False Leukemia-Lymphoma-Cell Lines

by Hans G. Drexler

DSMZ, Braunschweig

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False, Virtual, Misidentified, Misclassified Leukemia-Lymphoma Cell Lines

This chapter represents a modified version of the following publication:

- Drexler HG, Dirks WG, Matsuo Y, MacLeod RAF: False leukemia-lymphoma cell lines: An update on over 500 cell lines. Leukemia 17: 416-426 (2003).

In 96 of the 727 (13%) cell lines analyzed at DSMZ, we found unequivocal evidence of misidentification (compare **Figures 22 and 23;** details in **Tables 20-23**). Typical examples are a B-cell instead of a T-cell immunoprofile (e.g. cell line Karpas 45), untoward similarities in cytogenetic marker chromosomes (e.g. cell line SPI-801), isoenzyme patterns specific for murine rather than human cells (e.g. Reh-6), and discrepancies between cell lines allegedly established from the same patient (e.g. cell lines KMS-21-BM and KMS-21-PE). In most instances, the false cell lines showed the same DNA fingerprints as another, yet clearly authenticated cell line. In a few cases we could not determine the correct identity of the cross-contaminated cell line but had clear evidence that the cells at hand were false. Initially prior to the routine use of DNA fingerprinting, our cytogenetic analysis alone uncovered many cases of cross-contaminated cell lines based on the detection of marker chromosomes (e.g. cell lines LR10.6 and PBEI) and later as an adjunct to the DNA fingerprinting confirmed the identity of the impostor cells as the majority of the cross-contaminating cell lines were restricted to a handful of well-known and karyotypically well-characterized cell lines (**Table 24**).

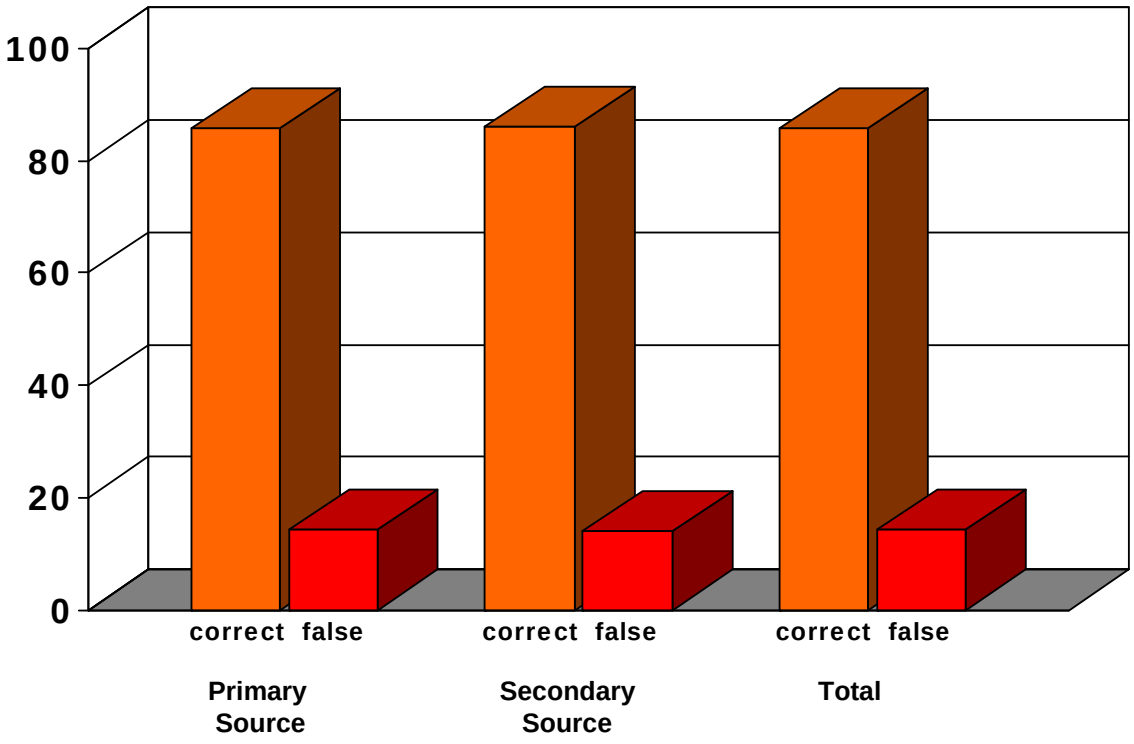

Figure 22: Percentages of false and correct leukemia-lymphoma cell lines obtained from the original (primary) source and from secondary sources.
Details: 497 cell line cultures from original sources (87% correct versus 13% false); 230 cell lines from secondary sources (87% correct versus 13% false); 727 cell lines in total (87% correct versus 13% false).
Drexler HG, Dirks W, MacLeod RAF, unpublished 2010.

