Journal of Medical Genetics 1988, **25**, 294–306

Genetic markers on chromosome 7

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SUMMARY Chromosome 7 is frequently associated with chromosome aberrations, rearrangements, and deletions. It also contains many important genes, gene families, and disease loci. This brief review attempts to summarise these and other interesting aspects of chromosome 7. With the rapid accumulation of cloned genes and polymorphic DNA fragments, this chromosome has become an excellent substrate for molecular genetic studies.

Chromosome 7 has been a subject of interest in medical genetics because of its frequent association with chromosome aberrations, rearrangements, and deletions, and because of the localisation of several important genes, gene families, and disease loci on this chromosome. It accounts for approximately 5% of the human genome[1] and it is estimated to be 136 centimorgans (cM) in length[2] and to contain 150 million basepairs of DNA. The short (p) and long (q) arm size ratio is approximately 1 to 1·5 (figure), but it varies significantly between prometaphase and midmetaphase.[3]

Approximately 40 genetic loci were reported to be on chromosome 7 at the Eighth Human Gene Mapping Workshop (Helsinki, September 1985).[4 5] There has been a dramatic increase of markers on this chromosome since then. The total number of gene loci and DNA markers characterised now well exceeds 400[6] (figure, table, K-H Grzeschik, 1987, personal communication, L-C Tsui *et al,* unpublished data). In addition, a chromosome 7 specific genomic library has been constructed by the Los Alamos and Lawrence Livermore National Laboratories using flow sorted chromosome 7 from a hamster-human hybrid cell line.[7]

Genes and gene families

More than 35 genes of assigned functions have been localised to chromosome 7. They are listed in the table. Some of the genes, such as β-actin[13] and proα2(I) collagen,[39–43] have been cloned and the gene structure analysed in detail; some have only been assigned by enzyme activities, such as biliverdin reductase[22] and paraoxanase.[51–54]

There are several gene families identified on chromosome 7. The T cell receptor β and γ chain genes have received most of the attention in the past few years. Both genes have complex gene structures and belong to the large immunoglobulin multigene family.[94 95] Expression of these genes requires proper chromosome rearrangement joining different DNA segments together during T cell differentiation.[96] The γ chain gene has been localised to the short arm, region p15.[20 21] The β chain gene has been mapped to 7q3 but different subregion localisation data have been obtained by different investigators; it is still unclear whether the gene maps to q32[68] or q35.[67 69]

The multidrug resistant phenotype of tumour cells is correlated with increased expression and amplification of the PGY-1 (*mdr-1*) locus which encodes a membrane glycoprotein called P glycoprotein.[97 98] This protein is thought to function as an energy dependent export pump to reduce intracellular levels of anticancer drugs.[99] Molecular cloning and mapping studies showed that the PGY-1 locus probably contains a number of related genes clustered within 7q21→q22.[45 46]

The β-actin[13] and the proα2(I) collagen[39–43] genes also belong to multigene families but their related members are dispersed elsewhere in the genome.

Disease loci

A number of disease loci have been mapped to chromosome 7. The metabolic defects argininosucci-

Received for publication 10 August 1987.
Accepted for publication 11 August 1987.

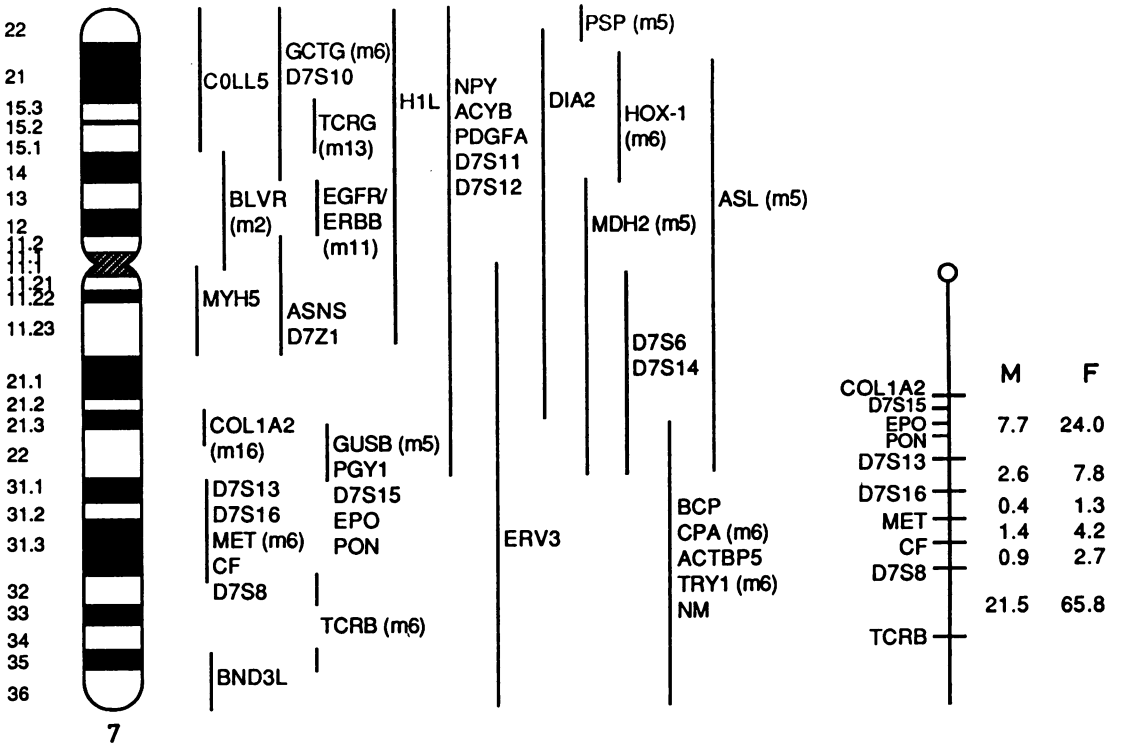

FIGURE *Genetic markers on chromosome 7. Regional localisations are indicated by vertical bars. The homologous mouse chromosomes are shown in brackets. The male and female genetic linkage maps for selected loci are shown on the right with numbers in centimorgans.*

nicaciduria, mucopolysaccharidosis VII, and trypsinogen deficiency are probably the result of mutations within the genes encoding the enzymes that are involved in the biochemical pathway, namely argininosuccinic lyase, β-glucuronidase, and trypsinogen, respectively.[100] Tritan colour blindness probably results from mutation of the blue-opsin gene.[57]

Osteogenesis imperfecta describes a group of fragile bone disorders which are clinically heterogeneous.[101] The syndrome can be divided into four phenotypic groups depending on the presence or absence of short stature, joint laxity, easy bruising, blue sclerae, presenile hearing loss, and dentinogenesis imperfecta. All of these symptoms are presumed to be the result of mutations in one of the six collagen genes. The genetics of OI have recently been reviewed in this Journal.[102] The proα2(I) collagen gene which is involved in all four groups is located on the long arm of chromosome 7 (7q21·3→q22·1).

The collagen genes have also been prime candidates for the gene mutation in Marfan syndrome, which is a group of autosomal dominant connective tissue disorders with skeletal, cardiac, and ocular defects.[103] A 20 amino acid insertion was found in 50% of the proα2(I) chains in one patient and was thought to be the cause of the disease.[104] DNA sequence analysis of the proα2(I) collagen gene revealed a 38 bp insertion in this patient.[105] However, subsequent studies showed that this 38 bp insertion is a common DNA fragment length polymorphism among random normal subjects[106] and there is no linkage relationship between the disease locus and the proα2(I) collagen gene in family studies.[107 108]

Ehlers-Danlos syndrome is another heterogeneous group of disorders with collagen defects.[100] Their clinical manifestations include laxity of joints, soft and extensible skin which may also be fragile, and excessive bruising. At least one form of this syndrome has been proposed to result from abnormal cleavage of the N-peptide of the proα2(I) chain.[109]

