Reports of NAS RA
ՀՀ ԳԱԱ Զեկույցներ
NATIONAL ACADEMY OF SCIENCES OF THE REPUBLIC OF ARMENIA
ՀԱՅԱՍՏԱՆԻ ՀԱՆՐԱՊԵՏՈՒԹՅԱՆ ԳԻՏՈՒԹՅՈՒՆՆԵՐԻ ԱԶԳԱՅԻՆ ԱԿԱԴԵՄԻԱ

2026 **Volume 126** **Issue 2** **Article No. 4**

Category: Biology
Type of Paper: Review Article
Received: April 15, 2026, **Revised:** June 25, 2026, **Accepted:** June 25, 2026
Published: July 6, 2026

DOI: 10.54503/0321-1339-2026.126.2-4

Cancer type specific copy number variants are identical over species borders

Thomas Liehr[1,*]

[1]Jena University Hospital, Friedrich Schiller University, Institute of Human Genetics, Am Klinikum 1, D-07747 Jena, Germany

[*]Correspondence: Thomas.Liehr@med.uni-jena.de

Abstract
Over the past two decades, we have studied dozens of murine and a few rat-derived tumor cell lines. While these cell lines are commonly used as models for human cancer, their cytogenetic and genetic properties are not fully understood. Therefore, we characterized approximately 40 rodent cancer cell lines using molecular cytogenetics, including multicolor fluorescence *in situ* hybridization and array-comparative genomic hybridization. We then *in silico* translated the observed acquired gains and losses of chromosomal regions into the human genome. During these studies, we found that rodent cancer cell lines carried chromosomal imbalances similar to those observed in human cancer cells. In this review, we discuss the cell lines studied, the percentage of gains and losses that agreed with the corresponding human cancer type, and the human cancer subtypes for which each cell line can serve as a suitable model. Additionally, we discuss the fact that polyploidy can only be reliably accessed by cytogenetics and that male cell lines tend to lose their Y chromosome. Overall, the reviewed findings highlight the need for an in-depth cytogenomic characterization before using a cell line in research. Furthermore, the data support the idea that copy number changes are more important than point mutations, at least in advanced tumors, as they are similar across species boundaries.

Keywords: murine tumor, rat tumor, cell lines, multicolor-fluorescence *in situ* hybridization – mFISH, murine multicolor banding, array-based comparative genomic hybridization – aCGH

Introduction

Cancer patients are identified at different ages within the human population; however, the risk of cancer is known to increase with age [1]. Some cancer types are associated with gender [2-3], while others are associated with the types of food, spices [4] or drugs used in daily life [5]. In general, the risk of cancer is increased by radiation or mutagenic influences [6]. Early diagnosis is imperative for adequate treatment of patients [7]. Subgrouping specific tumors based on genetic, immunohistochemical, and/or other factors impacts treatment, prognosis, and follow-up strategies [8].

Both basic and applied cancer research largely depends on in vitro models [9]. Although many model systems are available nowadays, such as tumor spheroids, genetically engineered cells (knock-in or knock-out models, including corresponding murine strains), 3D cell culture, microfluidics and all Crispr/Cas modeling variants, tumor cell lines remain popular as established tools [9]. Over the last 20

years, we have performed cytogenomic characterizations of approximately 30 human cancer cell lines [10]. We also characterized 36 murine [11-33] and two rat cancer cell lines [34-35]. Specifically, the latter were not characterized at the chromosomal level beforehand. This is because murine (and rat) chromosomes are difficult to distinguish using cytogenetic banding approaches [27]. Since 1996, multicolor fluorescence in situ hybridization with all 20 murine whole chromosome paints in different color combinations (M-FISH) became available [36]. However, it was only after establishing murine multicolor banding that besides inter- intrachromosomal aberrations became accessible as well [27]. Interestingly, most of the studied cell lines were established between 40 and over 100 years ago and some of them are now standard reference systems. Nevertheless, their chromosomal constitutions remained unknown until recently [11-35]. Thus, our results were partly surprising and called into question the justification for using some cell lines as corresponding model systems based only on histological parameters of a primary lesion [11-35].