Upon detection of a cross-contaminated cell line displaying a fingerprint identical to that of an older well-authenticated cell line, the question arises whether an authentic prototype of the cross-contaminated cell line ever existed: i.e. did cross-contamination give rise to a virtual cell line only, or replace cultures of one which actually enjoyed independent existence and of which uncontaminated material may still exist?

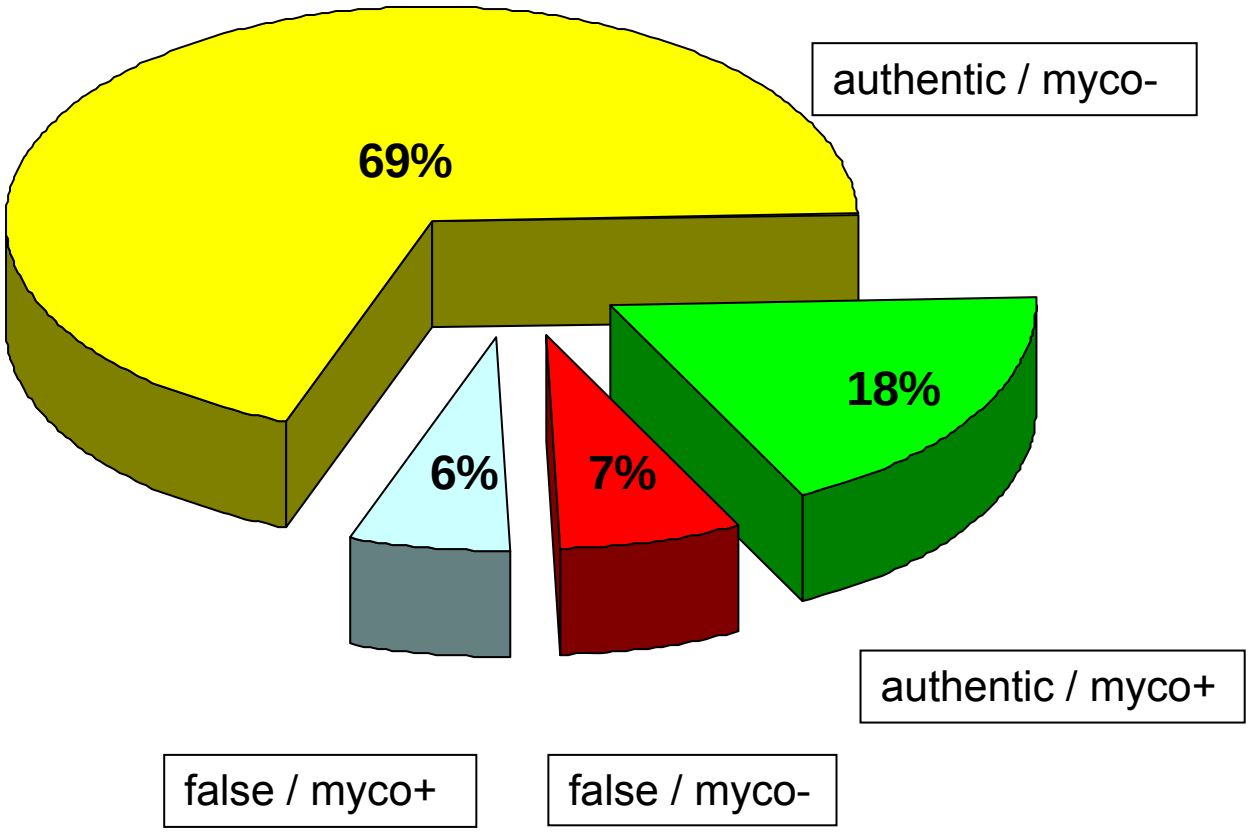

Figure 23: Analysis of 604 leukemia-lymphoma cell line cultures for the two parameters authentic/false and mycoplasma-positive/negative. Details: 417 authentic/myco-negative cell lines; 108 authentic/myco-positive cell lines; 41 false/myco-negative cell lines; 38 false/myco-positive cell lines.

