

Original Article

Tumor-specific trisomy signatures shaped by lineage-dependent selection: evidence from 76,590 cases

Felix Mitelman¹, Jakob Hofvander¹, Jonas Björk^{2,3}, Nils Mandahl¹

¹Division of Clinical Genetics, Department of Laboratory Medicine, Lund University, Lund, Sweden; ²Division of Occupational and Environmental Medicine, Lund University, Lund, Sweden; ³Clinical Studies Sweden, Forum South, Skåne University Hospital, Lund, Sweden

Received April 29, 2026; Accepted June 10, 2026; Epub October 15, 2026; Published October 30, 2026

Abstract: Whole-chromosome gains (trisomies) are the most frequent cytogenetic abnormalities in human cancer, yet their overall distribution and biological significance across tumor types remain incompletely understood. Using the Mitelman Database of Chromosome Aberrations and Gene Fusions in Cancer, we performed a comprehensive analysis of trisomy patterns in 76,590 tumors representing 28 tumor types across major hematologic and solid tumor groups, encompassing myeloid, lymphoid, epithelial, mesenchymal, and neuroectodermal tumors. In total, 64,947 trisomies involving all autosomes and the X chromosome were analyzed. Pairwise comparison and hierarchical clustering analyses of trisomy frequency distributions revealed that 301 of 378 (79.6%) tumor-type pairs display distinct, tumor-specific trisomy signatures. Apparent cross-tumor similarities were largely attributable to shared high-frequency gains of chromosomes 7, 8, and 12; exclusion of these trisomies abolished 44 of 60 (73%) significant associations, indicating that most similarities reflect single dominant gains. Trisomy patterns were not influenced by the presence of common sole trisomies. Moreover, trisomy distributions were highly reproducible, showing strong concordance between sexes and remarkable stability across decades. Together, these findings demonstrate that trisomies are non-randomly distributed and form robust, tumor-characteristic signatures. The results are in line with comprehensive genomic studies of multiple cancer types from The Cancer Genome Atlas demonstrating that copy number alteration patterns are strongly influenced by the cell of origin. The present cytogenetic analysis of whole-chromosome gains extends these observations and supports a model in which trisomy patterns are shaped by lineage-dependent selective constraints imposed by the cellular origin of the tumor, rather than representing stochastic by-products of chromosomal instability.

Keywords: Cancer, trisomy, chromosomes, lineage, aneuploidy

Introduction

Numerical chromosomal abnormalities are among the most pervasive cytogenetic alterations in human cancer, and trisomies, i.e., the gain of an entire chromosome, represent the most frequently observed cytogenetic changes. They have been documented in virtually all types of human neoplasms, spanning hematologic malignancies and solid tumors of various morphologic entities and organ localizations, reported in 19% of benign tumors and 37% of malignant neoplasms [1]. Trisomies may occur as sole anomalies, in which case they are often considered primary pathogenetic events, or they may arise as secondary aberrations acquired during clonal evolution [2]. Their abundance and dual

potential - as early drivers or later progression-related events - make them highly interesting for detailed studies to understand their role in tumorigenesis.

Despite their ubiquity, the distribution of gained chromosomes is far from random. Certain chromosomes are characteristically and recurrently gained in specific tumor types. Trisomy 7 is repeatedly observed in several solid tumors including, for example, astrocytomas, chondrosarcomas, and renal and colonic adenocarcinomas; trisomy 8 is a hallmark of myeloid malignancies and often seen in Wilms tumors and Ewing sarcomas; and trisomy 12 typifies many cases of chronic lymphocytic leukemia [1]. Such consistent patterns underscore that chromo-

somal gains may play a pathogenetically important role in a wide variety of biologically diverse tumor classes. However, these characteristic trisomies represent only a small subset of the numerical abnormalities observed across cancers. Thus, they provide limited insight into the overall patterns and biological significance of trisomies in general. The gain of a single chromosome can have profound and diverse consequences, ranging from promotion of genomic instability to widespread gene expression deregulation. Consequently, the functional impact of trisomies on cancer initiation and progression is complex and remains incompletely understood [3-7]. Defining the distribution of trisomies across tumor types will be essential for elucidating their biological role in cancer.

Although numerous studies have explored individual trisomies within selected cancers, a comprehensive examination of trisomy distribution across the full spectrum of human neoplasms is lacking. The Mitelman Database of Chromosome Aberrations and Gene Fusions in Cancer [1] - comprising more than 80,000 karyotypically abnormal tumors - provides a unique opportunity to perform such a systematic survey. By utilizing this resource, the present study - the most extensive evaluation to date of trisomy landscapes in human cancer - evaluates 64,947 trisomies in 76,590 malignant tumors in 28 tumor types, representing six major groups: myeloid, lymphoid, epithelial, mesenchymal, neuroectodermal, and germ-cell tumors. The central question addressed is if some tumors share trisomy signatures, suggesting common pathogenetic mechanisms or if the trisomy patterns are tumor-specific. To investigate this question, we conducted pairwise comparisons of trisomy profiles in the 28 tumor types in order to identify and quantify similarities and differences. The results reveal that most cancers exhibit unique, tumor-constrained trisomy distributions, with only limited cross-tumor similarities.

Materials and methods

Clonal chromosome abnormalities were obtained from the Mitelman Database [1]. When queried on 10 January 2026, the database contained 87,719 neoplasms, including 8,147 unpublished cases from our laboratory. For the assessment of trisomy frequencies, only tumor types with at least 200 reported cases and a

minimum of 200 identified trisomies were included. This selection yielded 76,590 cases across 28 tumor entities, comprising a total of 64,947 trisomies for analysis. The 28 tumor types included: acute myeloid leukemia (AML), chronic myeloid leukemia (CML), myelodysplastic syndromes/myeloproliferative neoplasms (MDS/MPN), myeloproliferative neoplasms (MPN), myelodysplastic syndromes (MDS), acute lymphoblastic leukemia/lymphoblastic lymphoma (ALL), plasma cell neoplasms (PCN), mature B-cell neoplasms (B-ML), mature T- and NK-cell neoplasms (T-ML), Hodgkin disease (HD), adenocarcinomas of the kidney (CA ad kidney), breast (CA ad breast), ovary (CA ad ovary), large intestine (CA ad colon), Wilms tumor (Wilms), head and neck squamous cell carcinoma (SQC H&N), germ cell and gonadal stromal cell neoplasms (GCT), astrocytoma (Astcyt), neuroblastoma (NB), primitive neuroectodermal tumors (PNET), malignant melanoma (Mal mel), malignant fibrous tumors (Mal fibr), malignant myogenic tumors (Mal myo), synovial sarcoma (SA syn), chondrosarcoma (SA chondr), osteosarcoma (SA ost), Ewing sarcoma (SA Ewing), and meningioma (MNG). With the exception of GCT and MNG, the remaining 26 tumor types could be categorized into five major groups: Myeloid disorders (MYEL), lymphatic disorders (LYMPH), malignant epithelial tumors (MET), malignant neuroectodermal tumors (MNET), and malignant mesenchymal tumors (MMT).

A program was developed to identify all tumors with numerical chromosome abnormalities. This involved an initial SQL query to retrieve all relevant cases from the database, followed by processing with a custom algorithm that filtered out non-informative data and extracted the required cytogenetic information. From this curated dataset, cases containing at least one trisomy - defined as the gain of an entire chromosome - were selected for further analysis. In accordance with the internationally accepted ISCN nomenclature, all numerical chromosome abnormalities in the database are described relative to the appropriate ploidy level. Thus, in near-haploid karyotypes (< 35 chromosomes) numerical changes are expressed relative to a haploid complement of 23 chromosomes; in near-triploid karyotypes (58-80 chromosomes) relative to 69 chromosomes; in near-tetraploid karyotypes (81-103 chromosomes) relative to 92 chromosomes; and so forth. Trisomy fre-

Tumor-specific trisomy signatures

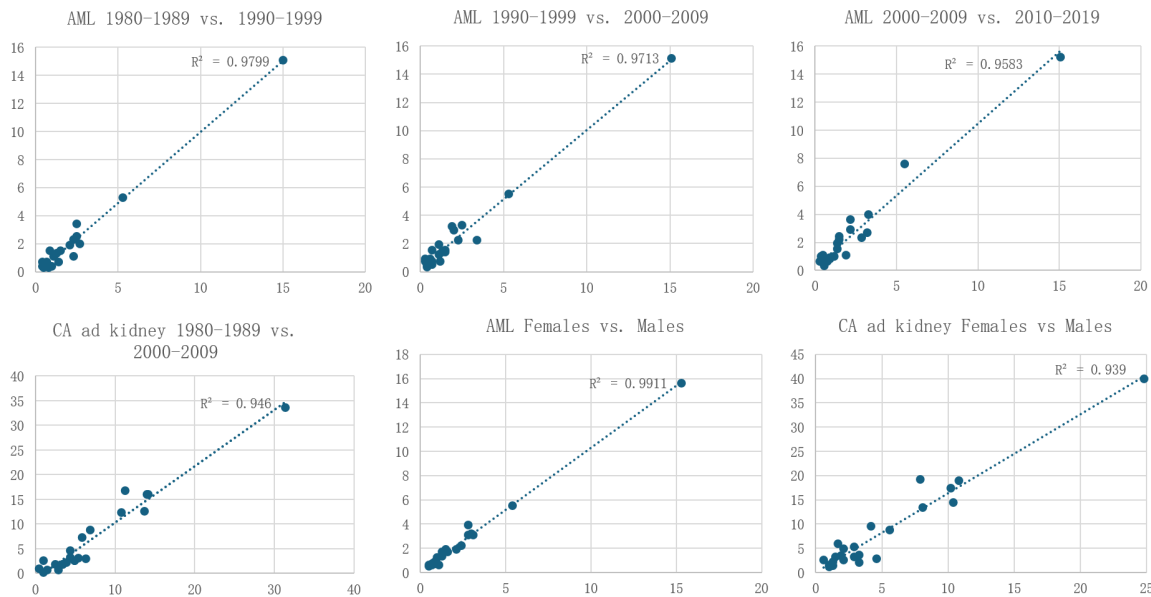


Figure 1. Trisomy frequency profiles in acute myeloid leukemia (AML) and kidney adenocarcinoma (CA ad kidney) stratified by 10-year periods and gender. Highly concordant distributions are observed between females and males and across all decades for both tumor types ($P < 10^{-15}$ for all comparisons).

quencies were calculated for each of the 23 chromosomes, excluding the Y chromosome, and expressed as percentages of the total number of cases within each tumor entity. To evaluate the similarity of trisomy distribution patterns between tumor types, Pearson correlation coefficients (R) and their squares (coefficient of determination: R^2) were computed for all pairwise combinations ($28 \times 27/2 = 378$). This analysis measures the degree of linear relationship between chromosomal alteration profiles, regardless of scale. For each pair, the correlation was calculated across all 23 chromosomes. Statistical significance was determined using a two-tailed test, with P -values derived under the null hypothesis of no correlation ($R = 0$). All analyses were conducted using Python. Significance thresholds for Pearson correlations were defined as follows: $P < 0.01$ (significant), and $P < 0.001$ (highly significant).