In previous papers [11-35], we focused on whether cytogenomic characterization could help define the cancer subtype for which an individual cell line could serve as a model (see Table 1). Here, we discuss what can be learned from these studies in terms of a more general view of the results obtained.

Literature Review and Analysis

We focused on a cumulative analysis of data obtained from 37 murine and two rat-derived tumor cell lines (see Tables 1 and 2). The inclusion criteria for the cell lines in Table 1 were that they were studied using M-FISH, murine multicolor banding, and array comparative genomic hybridization (aCGH), and imbalances were translated into the human genome according to [13]. Table 2 only includes male-derived rodent cancer cell lines that were characterized by M-FISH and examined for the presence or absence of the Y chromosome.

Accordingly, we can deduce the following:

1. Of the studied murine cancer cell lines, only 14 out of 33 (42%) have not formed subclones, as have all two (100%) of the rat-derived ones. The remaining 19 murine cell lines had between one and five subclones besides a main clone. However, it should be noted that the almost all cell lines exhibited single-cell aberrations to some extent.
2. Nearly 80% of the studied cell lines are hyperdiploid (Figure 1A). Approximately 70% of the cell lines are triploid to tetraploid, while about 9% have a chromosome complement ranging from 4.5n to 6n (Figure 1B).
3. As shown in Table 1, the observed copy number alterations in murine cell lines were consistent with those in the corresponding human cancer counterparts between 40% and 91%. On average, there was concordance for 64% of the detected gains and losses. The range was similar for the two rat cell lines, i.e., between 38% and 55%.
4. According to Table 2, Y chromosome loss was observable in seven of eleven cell lines (63%), gain was present in two of eleven cell lines (18%), and gain and loss in a mosaic pattern was observed in one of eleven cell lines (9%). Only one remaining triploid cell line had correctly a Y chromosome alongside two X chromosomes. The Y chromosome remained stable in the only male rat cell line studied. Interestingly, Y-chromosome loss led to complete erasure of this gonosome in six out of seven cell lines; these cell lines retained a “Turner syndrome-like” gonosomal constitution.

Discussion

We live in a time when the end of cytogenetics is loudly proclaimed once again — it is stated that it will all be replaced by a single approach yet to be introduced that can identify all possible genetic aberrations in a single experiment [37]. This review clearly shows that this idea is misleading.

Table 1. Summary of studied cancer cell lines derived from two different rodent species, for which a chromosomal gains and losses have been in silico translated to human genome.

Cell line	Ploidy/ subclones	Cancer type	Imbalances in agreement with human cancer type [%]	Reference
mouse				
A-20	2n / yes	Burkitt's lymphoma	63	11
AB1	3.5n / no	malignant epithelioid mesothelioma	88	12
AB22	4n / no	malignant epithelioid mesothelioma	90	12
AC29	3n / yes	malignant epithelioid mesothelioma	75	12
AE17	2n / yes	malignant sarcomatoid mesothelioma	75	13
AML12	4n / yes	advanced hepatocellular CA	68	14
B16-F0	4n / yes	malignant skin melanoma	67	15
B16-F1-ATCC	3.5n / yes	malignant skin melanoma	67	16
B16-4A5	3.5n / yes	malignant skin melanoma	70	16
C-127I	5n / yes	breast CA (late stage)	60	17
C57/B1	2n / yes	malignant skin melanoma	50	15
CMT-93	3n / yes	advanced type of metastatic colorectal CA	79	18
CT26	3n / yes	advanced type of metastatic colorectal CA	79	18
EMT6/P	3n / yes	breast CA (early stage)	42	17
Hepa 1-6	3n / no	advanced hepatocellular CA	66-78	19
I-10	2n / no	testicular germ cell tumor (early)	63	20
JC	4n / yes	breast CA luminal B, basal-like	57	21
KLN 205	4n / yes	lung squamous cell CA	43	22
LA-4	4.5n / no	lung squamous cell CA	58	23
MH-22A	3n / no	hepatoblastoma or early hepatocellular carcinoma	50-55 53	19
MLTC-1	6n / no	Leydig cell tumor (late)	40	24
MMT 060562	2n / yes	breast CA (early stage)	43	25
Neuro-2a	4n / no	neuroblastoma (late)	75	26
Neuro-2a TR-a	4n / no	neuroblastoma (late)	75	26
Neuro-2a TR-ß	4n / no	neuroblastoma (late)	75	26
NIH3T3	3.75n / no	ectodermal tumor	66	27
S91 clone M3	3n / no	malignant skin melanoma	61	16
S-180	4n / yes	mesenchymal chondrosarcoma	70	28
SCA-9	3.5n / no	mucoepidermoid or myoepithelioma salivary gland tumor	40	29
SEWA	2n / no	osteosarcoma (late)	91	30
TA3 Hauschka	3n / yes	breast CA (early stage)	40	17
TSC2ang1	3.5n / yes	cutaneous sarcoma	55	31
WR21	2n / yes	myoepithelioma salivary gland tumor	69	29
rat				
H9c2	3n / no	sarcoma	55	34
RAT-1	2n / no	epithelioid sarcoma or liposarcoma	43 38	35