Cystic fibrosis (CF) is the most common severe

TABLE *Genes and DNA probes.*

Regional assignment	*Gene symbol*	*Name or probe*	*Mode* RFLP*	*References*
Genes				
pter→p15	COLL5	Collagen-like 5	A	8
pter→p14	GCTG	γ-glutamylcyclotransferase	S	9
pter→p22	PSP	Phosphoserine phosphatase	S	10
pter→q11	H1L	Histone H1-like genes	A	11
pter→q22	NPY	Neuropeptide Y	S,RE	12
pter→q22	ACTB	β-actin	S,RE	13
pter→q22	PDGFA	Platelet derived growth factor, A chain	RE,S	14
p22·1→q21	DIA2	Diaphorase	S	15, 16
p21→p14	HOX-1	Mouse Hox-1 homologue	RE,S	17
p21→q22	ASL	Arginosuccinate lyase	S,RE	18, 19
p15	TCRG	T cell receptor, γ chain	S,RE +	20, 21
p14→cen	BLVR	Biliverdin reductase	S,RE	22, 23
p13→p11	EGFR	Epidermal growth factor receptor (=ERBB)	S	24–28
p12→p14	ERBB	v-*erb*B oncogene homologue (avian erythroblastic leukaemia virus)	S,RE	29–32
p13→q22	MDH2	NAD malate dehydrogenase (mitochondrial)	S	33
p11→q11	ASNS	Asparagine synthetase	S,RE +	34, 35
cen→q11·2	MYH5	Myosin heavy polypeptide, adult skeletal muscle	A	36
q21→q22	ERV3	v-*erv*3 homologue	S,RE	37, Tsui *et al*, unpublished data
q21·3→q22·1	COL1A2	Collagen, type I α2	S,RE +	38–43
q22	PGY1	P glycoprotein	S,RE,A	44–46
q22	GUSB	β-glucuronidase	S,RE	33, 45, 47, Tsui *et al*, unpublished data
q22		G protein, αH subunit	S,RE	FS Collins, J Seidman, 1987, personal communication
q22	EPO	Erythropoietin	S,RE +	48–50
q22	PON	Paraoxonase	F	51–54
q22→q31	MET	met proto-oncogene	RE,A,S +	45, 55, 56
q22→qter	BCP	Blue cone pigment	RE,S	57
q22→qter	CPA	Carboxypeptidase A	RE	58
q22→qter	ACTBP5	β-actin pseudogene 5	S,RE	13
q22→qter	TRY1	Trypsin 1	S,RE +	58, 59
q31	CF	Cystic fibrosis	F	53, 60–63
q32 or q35	TCRB	T cell receptor, B chain	RE,S,A +	64–69
q35→q36	BND3L	Band 3-like, fibroblast	RE,S +	70
	ASSP11	Arginosuccinate synthetase pseudogene 1	RE,S +	71, 72
	PAI-1	Plasminogen activator inhibitor	S,RE	73
		Cytochrome P450 protein glucocorticoid inducible	S,RE +	74
	INFB2	β2 interferon	S,RE +	75
	UP	Uridine phosphorylase	S	76
	HADH	Hydroyacyl-CoA dehydrogenase	S	77
	PO4DB	Procollagen-proline, 2-oxoglutarate: 4-dioxygenase, β chain	S	78
DNA segments				
pter→p14	*D7S10*	pJ5·11	RE,S +	79
pter→q22	*D7S11*	Phage 6	RE,S +	20
pter→q22	*D7S12*	pB78	RE,S	79,80
p12→qter	*DNF9*	pA-8		81
p11→q11	*D7Z1*	Chromosome 7 specific alpha satellite sequences	RE,S +	82, 83
q22→q31	*D7S8*	pJ3·11	RE,S +	45, 79
cen→q22	*D7S6*	pJu48	RE,S +	79
cen→q22	*D7S14*	pJu28	RE,S	79, 80
q22	*D7S15*	Lam4–917	RE,S +	45, 60
q22	*D7S13*	B79a	RE,S,A +	79, 84, V Buckle, 1987, personal communication
q22→q31	*D7S16*	7C22	RE,S,A +	85, 86
	D7S1	pA2H3	RE,S +	87, 88
	D7S2	p7–15	RE,S	81
	D7S3	p7–11	RE,S	81
	D7S4	p7–12	RE,S	81
	D7S5	p7–13	RE,S	81

(*Continued*)

TABLE *Genes and DNA probes (continued).*

Regional assignment	*Gene symbol*	*Name or probe*	*Mode* RFLP*	*References*
Other determinants				
q22→qter	NM	Neutrophil migration	D	89
	GCF1	Growth control factor 1	S	90
	NHCP2	Non-histone chromosome protein 2	S	91
	PYHG3	Protein spot in 2D gel MW 106 000	S	92
	YHG3	Protein spot in 2D gel MW 80 000	S	92
	S7	Surface antigen	S	93

*A=in situ hybridisation; S=somatic cell genetics; F=family studies; RE=recombinant DNA techniques; D=deletion mapping.

autosomal recessive disorder in the Caucasian population.[110] The frequency of the mutant gene is estimated to be as high as 5% in some studies.[111] Patients with CF have major symptoms including obstructive pulmonary disease, pancreatic enzyme insufficiency, and raised sweat electrolyte levels. Although a general malfunction in the secretory epithelia seems to be a consistent finding in CF,[112–115a] the basic defect has yet to be discovered. Mapping of the CF locus has been the subject of intensive investigations in the past two years.[51–54 59–63 85 116–125a] Through the mapping of a large number of DNA markers surrounding the disease locus, the most probable chromosome location for CF is within band 7q31.[45 56 62 63 85 86 126] Genetic diagnosis has also been possible with the tightly linked DNA markers.[127–129]

Chromosome aberrations

There is a large number of documented cases of patients with short arm (7p–) deletions and translocations,[130–147] and most of them have been summarised recently.[147] The majority (60%) of deletions involve the terminal band p22 and about half of these also have band p21 or p15 deleted. Two cases of recurrent spontaneous abortions seem to indicate the involvement of 7p.[148 149] The remaining cases are interstitial deletions of various lengths, spanning between p21 to p13. The most frequent clinical abnormalities associated with 7p deletions are cranial dysmorphism, cleft palate, saddle nose, small, dysplastic, and low set ears, foot malformations, congenital heart disease, and genital malformations. However, the size and location of the chromosome segments involved do not show any obvious correlation with these or other symptoms.

Chromosome abnormalities have also been reported for the long (q) arm of chromosome 7. In contrast to the 7p– condition, the 7q– syndrome exhibits relatively consistent dysmorphic findings. Patients with terminal 7q deletions[150–162] have growth deficiency, developmental delay, microcephaly, low birth weight, bulbous nasal tip, anomalous auricles, cleft lip/palate, genital malformations in males, and ocular anomalies. One-third of terminal del(7q) patients also have a history of perinatal feeding problems, abnormal EEG with or without seizures, ocular hypertelorism, micrognathia, and chest abnormalities.

Interstitial 7q deletions can be divided into three classes based on the chromosomal regions involved, namely, q11→q21 (or q22), q21→q31 (or q32), and q32→q34, all of which show delayed development as their common feature. The clinical presentations seem to agree well with this classification.[160] In the q11→q21 (or q22) class, patients[139 163–167] generally show feeding problems and some malformation in addition to their developmental delay. The main features in the del(7)(q21→q31 or q32) patients[160 165 168–175] are delayed development, feeding difficulties, ear malformations, simian creases, low birth weight, and an unusual 'cat like' cry. A relatively small number of patients have been studied for the q32→q34 deletion,[160 176 177] but developmental delay, broad nasal bridge, bulbous nasal tip, ocular hypertelorism, and large mouth seem to be consistent within this last class.

Heritable partial trisomy 7q can also be divided on the basis of the chromosomal regions involved,[178] which are q22→q31 or qter,[179–182] q31→qter,[183–186] and q32 (or q33)→qter.[136 178–194] Retardation of development and low set ears are the common clinical features of all three groups but significant differences are noted among them.[178] Strabismus, epicanthus, and frontal bossing seem to be unique to trisomy 7(q22→q31), wide open fontanelle, cleft palate, microretrognathia, and early prenatal death to q31→qter, and kyphoscoliosis and hypotonia to q32 (or q33)→qter. Hypertelorism and small palpebral fissures appear to be common in the first and second groups, and low birth weight, small nose,

and skeletal anomalies in the second and the third groups. Thus, trisomy 7(q31→qter) is more serious than the other two.[178]

Paracentric inversion is infrequent in man but chromosome 7 appears to be affected the most often. This topic has been reviewed recently.[195 196] Most paracentric inversions are apparently harmless and the heterozygous carriers of inv(7) were primarily detected in cases involving infertility, repeated abortions, malformation, and advanced maternal age.[195–207] As in the other chromosome 7 aberrations, the paracentric inversions appear to be non-randomly distributed within this chromosome. The majority of the breakpoints are located in bands q11 and q22, and the inverted segment was q11→q22 in half of the cases.