VIRTUAL (FALSE) CELL LINES: NON-EXISTENT PROTOTYPE

Table 20 summarizes those "virtual" cell lines where there is unambiguous and sufficient evidence to exclude the existence of any authentic prototype with reasonable certainty. The best evidence comes from those original publications in which a sufficiently informative karyotype of the purportedly new cell line (but displaying in fact the karyotype of the cross-contaminant) is available. Examples include the following cell lines: AG-F, Co, Dami, JOSK-series, SPI-801/SPI-802, SR-91, TI-1, WSU-CLL, YJ and others. In other instances the original investigators could not provide authentic prototype cells.

Table 20: Virtual (False) Cell Lines: Non-Existent Prototype

Cell Line	Purported Malignancy	Real Identity	Actual Malignancy	Evidence [a] Author`s Karyotype	Confirmation at DSMZ [b]	Ref. [c]
207 = EU-2	BCP-ALL	**Reh/SUP-B2**	BCP-ALL		+ [d]	1-3
AG-F	Hogdkin	**CCRF-CEM**	T-ALL	+ [e]		4,5
Be13	T-ALL	**Peer**	T-ALL	+	+ [f]	6,7
BLIN-1 (1E8) [g]	BCP-ALL	**NALM-6**	BCP-ALL	+	+ [h]	8,9
Co (= **Cole**)	Hodgkin	**CCRF-CEM**	T-ALL	+	+	5,10
CTV-1	AML M5	not known	probably T-ALL	+	+	11
Dami	AML M7	**HEL**	AML M6	+	+	12-14
DD	malignant histiocytosis	**K-562**	CML-BC		+ [i]	15,16
EH	HCL	**HK**	HCL		+ [j]	17
EU-1	BCP-ALL	**Reh**	BCP-ALL	+	+	2,18
EU-7	T-ALL	**CCRF-CEM**	T-ALL		+	5,19
HKB-1	Hodgkin	**BJA-B**	Burkitt	+	+ [k]	20,21
HPB-MLT	T-ALL	**HPB-ALL**	T-ALL	original clinical data	+ [l]	22
HS-SULTAN	Myeloma	**Jiyoye**	Burkitt	known at ATCC	+	23-25
J-111	AML M5	**HeLa**	cervix carcinoma	known since 1976	+	26,27
JOSK-I	AML M4	**U-937**	histiocytic lymphoma	+	+	28,29
JOSK-K	AML M5	**U-937**	histiocytic lymphoma	+	+	28,29
JOSK-M	CML-BC	**U-937**	histiocytic lymphoma	+	+	28,29
JOSK-S	AML M5	**U-937**	histiocytic lymphoma	+	+	28,29
KPB-M15	CML-BC	**KYO-1**	CML-BC	+	+	30,31
LR10.6	BCP-ALL	**NALM-6**	BCP-ALL	+	+	8,32
MDS	CMML	**JURKAT**	T-ALL		+ [i]	33,34
MKB-1	AML	**CCRF-CEM**	T-ALL	+	+	5,35
MOBS-1	AML M5	**U-937**	histiocytic lymphoma		+	29
MOLT 15	T-ALL	**CTV-1**	T-ALL?	+	+	36,37
MUTZ-1	BCP-ALL	**Namalwa**	Burkitt	+	+	38,39
NOI-90	NK-NHL	**Reh**	BCP-ALL		+ [i]	2,40
OU-AML-1	AML M4	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-2	AML M2	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-3	AML M4	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-4	AML M2	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-5	AML M5	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-6	AML M1	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-7	AML M4	**OCI/AML2**	AML M4		+ [i]	41,42
OU-AML-8	AML M4	**OCI/AML2**	AML M4		+ [i]	41,42
PBEI	BCP-ALL	**NALM-6**	BCP-ALL		+ [i]	8,43
PC-MDS	t-MDS	**K-562**	CML-BC	+	+	16,44
PLB-985	AML M4	**HL-60**	AML M2		+ [i]	45,46
RED-3	AML	**HL-60**	AML M2	+ [m]		46,47
RM10	CML-BC	**K-562**	CML-BC	+ [n]		16,48