Table 2. Y chromosome presence in the studied male rodent cancer cell lines.

Cell line	Ploidy/ subclones	Cancer type	Y-chromosome status	Reference
mouse				
AML12	4n / yes	advanced hepatocellular CA	XX,-Y,-Y	14
CMT-93	3n / yes	advanced type of metastatic colorectal CA	XXY	18
Col-GFP HSC	4n / yes	hepatic stellate cells	XXY,-Y/XXYY/ XXYY,+Y	32
I-10	2n / no	testicular germ cell tumor (early)	X,-Y	20
KLN 205	4n / yes	lung squamous cell CA	XXYY,+Y,+Y	22
NIH3T3	3.75n / no	ectodermal tumor	XXY,-Y	27
MLTC-1	6n / no	Leydig cell tumor (late)	XX,-X,-Y,-Y,-Y	24
Panc02	4.5n / no	pancreatic cancer	XX,-Y,-Y	33
S-180	4n / yes	mesenchymal chondrosarcoma	XX,-Y,-Y	28
SEWA	2n / no	osteosarcoma (late)	X,-Y	30
WR21	2n / yes	myoepithelioma salivary gland tumor	XY,+Y	29
rat				
RAT-1	2n / no	epithelioid sarcoma or liposarcoma	XY	35

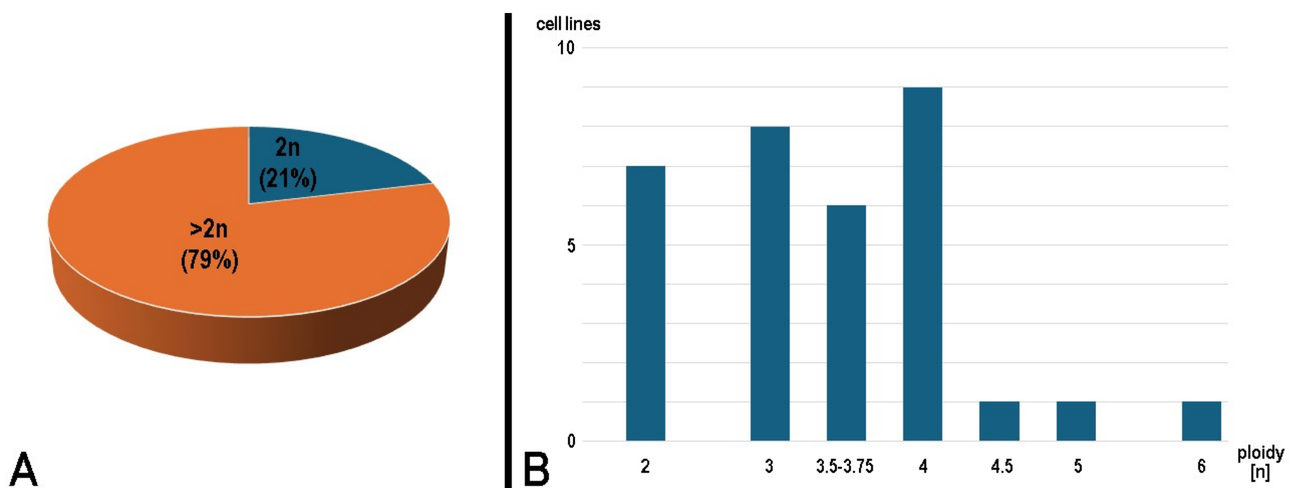

Figure 1. Graphic summary on the murine cell lines reviewed here. **A)** 21% of the cell lines were diploid, while 79% were hyperdiploid. **B)** Ploidylevels of the studied murine cell lines are detailed.