More recently, uniparental disomy has been proposed as a mechanism for human genetic disease.[208] In the reported case, the presence of two identical copies of the maternal chromosome 7 and the absence of paternal chromosome 7 were thought to be the cause of cystic fibrosis in a patient who also had short stature.

Somatic cell chromosome aberrations

Monosomy 7 is frequently associated with secondary blood disorders, namely, acute non-lymphocytic leukaemia (ANLL) or myelodysplastic syndrome (MDS), which often occurs in patients undergoing aggressive therapy (irradiation or chemotherapy or both) for a previous malignant disease (for example, Hodgkin's disease).[209–220] A recent review[220] has indicated that more than 90% of these patients show hypodiploidy and monosomy 7 was found in more than 60% of the abnormal cases. There is a highly specific correlation between chromosome 7 abnormality and chemotherapy ($p<0{\cdot}01$).[220] A correlation has also been shown between monosomy 7 and haematological disorders in persons exposed to chemical carcinogens in the work place.[221–225] Deletion and translocation of 7q accounts for all of the cases with partial monosomy 7 in the latter category, the common deleted segment being 7q32 or q34.[226] Monosomy 7 and deletion of 7q also seem to be involved in de novo ANLL (or MDS) according to some[227–229] but not all studies.[219 227]

Chromosome 7 abnormalities have been implicated in several other cancer conditions, including ataxia telangiectasia,[230–232] erythroleukaemia,[233 234] gamma heavy chain disease,[235] lung carcinoid tumour,[236] prostate carcinoma,[237] transitional cell carcinoma of the ureter,[238] bilateral renal oncocytoma,[239] and malignant melanoma.[240–243] In vitro cell culture studies suggest that chromosome 7 is particularly unstable and vulnerable.[244–246] Interestingly, the translocation and deletion breakpoints discussed above seem to coincide well with the fragile sites observed on this chromosome, namely, FRA7A (rare, $7p11{\cdot}2^{247\ 248}$), FRA7B ($7p22^{249}$), FRA7C ($7p14{\cdot}2^{249}$), FRA7D ($7p13^{250\ 251}$), FRA7E ($7p21{\cdot}2^{249}$), FRA7F ($7q22^{250}$), FRA7G ($7q31{\cdot}2^{249\ 251}$), and FRA7H ($7q32{\cdot}3^{249\ 250}$). In a recent classification of fragile sites, 7q32 is listed as one of the most common fragile sites.[252]

Oncogenes

Direct involvement of genes on chromosome 7 in tumour formation or malignancy has been suggested. Enhanced expression of the epidermal growth factor (EGF) receptor (cellular homologue of the viral *erb*B gene) in malignant melanoma,[253] epidermal carcinoma,[254–257] pancreatic carcinoma,[258] and glioblastoma[259] cell lines has been correlated with 7p abnormalities and polysomy 7. A strong correlation has been observed between the presence of a 7q abnormality and the expression of the T cell growth factor receptor (Tac) in a group of patients with T cell leukaemia.[260] The possibility of activation of the Tac gene via the T cell receptor β chain gene has been proposed. Chromosome 7 abnormalities have also been detected in some patients with chronic T lymphocyte tumours, but the breaks are located in different bands.[261]

The *met* proto-oncogene, located within the 7q31 region,[56] was shown to have transforming potential in vitro when activated.[55] The transforming activity was first discoverd in a mouse NIH/3T3 cell transformation assay with DNA isolated from a MNNG treated human osteosarcoma (HOS) cell line,[262] and it is therefore unlike most other proto-oncogenes, which were initially identified through viral oncogenes. DNA sequence analysis showed that the *met* gene belongs to the tyrosine kinase gene family and is closely related to the human insulin receptor and the viral *abl* gene.[56] Subsequent studies revealed that the activation of *met* in the MNNG-HOS cell line involved a gene fusion event which resulted in the replacement of the 5′ portion of the proto-oncogene by sequences originating from chromosome 1.[263 264] The normal cellular function of *met* is as yet unknown.

The integration of the simian virus 40 genome in human chromosome 7 has been associated with cellular transformation.[265] The site of viral integration appears to be non-random[265] and maps to region 7q31.[266] Whether the transformation is the result of viral gene (T antigen) expression or disruption of cellular genes remains to be investigated.

Gene loci previously mapped to chromosome 7

There are a number of gene loci previously assigned to chromosome 7 but subsequently reassigned to a different chromosome. The two classical markers, the Colton (Co) and the Kidd (Jk) blood groups, were thought to be on chromosome 7 based on several lines of direct and indirect evidence.[151 267 268] Recent data suggest that they are located elsewhere[160] and probably unlinked (J Mohr, 1987, personal communication).

The mapping of the histone genes is interesting. Unlike the sea urchin and *Drosophila* histone genes which are clustered and tandemly repeated,[269] the human genes,[270 271] as well as those in yeast,[272] mouse,[273 274] and chicken,[275 276] are clustered but have no apparent repeats. Previous investigations, based on in situ hybridisation with probes derived from a histone mRNA enriched RNA fraction from human cells or sea urchin histone cDNA, showed that the human histone genes are either located within band 7q22 or 7q32→36.[277 278] However, when specific gene probes were used, these genes were found to be in several different chromosome regions; one cluster containing H3 and H4 has been localised to 1cen→q25[279] and other clusters containing core (H3, H4, H2A, and H2B) alone or core together with H1 have been assigned to 1cen→q31, 6p12→q16, and 12p11·2→q21.[11] A small number of grains was observed between the 7pter→q11 when a H1 specific probe was used in the latter in situ hybridisation experiment, but no signal was detectable by gel blot hybridisation analysis with two somatic hybrid cell lines, each containing chromosome 7 as the only human chromosome. The sequences detected on chromosome 7 by in situ hybridisation may represent cross hybridisation of other histone genes, a possibility that remains to be tested.

Genetic linkage map

Since the localisation of CF to 7q31, joint studies have been carried out to examine the linkage relationship among markers in this region.[117 280] Six loci have been analysed further and the gene order with greatest statistical support is *COL1A2–D7S13–D7S16–met–D7S8–TCRB*.[280] The most likely location of the CF gene on this map is between *met* and *D7S8*. A linkage map has also been generated for almost the entire length of chromosome 7 with randomly isolated DNA markers[281]; the map spans 250 cM in females and 170 cM in males, values much greater than previously estimated.[2] The most significant differences between the male and female genetic map appears to be within the portion of 7q bound by *COL1A2* and *TCRB*.[280 281]

Comparative mapping

The use of conserved linkage groups among mammals and primates has been an alternative approach to understanding the human gene map and evolution of chromosomes. Comparative mapping studies showed that human chromosome 7 markers are distributed over six different mouse chromosomes and that the mouse chromosome 5 and 6 sequences are found in both the long and short arms of human chromosome 7[282 283] (table) (S Naylor, 1987, personal communication).

I wish to thank Manuel Buchwald and Ron Worton for their continued encouragement; Arthur Beaudet, Anne Bowcock, Veronica Buckle, Ben Carritt, Francis Collins, Michael Dean, Helen Donis-Keller, John Edwards, Bert La Du, Karl-Heinz Grzeschik, Mark Lathrop, Victor Ling, Tak Mak, Jan Mohr, Sue Naylor, Peter O'Connell, John Seidman, Jorg Schmidtke, and George Vande Woude who provided preprints and discussed their findings before publication; and Shirley Sauvé for assistance in preparing the manuscript.