Unsupervised hierarchical clustering of tumor types was performed using the R environment (v4.5.3). The dendrogram was generated using pvclust (v2.2.0) (distance = 1 - Pearson correlation, clustering method = ward.D2) and visualized together with the trisomy numbers using pheatmap (v1.0.13).

To assess the robustness of the analytical approach and to evaluate the potential influ-

ence of random events or technical biases on trisomy distributions, we performed a pilot analysis comparing trisomy patterns within the same tumor entity between sexes and across different time periods. The two most extensively studied tumor types representing hematologic and solid tumors were selected: AML (24,073 cases) and CA ad kidney (1,347 cases). Trisomy distributions were compared between males and females and across successive publication periods (**Figure 1**). For AML, four decades were analyzed (1980-1989, 1990-1999, 2000-2009, and 2010-2019), and for CA ad kidney three decades (1980-1989, 1990-1999, and 2000-2009). In both tumor types, the trisomy profiles showed very high concordance between sexes and across time periods ($R^2 = 0.94$ - 0.99 ; $P < 10^{-15}$ in all comparisons), indicating that the observed trisomy distributions are highly stable and supporting the robustness of the analytical framework.

Results

Table 1A and **1B** show the frequency distributions of all trisomies in 10 hematologic disorders and 18 solid tumor types, respectively. The mean number of trisomies per case ranged from 0.36 in MPN to 2.33 in GCT. Results of pairwise comparisons of trisomy distributions in the 28 tumor types, including R , R^2 , exact

Tumor-specific trisomy signatures

Table 1A. Distribution of trisomy frequencies (%) for chromosomes 1 - 22 and X among 10 hematologic malignant disorders

Tumor type	AML	CML	MDS/MPN	MPN	MDS	ALL	PCN	B-ML	T-ML	HD
No. cases	24073	5913	955	1817	6699	12988	2526	10542	1472	259
No. trisomies	12660	2827	360	658	2958	17562	5804	7855	1290	442
No. trisomies per case	0.53	0.48	0.38	0.36	0.44	1.35	2.30	0.75	0.88	1.71
Trisomy 1	1.7	0.6	1.4	2.8	4.4	2	5.7	1	1.6	5
Trisomy 2	0.8	0.3	0.5	0.6	0.5	1.4	2.9	1.8	2.6	14.3
Trisomy 3	0.6	0.5	0.1	0.2	0.4	0.8	21	5.2	9.9	7.7
Trisomy 4	2	0.6	0.8	0.2	0.5	9.4	3.2	0.7	2.4	2.7
Trisomy 5	0.7	0.5	0.6	0.3	0.5	4.2	19.4	2.9	7	16.2
Trisomy 6	2.3	1.6	0.9	0.8	1.3	10.8	6.6	1.2	2.2	3.5
Trisomy 7	0.6	0.5	0.2	0.3	0.3	1.6	20.5	8.2	7.7	5.4
Trisomy 8	15.4	20.5	16.1	12.6	16.4	9.3	5.3	3.4	8.4	8.9
Trisomy 9	1.6	0.9	1.2	7.6	1.8	3	27.1	1.7	3.5	13.1
Trisomy 10	1.7	1.7	0.2	0.4	0.7	10.3	1.8	1.2	1.4	3.9
Trisomy 11	3.1	0.8	1	0.7	2.1	2.9	20.3	2.4	2.3	4.6
Trisomy 12	0.8	1.2	0.6	0.3	0.5	2	2.4	15.6	2.6	15.4
Trisomy 13	3.4	1	1.9	1.2	1.3	1.8	1.4	1.5	1.1	0.8
Trisomy 14	1.5	1.3	1.4	0.6	1.1	11.2	2.8	1	2.6	5.8
Trisomy 15	1.1	1	0	0.8	1.5	1.8	25.9	1.2	2	3.5
Trisomy 16	0.5	0.5	0	0.2	0.4	1.5	1.5	1.5	1.4	6.6
Trisomy 17	0.4	1.6	0.1	0.2	0.2	7.6	4.1	1.2	2.1	5
Trisomy 18	0.9	0.7	0.6	0.3	0.7	10.1	11.4	6.1	4.3	8.1
Trisomy 19	2.9	5.4	2.7	1.3	2.1	3	22.4	2.2	4.8	8.1
Trisomy 20	1.3	0.9	0.5	0.6	0.8	2.1	4.2	2.5	3.1	8.5
Trisomy 21	5.4	3.6	5.5	3.4	4	20	16	3.9	6.3	10.4
Trisomy 22	3.1	1	0.5	0.6	1.9	3.8	2.7	1.6	3.2	6.6
Trisomy X	0.8	0.9	0.6	0.4	0.6	14.6	1.1	6.5	5.2	6.6

For abbreviations, see Materials and Methods.

P-values, and corresponding significance levels, are presented in [Supplementary Table 1](#). The results of these comparisons are illustrated in **Figure 2**. Of 378 comparisons, 301 (79.6%) showed no significant similarity, whereas only 77 (20.4%) were significant ($P < 0.01$), including 43 (11.4%) that were highly significant ($P < 0.001$). Thus, the overwhelming majority of tumor entities display distinct distributions of chromosomal trisomies rather than a common pattern shared across tumor classes. Unsupervised hierarchical clustering of all tumor types and tumor groups (**Figure 3**) further illustrates the diversity of trisomy patterns and demonstrates that the five myeloid disorders form a tightly clustered group. In contrast, the remaining tumor groups are, with a few exceptions (see below), widely dispersed. Notably, as is evident from **Table 1** and **Figure 3**, chromosomes 7, 8, and 12 are nonrandomly involved

in a large number of tumor types spanning several different tumor groups.

A potential source of bias relates to the ISCN convention that numerical chromosome abnormalities are described relative to the prevailing ploidy level rather than to the diploid state. Consequently, trisomy patterns might theoretically differ between diploid and polyploid tumors. To examine this possibility, we compared trisomy distributions in diploid tumors (35-57 chromosomes) with those in polyploid tumors (> 57 chromosomes) in four tumor types with relatively high frequencies of polyploid cases: kidney adenocarcinoma (20%), colon adenocarcinoma (41%), osteosarcoma (53%), and germ-cell tumors (62%). In all comparisons (data not shown), the trisomy patterns were highly similar ($R^2 = 0.64-0.84$; $P < 0.001$). These findings indicate that variation in ploidy level does not

Tumor-specific trisomy signatures

Table 1B. Distribution of trisomy frequencies (%) for chromosomes 1 - 22 and X among 18 solid tumor types

Tumor type	CA ad kidney	Wilms	CA ad breast	CA ad ovary	SQC H&N	CA ad colon	GCT	Ast cyt	NB	PNET	Mal mel	Mal fibr	Mal myo	SA syn	SA chondr	SA ost	SA Ewing	MNG
No. cases	1347	516	796	446	452	349	540	772	313	200	386	406	456	296	297	303	518	953
No. trisomies	2157	1052	803	496	512	534	1260	940	462	235	548	305	685	302	535	339	862	504
No. trisomies per case	1.60	2.04	1.01	1.11	1.13	1.53	2.33	1.22	1.48	1.18	1.42	0.75	1.50	1.02	1.80	1.12	1.66	0.53
Trisomy 1	1.3	3.5	9	8.5	4	2.3	6.5	4.9	5.4	6.5	5.4	1	4.2	1.4	3.7	3.6	4.1	0.8
Trisomy 2	7.6	7.9	2.9	8.5	4.6	5.7	8.5	4	5.8	3	6.7	2.2	16.4	9.8	11.4	9.2	11.6	0.9
Trisomy 3	7.5	7.2	4.1	9.6	4.2	4	12.6	4.7	4.5	4	4.9	1	2.9	0.7	4	4.3	2.7	3.3
Trisomy 4	2.5	1.2	2.1	2	2.2	1.1	3	3.2	3.8	3	4.1	1.5	1.1	3	2	4.3	6.9	1.6
Trisomy 5	11.6	2.9	4.3	5.4	4.6	4.3	3.7	4.3	5.4	6	3.6	5.7	9	3	11.8	5.3	9.1	4.7
Trisomy 6	1.8	21.5	3.6	2.9	5.1	5.4	7	4.1	8	8.5	8.5	1.2	4.6	1.4	2.4	5.9	3.5	0.7
Trisomy 7	34.6	16.7	10.4	9.2	11.5	27.8	23	34.2	13.4	15	17.4	7.4	11.8	8.8	23.6	14.5	8.3	3.3
Trisomy 8	3.8	25.6	7.4	4.9	8.6	8.6	26.7	5.2	6.4	7.5	15.3	10.8	16	13.2	12.1	7.6	33.8	2.3
Trisomy 9	1.9	10.1	4.1	2.9	7.5	4.6	4.8	3.2	7	4.5	4.1	1.7	1.8	6.4	2.4	2.3	5.8	2.7
Trisomy 10	3.4	7.9	3.1	4	4.6	2.9	3.9	1.6	4.2	2	1.3	0.7	3.1	0.7	0.7	0.7	4.2	1.4
Trisomy 11	2.8	1.2	2.9	4.3	5.3	5.2	2.6	4	4.8	6	3.1	7.4	9.6	1	4	3.6	3.1	1.4
Trisomy 12	15.3	37	4	15	5.1	8	21.9	5.7	9.9	5.5	4.4	3.2	11.6	14.2	13.8	7.3	18	4.8
Trisomy 13	4.2	16.5	2.3	3.4	2.7	16.9	4.6	1.2	8.6	5.5	6.2	1	6.8	4.1	1.3	2.3	3.7	2.4
Trisomy 14	1	1.2	3	3.4	2.7	3.7	10	1.7	4.2	3	2.1	2.2	2.9	3.4	1.7	4	7.9	0.9
Trisomy 15	1.3	3.5	2.4	2	2.7	2.9	9.3	3	2.9	2	5.7	3.4	3.3	6.1	8.4	2.3	6	2.5
Trisomy 16	16	0.6	5.7	3.1	4.4	8.3	6.1	2.1	4.8	0.5	3.9	0	2.9	2.7	6.1	3	2.7	1.5
Trisomy 17	14.7	6.8	2.4	1.8	5.3	1.4	11.1	3.2	14.4	3.5	3.4	3.2	3.9	3.4	4.7	2	1.9	3.7
Trisomy 18	3	13.6	6.3	1.8	3.3	1.4	3.3	4	10.9	10.5	4.1	3.7	4.4	1.7	7.4	2	9.1	1.9
Trisomy 19	4.3	2.5	2.9	3.4	2.4	4.9	5.9	7.8	2.6	5.5	5.4	2	7.9	3.4	15.2	5.3	1.7	2.8
Trisomy 20	13	8.9	7.4	8.5	10	14.6	8.7	8.4	8	6.5	13.5	8.4	13.6	2.7	18.5	8.9	11.4	6.1
Trisomy 21	3.5	3.9	3.1	3.6	0.4	3.7	25.6	5.1	7	5.5	7.3	2.2	3.9	7.8	13.8	6.9	7.1	2
Trisomy 22	2.4	1.2	2.5	1.1	9.1	3.4	8.1	3	4.8	2	8.3	3.2	5.3	2	7.4	2.6	1	0.5
Trisomy X	2.4	2.7	4.8	1.8	2.9	11.7	16.5	3.2	1	1.5	3.1	2	3.3	1.4	3.7	4	2.9	0.6