The first finding mentioned in this review is that 58% of murine cell lines have one to five additional subclones besides one main clone. This cannot be detected or characterized in detail by any approach that is not single-cell directed. Hints for mosaic situations may be present in next-generation sequencing (NGS) or aCGH. However, the nature of the underlying chromosomal aberrations cannot be determined this way, nor can mosaics. This is due to technical limitations, such as the blindness of NGS and aCGH toward heterochromatic regions and their goal to analyze than ten thousands cells at a time; thus the same is true for optical genome mapping [38-40]. Our group recently discovered amplification of satellite DNAs in advanced cancers [41], which has been found in at least one murine cell line [26], to and cannot be found by any other approach than FISH yet. The high rates of chromosomal instability detected in our studies, a typical feature of cancer, cannot be accessed by high-throughput approaches either [42].

As shown here, 80% of murine cancer cell lines are hyperdiploid with 3n to 6n. This information can only be obtained through (molecular) cytogenetics. Knowing the ploidy of a cancer cell line model

is essential. When studying the effects of knocking out a tumor suppressor gene, for example, the necessity of this knowledge is clear. Furthermore, a highly polyploid cell line can only model a late-stage tumor, which is important to know when studying a cell line as a model for tumor treatment, for example.

For the cancer cell lines derived from the Rodentia family that were studied here, 64% of the detected gains and losses were in concordance with those of their human cancer counterparts, ranging from 40% to 91%. As expected, these results demonstrate that mice and rats are not human [43]. However, it also suggests that tumorigenesis in the same tissue of different species involves similar environmental factors that favor specific imbalances. During tumor progression, chromosomal imbalances at least trigger tumor evolution. These environmental conditions may be the same in all mammals. Thus, this finding reinforces the idea that murine cancer cell lines are suitable models for human cancer [43]. At the same time, it demonstrates that cancer development may have more similarities across mammals than previously suggested [44].

Finally, this study demonstrated that cytogenomic analysis of a cell line's karyotype is particularly important in male murine cell lines. More than half of these cell lines lost their sex-determining Y chromosome, and in approximately 90% of the affected lines, the loss is complete. Therefore, these cell lines are better models for individuals with Turner syndrome [45] than for males. This must be considered when choosing cell lines based on their original sex.

Conclusions

Before using a murine cancer cell line in a larger research project, conduct a molecular cytogenetic study to confirm its ploidy, characterize its chromosomal rearrangements, and determine its gonosomal constitution. This will help avoid using an unsuitable model system.

After studying ~40 murine and rat-derived cancer cell lines, it was found that they carry similar chromosomal imbalances to those observed in human cancer cells. This supports the hypothesis that gains and losses, together with chromosomal instability, are driving factors in cancer progression [44]. Their impact on cancer initiation remains to be determined.

A whole genomic view of tumor cell lines is imperative and cannot be replaced by high-throughput approaches. Short-read NGS cannot reliably access translocations, complex rearrangements, or mosaic situations. Although long-range sequencing and optical genomic mapping can detect a certain percentage of gene fusions or fissions, the nature of chromosomal rearrangements is difficult to determine without a chromosomal view. Both approaches are blind to heterochromatic regions, and their role may be underestimated [41]. Overall, no approach should be dismissed simply because it is “old,” such as banding cytogenetics, when it provides a whole genomic view combined with single-cell analysis.