References

1. Renwick JH. The mapping of human chromosomes. *Annu Rev Genet* 1971;**5**:81–120.
2. Morton NE, Lindsten J, Iselius L, Yee S. Data and theory for a revised chiasma map of man. *Hum Genet* 1982;**62**:266–70.
3. Van Dyke DL, Worsham MJ, Fisher LJ, Weiss L. The centromere index and relative length of human high-resolution G-banded chromosomes. *Hum Genet* 1986;**73**:130–2.
4. Smith M, Spence A. Report of the committee on the genetic constitution of chromosomes 7, 8 and 9. *Cytogenet Cell Genet* 1985;**40**:156–78.
5. Willard HF, Skolnick M, Pearson PL, Mandel JL. Human gene mapping by recombinant DNA techniques. *Cytogenet Cell Genet* 1985;**40**:360–98.
6. Donis-Keller H, Barker DF, Knowlton RG, Schumm JW, Braman JC, Green P. Highly polymorphic RFLP probes as diagnostic tools. *Cold Spring Harbor Symp Quant Biol* 1986;**51**:317–24.
7. Deaven LL, Van Dilla MA, Bartholdi MF, *et al.* Construction of human chromosome-specific DNA libraries from flow-sorted chromosomes. *Cold Spring Harbor Symp Quant Biol* 1986;**50**:159–67.
8. Retief AE, Parker MI, Fox MF, *et al.* Chromosome mapping of collagen-genes by low stringency in situ hybridization conditions. HGM8. *Cytogenet Cell Genet* 1985;**40**:730.
9. Bissbort S, Bender K, Grzeschik KH. Assignment of the human gene for gamma-glutamyl-cyclotransferase (GCTG) to chromosome 7p. HGM7 *Cytogenet Cell Genet* 1984;**37**:421.
10. Koch CA, Eddy RL, Haley LL, Byers MC, McAvoy M, Shows TB. Assignment of the human phosphoserinephosphatase gene (PSP) to pter-q22 region of chromosome 7. *Cytogenet Cell Genet* 1983;**35**:67–9.
11. Tripputi P, Emanuel BS, Croce CM, Green LG, Stein GS, Stein JL. Human histone genes map to multiple chromosomes. *Proc Natl Acad Sci USA* 1986;**83**:3185–8.
12. Takeuchi T, Gumucio DL, Yamada T, *et al.* Genes encoding pancreatic polypeptide and neuropeptiode Y are on human chromosomes 17 and 7. *J Clin Invest* 1986;**77**:1038–41.
13. Ng SY, Gunning P, Eddy R, *et al.* Evolution of the functional

human β-actin gene and its multi-pseudogene family: conservation of noncoding regions and chromosomal dispersion of pseudogenes. *Mol Cell Biol* 1985;**5**:2720–32.

[14] Betsholtz C, Johnsson A, Heldin CH, *et al.* cDNA sequence and chromosomal localization of human platelet-derived growth factor A-chain and its expression in tumour cell lines. *Nature* 1986;**320**:695–9.

[15] Astrin KH, Arredondo-Vega FX, Desnick RJ, Smith M. Studies on the expression of human diaphorases in rat x human hybrids. HGM6. *Cytogenet Cell Genet* 1982;**32**:249.

[16] Baeteman MA, Philip N, Mattei MG, Mattei JF. Clinical, chromosomal and enzymic studies in four cases of rearrangement of chromosome 7. *Clin Genet* 1985;**27**:564–9.

[17] Rabin M, Ferguson-Smith MA, Hart CP, Ruddle FH. Cognate homeo-box loci mapped on homologous human and mouse chromosomes. *Proc Natl Acad Sci USA* 1986;**83**:9104–8.

[18] Naylor SL, Klebe RJ, Shows TB. Argininosuccinic aciduria: assignment of the argininosuccinatelyase gene to the pter-q22 region of human chromosome 7 by bioautography. *Proc Natl Acad Sci USA* 1978;**75**:6159–62.

[19] O'Brien WE, McInnes R, Kalumuck K, Adcock M. Cloning and sequence analysis of cDNA for human argininosuccinate lyase. *Proc Natl Acad Sci USA* 1986;**83**:7211–5.

[20] Spurr NK, Solomon E, Goodfellow P, *et al.* Genetics of the T cell receptor. HGM8. *Cytogenet Cell Genet* 1985;**40**:754.

[21] Morton CC, Duby AD, Murre C, Waldman RA, Shows TB, Seidman JG. The human T-cell receptor beta chain and the T-cell gamma gene are assigned to chromosome 7. HGM8. *Cytogenet Cell Genet* 1985;**40**:703.

[22] Meera Khan P, Wijnen JT, Grzeschik KH. Electrophoretic characterization and genetics of human biliverdin reductase (BLVR;EC.1.3.1.24); assignment of BLVR to the p14-cen region of human chromosome 7 in mouse-human somatic cell hybrids. *Biochem Genet* 1983;**21**:123–33.

[23] Parker M, Jeremiah SJ, Povey S, *et al.* Confirmation of the assignment of human biliverdin reductase to chromosome 7. *Ann Hum Genet* 1984;**48**:57–60.

[24] Shimizu N, Behzadian MA, Shimizu Y. Genetics of cell surface receptors for bioactive polypeptides: binding of epidermal growth factor is associated with the presence of human chromosome 7 in human-mouse cell hybrids. *Proc Natl Acad Sci USA* 1980;**77**:3600–4.

[25] Davies RL, Grosse VA, Kucherlapati R, Bothwell M. Genetic analysis of epidermal growth factor action: assignment of human epidermal growth factor receptor gene to chromosome 7. *Proc Natl Acad Sci USA* 1980;**77**:4188–92.

[26] Kondo I. Regional mapping of the human gene for epidermal growth factor receptor (EGFR). HGM6. *Cytogenet Cell Genet* 1982;**32**:292.

[27] Goodfellow PN, Banting G, Waterfield M, Ozanne B. Assignment of a gene controlling human EGF receptor expression to chromosome 7: confirmation using a species-specific monoclonal antibody. HGM6. *Cytogenet Cell Genet* 1982;**32**:283.

[28] Lin CR, Chen WS, Kruiger W, *et al.* Expression cloning of human.EGF receptor complementary DNA: gene amplification and three related messenger RNA product in A431 cells. *Science* 1984;**224**:843–8.

[29] Spurr NK, Goodfellow PN, Solomon E, Parkar M, Vennstrom B, Bodmer WF. Mapping of cellular oncogenes: erb B on chromosome 7. HGM7. *Cytogenet Cell Genet* 1984;**37**:591.

[30] Sakaguchi AY, Zabel BU, Grzeschik KH, Law ML, Naylor SL. Human proto-oncogene assignments. HGM7. *Cytogenet Cell Genet* 1984;**37**:572–3.

[31] Yang-Feng TL, Schechter AL, Weinberg RA, Francke U. Oncogene from rat neuro/glioblastomas (human gene symbol *NGL*) is located on the proximal long arm of human chromosome 17 and *EGFR* is confirmed at 7p13–q11·2. HGM8. *Cytogenet Cell Genet* 1985;**40**:784.

[32] Zabel BU, Fournier REK, Lalley PA, Naylor SL, Sakaguchi AY. Cellular homologs of the avian erythroblastosis virus *erb-A* and *erb-B* genes are syntenic in mouse but asyntenic in man. *Proc Natl Acad Sci USA* 1984;**81**:4874–8.

[33] Bootsma D, Ruddle FH. Report of the committee on the genetic constitution of chromosomes 2,3,4,5,7,8,10,11 and 12. HGM4. *Cytogenet Cell Genet* 1984;**22**:74–91.

[34] Arfin SM, Cirullo RE, Arredondo-Vega FX, Smith M. Assignment of structural gene for asparagine synthetase to human chromosome 7. *Somat Cell Genet* 1983;**9**:517–31.

[35] Andrulis IL, Chen J, Ray PN. Isolation of human cDNAs for asparagine synthetase and expression in Jensen rat sarcoma cells. *Mol Cell Biol* 1987;**7**:2435–43.

[36] Barton P, Weydert A, Daubas P, Buckingham M, Stone M, Ferguson-Smith MA. Localization of a cluster of myosin heavy chain genes on human chromosome 7 by *in situ* hybridisation. HGM7. *Cytogenet Cell Genet* 1984;**37**:414.

[37] O'Connell C, O'Brien S, Nash WG, Cohen M, ERV3, a full-length human endogenous provirus: chromosome localization and evolutionary relationships. *Virology* 1984;**138**:225–35.

[38] Sykes B, Solomon E. Assignment of a type I collagen structural gene to human chromosome 7. *Nature* 1978;**272**:548–9.

[39] Junien C, Weil D, Myers JC, *et al.* Assignment of the human proα2 (I) collagen structural gene to chromosome 7 by molecular hybridization. HGM7. *Cytogenet Cell Genet* 1982;**32**:289.

[40] Strom CM, Upholt WB. Isolation and characterization of genomic clones corresponding to the human type II procollagen gene. *Nucleic Acids Res* 1984;**12**:1025–38.

[41] Grobler-Rabie A, Brebner D, Vandenplas S, *et al.* Polymorphism of DNA sequences in the human proα2 (I) collagen gene. *J Med Genet* 1985;**22**:182–6.