Tumor-specific trisomy signatures

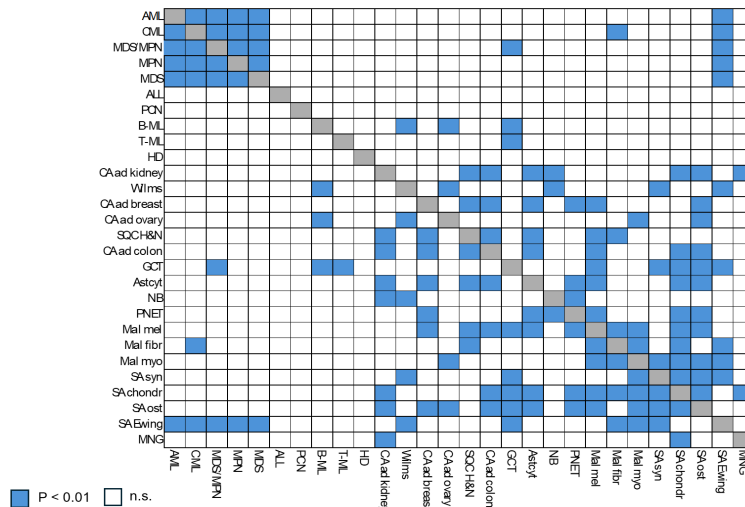


Figure 2. Dichotomized similarity matrix showing pairwise relationships among all tumor types after classification of correlations as either significant ($P < 0.01$) or non-significant ($P > 0.01$). This threshold highlights the most robust similarities and shows that most tumor-type comparisons fall into the non-significant category, underscoring the predominance of tumor type-specific trisomy profiles. For abbreviations of neoplasms, see Materials and Methods.

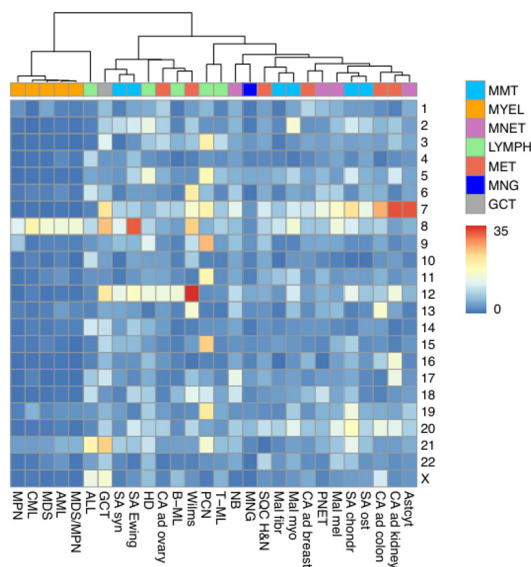


Figure 3. Unsupervised hierarchical clustering (distance = 1 - Pearson correlation, method = ward.D2) of 28 tumor types based on the frequency of trisomy per chromosome. Color-coded tumor groups from top to bottom: MMT, malignant mesenchymal tumors; MYEL, myeloid disorders; MNET, malignant neuroectodermal tumors; LYMPH, lymphoid neoplasms; MET, malignant epithelial tumors; MNG, meningiomas; GCT, germ cell tumors.

materially influence the trisomy distributions when chromosome gains are expressed according to ISCN conventions.

The major conclusions emerging from the present study with respect to trisomy patterns may be summarized as follows.

Hematologic disorders

Among hematologic disorders, 35 of 45 (77.8%) pairwise comparisons did not show significant similarities in trisomy profiles. Notably, all 10 comparisons among myeloid neoplasms (AML, CML, MDS/MPN, MPN, MDS) displayed highly similar distributions, whereas none of the 10 comparisons among lymphoid malignancies (ALL, PCN, B-ML, T-ML, HD) showed similar profiles. Furthermore, none of the 25 comparisons between myeloid and lymphoid disorders demonstrated significant similarity,

underscoring clear lineage-specific cytogenetic signatures.

Solid tumors

Among solid tumors, considerable heterogeneity was observed: 97 of 153 (63.4%) pairwise comparisons showed no significant similarity in trisomy profiles. Within tumor groups, no significant similarity was observed in 9 of 15 (60%) comparisons among malignant epithelial tumors, 2 of 6 (33.3%) among malignant neuroectodermal tumors, and 5 of 15 (33.3%) among malignant mesenchymal tumors. In cross-group comparisons, no significant similarity was observed between epithelial and neuroectodermal tumors in 11 of 18 (61%) comparisons, between epithelial and mesenchymal tumors in 16 of 36 (44%), and between neuroectodermal and mesenchymal tumors in 14 of 18 (78%). These findings indicate that most solid tumors exhibit distinct tumor-specific trisomy profiles.

Hematologic disorders - solid tumor comparisons

Comparisons between hematologic and solid tumors revealed that 169 of 180 (93.9%) pairs showed no significant similarity, indicating that their trisomy patterns are largely distinct.

Tumor-specific trisomy signatures

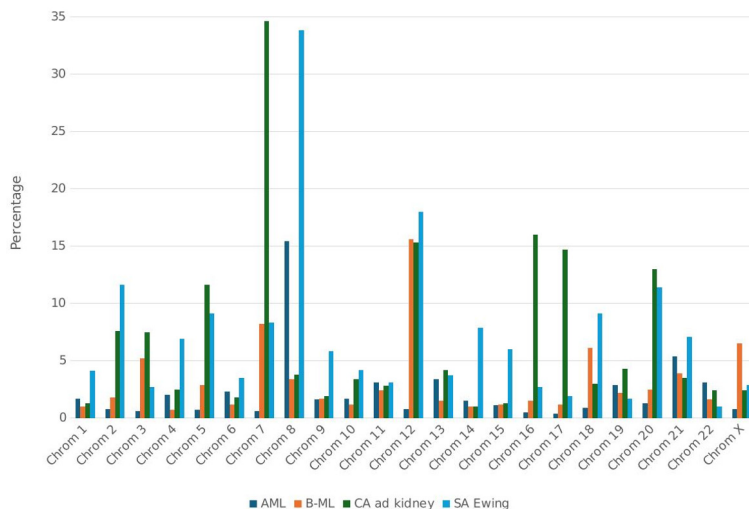


Figure 4. Histogram showing the frequency distribution of trisomies across all chromosomes in four tumor types representing different cellular lineages and biological contexts: acute myeloid leukemia (AML), mature B-cell neoplasms (B-ML), kidney adenocarcinoma (CA ad kidney), and Ewing sarcoma (SA Ewing). The marked differences between tumor types, despite occasional shared trisomies, illustrate the non-random and lineage-dependent nature of trisomy signatures.

Myeloid disorders showed significant similarity exclusively with SA Ewing (5 of 5 comparisons), but not with other malignant mesenchymal tumors (24 of 25 comparisons), nor with malignant epithelial tumors (30 of 30) or malignant neuroectodermal tumors (15 of 15). Among lymphoid disorders, no significant similarity was observed in any comparison with malignant neuroectodermal tumors (15 of 15) or malignant mesenchymal tumors (30 of 30), and only rarely with malignant epithelial tumors (28 of 30 comparisons showing no similarity). Notably, three lymphoid malignancies (ALL, PCN, and HD) showed no significant similarity to any other hematologic or solid tumor type. Thus, although occasional lineage-transcending similarities occur, they are rare. The distinct trisomy distributions observed across tumor types are illustrated in **Figure 4**, which shows representative frequency profiles for AML, B-ML, CA ad kidney, and SA Ewing.

Exclusion of shared frequent trisomies

To assess whether the observed associations in the pairwise comparisons were driven by, or largely influenced by, individual trisomies occurring at high frequencies in both tumor types, partial correlation analyses were performed by selectively excluding the most common shared

trisomy in each pair. Among the 77 pairs showing significant associations, this analysis was feasible in 60 pairs; no shared frequent trisomy occurred in the remaining 17 pairs. The shared gains were restricted to only three chromosomes: 7, 8, and 12. The results are presented in **Table 2**. In 44 of the 60 cases (73%), the associations became non-significant once the shared trisomy was excluded, highlighting that most apparent cross-tumor similarities arise from a single recurrent gain rather than an overall pattern. Illustrative examples include the strong association between all myeloid disorders and SA Ewing, entirely attributable to trisomy 8, and the correlation between CA ad kidney and CA

ad colon/Astcyt/SA chondr/SA ost, which disappeared when trisomy 7 was omitted. Similarly, the association between B-ML and Wilms/CA ad ovary vanished after exclusion of trisomy 12.

Effect of sole anomalies

To evaluate the influence of very common sole trisomies, cases with a sole abnormality were identified, and their trisomy profiles were compared with those of the remaining cases with multiple trisomies. Three tumor types (AML, B-ML, and CA ad kidney) representing different tumor groups (MYEL, LYMPH, MET) were selected because they contained sufficient numbers of cases with sole anomalies and allowed assessment of the frequently occurring trisomies of chromosomes 7, 8, and 12. In all three tumor types, the trisomy patterns among cases with sole anomalies were identical to those observed in cases with multiple trisomies ($P < 0.001$ in all comparisons). These findings demonstrate that even very frequent sole trisomies do not decisively influence the overall trisomy distribution that characterizes each tumor type.