References

1. L Montégut, C López-Otín, G Kroemer. Aging and cancer. Mol Cancer 2024, 23:106. https://doi.org/10.1186/s12943-024-02020-z.
2. V Valentini, A Bucalo, G Conti, L Celli, V Porzio, C Capalbo, V Silvestri, L Ottini. Gender-specific genetic predisposition to breast cancer: *BRCA* genes and beyond. Cancers (Basel) 2024, 16:579. https://doi.org/10.3390/cancers16030579.
3. MM Muermann, RJ Wassersug. Prostate cancer from a sex and gender perspective: A review. Sex Med Rev 2022, 10:142-154. https://doi.org/10.1016/j.sxmr.2021.03.001.
4. M Chandra. 2025. Top 10 foods that can cause cancer – and what’s just a myth. https://everhope.care/blogs/foods-that-cause-cancer (accessed on 14/04/2026).
5. JA Kwon, N Kim, JK Oh, B Park, YJ Choi, Y Choi, B Kim B. Longitudinal assessment of smoking-related knowledge, attitude, and practice for cancer prevention: an analysis of data from the Korean

National Cancer Prevention Awareness and Practice Survey. BMC Public Health 2025, 25:1134. https://doi.org/10.1186/s12889-025-22258-6.

6. S Kumari, S Sharma, D Advani, A Khosla, P Kumar, RK Ambasta. Unboxing the molecular modalities of mutagens in cancer. Environ Sci Pollut Res Int 2022, 29:62111-62159. https://doi.org/10.1007/s11356-021-16726-w.
7. V Butnari, P Scantlebury, T Green, D Pattni, A Mansuri, W Chinnery, N Rajendran, M Brannan, M Cole, S Banerjee. The crucial role of early diagnosis for patients and the nation, understanding the costs of late-stage cancer diagnosis from a large district general hospital in England. Cost Eff Resour Alloc 2025, 23:60. https://doi.org/10.1186/s12962-025-00657-1.
8. S Ray, NK Chaturvedi, KK Bhakat, A Rizzino, S Mahapatra. Subgroup-specific diagnostic, prognostic, and predictive markers influencing pediatric medulloblastoma treatment. Diagnostics (Basel) 2021, 12:61. https://doi.org/10.3390/diagnostics12010061.
9. ME Katt, AL Placone, AD Wong, ZS Xu, PC Searson. *In vitro* tumor models: Advantages, disadvantages, variables, and selecting the right platform. Front Bioeng Biotechnol 2016, 4:12. https://doi.org/10.3389/fbioe.2016.00012.
10. T Liehr. 2026. Detailed karyotypes of tumor cell lines. https://cs-tl.de/DB/TC/CLs/0-Start.html, (accessed on 14/04/2026).
11. K Guja, T Liehr, M Rincic, N Kosyakova, SS Hussein Azawi. Molecular cytogenetic characterization identified the murine B-Cell lymphoma cell line A-20 as a model for sporadic Burkitt's lymphoma. J Histochem Cytochem 2017, 65: 669-677. https://doi.org/10.1369/0022155417731319.
12. E Wahlbul, T Liehr, M Rincic, S Azawi. Cytogenomic characterization of three murine malignant mesothelioma tumor cell lines. Mol Cytogenet 2020, 13:43. https://doi.org/10.1186/s13039-020-00511-4.
13. E Kubicova, V Trifonov, F Borovecki, T Liehr, M Rincic, N Kosyakova, SS Hussein. First molecular cytogenetic characterization of murine malignant mesothelioma cell line AE17 and in silico translation to the human genome. Curr Bioinform 2017, 12:11-18. https://doi.org/10.2174/1574893611666160606164459.
14. DY Asar, S Kankel, DT Keller, KS Hardt, SK Schröder-Lange, EM Buhl, T Liehr, R Weiskirchen. Comprehensive cytogenetic and genomic profiling of the murine AML12 (Alpha Mouse Liver 12) hepatocyte cell line. Cells 2026, 15:390. https://doi.org/10.3390/cells15050390.
15. M Manferrari, M Rincic, T Liehr, S Azawi. Cytogenomics of murine melanoma cell lines C57/B1 and B16-F0. Mol Exp Biol Med 2020, 3:39-44.
16. K Piaszinski, M Rincic, T Liehr, S Azawi. Molecular cytogenetic characterization of the murine melanoma cell lines S91 clone M3 and B16-F1 with variant B16-4A5. Cytogenet Genome Res 2021, 161:82-92. https://doi.org/10.1159/000513174.
17. A Azawi, T Liehr, M Rincic, M Manferrari. Molecular cytogenomic characterization of the murine breast cancer cell lines C-127I, EMT6/P and TA3 Hauschka. Int J Mol Sci 2020, 21:4716. https://doi.org/10.3390/ijms21134716.
18. H Rhode, T Liehr, N Kosyakova, M Rinčic, SSH Azawi. Molecular cytogenetic characterization of two murine colorectal cancer cell lines. OBM Genetics 2018, 2:037. https://doi.org/10.21926/obm.genet.1803037.
19. S Azawi, K Piaszinski, M Balachandran, T Liehr, M Rincic. Molecular cytogenomic characterization of two murine liver cancer cell lines: MH-22A and Hepa 1-6. J Genet Genomes 2021, 5:1.
20. S Azawi, L-M Barf, T Liehr, S Kankel, M Rincic. First cytogenomic characterization of murine testicular tumor cell line I-10. OBM Genetics 2022, 6:159. https://doi.org/10.21926/obm.genet.2202159.