SAM-1	CML-BC	**K-562**	CML-BC		+ [l]	16,49
SAML-1	AML	**U-937**	histiocytic lymphoma	+ [o]		29,50
SPI-801	T-ALL	**K-562**	CML-BC	+	+	16,51
SPI-802	T-ALL	**K-562**	CML-BC	+	+	16,51
SR-91	T-ALL	**AML-193**	AML M5	+	+	52,53
ST-4	T-NHL	**PF-382**	T-ALL		+ [l]	54,55
TI-1	AML M2	**K-562**	CML-BC	+	+	16,56,57
UCONN L2	ALCL	**JB6**	ALCL		+	58,59
UTMB-460	B-cells	**CCRF-CEM**	T-ALL	+		5,60
WSU-ALCL	ALCL	**CCRF-CEM**	T-ALL		+ [l]	5,61
WSU-CLL	CLL	**Reh**	BCP-ALL	+	+	2,62,63
YAA	Monocytes	**U-937**	histiocytic lymphoma	+	+	29,64
YAP	Monocytes	**U-937**	histiocytic lymphoma	+	+	29,65
YJ	CMML	**HL-60**	AML M2	+	+	46,66

[a] Evidence in the original publication for the real identity, e.g. unequivocal karyotypic description or image of sufficient quality.
[b] Confirmation of real identity at DSMZ by DNA fingerprinting and comparison with DNA fingerprint databank and/or cytogenetic analysis.
[c] References for false and correct cell lines and relevant related publications.
[d] 207 cells were obtained directly from original author twice and turned out to be cross-contaminated with different cell lines; informative karyotype not provided in the literature; a 207 aliquot obtained from a secondary source was found by DNA fingerprinting to be CCRF-CEM (not listed).
[e] Karyotypic identity between original author's report and existing CCRF-CEM variant karyotypes; original author declined to provide cell line for DNA fingerprinting.
[f] Cell lines Be13 and Peer show identical DNA fingerprints and hence share common origin, presumably due to cross-contamination; however, their diploid and tetraploid karyotypes indicative of earlier and later passage numbers, respectively, suggest that Be13 is derived from Peer, rather than vice versa.
[g] 1E8 is a subclone of BLIN-1.
[h] Both the parental cell line BLIN-1 and the subclone 1E8 were found to be NALM-6; another BLIN-1 culture was cross-contaminated with K-562 (not listed).
[i] Cells were obtained directly from original author; informative karyotype not provided in the literature.
[j] Cell lines EH and HK are supposed to be derived from two individual patients; presumably rather EBV-positive B-LCLs (see **Table 23**).
[k] Different karyotype than original description of cell line and different from patient material (confirmed by Dr. R. Siebert, Kiel, Germany).
[l] Cell line HPB-MLT is taken to be HPB-ALL based on (i) gender and (ii) clinical diagnosis, both of which are compatible with HPB-ALL only.
[m] Based on molecular biological description of RED-3 cells (*MYC* and *NRAS* mutations); cells were not provided by original investigators.
[n] *ABL* amplification and globin expression identical with that of K-562; original author declined to provide cell line.
[o] Original authors detected themselves the cross-contamination (personal communication).

MISIDENTIFIED (FALSE) CELL CULTURES: PROTOTYPE (CORRECT) CELL LINE EXISTS OR MAY EXIST

In **Tables 21** and **22** I listed those cell lines which were found to be false but of which the correct prototypes may still exist. However, in a number of cases, it appears improbable that the prototype cells may emerge (**Table 21**). Fortunately, in a number of cases I know that the correct cell lines (the prototypes) still exist and are available (**Table 22**). These tables should alert the reader that while for example the real cell lines HPB-ALL, KE-37, L 540, U-937 and UT-7 (to name a few of the widely distributed lines) are certainly available, there are also impostor cultures under these designations "going the rounds".

Table 21: Misidentified (False) Cell Cultures: Prototype (Correct) Cell Line May Exist or May Be Lost