Discussion

By exploiting the extensive dataset of the Mitelman Database [1], the present study pro-

Tumor-specific trisomy signatures

Table 2. Effects of removal of most frequent shared trisomic chromosomes in pairwise comparisons of significant associations

Tumor A	Tumor B	P value	-7	-8	-12	Tumor A	Tumor B	P value	-7	-8	-12
AML	CML	0.001		0.01		CA ad breast	CA ad colon	0.01	n.s.		
AML	MDS/MPN	0.001		0.001		CA ad breast	Astcyt	0.001	n.s.		
AML	MPN	0.001		n.s.		CA ad breast	PNET	0.001	n.s.		
AML	MDS	0.001		0.001		CA ad breast	Mal mel	0.01	n.s.		
AML	SA Ewing	0.001		n.s.		CA ad breast	SA ost	0.01	n.s.		
CML	MDS/MPN	0.001		0.001		CA ad ovary	Mal myo	0.01			0.01
CML	MPN	0.001		n.s.		SQC H&N	CA ad colon	0.01	n.s.		
CML	MDS	0.001		n.s.		SQC H&N	Astcyt	0.01	n.s.		
CML	Mal fibr	0.01		n.s.		SQC H&N	Mal mel	0.001	0.01		
CML	SA Ewing	0.001		n.s.		CA ad colon	Astcyt	0.001	n.s.		
MDS/MPN	MPN	0.001		n.s.		CA ad colon	Mal mel	0.001	n.s.		
MDS/MPN	MDS	0.001		0.001		CA ad colon	SA chondr	0.01	n.s.		
MDS/MPN	GCT	0.01		n.s.		CA ad colon	SA ost	0.001	n.s.		
MDS/MPN	SA Ewing	0.001		n.s.		GCT	Mal mel	0.01		n.s.	
MPN	MDS	0.001		0.01		GCT	SA ost	0.01	n.s.		
MPN	SA Ewing	0.001		n.s.		GCT	SA Ewing	0.01		n.s.	
MDS	SA Ewing	0.001		n.s.		Astcyt	PNET	0.001	n.s.		
B-ML	Wilms	0.001			n.s.	Astcyt	Mal mel	0.001	n.s.		
B-ML	CA ad ovary	0.001			n.s.	Astcyt	SA chondr	0.001	0.001		
B-ML	GCT	0.01			0.01	Astcyt	SA ost	0.001	0.001		
CA ad kidney	SQC H&N	0.01	n.s.			NB	PNET	0.01	n.s.		
CA ad kidney	CA ad colon	0.001	n.s.			PNET	Mal mel	0.01	n.s.		
CA ad kidney	Astcyt	0.001	n.s.			PNET	SA chondr	0.01	n.s.		
CA ad kidney	NB	0.01	n.s.			PNET	SA ost	0.01	n.s.		
CA ad kidney	SA chondr	0.001	n.s.			Mal mel	SA chondr	0.001	0.01		
CA ad kidney	SA ost	0.001	n.s.			Mal mel	SA ost	0.001	0.01		
Wilms	CA ad ovary	0.01			n.s.	Mal fibr	Mal myo	0.001		0.01	
Wilms	SA syn	0.001			n.s.	Mal fibr	SA Ewing	0.001		n.s.	
Wilms	SA Ewing	0.01		0.01		SA syn	SA chondr	0.01	n.s.		
CA ad breast	SQC H&N	0.01	n.s.			SA chondr	SA ost	0.001	0.001		

-7, -8, and -12 denote removal of chromosomes 7, 8, and 12, respectively. For abbreviations, see Materials and Methods.

vides the most comprehensive survey to date of trisomy distributions across neoplastic disorders and demonstrates that trisomy patterns are largely tumor type-specific. In fact, using a stringent significance threshold ($P < 0.01$), we show that approximately 80% of 378 pairwise comparisons among 28 tumor types, encompassing both hematologic malignancies and malignant solid tumors, showed no significant similarity in trisomy profiles, indicating that most tumor types harbor distinct and largely non-overlapping trisomy signatures.

A particularly striking observation is that only three chromosomes - 7, 8, and 12 - account for all shared high-frequency gains observed across tumor-type comparisons. These trisomies are well-established recurrent abnormalities [1, 2]: Trisomy 7 in a broad range of epithe-

lial cancers, trisomy 8 in myeloid neoplasms and certain sarcomas, and trisomy 12 in chronic lymphocytic leukemia. The extent to which these common trisomies contribute to the observed similarities in trisomy patterns between tumor types is therefore an important question. To address this, we selectively excluded chromosomes 7, 8, or 12 in the 60 pairwise comparisons in which these trisomies were shared. Removal of these frequent gains abolished no less than 44 of the 60 significant cross-tumor correlations. These findings indicate that many apparent similarities between tumor types are driven by a single highly recurrent gain rather than reflecting broader shared cytogenetic programs.

Another important aspect of the role of common trisomies concerns their frequent occur-

rence as sole cytogenetic abnormalities in several tumor types, most notably trisomy 7 in kidney adenocarcinoma, trisomy 8 in AML, and trisomy 12 in B-cell neoplasms [1]. A key question, therefore, is whether such sole trisomies exert a disproportionate influence on the overall trisomy signatures that characterize these malignancies. Our findings show that even very common sole trisomies do not decisively shape or bias the overall trisomy distributions within a tumor type.

The present study emphasizes that trisomy patterns form distinct signatures characteristic of individual malignancies across different cancer types defined by morphology, cell lineage, tissue type, and organ localization. The results are in line with comprehensive genomic studies of multiple cancer types from The Cancer Genome Atlas demonstrating that copy number alteration patterns are strongly shaped by the cell of origin [8-11]. Direct comparisons between cytogenetic and genomic data are challenging owing to differences in resolution, the inclusion of partial chromosomal gains in sequencing-based studies, and the fact that cytogenetics captures abnormalities at the single-cell and clonal level rather than as population averages. Still, both approaches converge on the same central conclusion: aneuploidy patterns are tumor-dependent. Interestingly, our recent cytogenetic study of almost 60,000 translocations across 58 tumor types [12] has demonstrated that also rare and even exceedingly rare translocation in a wide range of both hematologic disorders and solid tumors give rise to tumor-specific cytogenetic signatures, further emphasizing the important role of tumor cell origin in shaping cytogenetic tumor signatures.

Despite the very large amount of data available on trisomies in practically all tumor types, their pathogenetic significance remains unclear [4, 6, 7, 13]. Data have been provided that gains of a characteristic set of chromosomes are early events, possibly arising simultaneously before birth, in high hyperdiploidy acute lymphoblastic leukemia in children [14]. However, there is no convincing experimental evidence that trisomies are primary events in the sense that one copy number change may be directly responsible for cancer initiation [15]. One argument in favor of a primary pathogenetic role is that trisomies have been observed as the sole cytoge-

netic abnormality at diagnosis in several tumor types. However, it is well established that genomic analyses of tumors with sole cytogenetic numerical anomalies frequently reveal submicroscopic alterations, including pathogenic gene fusions, e.g., trisomy 5 in B-ALL with *ETV6::ABL1* [16]; trisomy 8 in Ewing sarcoma with *EWSR1::FLI1* [17], in AML with *KMT2A::AFDN* [18], and in acute promyelocytic leukemia with *PML::RARA* [19, 20]; trisomy 10 in B/myeloid mixed-phenotype acute leukemia with *KMT2A::USP2* [21]; and trisomy 21 in B-ALL with *ETV6::RUNX1* [22, 23]. Furthermore, trisomies described as sole abnormalities in a given tumor type may occur as secondary changes during tumor progression in the same disease entity characterized by a well-established primary structural cytogenetic abnormality [1]. Examples include trisomy 8 as a secondary change in CML with t(9;22)(q34;q11), in Ewing sarcoma with t(11;22)(q24;q12), and in myxoid liposarcoma with t(12;16)(q13;p11); trisomy 6 in ALL with t(4;11)(q21;q23); and trisomy 22 in AML with inv(16)(p13q22). In these settings, the trisomies are clearly secondary events. Additional evidence supporting a secondary role for trisomies comes from their limited clinical impact when present as sole abnormalities. For example, trisomies 4, 8, 11, 13, and 21 as isolated findings in AML do not define specific disease characteristics across AML subgroups and do not influence prognosis [24-26]. Similarly, the presence of a single additional trisomy does not alter the favorable prognosis associated with established primary lesions such as t(8;21), t(15;17), or inv(16) in AML [27, 28]. A final argument against a primary pathogenetic role in tumorigenesis is the observation that trisomies may occur as sole abnormalities in normal and non-neoplastic tissues. A well-known example is trisomy 7, which is common as a sole cytogenetic abnormality in several tumor types, including kidney and brain tumors, but has also been documented in normal kidney cells [29], normal brain tissue [30], and a variety of non-neoplastic conditions such as Dupuytren's contracture, Peyronie's disease, and synovia, cartilage, and osteophytes from patients with osteoarthritis [31-33]. Neoplastic transformation of such cells through, for example, the acquisition of a fusion gene would therefore by necessity include trisomy 7.

Available data thus indicate that trisomies are, in most cases, secondary to well-established

pathogenetically important cytogenetic abnormalities or to cryptic genomic alterations. Irrespective of their mode of origin, their biological consequences may differ fundamentally. Trisomies may represent neutral passengers tolerated by established tumor cells, but they may also contribute to tumor progression by modulating key phenotypic parameters such as proliferative capacity, invasiveness, therapy resistance, or metastatic potential. The clonal evolution characteristic of malignant disease, involving continuous acquisition of new abnormalities during tumor development and resulting in increasingly complex karyotypes in advanced stages, provides a well-established framework for the emergence and selection of secondary genomic changes [2, 34].

The trisomy signatures identified here likely reflect a composite of gains that confer selective advantage and gains that impose no significant selective disadvantage within a given cellular context, while strong negative selection pressures act against trisomies that are incompatible with cell survival or clonal expansion. It is also conceivable that the functional role of a given trisomy may change over time, such that a chromosome gain that initially provides a selective advantage may later become effectively neutral as additional driver events accumulate, or conversely, that a previously tolerated gain may become advantageous under altered selective conditions. Selection pressures within an evolving tumor are expected to fluctuate over time as a consequence of internal factors such as variable vascular supply, hypoxia, and spatially heterogeneous microenvironments, as well as external influences including therapy. It is therefore plausible that the forces shaping secondary chromosomal changes vary both temporally within a tumor and, even more markedly, between different tumor types and lineages, reflecting differences in, e.g., cellular differentiation state, signaling pathways, and mutational landscapes.