[42] Brebner D, Grobler-Rabie A, Bester A, Mathew C, Boyd C. Two new polymorphic markers in the human proalpha2 (I) collagen gene. *Hum Genet* 1985;**70**:25–7.

[43] Retief E, Parker ML, Retief AE. Regional chromosome mapping of human collagen genes alpha 2 (I) and alpha 1 (I) (COL1A2 and COL1A1). *Hum Genet* 1985;**69**:304–8.

[44] Fojo A, Lebo R, Shimizu N, *et al.* Localization of multidrug resistance-associated DNA sequences to human chromosome 7. *Somat Cell Mol Genet* 1986;**12**:415–20.

[45] Zengerling S, Tsui LC, Grzeschik KH, Olek K, Riordan JR, Buchwald M. Mapping of DNA markers linked to the cystic fibrosis locus on the long arm of chromosome 7. *Am J Hum Genet* 1987;**40**:228–36.

[46] Trent J, Witkowski CM. Clarification of the chromosomal assignment of the human P-glycoprotein/mdrl gene: possible coincidence with the cystic fibrosis and c-met oncogene. *Cancer Genet Cytogenet* 1987;**26**:187–90.

[47] Danesino C, Gimelli G, Cuoco C, Ciccone MO. Triplex gene dosage effect for β-glucuronidase and possible assignment to band q22 in a partial duplication 7q. *Hum Genet* 1981;**56**:371–3.

[48] Lin FK, Suggs S, Lin CH, *et al.* Cloning and expression of the human erythropoietin gene. *Proc Natl Acad Sci USA* 1985;**82**:7580–4.

[49] Law ML, Cai GY, Lin FK, *et al.* Chromosomal assignment of the human erythropoietin gene and its DNA polymorphism. *Proc Natl Acad Sci USA* 1986;**83**:6920–4.

[50] Watkins PC, Eggy R, Hoffman N, *et al.* Regional assignment of the erythropoietin gene to human chromosome region 7pter→q22. *Cytogenet Cell Genet* 1986;**42**:214–8.

[51] Eiberg H, Mohr J, Schmiegelow K, Nielsen LS, Williamson R. Linkage relationships of paraoxonase (PON) with other markers. Indication of PON-cystic fibrosis synteny. *Clin Genet* 1985;**28**:265–71.

[52] Eiberg H, Schmiegelow K, Tsui LC, *et al.* Cystic fibrosis, linkage with paraoxonase. HGM8. *Cytogenet Cell Genet* 1985;**40**:429.

[53] Tsui LC, Buchwald M, Barker D, *et al.* Cystic fibrosis locus defined by a genetically linked polymorphic DNA marker. *Science* 1985;**230**:1054–7.

[54] Schmiegelow K, Eiberg H, Tsui LC, *et al.* Linkage between the loci for cystic fibrosis and paraoxonase. *Clin Genet* 1986;**29**:374–7.

[55] Cooper CS, Park M, Blair DG, *et al.* Molecular cloning of a new transforming gene from a chemically transformed human cell line. *Nature* 1984;**311**:29–33.

[56] Dean M, Park M, Le Beau MM, *et al.* The human met oncogene is related to the tyrosine kinase oncogenes. *Nature* 1985;**318**:385–8.

[57] Nathans J, Thomas D, Hogness DS. Molecular genetics of human color vision: the genes encoding blue, green, and red pigments. *Science* 1986;**232**:193–202.

[58] Honey NK, Sakaguchi AV, Quinto D, MacDonald RJ, Rutter WJ, Naylor SL. Assignment of the human genes for elastase to chromosome 12, and for trypsin and carboxypeptidase A to chromosome 7. HGM7. *Cytogenet Cell Genet* 1984;**37**:492.

[59] Bowcock AM, Crandall J, Daneshvar L, *et al.* Genetic analysis of cystic fibrosis: linkage of DNA and classical markers in multiplex families. *Am J Hum Genet* 1986;**39**:699–706.

[60] Knowlton RG, Cohen-Haguenauer O, Nguyen VC, *et al.* A polymorphic DNA marker linked to cystic fibrosis is located on chromosome 7. *Nature* 1985;**318**:380–2.

[61] White R, Woodward S, Leppert M, *et al.* A closely linked genetic marker for cystic fibrosis. *Nature* 1985;**318**:382–4.

[62] Wainwright BJ, Scambler PJ, Schmidtke J, *et al.* Localization of cystic fibrosis locus to human chromosome 7cen–q22. *Nature* 1985;**318**:384–5.

[63] Tsui LC, Zengerling S, Willard HF, Buchwald M. Mapping of the cystic fibrosis locus on chromosome 7. *Cold Spring Harbor Symp Quant Biol* 1986;**51**:325–35.

[64] Barker PE, Ruddle FH, Royer HD, Acuto O, Reinherz EL. Chromosomal location of human T-cell receptor gene TiBeta. *Science* 1984;**23**:348–9.

[65] Collins MKL, Goodfellow PN, Dunne MJ, Spurr NK, Solomon E, Owen MJ. A human T-cell antigen receptor beta chain gene maps to chromosome 7. *EMBO J* 1984;**3**:2347–9.

[66] Caccia N, Kronenberg M, Saxe D, *et al.* The T cell receptor β chain genes are located on chromosome 6 in mice and chromosome 7 in humans. *Cell* 1984;**37**:1091–9.

[67] Isobe M, Erikson J, Emanuel BS, Nowell PC, Croce CM. Location of gene for β subunit of human T-cell receptor at band 7q35, a region prone to rearrangements in T cells. *Science* 1985;**228**:580–2.

[68] Morton CC, Duby AD, Eddy RL, Shows TB, Seidman JG. Genes for β chain of human T-cell antigen receptor map to regions of chromosomal rearrangement in T cells. *Science* 1985;**228**:582–5.

[69] Le Beau MM, Diaz MO, Rowley JD, Mak TW. Chromosomal localization of the human T cell receptor β-chain genes. *Cell* 1985;**41**:335.

[70] Palumbo AP, Isobe M, Huebner K, *et al.* Chromosomal localization of a human band 3-like gene to region 7q35–7q36. *Am J Hum Genet* 1986;**39**:307–16.

[71] Daigar S, Hoffman N, Wilden R, Su T. Multiple, independent RFLPs in human DNA detected with a cDNA probe to argininosuccinate synthetase. *Am J Hum Genet* 1984;**36**:736–49.

[72] Beaudet AL, Rosenbloom CL, Spence JE, O'Brien WE, Cole S, Ferrell RE. Gene mapping for cystic fibrosis. HGM8. *Cytogenet Cell Genet* 1985;**40**:579.

[73] Ginsburg D, Zeheb R, Yang AY, *et al.* cDNA cloning of human plasminogen activator-inhibitor from endothelial cells. *J Clin Invest* 1986;**78**:1673–80.

[74] Molawa DT, Schuetz EG, Wrighton SA, *et al.* Complete cDNA sequence for a cytochrome P-450 inducible by glucocorticoids in human liver. *Proc Natl Acad Sci USA* 1986;**83**:5311–5.

[75] Sehgal PB, Zilberstein A, Ruggieri RM, *et al.* Human chromosome 7 carries the β2 interferon gene. *Proc Natl Acad Sci USA* 1986;**83**:5219–22.

[76] Robson EB, Meera Khan P. Report of the committee on the genetic constitution of chromosomes 7, 8 and 9. HGM6. *Cytogenet Cell Genet* 1982;**32**:144–52.

[77] Craig IW, Tolley E, Bobrow M. A preliminary analysis of the segregation of human hydroxyacylcoenzyme A dehydrogenase in human-mouse somatic cell hybrids. HGM3. *Cytogenet Cell Genet* 1976;**12**:115–7.

[78] Pajunen L, Höyhtyä M, Tryggvason K, Kivirikko KI, Myllylä R. Species-specific antibodies in the assignment of the gene for the beta-subunit of human prolyl 4-hydroxylase. HGM8. *Cytogenet Cell Genet* 1985;**40**:719.

[79] Bartels I, Grzeschik KH, Cooper DN, Schmidtke J. Regional mapping of six cloned DNA sequences on human chromosome 7. *Am J Hum Genet* 1986;**38**:280–7.

[80] Cooper D, Smith B, Cooke HJ, Niemann S, Schmidtke J. An esimate of unique DNA sequence heterozygosity in the human genome. *Hum Genet* 1985;**69**:201–15.