Cell Culture	Purported Malignancy	Real Identity	Actual Malignancy	Confirmed at DSMZ [a]	Existent Prototype [b]	Ref. [c]
HIMEG-1	CML	**HL-60**	AML M2	+	not known, but unlikely [d]	46,67
K051	AML M2	**K-562**	CML-BC	+	not known;unique karyotype published [d]	16,68
KM-3	BCP-ALL	**Reh**	BCP-ALL	+	not known	2,34
KMS-21-BM	myeloma	unknown	unknown	+	not known [d,e]	69
MHH 225	AML M7	**JURKAT**	T-ALL	+	not known;unique karyotype published [d]	34,70
P39/Tsugane	AML M2	**HL-60**	AML M2	+	not known;unique karyotype published	46,71
RC-2A	AML M4	**CCRF-CEM**	T-ALL	+	not known, but unlikely	5,72
RS-1	AML M7	**K-562**	CML-BC	+	not known;unique karyotype published [d]	16,73
SKW-3	T-CLL	**KE-37**	T-ALL	+	not known	74
SU-DHL-7	B-NHL	**SU-DHL-8**	B-NHL	+	not known;unique karyotype published [d]	75
SU-DHL-9	B-NHL	**SU-DHL-8**	B-NHL	+	not known;unique karyotype published [d]	75
T-33	CML-BC	**K-562**	CML-BC	+	not known;unique karyotype published [d]	16,76

[a] Confirmation at DSMZ by DNA fingerprinting and comparison with DNA fingerprint databank.
[b] If the prototype cell line was not available for our analysis (e.g. from the original investigator) or the original publication was not sufficiently informative (e.g. full karyotype), it remains uncertain whether a prototype cell line truly exists.
[c] References for false and correct cell lines and relevant related publications.
[d] Cell line obtained from laboratory of the originator.
[e] KMS-21-BM and KMS-21-PE are supposedly sister cell lines, but showed different DNA fingerprints.

Table 22: Misidentified (False) Cell Cultures: Prototype (Correct) Cell Line Does Exist

Cell Culture	Purported Malignancy	Real Identity	Actual Malignancy	Confirmed at DSMZ [a]	Prototype Exists [b]	Ref. [c]
BJA-B	Burkitt	**Reh**	BCP-ALL	+	yes	2,21
DoHH2	B-NHL	**SU-DHL-1**	ALCL	*	yes (e.g. correct DoHH2 at DSMZ)	77,78
HPB-ALL	T-ALL	**JURKAT**	T-ALL	+	yes (e.g. correct HPB-ALL at DSMZ)	22,34
Karpas 45	T-ALL	unknown	unknown	+	yes (e.g. correct Karpas 45 at DSMZ)	79
KBM-3	AML M4	**HL-60**	AML M2	+	Yes	46,80
KE-37	T-ALL	**CCRF-CEM**	T-ALL	+	yes (e.g. correct KE-37 at DSMZ)	5,81
L 540	Hodgkin	**CCRF-CEM**	T-ALL	+	yes (e.g. correct L 540 at DSMZ)	5,82
MB-02	AML M7	**HU-3**	AML M7	+	yes	83,84
RPMI 8402	T-ALL	unknown	unknown	+	yes (e.g. correct RPMI 8402 at DSMZ)	85
SU-DHL-4	B-NHL	unknown	unknown	+	yes, (e.g. correct SU-DHL-4 at DSMZ)	75
U-937	histiocytic lymphoma	unknown	unknown	+	yes (e.g. correct U-937 at DSMZ)	29
UT-7	AML M7	**U-937**	histiocytic lymphoma	+	yes (e.g. correct UT-7 at DSMZ)	29,86

[a] Confirmation at DSMZ by DNA fingerprinting and comparison with DNA fingerprint databank.
[b] The correct cell line does exist, e.g. in a cell lines bank.
[c] References for false and correct cell lines and relevant related publications.

MISCLASSIFIED (NON-MALIGNANT) CELL LINES

Not every cell line derived from a tumor patient is necessarily a tumor cell line as non-malignant cells which are independent of the tumor cells may sometimes be immortalized as well. In a leukemic context, such cell lines are usually normal B-cells which become immortalized through incorporation of the Epstein-Barr virus genome (so-called EBV^+ B-lymphoblastoid cell lines, B-LCL) (see also chapter: **III. EBV- and HTLV-Positive Cell Lines**).

There are several hematopoietic diseases from which it is notoriously difficult to establish cell lines, in particular the mature B-cell malignancies such as chronic lymphocytic leukemia (CLL), hairy cell leukemia (HCL), plasma cell leukemia (PCL) and multiple myeloma and Hodgkin lymphoma (**Table 23**). While there are number of *bone fide* myeloma-, PCL- and Hodgkin lymphoma-derived cell lines, various cell lines which are in reality EBV^+ B-LCLs have been described and are still being used as model systems for these diseases. The most difficult group are certainly the alleged B-CLL and HCL cell lines of which the majority are EBV^+ B-LCLs.