Our observation that trisomy patterns are in general characteristic of individual tumor types, together with the findings that these patterns are essentially identical in early-stage cases with trisomies as sole anomalies and in advanced cases with multiple additional abnormalities, indicates that trisomy signatures are not simply stochastic by-products of chromosomal instability. The striking concordance between

males and females and across successive publication periods as revealed by our pilot study demonstrate that trisomy signatures represent highly reproducible features of individual tumor types. A plausible explanation is that the selective constraints governing which trisomies can be acquired and maintained differ between tumors because they arise in cells with distinct developmental origins and regulatory programs, i.e., different cellular lineages. Thus, the strong concordance of trisomy profiles observed among myeloid neoplasms, which constitute a single well-defined lineage, contrasts with the greater heterogeneity seen among lymphoid, epithelial and mesenchymal tumors arising from diverse cellular origins (**Figure 3**).

Taken together, these findings provide important insights into the role of aneuploidy in cancer. Rather than representing random consequences of chromosomal instability, trisomies appear to reflect lineage-dependent selective constraints that shape the genomic landscape of tumor cells. As such, trisomy signatures may be viewed as integrated readouts of the cellular states established by primary oncogenic events and the selective environments in which tumor evolution occurs. Although most trisomies are likely secondary alterations, potentially influencing clinical parameters such as growth, invasiveness, metastasizing ability, and therapeutic response, their non-random distribution patterns in distinct tumor types suggest that they contribute to tumor adaptation in a context-dependent manner.

Acknowledgements

This study was supported by grants from The Swedish Cancer Society and The Swedish Childhood Cancer Foundation.

Disclosure of conflict of interest

None.

Address correspondence to: Felix Mitelman, Division of Clinical Genetics, Department of Laboratory Medicine, Lund University, BMC D14, SE-221 84 Lund, Sweden. Tel: +46-708245342; ORCID: 0000-0002-3757-7212; E-mail: felix.mitelman@med.lu.se

References

- [1] Mitelman F, Johansson B and Mertens F. Mitelman database of chromosome aberrations

- and gene fusions in cancer. 2026. Available from: <https://mitelmandatabase.isb-cgc.org>.
- [2] Heim S and Mitelman F. *Cancer Cytogenetics*. 4th ed. New York: Wiley-Blackwell; 2015.
- [3] Santaguida S and Amon A. Short- and long-term effects of chromosome mis-segregation and aneuploidy. *Nat Rev Mol Cell Biol* 2015; 16: 473-485.
- [4] Ben-David U and Amon A. Context is everything: aneuploidy in cancer. *Nat Rev Genet* 2020; 21: 44-62.
- [5] Torres EM. Consequences of gaining an extra chromosome. *Chromosome Res* 2023; 31: 24.
- [6] Baker TM, Waise S, Tarabichi M and Van Loo P. Aneuploidy and complex genomic rearrangements in cancer evolution. *Nat Cancer* 2024; 5: 228-239.
- [7] Sdeor E, Okada H, Saad R, Ben-Yishay T and Ben-David U. Aneuploidy as a driver of human cancer. *Nat Genet* 2024; 56: 2014-2026.
- [8] Beroukhi R, Mermel CH, Porter D, Wei G, Raychaudhuri S, Donovan J, Barretina J, Boehm JS, Dobson J, Urashima M, Mc Henry KT, Pinchback RM, Ligon AH, Cho YJ, Haery L, Greulich H, Reich M, Winckler W, Lawrence MS, Weir BA, Tanaka KE, Chiang DY, Bass AJ, Loo A, Hoffman C, Prensner J, Liefeld T, Gao Q, Yecies D, Signoretti S, Maher E, Kaye FJ, Sasaki H, Tepper JE, Fletcher JA, Tabernero J, Baselga J, Tsao MS, Demichelis F, Rubin MA, Janne PA, Daly MJ, Nucera C, Levine RL, Ebert BL, Gabriel S, Rustgi AK, Antonescu CR, Ladanyi M, Letai A, Garraway LA, Loda M, Beer DG, True LD, Okamoto A, Pomeroy SL, Singer S, Golub TR, Lander ES, Getz G, Sellers WR and Meyer-son M. The landscape of somatic copy-number alteration across human cancers. *Nature* 2010; 463: 899-905.
- [9] Hoadley KA, Yau C, Hinoue T, Wolf DM, Lazar AJ, Drill E, Shen R, Taylor AM, Cherniack AD, Thorsson V, Akbani R, Bowlby R, Wong CK, Wizerowicz M, Sanchez-Vega F, Robertson AG, Schneider BG, Lawrence MS, Noushmehr H and Malta TM; Cancer Genome Atlas Network, Stuart JM, Benz CC and Laird PW. Cell-of-origin patterns dominate the molecular classification of 10,000 tumors from 33 types of cancer. *Cell* 2018; 173: 291-304, e6.
- [10] Taylor AM, Shih J, Ha G, Gao GF, Zhang X, Berger AC, Schumacher SE, Wang C, Hu H, Liu J and Lazar AJ; Cancer Genome Atlas Research Network, Cherniack AD, Beroukhi R and Meyer-son M. Genomic and functional approaches to understanding cancer aneuploidy. *Cancer Cell* 2018; 33: 676-689, e3.
- [11] Steele CD, Abbasi A, Islam SMA, Bowes AL, Khandekar A, Haase K, Hames-Fathi S, Ajayi D, Verfaillie A, Dhami P, McLatchie A, Lechner M, Light N, Shlien A, Malkin D, Feber A, Proszek P, Lesluyes T, Mertens F, Flanagan AM, Tarabichi M, Van Loo P, Alexandrov LB and Pillay N. Signatures of copy number alterations in human cancer. *Nature* 2022; 606: 984-991.
- [12] Mitelman F and Mandahl N. Distinct signatures of chromosomal involvement in 59,251 translocations across 58 tumor types. A novel perspective. *Genes Chromosomes Cancer* 2025; 64: e70053.
- [13] Girish V, Lakhani AA, Thompson SL, Scaduto CM, Brown LM, Hagenson RA, Sausville EL, Mendelson BE, Kandikuppa PK, Lukow DA, Yuan ML, Stevens EC, Lee SN, Schukken KM, Akalu SM, Vasudevan A, Zou C, Salovska B, Li W, Smith JC, Taylor AM, Martienssen RA, Liu Y, Sun R and Sheltzer JM. Oncogene-like addiction to aneuploidy in human cancers. *Science* 2023; 381: eadg4521.
- [14] Woodward EL, Yang M, Moura-Castro LH, van den Bos H, Gunnarsson R, Olsson-Arvidsson L, Spierings DCJ, Castor A, Duployez N, Zaliowa M, Zuna J, Johansson B, Fojier F and Paulsson K. Clonal origin and development of high hyperdiploidy in childhood acute lymphoblastic leukaemia. *Nat Commun* 2023; 14: 1658.
- [15] Klockner TC and Campbell CS. Selection forces underlying aneuploidy patterns in cancer. *Mol Cell Oncol* 2024; 11: 2369388.
- [16] Song JS, Shin SY, Lee ST, Kim HJ and Kim SH. A cryptic ETV6/ABL1 rearrangement represents a unique fluorescence in situ hybridization signal pattern in a patient with B acute lymphoblastic leukemia. *Ann Lab Med* 2014; 34: 475-477.
- [17] Batanian JR, Bridge JA, Wickert R, Vogler C, Gaudre B and Huang Y. EWS/FLI-1 fusion signal inserted into chromosome 11 in one patient with morphologic features of Ewing sarcoma, but lacking t(11;22). *Cancer Genet Cytogenet* 2002; 133: 72-75.
- [18] Berg HE, Greipp PT, Baughn LB, Falcon CP, Jackson CC and Peterson JF. Detection of a cryptic KMT2A/AFDN gene fusion [ins(6;11)(q27;q23q23)] in a pediatric patient with newly diagnosed acute myeloid leukemia. *Lab Med* 2022; 53: e95-e99.
- [19] Greenfield G, Michail O, Merron B, McGimpsey J, Catherwood M and McMullin MF. Acute promyelocytic leukaemia (APML) with cryptic PML-RARA fusion has a clinical course comparable to classical APML with t(15;17)(q24.1;q21.2) translocation. *Br J Haematol* 2019; 186: 155-157.
- [20] Mohebnasab M, Li P, Hong B, Dunlap J, Traer E, Fan G, Press RD, Moore SR and Xie W. Cytogenetically cryptic acute promyelocytic leukemia: a diagnostic challenge. *Int J Mol Sci* 2023; 24: 13075.