21. S Azawi, M Rincic, T Liehr. Cytogenomic characteristics of murine breast cancer cell line JC. Mol Cytogenet 2021, 14:7. https://doi.org/10.1186/s13039-020-00524-z.
22. S Azawi, T Liehr, M Rincic. First molecular cytogenetic characterisation of tracheal squamous cell carcinoma cell line KLN 205. J Cancer Metast Treatm 2021, 7:38. http://dx.doi.org/10.20517/2394-4722.2021.59.
23. S Azawi, M Balachandran, F Kramer, S Kankel, M Rincic, T Liehr. Molecular cytogenetic characterization of urethane induced murine lung cell line LA-4 as model for human squamous cell lung cancer. Mol Clin Oncol 2022, 16:9. https://doi.org/10.3892/mco.2021.2440.
24. S Azawi, S Kankel, T Liehr, M Rincic. First cytogenomic characterization of murine testis tumor cell line MLTC-1. Mol Exp Biol Med 2022, 4:10-14.
25. S Azawi, L-M Barf, T Liehr. First molecular cytogenetic characterization of the MMT 060562 murine breast cancer cell line. Research Results Biomedicine 2021, 7:4-14. https://doi.org/10.18413/2658-6533-2020-7-1-0-1.
26. L Hergenhahn, N Padutsch, S Azawi, R Weiskirchen, T Liehr, Martina Rinčic. Cytogenomic characterization of murine neuroblastoma cell line Neuro-2a and its two derivatives Neuro-2a TR-alpha and Neuro-2a TR-beta. Cells 2024, 13:1889. https://doi.org/10.3390/cells13221889.
27. C Leibiger, N Kosyakova, H Mkrtchyan, M Glei, V Trifonov, T Liehr. First molecular cytogenetic high resolution characterization of the NIH 3T3 cell line by murine multicolor banding. J Histochem Cytochem 2013, 61:306-312. https://doi.org/10.1369/0022155413476868.
28. T Liehr, M Rincic. Cytogenomic characterization of murine cell line Sarcoma 180 = S-180. Int J Mol Sci 2025, 26:1127. https://doi.org/10.3390/ijms26031127.
29. R Steinacker, T Liehr, N Kosyakova, M Rincic, SS Hussein Azawi. Molecular cytogenetic characterization of two murine cancer cell lines derived from salivary gland. Biol Commun 2019, 63: 243-255. https://doi.org/10.21638/spbu03.2018.403.
30. T Liehr, M Rincic. Cytogenomic characterization of murine osteosarcoma cell line SEWA. Cytogenet Genome Res 2024, 164:236-242. https://doi.org/10.1159/000543063.
31. S Azawi, F Kramer, T Liehr, M Rincic. First cytogenomic characterization of murine cutaneous sarcoma cell line TSC2ang1. Science Technology and Engineering Journal (STEJ) 2022, 8:1-16. https://ph02.tci-thaijo.org/index.php/stej/article/view/246689.
32. LF Buitkamp, T Liehr, S Kankel, EM Buhl, KS Hardt, DT Keller, SK Schröder-Lange, R Weiskirchen. Comprehensive genetic and molecular characterization confirms hepatic stellate cell origin of the immortal Col-GFP HSC line. Int J Mol Sci 2025, 26:7764. https://doi.org/10.3390/ijms26167764.
33. E Arlt, N Bley, A Rausch, S Hauser, J Pietzsch, D Misiak, T Liehr, S Kankel, T Dautz, L Dreher, X Kŏng, H Kielstein, I Bazwinsky-Wutschke. Sex determines the natural killer cell-mediated immunity against pancreatic cancer. Oncoimmunology 2026, 15:2645298. https://doi.org/10.1080/2162402X.2026.2645298.
34. T Liehr, S Kankel, KS Hardt, EM Buhl, H Noels, DT Keller, SK Schröder-Lange, Ralf Weiskirchen. Genetic and molecular characterization of H9c2 rat myoblast cell line. Cells 2025, 14:502. https://doi.org/10.3390/cells14070502.
35. T Liehr, S Kankel, EM Buhl, SK Schröder-Lange, R Weiskirchen. Genetic characteristics of the rat fibroblast cell line Rat-1. Cells 2025, 14:21. https://doi.org/10.3390/cells14010021.
36. M Liyanage, A Coleman, S du Manoir, T Veldman, S McCormack, RB Dickson, C Barlow, A Wynshaw-Boris, S Janz, J Wienberg, MA Ferguson-Smith, E Schröck, T Ried T. Multicolour spectral karyotyping of mouse chromosomes. Nat Genet 1996, 14:312-315. https://doi.org/10.1038/ng1196-312.
37. Y Wang, T Liehr. The need for a concert of cytogenomic methods in chromosomic research and diagnostics. Genes (Basel) 2025, 16:533. https://doi.org/10.3390/genes16050533.