Table 23: Misclassified (Non-Malignant) Cell Lines

Cell Line	Purported Malignancy	Actual Cell Type	Comments	References [a]
ARH-77	myeloma	EBV^+ B-LCL?	controversial [b]	87,88
EH/HK	HCL	EBV^+ B-LCL	see also Table 1	17
EHEB	B-CLL	EBV^+ B-LCL	DSMZ ACC 87	89,90
FQ	Hodgkin	monkey cell line	see below [c]	91-93
GM1312	myeloma	EBV^+ B-LCL		94
GM1500	myeloma	EBV^+ B-LCL		94
Hs 445	Hodgkin	EBV^+ B-LCL	ATCC [d]	95
IM-9	myeloma	EBV^+ B-LCL	DSMZ ACC 117	88,96
L 591	Hodgkin	EBV^+ B-LCL?	controversial [e]	97,98
MC/CAR	myeloma	EBV^+ B-LCL		88,99
RB	Hodgkin	monkey cell line	see below [c]	91-93
RPMI 6666	Hodgkin	EBV^+ B-LCL	ATCC [d]	100
Rsp	Hodgkin	EBV^+ B-LCL		101
RY	Hodgkin	monkey cell line	see below [c]	91-93
SpR	Hodgkin	monkey cell line	see below [c]	91-93
TMM	CML-BC	EBV^+ B-LCL	DSMZ ACC 95	102
UMJF-2	myeloma	EBV^+ B-LCL		103

[a] References for misclassified cell lines and relevant related publications.
[b] Cell line is often used as myeloma model system; while cells carry various clonal cytogenetic abnormalities, cells are definitely EBV^+ (see also above in **Cell Lines Chapter 3: Plasma Cell Lines**).
[c] For a discussion of this notorious case, see the book “Betrayers of the Truth, Fraud and Deceit in Science” (ref. 93).
[d] Misleadingly indicated at ATCC as being derived from “tissue Hodgkin’s disease”.
[e] Cell line is often used as Hodgkin lymphoma model system, but not proven to be derived from Hodgkin-Reed-Sternberg cells.

MOST PROLIFIC CONTAMINANTS

In the majority of cases, the cross-contaminating intruder could be identified. In most instances, these cross-contaminating cells were well-known “classic” cell lines (**Table 24**). As these cell lines have all been established

more than 25 years ago, they are now widely distributed throughout the scientific community and may be found in many laboratories working with cell lines. Furthermore, most are available from the major public cell line banks in Europe, USA and Japan (and from minor cell line banks in other countries). Finally, these cell lines grow very well, exacerbating dispersal, and have short doubling times leading to rapid overgrowth of the initial culture into which these cell lines were introduced.

Besides DNA fingerprints, distinguishing chromosomal features when present may be used to identify human tumor cell lines. Cytogenetic identifiers for each of the seven most prolific cross-contaminants are presented in **Table 24** and are uniquely represented *in vitro*, excepting del(2)(p23) in JURKAT which recurs in several cell lines. A further problem for cytogenetic authentication is posed by cell lines originally displaying normal or near-normal karyotypes of which CCRF-CEM is a prime example. Several subclones of the originally near-normal CCRF-CEM exist under a bewildering variety of aliases, each with its own apparently unique acquired chromosome rearrangement allowing these to pose as truly distinct cell lines. Although recurrent primary translocations are usually less informative for authentication, a notable exception involves the classic CML cell line K-562 which uniquely carries two marker chromosomes in which *BCR-ABL* fusion is effected by a cryptic t(9;22)(q34;q11) regionally amplified in tandem, while U-937 (*PICALM-AF10*) and REH (*ETV6/TEL-RUNX1/AML1*) are, respectively, unique and almost unique *in vitro* models for their respective gene fusions which, therefore, comprise useful descriptors.

Table 24: Most Prolific Contaminants

Cell Line	Year	Malignancy	Doubling Time [a]	Contamin-ations	Karyotypic Descriptors
CCRF-CEM	1964	T-ALL	24 h	9 x	t(8;9)(p11;p24) [b]
K-562	1970	CML-BC	30-40 h	9 x	2-3 markers comprising tandem *BCR-ABL1* fusion repeats
U-937	1974	histiocytic lymphoma	30-40 h	8 x	t(1;5)(p22;q3?), t(10;11)(p13;q14-21) [c]
HL-60	1976	AML M2	25 h	6 x	dic(5;17)(q11;q11)
Reh	1974	BCP-ALL	30-50 h	5 x	t(4;12;21;16)(q32;p13;q21;q24) [d]
NALM-6	1976	BCP-ALL	36 h	5 x	t(5;12)(q33;p13)
JURKAT	1976	T-ALL	25-35 h	3 x	del(2)(p23)

[a] Doubling time according to experience at DSMZ.
[b] Present in most but not all subclones.
[c] t(10;11) effects *AF10-PICALM* fusion; detectible also by RT-PCR.
[d] Occult t(12;21) effects *ETV6/TEL-RUNX1/AML1* fusion; detectible also by RT-PCR.