- [21] Blackburn PR, Smadbeck JB, Znoyko I, Webley MR, Pitel BA, Vasmatzis G, Xu X, Greipp PT, Hoppman NL, Ketterling RP, Baughn LB, Lindsey KG, Schandl CA, Wolff DJ and Peterson JF. Cryptic and atypical KMT2A-USP2 and KMT2A-USP8 rearrangements identified by mate pair sequencing in infant and childhood leukemia. *Genes Chromosomes Cancer* 2020; 59: 422-427.
- [22] Loncarevic IF, Roitzheim B, Ritterbach J, Viehmann S, Borkhardt A, Lampert F and Harbott J. Trisomy 21 is a recurrent secondary aberration in childhood acute lymphoblastic leukemia with TEL/AML1 gene fusion. *Genes Chromosomes Cancer* 1999; 24: 272-277.
- [23] Karrman K, Forestier E, Andersen MK, Autio K, Borgström G, Heim S, Heinonen K, Hovland R, Kerndrup G and Johansson B; Nordic Society of Paediatric Haematology and Oncology (NOPHO) and the NOPHO Leukaemia Cytogenetic Study Group (NLCSG). High incidence of the ETV6/RUNX1 fusion gene in paediatric precursor B-cell acute lymphoblastic leukaemias with trisomy 21 as the sole cytogenetic change: a Nordic series of cases diagnosed 1989-2005. *Br J Haematol* 2006; 135: 352-354.
- [24] Schoch C, Kohlmann A, Dugas M, Kern W, Schnittger S and Haferlach T. Impact of trisomy 8 on expression of genes located on chromosome 8 in different AML subgroups. *Genes Chromosomes Cancer* 2006; 45: 1164-1168.
- [25] Lazarevic VL, Rosso A, Juliusson G, Antunovic P, Derolf ÅR, Deneberg S, Möllgård L, Uggla B, Wennström L, Wahlin A, Höglund M, Lehmann S and Johansson B. Incidence and prognostic significance of isolated trisomies in adult acute myeloid leukemia: a population-based study from the Swedish AML registry. *Eur J Haematol* 2017; 98: 493-500.
- [26] Bhatnagar B, Eisfeld AK, Kohlschmidt J, Mrózek K, Nicolet D, Papaioannou D, Walker CJ, Orwick S, Blachly JS, Kolitz JE, Powell BL, Carroll AJ, Stone RM, Byrd JC and Bloomfield CD. Clinical and molecular characterization of patients with acute myeloid leukemia and sole trisomies of chromosomes 4, 8, 11, 13 or 21. *Leukemia* 2020; 34: 358-368.
- [27] Paulsson K and Johansson B. Trisomy 8 as the sole chromosomal aberration in acute myeloid leukemia and myelodysplastic syndromes. *Pathol Biol (Paris)* 2007; 55: 37-48.
- [28] Grimwade D, Hills RK, Moorman AV, Walker H, Chatters S, Goldstone AH, Wheatley K, Harrison CJ and Burnett AK; National Cancer Research Institute Adult Leukaemia Working Group. Refinement of cytogenetic classification in acute myeloid leukemia: determination of prognostic significance of rare recurring chromosomal abnormalities among 5876 younger adult patients treated in the United Kingdom Medical Research Council trials. *Blood* 2010; 116: 354-365.
- [29] Elfving P, Cigudosa JC, Lundgren R, Limon J, Mandahl N, Kristoffersson U, Heim S and Mitelman F. Trisomy 7, trisomy 10, and loss of the Y chromosome in short-term cultures of normal kidney tissue. *Cytogenet Cell Genet* 1990; 53: 123-125.
- [30] Heim S, Mandahl N, Jin Y, Strömblad S, Lindström E, Salford LG and Mitelman F. Trisomy 7 and sex chromosome loss in human brain tissue. *Cytogenet Cell Genet* 1989; 52: 136-138.
- [31] Johansson B, Heim S, Mandahl N, Mertens F and Mitelman F. Trisomy 7 in nonneoplastic cells. *Genes Chromosomes Cancer* 1993; 6: 199-205.
- [32] Broberg K, Toksvig-Larsen S, Lindstrand A and Mertens F. Trisomy 7 accumulates with age in solid tumors and non-neoplastic synovia. *Genes Chromosomes Cancer* 2001; 30: 310-315.
- [33] Karunya B BM, Chacko MP, Parasuraman G, Livingston A, Ramasamy B, Sathishkumar S and Vinod E. Cytogenetic profiling of human articular cartilage chondrocytes, fibronectin adhesion assay derived chondroprogenitors and migratory chondroprogenitors. *Connect Tissue Res* 2026; 67: 13-26.
- [34] Turajlic S, Sottoriva A, Graham T and Swanton C. Resolving genetic heterogeneity in cancer. *Nat Rev Genet* 2019; 20: 404-416.

Tumor-specific trisomy signatures

Supplementary Table 1. 378 pairwise comparisons of trisomy patterns in 28 tumor types

Tumor A	Tumor B	R	R ²	P-value	P-value category
AML	CML	0.94547	0.894	0.0000	<0.001
AML	MDS/MPN	0.97571	0.952	0.0000	<0.001
AML	MPN	0.83357	0.695	0.0000	<0.001
AML	MDS	0.95927	0.920	0.0000	<0.001
AML	ALL	0.28386	0.081	0.1893	n.s.
AML	PCN	-0.07077	0.005	0.7483	n.s.
AML	B-ML	-0.08901	0.008	0.6863	n.s.
AML	T-ML	0.33893	0.115	0.1136	n.s.
AML	HD	-0.00806	0.000	0.9709	n.s.
AML	CA ad kidney	-0.25445	0.065	0.2413	n.s.
AML	Wilms	0.30620	0.094	0.1553	n.s.
AML	CA ad breast	0.12986	0.017	0.5548	n.s.
AML	CA ad ovary	-0.11402	0.013	0.6045	n.s.
AML	SQC H&N	0.15653	0.025	0.4757	n.s.
AML	CA ad colon	0.01953	0.000	0.9295	n.s.
AML	GCT	0.46553	0.217	0.0252	n.s.
AML	Astcyt	-0.06970	0.005	0.7520	n.s.
AML	NB	-0.06991	0.005	0.7513	n.s.
AML	PNET	0.14893	0.022	0.4976	n.s.
AML	Mal mel	0.46150	0.213	0.0266	n.s.
AML	Mal fibr	0.50883	0.259	0.0132	n.s.
AML	Mal myo	0.37331	0.139	0.0793	n.s.
AML	SA syn	0.43396	0.188	0.0386	n.s.
AML	SA chondr	0.11638	0.014	0.5969	n.s.
AML	SA ost	0.13850	0.019	0.5285	n.s.
AML	SA Ewing	0.69834	0.488	0.0002	<0.001
AML	MNG	-0.11041	0.012	0.6160	n.s.
CML	MDS/MPN	0.96688	0.935	0.0000	<0.001
CML	MPN	0.82214	0.676	0.0000	<0.001
CML	MDS	0.94461	0.892	0.0000	<0.001
CML	ALL	0.22952	0.053	0.2921	n.s.
CML	PCN	-0.04849	0.002	0.8261	n.s.
CML	B-ML	-0.00674	0.000	0.9757	n.s.
CML	T-ML	0.40871	0.167	0.0528	n.s.
CML	HD	0.07713	0.006	0.7265	n.s.
CML	CA ad kidney	-0.13747	0.019	0.5316	n.s.
CML	Wilms	0.35860	0.129	0.0929	n.s.
CML	CA ad breast	0.20034	0.040	0.3594	n.s.
CML	CA ad ovary	-0.04992	0.002	0.8210	n.s.
CML	SQC H&N	0.18851	0.036	0.3890	n.s.
CML	CA ad colon	0.02151	0.000	0.9224	n.s.
CML	GCT	0.50701	0.257	0.0135	n.s.
CML	Astcyt	-0.01051	0.000	0.9620	n.s.
CML	NB	-0.03821	0.001	0.8626	n.s.
CML	PNET	0.15266	0.023	0.4868	n.s.
CML	Mal mel	0.45442	0.206	0.0294	n.s.
CML	Mal fibr	0.52814	0.279	0.0096	<0.01

Tumor-specific trisomy signatures

CML	Mal myo	0.41527	0.172	0.0488	n.s.
CML	SA syn	0.48331	0.234	0.0195	n.s.
CML	SA chondr	0.20424	0.042	0.3499	n.s.
CML	SA ost	0.18027	0.032	0.4105	n.s.
CML	SA Ewing	0.75749	0.574	0.0000	<0.001
CML	MNG	0.00707	0.000	0.9744	n.s.
MDS/MPN	MPN	0.86193	0.743	0.0000	<0.001
MDS/MPN	MDS	0.96588	0.933	0.0000	<0.001
MDS/MPN	ALL	0.31121	0.097	0.1483	n.s.
MDS/MPN	PCN	-0.04635	0.002	0.8337	n.s.
MDS/MPN	B-ML	-0.01264	0.000	0.9543	n.s.
MDS/MPN	T-ML	0.42225	0.178	0.0447	n.s.
MDS/MPN	HD	0.10731	0.012	0.6260	n.s.
MDS/MPN	CA ad kidney	-0.18637	0.035	0.3945	n.s.
MDS/MPN	Wilms	0.33407	0.112	0.1192	n.s.
MDS/MPN	CA ad breast	0.20995	0.044	0.3363	n.s.
MDS/MPN	CA ad ovary	-0.04081	0.002	0.8533	n.s.
MDS/MPN	SQC H&N	0.11730	0.014	0.5940	n.s.
MDS/MPN	CA ad colon	0.03136	0.001	0.8871	n.s.
MDS/MPN	GCT	0.54888	0.301	0.0067	<0.01
MDS/MPN	Astcyt	-0.02815	0.001	0.8985	n.s.
MDS/MPN	NB	-0.03061	0.001	0.8897	n.s.
MDS/MPN	PNET	0.18317	0.034	0.4028	n.s.
MDS/MPN	Mal mel	0.45808	0.210	0.0279	n.s.
MDS/MPN	Mal fibr	0.50620	0.256	0.0137	n.s.
MDS/MPN	Mal myo	0.39469	0.156	0.0624	n.s.
MDS/MPN	SA syn	0.50992	0.260	0.0129	n.s.
MDS/MPN	SA chondr	0.18819	0.035	0.3898	n.s.
MDS/MPN	SA ost	0.20733	0.043	0.3425	n.s.
MDS/MPN	SA Ewing	0.76283	0.582	0.0000	<0.001
MDS/MPN	MNG	-0.03797	0.001	0.8635	n.s.
MPN	MDS	0.88277	0.779	0.0000	<0.001
MPN	ALL	0.14581	0.021	0.5068	n.s.
MPN	PCN	0.13946	0.019	0.5257	n.s.
MPN	B-ML	-0.08857	0.008	0.6878	n.s.
MPN	T-ML	0.32379	0.105	0.1318	n.s.
MPN	HD	0.20757	0.043	0.3419	n.s.
MPN	CA ad kidney	-0.23343	0.054	0.2837	n.s.
MPN	Wilms	0.31001	0.096	0.1500	n.s.
MPN	CA ad breast	0.25423	0.065	0.2418	n.s.
MPN	CA ad ovary	-0.05727	0.003	0.7952	n.s.
MPN	SQC H&N	0.25450	0.065	0.2412	n.s.
MPN	CA ad colon	-0.01236	0.000	0.9554	n.s.
MPN	GCT	0.39487	0.156	0.0622	n.s.
MPN	Astcyt	-0.05390	0.003	0.8070	n.s.
MPN	NB	-0.01052	0.000	0.9620	n.s.
MPN	PNET	0.14200	0.020	0.5181	n.s.
MPN	Mal mel	0.37934	0.144	0.0742	n.s.
MPN	Mal fibr	0.39633	0.157	0.0612	n.s.