[81] Balazs I, Purrello M, Grzeschik KH. Properties of random human cDNA clones homologous to DNA sequences from chromosome 7. HGM7. *Cytogenet Cell Genet* 1984;**37**:407–8.

[82] Jørgensen AL, Bostock CJ, Bak AL. Chromosome-specific subfamilies within human alphoid repetitive DNA. *J Mol Biol* 1986;**187**:185–96.

[83] Waye JS, England SB, Willard HF. Genomic organization of alpha satellite DNA on human chromosome 7: evidence for two distinct alphoid domains on single chromosome. *Mol Cell Biol* 1987;7:349–56.

[84] Estivill X, Schmidtke J, Williamson R, Wainwright B. Chromosome assignment and restriction fragment length polymorphism analysis of the anonymous DNA probe B79a at 7q22 (HGM* assignment D7S13). *Hum Genet* 1986;**74**:320–2.

[85] Scambler PJ, Wainwright BJ, Watson E, *et al.* Isolation of a further anonymous informative DNA sequence from chromosome seven closely linked to cystic fibrosis. *Nucleic Acids Res* 1986;**14**:1951–6.

[86] Buckle VJ, Scambler PJ, Wainwright BJ. Localisation of a sequence, 7C22, showing close linkage to the cystic fibrosis locus. *Cytogenet Cell Genet* 1987;**44**:41–2.

[87] Humphries P, Barton D, McKay AM, Humphries MM, Carritt B. Isolation of a polymorphic segment unique to human chromosome 7 by molecular cloning of hybrid cell DNA. *Mol Gen Genet* 1983;**190**:143–9.

[88] Carritt B. Restriction fragment length polymorphisms in the D7S1 region of human chromosome 7. *Hum Genet* 1986;**72**:83–5.

[89] de la Chapelle A, Ruutu P, Ruutu T, *et al.* The locomotion defect of granulocytes in monosomy 7 involves random locomotion, chemotaxis and chemokinesis. HGM6. *Cytogenet Cell Genet* 1982;**32**:264.

[90] Donald LJ, Wang HS, Holliday NJ, Hamerton JL. Are there growth-rate-controlling factors on chromosomes 7 and 16? HGM6. *Cytogenet Cell Genet* 1982;**32**:268–9.

[91] Alevy YG, Fleischman JB. Immuospecific isolation of a human chromatin fraction from mouse-human hybrid cells. *Mol Immunol* 1980;**17**:275–80.

[92] Taggart RT, Francke U. Mapping of polypeptide genes by two-dimensional gel electrophoresis of hybrid cell extracts. HGM6. *Cytogenet Cell Genet* 1982;**32**:99–110.

[93] Cicurel L, Croce CM. Somatic cell hybrids between mouse peritoneal macrophages and SV40-tranformed human cells. III. Identification of surface antigens coded for by human chromosome 7 and 17. *J Immunol* 1977;**118**:1951–6.

[94] Hood L, Kronenberg M, Hunkapiller T. T cell antigen receptors and the immunoglobulin supergene family. *Cell* 1985;**40**:225–9.

[95] Mak TW, Caccia N, Reis M, *et al.* Genes encoding the alpha, beta, and gamma chains of the human T-cell antigen receptor. *J Infect Dis* 1987;**155**:418–22.

[96] Siu G, Clark SP, Yoshikai Y, *et al.* The human T cell antigen receptor is encoded by variable, diversity, and joining gene segments that rearrange to generate a complete V gene. *Cell* 1984;**37**:393–401.

[97] Riordan JR, Deuchars K, Kartner N, Alon N, Trent J, Ling V. Amplification of P-glycoprotein genes in multidrug-resistant mammalian cell lines. *Nature* 1985;**316**:817–9.

[98] Gerlach JH, Kartner N, Bell DR, Ling V. Multidrug resistance. *Cancer Survey* 1986;**5**:25–46.

[99] Gerlach JH, Endicott JA, Juranka PF, *et al.* Homology between P-glycoprotein and a bacterial haemolysin transport protein suggests a model for multidrug resistance. *Nature* 1986;**324**:485–9.

[100] McKusick VA. *Mendelian inheritance in man.* 6th ed. Baltimore: Johns Hopkins University Press, 1983.

[101] Sillence DO, Senn A, Danks DM. Genetic heterogeneity in osteogenesis imperfecta. *J Med Genet* 1979;**16**:101–16.

[102] Tsipouras P, Ramirez F. Genetic disorder of collagen. *J Med Genet* 1987;**24**:2–8.

[103] Pyeritz RE, McKusick VA. The Marfan syndrome: diagnosis and management. *N Engl J Med* 1979;**300**:772–7.

[104] Byers PH. Siegel RC, Peterson KE, *et al.* Marfan syndrome: abnormal α2 chain in type I collagen. *Proc Natl Acad Sci USA* 1981;**78**:7745–9.

[105] Henke E, Leader M, Tajima S, Pinnell S, Kaufman R. A 38 base pair insertion in the pro α2 (I) collagen gene of a patient with Marfan syndrome. *J Cell Biochem* 1985;**27**:161–74.

[106] Dalgleish R, Williams G, Hawkins JR. Length polymorphism in the pro α2 (I) collagen gene: an alternative explanation in case of Marfan syndrome. *Hum Genet* 1986;**73**:91–2.

[107] Tsipouras P, Børresen AL, Bamforth S, Harper PS, Berg K. Marfan syndrome: exclusion of genetic linkage to the COL1A2 gene. *Clin Genet* 1986;**30**:428–32.

[108] Dalgleish R, Hawkins JR, Keston M. Exclusion of the α2 (I) and α2 (III) collagen genes as the mutant loci in a Marfan syndrome family. *J Med Genet* 1987;**24**:148–51.

[109] Steinman B, Tuderman L, Peltonen L, Martin GR, McKusick VA, Prockop DJ. Evidence for a structural mutation of procollagen type I in a patient with Ehlers-Danlos syndrome type VII. *J Biol Chem* 1980;**255**:8887–93.

[110] Talamo RC, Rosenstein BJ, Berninger RW. Cystic fibrosis. In: Stanbury JB, Wyngaarden JB, Fredrickson DS, Goldstein JL, Brown MS, eds. *The metabolic basis of inherited disease.* 5th ed. New York: McGraw-Hill, 1983:1889–971.

[111] Thompson MW. Genetics of cystic fibrosis. In: Sturgess JM, ed. *Perspectives in cystic fibrosis.* Toronto: Canadian Cystic Fibrosis Foundation, 1980:281–91.

[112] Knowles MR, Stutts MJ, Spock A, Fischer N, Gatzy JT, Boucher RC. Abnormal ion permeation through cystic fibrosis respiratory epithelium. *Science* 1983;**221**:1067–70.

[113] Quinton PM. Chloride impermeability in cystic fibrosis. *Nature* 1983;**301**:421–2.

[114] Frizzell RA, Rechkemmer G, Shoemaker RL, Altered regulation of airway epithelial cell chloride channels in cystic fibrosis. *Science* 1986;**233**:558–60.

[115] Welsh MJ, Liedtke CM. Chloride and potassium channels in cystic fibrosis airway epithelial cells. *Nature* 1986;**322**:467–72.

[116] Buchwald M, Zsiga M, Markiewicz D, *et al.* Linkage of cystic fibrosis to the proα2 (I) collagen gene COL1A2, on chromosome 7. *Cytogenet Cell Genet* 1986;**41**:234–9.

[117] Beaudet AL, Bowcock A, Buchwald M, *et al.* Linkage of cystic fibrosis to two tightly linked DNA markers. Joint report from a collaborative study. *Am J Hum Genet* 1986;**39**:681–93.

[118] White R, Leppert M, O'Connell P, *et al.* Further linkage data on cystic fibrosis: the Utah study. *Am J Hum Genet* 1986;**39**:694–8.

[119] Tsui LC, Buetow K, Buchwald M. Genetic analysis of cystic fibrosis using linked DNA markers. *Am J Hum Genet* 1986;**39**:720–8.

[120] Spence JE, Rosenbloom CL, O'Brien WE, *et al.* Linkage of DNA markers to cystic fibrosis in 26 families. *Am J Hum Genet* 1986;**39**:729–34.

[121] Watkins PC, Schwartz R, Hoffman N, Stanislovitis P, Doherty R, Kilinger K. A linkage study of cystic fibrosis in extended multigenerational pedigrees. *Am J Hum Genet* 1986;**39**:735–43.