References

[1] Findley HW Jr et al., Blood 60: 1305-1309 (1982).
[2] Rosenfeld C et al., Nature 267: 841-843 (1977).
[3] Zhang LQ et al., Leukemia 7: 1865-1874 (1993).
[4] Gazitt Y et al., Leukemia 7: 2034-2044 (1993).
[5] Foley GE et al., Cancer 18: 522-529 (1965).
[6] Galili N et al., Human Lymph Diff 1: 123-130 (1981).
[7] Ravid Z et al., Int J Cancer 25: 705-710 (1980).
[8] Hurwitz R et al., Int J Cancer 23: 174-180 (1979).
[9] Wörmann B et al., J Immunol 142: 110-117 (1989).
[10] Jones DB et al., Hematol Oncol 3: 133-145 (1985).
[11] Chen PM et al., Gann 75, 660-664 (1984).
[12] MacLeod RAF et al., Leukemia 11: 2032-2038 (1997).
[13] Greenberg SM et al., Blood 72: 1968-1977 (1988).
[14] Handin RI, Blood 89: 4238 (1997).
[15] Kávai M et al., Cell Immunol 139: 531-540 (1992).
[16] Lozzio CB & Lozzio BB, J Natl Cancer Inst 50: 535-538 (1973).
[17] Faguet GB et al., Blood 71: 422-429 (1988).