Tumor-specific trisomy signatures

MPN	Mal myo	0.24819	0.062	0.2535	n.s.
MPN	SA syn	0.48902	0.239	0.0179	n.s.
MPN	SA chondr	0.05387	0.003	0.8071	n.s.
MPN	SA ost	0.08308	0.007	0.7063	n.s.
MPN	SA Ewing	0.65955	0.435	0.0006	<0.001
MPN	MNG	-0.02831	0.001	0.8980	n.s.
MDS	ALL	0.19810	0.039	0.3649	n.s.
MDS	PCN	-0.03621	0.001	0.8697	n.s.
MDS	B-ML	-0.07147	0.005	0.7459	n.s.
MDS	T-ML	0.34996	0.122	0.1016	n.s.
MDS	HD	0.04901	0.002	0.8243	n.s.
MDS	CA ad kidney	-0.23178	0.054	0.2872	n.s.
MDS	Wilms	0.29725	0.088	0.1684	n.s.
MDS	CA ad breast	0.29215	0.085	0.1762	n.s.
MDS	CA ad ovary	-0.02510	0.001	0.9095	n.s.
MDS	SQC H&N	0.19506	0.038	0.3724	n.s.
MDS	CA ad colon	-0.01808	0.000	0.9348	n.s.
MDS	GCT	0.47585	0.226	0.0217	n.s.
MDS	Astcyt	-0.04465	0.002	0.8397	n.s.
MDS	NB	-0.07201	0.005	0.7440	n.s.
MDS	PNET	0.16764	0.028	0.4445	n.s.
MDS	Mal mel	0.45870	0.210	0.0277	n.s.
MDS	Mal fibr	0.51668	0.267	0.0116	n.s.
MDS	Mal myo	0.37337	0.139	0.0793	n.s.
MDS	SA syn	0.44453	0.198	0.0336	n.s.
MDS	SA chondr	0.12703	0.016	0.5635	n.s.
MDS	SA ost	0.14116	0.020	0.5206	n.s.
MDS	SA Ewing	0.73291	0.537	0.0001	<0.001
MDS	MNG	-0.10199	0.010	0.6433	n.s.
ALL	PCN	-0.22963	0.053	0.2919	n.s.
ALL	B-ML	-0.04527	0.002	0.8375	n.s.
ALL	T-ML	0.11859	0.014	0.5899	n.s.
ALL	HD	-0.10077	0.010	0.6473	n.s.
ALL	CA ad kidney	-0.35961	0.129	0.0919	n.s.
ALL	Wilms	-0.07498	0.006	0.7338	n.s.
ALL	CA ad breast	-0.17535	0.031	0.4235	n.s.
ALL	CA ad ovary	-0.44925	0.202	0.0315	n.s.
ALL	SQC H&N	-0.40366	0.163	0.0561	n.s.
ALL	CA ad colon	-0.25604	0.066	0.2383	n.s.
ALL	GCT	0.30309	0.092	0.1598	n.s.
ALL	Astcyt	-0.21086	0.044	0.3341	n.s.
ALL	NB	-0.08134	0.007	0.7122	n.s.
ALL	PNET	-0.05082	0.003	0.8179	n.s.
ALL	Mal mel	-0.13911	0.019	0.5267	n.s.
ALL	Mal fibr	-0.09081	0.008	0.6803	n.s.
ALL	Mal myo	-0.32374	0.105	0.1318	n.s.
ALL	SA syn	-0.06887	0.005	0.7549	n.s.
ALL	SA chondr	-0.19323	0.037	0.3770	n.s.
ALL	SA ost	-0.11562	0.013	0.5993	n.s.

Tumor-specific trisomy signatures

ALL	SA Ewing	0.06947	0.005	0.7528	n.s.
ALL	MNG	-0.35123	0.123	0.1003	n.s.
PCN	B-ML	0.01384	0.000	0.9500	n.s.
PCN	T-ML	0.43263	0.187	0.0392	n.s.
PCN	HD	0.19690	0.039	0.3679	n.s.
PCN	CA ad kidney	0.05917	0.004	0.7886	n.s.
PCN	Wilms	-0.16148	0.026	0.4617	n.s.
PCN	CA ad breast	0.02887	0.001	0.8960	n.s.
PCN	CA ad ovary	-0.01132	0.000	0.9591	n.s.
PCN	SQC H&N	0.02471	0.001	0.9109	n.s.
PCN	CA ad colon	-0.01944	0.000	0.9299	n.s.
PCN	GCT	-0.02381	0.001	0.9141	n.s.
PCN	Astcyt	0.30806	0.095	0.1527	n.s.
PCN	NB	-0.04480	0.002	0.8392	n.s.
PCN	PNET	0.29862	0.089	0.1663	n.s.
PCN	Mal mel	0.06018	0.004	0.7850	n.s.
PCN	Mal fibr	0.17337	0.030	0.4289	n.s.
PCN	Mal myo	-0.07430	0.006	0.7362	n.s.
PCN	SA syn	0.02020	0.000	0.9271	n.s.
PCN	SA chondr	0.25768	0.066	0.2352	n.s.
PCN	SA ost	0.08706	0.008	0.6928	n.s.
PCN	SA Ewing	-0.14383	0.021	0.5126	n.s.
PCN	MNG	0.25950	0.067	0.2318	n.s.
B-ML	T-ML	0.34343	0.118	0.1086	n.s.
B-ML	HD	0.43822	0.192	0.0365	n.s.
B-ML	CA ad kidney	0.46346	0.215	0.0259	n.s.
B-ML	Wilms	0.64862	0.421	0.0008	<0.001
B-ML	CA ad breast	0.29219	0.085	0.1761	n.s.
B-ML	CA ad ovary	0.64680	0.418	0.0009	<0.001
B-ML	SQC H&N	0.15234	0.023	0.4877	n.s.
B-ML	CA ad colon	0.35585	0.127	0.0956	n.s.
B-ML	GCT	0.57548	0.331	0.0041	<0.01
B-ML	Astcyt	0.38769	0.150	0.0676	n.s.
B-ML	NB	0.33261	0.111	0.1210	n.s.
B-ML	PNET	0.35822	0.128	0.0933	n.s.
B-ML	Mal mel	0.15482	0.024	0.4806	n.s.
B-ML	Mal fibr	0.19780	0.039	0.3656	n.s.
B-ML	Mal myo	0.31190	0.097	0.1474	n.s.
B-ML	SA syn	0.51712	0.267	0.0115	n.s.
B-ML	SA chondr	0.45499	0.207	0.0292	n.s.
B-ML	SA ost	0.42320	0.179	0.0442	n.s.
B-ML	SA Ewing	0.35311	0.125	0.0984	n.s.
B-ML	MNG	0.39220	0.154	0.0642	n.s.
T-ML	HD	0.35767	0.128	0.0938	n.s.
T-ML	CA ad kidney	0.26583	0.071	0.2202	n.s.
T-ML	Wilms	0.12404	0.015	0.5728	n.s.
T-ML	CA ad breast	0.35468	0.126	0.0968	n.s.
T-ML	CA ad ovary	0.23046	0.053	0.2901	n.s.
T-ML	SQC H&N	0.21480	0.046	0.3250	n.s.

Tumor-specific trisomy signatures

T-ML	CA ad colon	0.23141	0.054	0.2881	n.s.
T-ML	GCT	0.55622	0.309	0.0058	<0.01
T-ML	Astcyt	0.42648	0.182	0.0424	n.s.
T-ML	NB	0.02915	0.001	0.8949	n.s.
T-ML	PNET	0.39214	0.154	0.0642	n.s.
T-ML	Mal mel	0.42456	0.180	0.0435	n.s.
T-ML	Mal fibr	0.41500	0.172	0.0489	n.s.
T-ML	Mal myo	0.24213	0.059	0.2657	n.s.
T-ML	SA syn	0.22842	0.052	0.2945	n.s.
T-ML	SA chondr	0.45230	0.205	0.0302	n.s.
T-ML	SA ost	0.45779	0.210	0.0280	n.s.
T-ML	SA Ewing	0.35039	0.123	0.1012	n.s.
T-ML	MNG	0.28066	0.079	0.1946	n.s.
HD	CA ad kidney	0.17792	0.032	0.4167	n.s.
HD	Wilms	0.23416	0.055	0.2822	n.s.
HD	CA ad breast	0.07967	0.006	0.7178	n.s.
HD	CA ad ovary	0.46111	0.213	0.0268	n.s.
HD	SQC H&N	0.12689	0.016	0.5640	n.s.
HD	CA ad colon	-0.09552	0.009	0.6646	n.s.
HD	GCT	0.24844	0.062	0.2530	n.s.
HD	Astcyt	0.00675	0.000	0.9756	n.s.
HD	NB	0.06283	0.004	0.7758	n.s.
HD	PNET	0.03670	0.001	0.8680	n.s.
HD	Mal mel	0.00241	0.000	0.9913	n.s.
HD	Mal fibr	0.18968	0.036	0.3860	n.s.
HD	Mal myo	0.42739	0.183	0.0419	n.s.
HD	SA syn	0.50327	0.253	0.0144	n.s.
HD	SA chondr	0.43444	0.189	0.0383	n.s.
HD	SA ost	0.32284	0.104	0.1330	n.s.
HD	SA Ewing	0.40903	0.167	0.0526	n.s.
HD	MNG	0.40947	0.168	0.0523	n.s.
CA ad kidney	Wilms	0.27836	0.077	0.1984	n.s.
CA ad kidney	CA ad breast	0.51105	0.261	0.0127	n.s.
CA ad kidney	CA ad ovary	0.49447	0.244	0.0165	n.s.
CA ad kidney	SQC H&N	0.53918	0.291	0.0079	<0.01
CA ad kidney	CA ad colon	0.68591	0.470	0.0003	<0.001
CA ad kidney	GCT	0.36750	0.135	0.0845	n.s.
CA ad kidney	Astcyt	0.78870	0.622	0.0000	<0.001
CA ad kidney	NB	0.60489	0.366	0.0022	<0.01
CA ad kidney	PNET	0.46201	0.213	0.0265	n.s.
CA ad kidney	Mal mel	0.47821	0.229	0.0210	n.s.
CA ad kidney	Mal fibr	0.32310	0.104	0.1326	n.s.
CA ad kidney	Mal myo	0.39276	0.154	0.0638	n.s.
CA ad kidney	SA syn	0.32565	0.106	0.1294	n.s.
CA ad kidney	SA chondr	0.65945	0.435	0.0006	<0.001
CA ad kidney	SA ost	0.65448	0.428	0.0007	<0.001
CA ad kidney	SA Ewing	0.10771	0.012	0.6247	n.s.
CA ad kidney	MNG	0.52963	0.281	0.0093	<0.01
Wilms	CA ad breast	0.25730	0.066	0.2359	n.s.