38. T Liehr. Chromosome microarray results to be verified by FISH. In: Liehr, T. (eds) Fluorescence In Situ Hybridization (FISH). Springer Protocols Handbooks. Humana, New York, NY 2026, pp. 689-694. https://doi.org/10.1007/978-1-0716-5150-6_63.
39. T Liehr. Next generation sequencing should consider molecular cytogenetic results. In: Liehr, T. (eds) Fluorescence In Situ Hybridization (FISH). Springer Protocols Handbooks. Humana, New York, NY 2026, pp. 703-707. https://doi.org/10.1007/978-1-0716-5150-6_65.
40. T Liehr. Optical genome mapping results need to be verified by FISH. In: Liehr, T. (eds) Fluorescence In Situ Hybridization (FISH). Springer Protocols Handbooks. Humana, New York, NY 2026, pp. 709-712. https://doi.org/10.1007/978-1-0716-5150-6_66.
41. NA Ariffen, AA Ornellas, G Alves, AM Shana'ah, S Sharma, S Kankel, E Jamali, B Theis, T Liehr. Amplification of different satellite-DNAs in prostate cancer. Pathol Res Pract 2024, 256:155269. https://doi.org/10.1016/j.prp.2024.155269.
42. IY Iourov, SG Vorsanova, YB Yurov, MA Zelenova, OS Kurinnaia, KS Vasin, SI Kutsev. The cytogenomic “Theory of Everything”: Chromohelkosis may underlie chromosomal instability and mosaicism in disease and aging. Int J Mol Sci 2020, 21:8328. https://doi.org/10.3390/ijms21218328.
43. M Fischer. Mice are not humans: The case of p53. Trends Cancer 2021, 7:12-14. https://doi.org/10.1016/j.trecan.2020.08.007.
44. T Liehr. Thorough discussion of cancer research – thoughts against the main stream. Europ J Hum Genet 2017, 25:902-903. https://doi.org/10.1038/ejhg.2017.7.
45. AC Huang, SB Olson, CL Maslen. A review of recent developments in Turner syndrome research. J Cardiovasc Dev Dis 2021, 8:138. https://doi.org/10.3390/jcdd8110138.

Conflicts of Interest: The author declares no conflicts of interest.

Disclaimer/Publisher’s Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of REPNAS and/or the editor(s). REPNAS and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.