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) 3xFLAG-TREX1 (IRESzeo, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) 3xFLAG-TREX1D18N (IRESzeo, pool)			
CRISPR TREX1 KOs of T2p1 H2B-mCherry mTurquoise2-RPA70 and derivatives				
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.2			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.2 3xFLAG-TREX1 (IRESzeo, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.2 3xFLAG-TREX1D18N (IRESzeo, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.25			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.25 3xFLAG-TREX1 (IRESzeo, pool)			
T2p1	H2B-mCherry (FACS, clone) mTurquoise2-RPA70 (FACS, clone) CRISPR TREX1-/- clone2.25 3xFLAG-TREX1D18N (IRESzeo, pool)			
T4 derivatives				
T4	van Steensel et al 1998			
T4	H2B-mCherry (FACS, clone) GFP-RPA70 (FACS, clone)			
T4	H2B-mCherry (FACS, clone) GFP-RPA70 (FACS, clone) CRISPR TREX1-/- clone5			

SUPPLEMENTAL TABLE S2. Karyotypes of sequenced cell lines

	Parental lines
	T2p1: 47,X,der(X)t(X;10)(qter;q21),i(12)(p10) RPE-1/Rbsh/p21sh/rTA: 47,X,der(X)t(X;10)(qter;q21),i(12)(p10)
	T2cl24 derivatives:
141	46,X,der(X)t(X;10)(qter;q21),-7,**psu dic(12;7)(q24;q22)**,i(12)(p10) [18]
	47,X,der(X)t(X;10)(qter;q21),+i(12)(p10),r(12)(p13q24)[2]
144	47,X,der(X)t(X;10)(qter;q21),dup(3)(~p21p25),+i(12)(p10)[17]
	47,X,der(X)t(X;10)(qter;q21),dup(3)(~p21p25),+i(12)(p10),der(6)t(3;6)(?q11;p25)[1]
	47,X,der(X)t(X;10)(qter;q21),dup(3)(~p21p25),+i(12)(p10),der(6)t(6;18)(p25;q11)[1]
	47,X,der(X)t(X;10)(qter;q21),dup(3)(~p21p25),r(12)(p13q24),+i(12)(p10)[1]
	T2p1 derivatives:
I.1	52,X,der(X)t(X;10)(qter;q21),+2,+7,+8,+11,+i(12)(p10),+15[14]
	52,X,der(X)t(X;10)(qter;q21),+2,del(3)(p11),+7,+8,+11,+i(12)(p10),+15[1]
	52,X,der(X)t(X;10)(qter;q21),+2,+der(7)t(2;7)(q11;q11),+8,+11,+i(12)(p10),+15[1]
	52,X,der(X)t(X;10)(qter;q21),+2,+7,+8,+11,+i(12)(p10),+del(15)(?q15)[1]
	54,X,der(X)t(X;10)(qter;q21),+2,+7,+8,+11,+i(12)(p10),+15,+17,+20[1]
X.25	48,X,der(X)t(X;10)(qter;q21),**psu dic(4;13)**,i(12)(p10),der(12)t(8;12)
X.29	48,X,der(X)t(X;10)(qter;q21),**der(11)**,i(12)(p10)
X.32	46,X,der(X)t(X;10)(qter;q21),i(12)(p10),-18
X.33	45,der(X)t(X;10)(qter;q21),**der(8)**,i(12)(p10),-18
X.35	47,X,der(X)t(X;10)(qter;q21),der(6),i(12)(p10)
X.36	45,der(X)t(X;10)(qter;q21),-2,-7,-10,i(12)(p10),der(18)
X.37	42~44,X,der(X)t(X;10)(qter;q21),**der(1)t(1;22)(p36;?q13)**,der(1)t(1;15)(q21;?)t(15;17)(?;q21),+i(12)(p10), der(13)t(13;14)(p11;q24),-14,der(15)t(1;15)(?;q24),**der(18)t(18;22)(?q11;?q11),-17,-22**[cp13]
	Bold: chromothriptic clones and their chromothriptic marker chromosomes. [n] indicates the number of cells showing the indicated karyotype. I.1 was kept in doxycycline for two weeks.

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