Tumor-specific trisomy signatures

Wilms	CA ad ovary	0.52730	0.278	0.0097	<0.01
Wilms	SQC H&N	0.31803	0.101	0.1392	n.s.
Wilms	CA ad colon	0.34384	0.118	0.1082	n.s.
Wilms	GCT	0.47742	0.228	0.0212	n.s.
Wilms	Astcyt	0.23078	0.053	0.2894	n.s.
Wilms	NB	0.53449	0.286	0.0086	<0.01
Wilms	PNET	0.48291	0.233	0.0196	n.s.
Wilms	Mal mel	0.39863	0.159	0.0596	n.s.
Wilms	Mal fibr	0.27456	0.075	0.2049	n.s.
Wilms	Mal myo	0.46610	0.217	0.0250	n.s.
Wilms	SA syn	0.64943	0.422	0.0008	<0.001
Wilms	SA chondr	0.26730	0.071	0.2176	n.s.
Wilms	SA ost	0.37333	0.139	0.0793	n.s.
Wilms	SA Ewing	0.62477	0.390	0.0014	<0.01
Wilms	MNG	0.32998	0.109	0.1241	n.s.
CA ad breast	CA ad ovary	0.41497	0.172	0.0490	n.s.
CA ad breast	SQC H&N	0.55195	0.305	0.0063	<0.01
CA ad breast	CA ad colon	0.54017	0.292	0.0078	<0.01
CA ad breast	GCT	0.34474	0.119	0.1072	n.s.
CA ad breast	Astcyt	0.64337	0.414	0.0009	<0.001
CA ad breast	NB	0.32299	0.104	0.1328	n.s.
CA ad breast	PNET	0.64152	0.412	0.0010	<0.001
CA ad breast	Mal mel	0.61795	0.382	0.0017	<0.01
CA ad breast	Mal fibr	0.44981	0.202	0.0313	n.s.
CA ad breast	Mal myo	0.34619	0.120	0.1056	n.s.
CA ad breast	SA syn	0.16277	0.026	0.4580	n.s.
CA ad breast	SA chondr	0.49203	0.242	0.0171	n.s.
CA ad breast	SA ost	0.53344	0.285	0.0088	<0.01
CA ad breast	SA Ewing	0.35207	0.124	0.0994	n.s.
CA ad breast	MNG	0.20676	0.043	0.3439	n.s.
CA ad ovary	SQC H&N	0.29203	0.085	0.1763	n.s.
CA ad ovary	CA ad colon	0.34194	0.117	0.1103	n.s.
CA ad ovary	GCT	0.38218	0.146	0.0719	n.s.
CA ad ovary	Astcyt	0.38845	0.151	0.0670	n.s.
CA ad ovary	NB	0.25449	0.065	0.2413	n.s.
CA ad ovary	PNET	0.31278	0.098	0.1462	n.s.
CA ad ovary	Mal mel	0.28149	0.079	0.1932	n.s.
CA ad ovary	Mal fibr	0.20289	0.041	0.3532	n.s.
CA ad ovary	Mal myo	0.55385	0.307	0.0061	<0.01
CA ad ovary	SA syn	0.47707	0.228	0.0213	n.s.
CA ad ovary	SA chondr	0.47591	0.226	0.0217	n.s.
CA ad ovary	SA ost	0.59042	0.349	0.0030	<0.01
CA ad ovary	SA Ewing	0.37616	0.141	0.0769	n.s.
CA ad ovary	MNG	0.49441	0.244	0.0165	n.s.
SQC H&N	CA ad colon	0.52827	0.279	0.0096	<0.01
SQC H&N	GCT	0.21406	0.046	0.3267	n.s.
SQC H&N	Astcyt	0.56459	0.319	0.0050	<0.01
SQC H&N	NB	0.41088	0.169	0.0515	n.s.
SQC H&N	PNET	0.42214	0.178	0.0448	n.s.

Tumor-specific trisomy signatures

SQC H&N	Mal mel	0.69944	0.489	0.0002	<0.001
SQC H&N	Mal fibr	0.62525	0.391	0.0014	<0.01
SQC H&N	Mal myo	0.50954	0.260	0.0130	n.s.
SQC H&N	SA syn	0.23125	0.053	0.2884	n.s.
SQC H&N	SA chondr	0.45499	0.207	0.0291	n.s.
SQC H&N	SA ost	0.48010	0.230	0.0204	n.s.
SQC H&N	SA Ewing	0.31053	0.096	0.1493	n.s.
SQC H&N	MNG	0.32715	0.107	0.1276	n.s.
CA ad colon	GCT	0.39060	0.153	0.0654	n.s.
CA ad colon	Astcyt	0.74372	0.553	0.0000	<0.001
CA ad colon	NB	0.34764	0.121	0.1041	n.s.
CA ad colon	PNET	0.51421	0.264	0.0121	n.s.
CA ad colon	Mal mel	0.66428	0.441	0.0005	<0.001
CA ad colon	Mal fibr	0.38075	0.145	0.0731	n.s.
CA ad colon	Mal myo	0.46074	0.212	0.0269	n.s.
CA ad colon	SA syn	0.28803	0.083	0.1826	n.s.
CA ad colon	SA chondr	0.53585	0.287	0.0084	<0.01
CA ad colon	SA ost	0.66194	0.438	0.0006	<0.001
CA ad colon	SA Ewing	0.15885	0.025	0.4691	n.s.
CA ad colon	MNG	0.29330	0.086	0.1744	n.s.
GCT	Astcyt	0.43224	0.187	0.0394	n.s.
GCT	NB	0.25723	0.066	0.2361	n.s.
GCT	PNET	0.27888	0.078	0.1975	n.s.
GCT	Mal mel	0.54773	0.300	0.0068	<0.01
GCT	Mal fibr	0.37951	0.144	0.0741	n.s.
GCT	Mal myo	0.36418	0.133	0.0876	n.s.
GCT	SA syn	0.70025	0.490	0.0002	<0.001
GCT	SA chondr	0.53622	0.288	0.0084	<0.01
GCT	SA ost	0.59493	0.354	0.0027	<0.01
GCT	SA Ewing	0.56624	0.321	0.0049	<0.01
GCT	MNG	0.18301	0.033	0.4033	n.s.
Astcyt	NB	0.45940	0.211	0.0274	n.s.
Astcyt	PNET	0.73759	0.544	0.0001	<0.001
Astcyt	Mal mel	0.68803	0.473	0.0003	<0.001
Astcyt	Mal fibr	0.42711	0.182	0.0421	n.s.
Astcyt	Mal myo	0.37694	0.142	0.0762	n.s.
Astcyt	SA syn	0.29293	0.086	0.1749	n.s.
Astcyt	SA chondr	0.71563	0.512	0.0001	<0.001
Astcyt	SA ost	0.78315	0.613	0.0000	<0.001
Astcyt	SA Ewing	0.11295	0.013	0.6079	n.s.
Astcyt	MNG	0.28707	0.082	0.1841	n.s.
NB	PNET	0.62685	0.393	0.0014	<0.01
NB	Mal mel	0.37529	0.141	0.0776	n.s.
NB	Mal fibr	0.27267	0.074	0.2081	n.s.
NB	Mal myo	0.25530	0.065	0.2397	n.s.
NB	SA syn	0.31102	0.097	0.1486	n.s.
NB	SA chondr	0.33381	0.111	0.1196	n.s.
NB	SA ost	0.32822	0.108	0.1263	n.s.
NB	SA Ewing	0.17276	0.030	0.4305	n.s.

Tumor-specific trisomy signatures

NB	MNG	0.42399	0.180	0.0438	n.s.
PNET	Mal mel	0.62640	0.392	0.0014	<0.01
PNET	Mal fibr	0.50810	0.258	0.0133	n.s.
PNET	Mal myo	0.40513	0.164	0.0551	n.s.
PNET	SA syn	0.23170	0.054	0.2874	n.s.
PNET	SA chondr	0.53387	0.285	0.0087	<0.01
PNET	SA ost	0.60351	0.364	0.0023	<0.01
PNET	SA Ewing	0.28939	0.084	0.1805	n.s.
PNET	MNG	0.27628	0.076	0.2019	n.s.
Mal mel	Mal fibr	0.65918	0.435	0.0006	<0.001
Mal mel	Mal myo	0.62053	0.385	0.0016	<0.01
Mal mel	SA syn	0.44883	0.201	0.0317	n.s.
Mal mel	SA chondr	0.70166	0.492	0.0002	<0.001
Mal mel	SA ost	0.76785	0.590	0.0000	<0.001
Mal mel	SA Ewing	0.49781	0.248	0.0156	n.s.
Mal mel	MNG	0.27252	0.074	0.2084	n.s.
Mal fibr	Mal myo	0.70812	0.501	0.0002	<0.001
Mal fibr	SA syn	0.37646	0.142	0.0766	n.s.
Mal fibr	SA chondr	0.59185	0.350	0.0029	<0.01
Mal fibr	SA ost	0.51925	0.270	0.0111	n.s.
Mal fibr	SA Ewing	0.64517	0.416	0.0009	<0.001
Mal fibr	MNG	0.43673	0.191	0.0372	n.s.
Mal myo	SA syn	0.60193	0.362	0.0024	<0.01
Mal myo	SA chondr	0.67763	0.459	0.0004	<0.001
Mal myo	SA ost	0.70608	0.499	0.0002	<0.001
Mal myo	SA Ewing	0.65962	0.435	0.0006	<0.001
Mal myo	MNG	0.37241	0.139	0.0801	n.s.
SA syn	SA chondr	0.53755	0.289	0.0082	<0.01
SA syn	SA ost	0.56206	0.316	0.0052	<0.01
SA syn	SA Ewing	0.75999	0.578	0.0000	<0.001
SA syn	MNG	0.28252	0.080	0.1915	n.s.
SA chondr	SA ost	0.81712	0.668	0.0000	<0.001
SA chondr	SA Ewing	0.39880	0.159	0.0594	n.s.
SA chondr	MNG	0.56241	0.316	0.0052	<0.01
SA ost	SA Ewing	0.44532	0.198	0.0332	n.s.
SA ost	MNG	0.33450	0.112	0.1187	n.s.
SA Ewing	MNG	0.27836	0.077	0.1984	n.s.