[122] Naylor SL, Barnett DR, Buchanan JM, *et al.* Linkage of cystic fibrosis locus and polymorphic DNA markers in 14 families. *Am J Hum Genet* 1986;**39**:707–12.

[123] Farrall M, Watson E, Bates G, *et al.* Further data supporting linkage between cystic fibrosis and the *met* oncogene and haplotype analysis with *met* and pJ3.11. *Am J Hum Genet* 1986;**39**:713–9.

[124] Vitale E, Devoto M, Mastella G, Romeo G. Homogeneity of cystic fibrosis in Italy. *Am J Hum Genet* 1986;**39**:832–6.

[125] Methy L, Kampmann W, Higuchi M, *et al.* Cystic fibrosis: typing 48 German families with linked DNA probes. *Hum Genet* 1987;**75**:359–61.

[125a] Schmidtke J, Krawczak M, Schwartz M, *et al.* Linkage relationships and allelic association of the cystic fibrosis locus and four marker loci. *Hum Genet* 1987;**76**:337–43.

[126] Estivill X, Farrall M, Scambler PJ, *et al.* A candidate for the cystic fibrosis locus isolated by selection for methylation-free islands. *Nature* 1987;**326**:840–5.

[127] Farrall M, Rodeck CH, Stanier P, *et al.* First-trimester prenatal diagnosis of cystic fibrosis with linked DNA probes. *Lancet* 1986;**i**:1402–6.

[128] Spence JE, Buffone GJ, Rosenbloom CL, *et al.* Prenatal diagnosis of cystic fibrosis using linked DNA markers and microvillar intestinal enzyme analysis. *Hum Genet* 1987;**76**:5–10.

[129] Harper PS. DNA prediction in cystic fibrosis. *J Med Genet* 1986;**23**:289–90.

[130] Zackai EH, Breg WR. Ring chromosome 7 with variable phenotypic expression. *Cytogenet Cell Genet* 1973;**12**:40–8.

[131] Wilson MG, Fujimoto A, Shinno NW, Towner JW. Giant satellites or translocation. *Cytogenet Cell Genet* 1973;**12**:209–14.

[132] Friedrich U, Lyngbye T, Øster J. A girl with karyotype 46,XX,del(7)(qter→p15). *Hum Genet* 1975;**26**:161–5.

[133] Nakagome Y, Teramura F, Kataoka K, Hosono F. Mental retardation, malformation syndrome and partial 7p monosomy (45,XX,tdic(7;15)(p21;p11)). *Clin Genet* 1976;**9**:621–4.

[134] Gong BT, Norwood TH, Hoehn H, McPherson E, Hall JG, Hickman R. Chromosome 7 short arm deletion and craniosynostosis, a 7p– syndrome. *Hum Genet* 1976;**35**:117–23.

[135] Marcallo FA, Werneck LC, Pilotto RF, Opitz JM. Hemihypotrophy in a girl with a translocation t(13q;7p). *Eur J Pediatr* 1977;**124**:167–71.

[136] Winsor EJT, Palmer CG, Ellis PM, Hunter JLP, Ferguson-Smith MA. Meiotic analysis of a pericentric inversion inv(7)(p22q32) in a father of a child with a duplication-deletion of chromosome 7. *Cytogenet Cell Genet* 1978;**20**:169–84.

[137] Carnevale A, Frias S, del Castello V. Partial trisomy of the short arm of chromosome 7 due to a familial translocation rcp (7;14) (p11;p11). *Clin Genet* 1978;**14**:202–6.

[138] Moedjono SJ, Funderburk SJ, Sparkes RS. Chromosome 7 short arm interstitial deletion (p14). *Hum Genet* 1978;**44**:51–7.

[139] Crawfurd Md'A, Kessel J, Liberman M, McKeown JA, Mandalia PY, Ridler MAC. Partial monosomy 7 with interstitial deletions in two infants with differing congenital abnormalities. *J Med Genet* 1979;**16**:453–60.

[140] Dhadial RK, Smith MF. Terminal 7p deletion and 1;7 translocation associated with craniosynostosis. *Hum Genet* 1979;**50**:285–9.

[141] Miller M, Kaufman G, Reed G, Bilenker R, Schinzel A. Familial balanced insertional translocation of chromosome 7 leading to offspring with deletion and duplication of the inserted segment, 7p15→7p21. *Am J Hum Genet* 1979;**4**:323–32.

[142] Bianchi DW, Cirillo-Silengo M, Luzatti L, Greenstein RM. Interstitial deletion of the short arm of chromosome 7 without craniosynostosis. *Clin Genet* 1981;**19**:456–61.

[143] Müller U, Staudt F, Hameister H. A patient with intersitial deletion 7(p13→p21). *Ann Genet (Paris)* 1981;**24**:239–41.

[144] Baccichetti C, Attifoni L, Zanardo V. Deletion of the short arm of chromosome 7 without craniosynostosis. *Clin Genet* 1982;**21**:348–55.

[145] Fryns JP, Haspelagh M, Agneessens A, van den Berghe H. De novo partial 2q3 trisomy/distal 7p22 monosomy in a malformed newborn with 7p deletion phenotype and craniosynostosis. *Ann Genet (Paris)* 1985;**28**:45–8.

[146] Marks K, Hill L, Chitham RG, Whitehouse WL. Interstitial deletion of chromosome 7p detected antenatally. *J Med Genet* 1985;**22**:316–8.

[147] Schömig-Springler M, Schmid M, Brosi W, Grimm T. Chromosome 7 short arm deletion, 7p21→pter. *Hum Genet* 1986;**74**:323–5.

[148] Korner H, Agricola H, Bayer H. Translocation t(7p+;13q−) associated with recurrent abortion. *Humangenetik* 1975;**28**: 83–6.

[149] Hatzissevastou-Loukidou H, Bouli-Kalachani A, Malaka-Zafiriou A, Mantalenakis S. Reciprocal balanced translocation of the long arm of chromosome 8 to the short arm of chromosome 7 in a woman with two spontaneous abortions. *Hum Genet* 1985;**70**:379.

[150] de Grouchy J, Veslot J, Bonnette J, Roidat M. A case of ?6p– chromosome aberration. *Am J Dis Child* 1968;**115**:93–9.

[151] Shokeir MHK, Ying LK, Pabello P. Deletion of the long arm of chromosome No 7: tentative assignment of the Kidd (Jk) locus. *Clin Genet* 1973;**4**:360–8.

[152] Harris EL, Wappner RS, Palmer CG, *et al.* 7q deletion syndrome (7q32–qter). *Clin Genet* 1977;**12**:233–8.

[153] Kousseff BG, Hsu LYF, Paciuc S, Hirschhorn K. A partial long arm deletion of chromosome 7: 46,XY,del(7)(q32). *J Med Genet* 1977;**14**:144–7.

[154] Biederman B, Bowen P. Balanced t(8;9)(q12;q33)pat carrier with phenotypic abnormalities attributable to a de novo terminal deletion of the long arm of chromosome 7. *Hum Genet* 1978;**41**:101–7.

[155] Francke U. Hageman (factor XII) locus on 7q. *Hum Genet* 1978;**45**:363–7.

[156] Turleau C, de Grouchy J, Perignon F, Lenoir G. Monosomy 7qter. *Ann Genet (Paris)* 1979;**22**:242–4.

[157] Friedrich U, Osterballe O, Stenbjerg S, Jorgensen J. A girl with karyotype 46,XX,del(7)(pter→q32). *Hum Genet* 1979;**51**:231–5.

[158] Bernstein R, Dawson B, Morcom G, Wagner J, Jenkins T. Two unrelated children with distal long arm deletion of chromosome 7: clinical features, cytogenetic and gene marker studies. *Clin Genet* 1980;**17**:228–37.

[159] Taysi K, Burde RM, Rohrbaugh JR. Terminal long arm deletion of chromosome 7 and retinocoroidal coloboma. *Ann Genet (Paris)* 1982;**25**:159–61.

[160] Young RS, Weaver DD, Kukolich MK, *et al.* Terminal and interstitial deletions of the long arm of chromosome 7: a review with five new cases. *Am J Med Genet* 1984;**17**:437–50.

[161] Reynolds JD, Golden WL, Zhang Y, Hiles DA. Ocular abnormalities in terminal deletions of the long arm of chromosome seven. *J Pediatr Ophthalmol Strabismus* 1984;**21**:28–32.