[18] Zhou M et al., Leukemia 8: 659-663 (1994).
[19] Zhou M et al., Leukemia 9: 1159-1161 (1995).
[20] Wagner HJ et al., Med Ped Oncol 31: 138-143 (1998).
[21] Menezes J et al., Biomedicine 22: 276-284 (1975).
[22] Morikawa S et al., Int J Cancer 21: 166-170 (1978).
[23] Drexler HG et al., Blood 98: 3495-3496 (2001).
[24] Harris NS, Nature 150: 507-509 (1974).
[25] Kohn G et al., J Natl Cancer Inst 38: 209-222 (1967).
[26] Nelson-Rees WA & Flandermeyer RR, Science 191: 96-97 (1976).
[27] Osgood EE & Brooke JH, Blood 10: 1010-1022 (1955).
[28] Ohta M et al., Cancer Res 46: 3067-3074 (1986).
[29] Sundström C & Nilsson K, Int J Cancer 17: 565-577 (1976).
[30] Kamamoto T et al., Jpn J Clin Oncol 16: 107-115 (1986).
[31] Ohkubo T et al., Leukemia Res 9: 921-926 (1985).
[32] Inglés-Esteve J et al., Leukemia 11: 1040-1044 (1997).
[33] Banerjee R et al., Proc Natl Acad Sci USA 89: 9996-10000 (1992).
[34] Schneider U et al., Int J Cancer 19: 621-626 (1977).
[35] Matsuo Y et al., Human Cell 2: 423-429 (1989).
[36] Drexler HG et al., Hematol Oncol 7: 115-125 (1989).
[37] Chen PM et al., Gann 75: 660-664 (1984).
[38] Steube K et al., Leukemia Lymphoma 25: 345-363 (1997).
[39] Nadkarni JS et al., Cancer 23: 64-79 (1969).
[40] Sahraoui Y et al., Leukemia 11: 245-252 (1997).
[41] Zheng A et al., Brit J Cancer 79: 407-415 (1999).
[42] Wang C et al., Leukemia 3: 264-269 (1989).
[43] Pirruccello SJ et al., Blood 78: 39a (1991).
[44] Bogdanovic G et al., Leukemia Res 31: 1097-1105 (2007).
[45] Tucker KA et al., Blood 70: 372-378 (1987).
[46] Collins SJ et al., Nature 270: 347-349 (1977).
[47] Mallet MK et al., Leukemia 3: 511-515 (1989).
[48] Hirata J et al., Leukemia 4: 365-372 (1990).
[49] Kamesaki H et al., Blood 87: 999-1005 (1996).
[50] Knutsen T et al., Genes Chromosomes Cancer 37: 270-281 (2003).
[51] Komiyama A et al., Blood 60: 1429-1436 (1982).
[52] Klingemann HG et al., Leukemia Lymphoma 12: 463-470 (1994).
[53] Lange B et al., Blood 70: 192-199 (1987).
[54] Arione R et al., Cancer Res 48: 1312-1318 (1988).
[55] Pegoraro L et al., J Natl Cancer Inst 75: 285-290 (1985).
[56] Rush J et al., Blood 99: 1874-1876 (2002).
[57] Taoka T et al., Blood 80: 46-52 (1992).
[58] Morris SW et al., Science 263: 1281-1284 (1994).
[59] Kadin ME et al., Blood 76: 354a (1990).
[60] Juneja HS et al., Leukemia Res 10: 1209-1219 (1986).
[61] Al-Katib A et al., Blood 84: 638a (1994).
[62] Drexler HG et al., Leukemia 16: 1868-1870 (2002).
[63] Mohammad RM et al., Leukemia 10: 130-137 (1996).
[64] Hamamoto Y et al., Exp Dermatol 6: 49-53 (1997).
[65] Hamamoto Y et al., Arch Dematol Res 288: 225-229 (1996).
[66] Yamaguchi Y et al., Leukemia 12: 1430-1439 (1998).
[67] Cheng T et al., Leukemia 9: 1257-1263 (1995).
[68] Abo J et al., Blood 82: 2829-2836 (1993).
[69] Otsuki T et al., Leukemia Lymphoma 42: 761-774 (2001).
[70] Hassan HT et al., Ann Hematol 71: 111-117 (1995).
[71] Nagai M et al., Gann 75: 1100-1107 (1984).
[72] Bradley TR et al., Brit J Haematol 51: 595-604 (1982).
[73] Skinnider LF et al., Acta Haematol 98: 26-31 (1997).
[74] Hirono T et al., J Immunol 123: 1133-1140 (1979).
[75] Epstein AL et al., Cancer 42: 2379-2391 (1978).
[76] Tange T et al., Cancer Res 48: 6137-6144 (1988).
[77] Kluin-Nelemans et al., Leukemia 5: 221-224 (1991).
[78] Epstein AL et al., Cancer 34: 1851-1872 (1974).
[79] Karpas A et al., Leukemia Res 1: 35-49 (1977).

[80] Andersson BS et al., Exp Hematol 20: 361-367 (1992).
[81] Drexler HG et al., Leukemia Res 9: 209-229 (1985).
[82] Diehl V et al., J Cancer Res Clin Oncol 101: 111-124 (1981).
[83] Morgan DA et al., Blood 78: 2860-2871 (1991).
[84] Morgan DA et al., Exp Hematol 25: 1378-1385 (1997).
[85] Moore GE et al., In Vitro 8: 434 (1973).
[86] Komatsu N et al., Cancer Res 51: 341-348 (1991).
[87] Drewinko B et al., Cancer 54: 1883-1892 (1984).
[88] Pellat-Deceunynk C et al., Blood 86: 4001-4002 (1995).
[89] Saltman D et al., Leukemia Res 14: 381-387 (1990).
[90] Drexler HG et al., 2010: http://www.cellines.de.
[91] Harris NL et al., Nature 289: 228-230 (1981).
[92] Long JC et al., J Exp Med 145: 1484-1500 (1977).
[93] Broad W & Wade N, Oxford University Press, 1985.
[94] Goldstein M et al., Blood 66: 444-446 (1985).
[95] Arnstein P et al., J Natl Cancer Inst 52: 71-84 (1974).
[96] Fahey JL et al., Ann NY Acad Sci 190: 221-234 (1971).
[97] Diehl V et al., Cancer Treat Rep 66: 615-632 (1982).
[98] Drexler HG, Leukemia Lymphoma 9: 1-26 (1993).
[99] Ritts RE Jr et al., Int J Cancer 31: 133-141 (1983).
[100] Moore GE et al., NY State J Med 66: 2757-2764 (1966).
[101] Friend C et al., Cancer Res 38: 2581-2591 (1978).
[102] McCarty TM et al., Blood 70: 1665-1672 (1987).
[103] Farnen JP et al., Leukemia 5: 574-584 (1991).