[162] Hara S, Yamada T, Nakai H, Ohtani A, Mizuno K. A case of partial 5q trisomy associated with partial 7q monosomy. *Br J Ophthalmol* 1986;**70**:630–3.

[163] Valentine H, Sergovich F. A syndrome associated with interstitial deletion of chromosome 7. *Birth Defects* 1979;**XIV**(3B):261–2.

[164] Seabright M, Lewis GM. Interstitial deletion of chromosome 7 detected in three unrelated patients. *Hum Genet* 1978;**42**:223–6.

[165] Klep-de-Pater JM, Bijlsma JB, Bleecker-Wagemakers EM, de France HF, de Vries-Ekkers CMAM. Two cases with different deletions of the long arm of chromosome 7. *J Med Genet* 1979;**16**:151–4.

[166] Gibson J, Ellis PM, Forsyth JS. Interstitial deletion of chromosome 7: a case report and review of literature. *Clin Genet* 1982;**22**:256–65.

[167] Pfeiffer RA. Interstitial deletion of a chromosome 7 (q11·2q22·1) in a child with splithand/splitfoot malformation. *Ann Genet (Paris)* 1984;**27**:45–8.

[168] Ayraud N, Rovinski J, Lambert JC, Galiana A. Interstitial deletion of the long arm of chromosome 7 in a leprechaun infant. *Ann Genet (Paris)* 1976;**19**:265–8.

[169] Higginson G, Weaver DD, Magenis RE, Prescott GH, Haag C, Hepburn DJ. Interstitial deletion of the long arm of chromosome no 7 (7q−) in an infant with multiple anomalies. *Clin Genet* 1976;**10**:307–12.

[170] Dennis NR, Neu RL, Bannermann RM. A partial 7q monosomy in an infant with multiple anomalies. *Am J Hum Genet* 1977;**29**:37A.

[171] Franceschini P, Silengo MC, Davi GF, Santoro MA, Prandi G, Fabris C. Interstitial deletion of the long arm of chromosome 7, 46,XX,del(7)(pter→2200::3200→qter). *Hum Genet* 1978; **44**:345–8.

[172] Johnson EW, Kennell JH, Macintyre MN, Van Dyke DL. An interstitial deletion on 7q in a female. *Am J Hum Genet* 1978;**30**:84A.

[173] Serum L. Interstitial deletion of the long arm of chromosome 7. *Hum Genet* 1980;**54**:19–23.

[174] Abuelo DN, Padre-Mendoza T. Cat-like cry and mental retardation owing to 7q interstitial deletion (7q22→7q32). *J Med Genet* 1982;**19**:473–8.

[175] Martin-Point B, Pilczer C, Dandine M, Tamboise A. De novo interstitial deletion of the long arm of chromosome 7: 46,XY, del(7)(q23;q32). *Ann Genet (Paris)* 1985;**28**:251–3.

[176] Nielson KB, Egede F, Mouridsen I, Mohr J. Familial partial 7q monosomy resulting from segregation of an insertional chromosome rearrangement. *J Med Genet* 1979;**16**:461–6.

[177] Stallard R, Juberg RC. Partial monosomy 7q syndrome due to distal interstitial deletion. *Hum Genet* 1981;**57**:210–3.

[178] Novales MA, Fernandez-Novoa C, Hevia A, San Martin V, Gelera H. Partial trisomy for the long arm of chromosome 7. Case report and review. *Hum Genet* 1982;**62**:378–81.

[179] Grace E, Sutherland GR, Stark GO, Bain AD. Partial trisomy of 7q resulting from a familial translocation. *Ann Genet (Paris)* 1973;**16**:51–4.

[180] Berger R, Derre J, Opitz MA. Les trisomies partielles du bras long du chromosome 7. *Nouv Presse Med* 1974;**3**:1801–4.

[181] Serville F, Broustet A, Sandler B, Bourdeau MJ, Leloup M. Trisomie 7q partielle. *Ann Genet (Paris)* 1975;**18**:67–70.

[182] Carpentier S, Rethoré MO, Lejeune J. Trisomie partielle 7q par translocation familiale t(7;12)(q22;q24). *Ann Genet (Paris)* 1972;**15**:283–6.

[183] Alfi OS, Donnel GN, Kramer SL. Partial trisomy of the long arm of chromosome No 7. *J Med Genet* 1973;**10**:187–9.

[184] Vogel W, Siebers JW, Reinwein H. Partial trisomy 7q. *Ann Genet (Paris)* 1973;**16**:277–80.

[185] Al Saadi A, Moghadan HA. Partial trisomy of the long arm of chromosome 7. *Clin Genet* 1976;**9**:250–4.

[186] Berger R, Ture C, Wachter H, Begue G. Partial 7q trisomy. *Clin Genet* 1977;**11**:39–42.

[187] Newton MS, Cunningham C, Jacob PA, Price WH, Fraser IA. Chromosome survey of a hospital for the mentally subnormal. Part II. Autosomal abnormalities. *Clin Genet* 1972;**3**:226–48.

[188] Bass HN, Crandall BF, Marcy SM. Two different chromosome abnormalities resulting from a translocation carrier father. *J Pediatr* 1973;**83**:1034–8.

[189] Moric-Petrovic S, Laca Z, Krajgher A. Translocation réciproque dans la famille de deux proposants avec une trisomie partielle du chromosome 7q. *Ann Genet (Paris)* 1976;**19**:133–6.

[190] Schmid M, Wolf J, Nestler H, Krone W. Partial trisomy for the long arm of chromosome 7 due to familial balanced translocation. *Hum Genet* 1979;**49**:283–9.

[191] Schinzel A, Tonz O. Partial trisomy 7q and probable partial

monosomy of 5p in the son of a mother with a reciprocal translocation between 5p and 7q. *Hum Genet* 1979;**53**:121–4.
[192] Felding I, Mitelman F. A child with partial trisomy 7 and 20 inherited from the mother. *Hereditas* 1979;**91**:91–5.
[193] Turleau C, Rossier A, de Montis G, Roubin M, Chavin-Colin F, de Grouchy J. Trisomie partielle 7q. Un ou deux syndrome? A propos d'une nouvelle observation. *Ann Genet (Paris)* 1976;**19**:37–42.
[194] Klasen M, Schmid M, Hansmann I, Grimm T. Partial trisomy 7q in two siblings. *Ann Genet (Paris)* 1983;**26**:100–2.
[195] Madan K, Seabright M, Lindenbaum RH, Bobrow M. Paracentric inversions in man. *J Med Genet* 1984;**21**:407–12.
[196] Schmid M, Haaf T, Zorn M. Paracentric inversions in human chromosome 7. *Hum Genet* 1986;**74**:197–9.
[197] Shimba H, Ohtaki K, Tanabe K, Sofuni T. Paracentric inversion of a human chromosome 7. *Hum Genet* 1976;**31**:1–7.
[198] Canki N, Dutrillaux B. Two cases of familial paracentric inversion in man associated with sex chromosome anomaly. *Hum Genet* 1979;**47**:261–8.
[199] Faed MJM, Robertson J, Lamont MA. A cytogenetic survey of men being investigated for subfertility. *J Reprod Fertil* 1979;**56**:209–16.
[200] Ridler MAC, Sutton SD. A case of a paracentric inv(7)(q11q22). Prenatal detection and counselling. *Prenat Diagn* 1981;**1**:81–4.
[201] Walker J, Lindenbaum RH. A four-break rearrangement of chromosome 2 and 13 with inversion and insertion, resulting in pure trisomy for the region 7q11.2 to q21.1. *Clin Genet* 1981;**20**:339–400.
[202] Hoo JJ, Lorenz R, Fischer A, Furhmann W. Tiny interstitial duplication of proximal 7q in association with a maternal paracentric inversion. *Hum Genet* 1982;**62**:113–6.
[203] Orye E, Van Bever H. Paracentric inversions: two new familial cases, inv(7) (q22q11) and inv(11) (q23q13). *J Med Genet* 1983;**20**:231.
[204] Stetten G, Rock JA. A paracentric chromosome inversion associated with repeated early prenatal wastage. *Fertil Steril* 1983;**40**:124–6.
[205] Callen DF, Woollatt E, Sutherland GR. Paracentric inversions in man. *Clin Genet* 1985;**27**:87–92.
[206] Martin RH. Sperm chromosome analysis in a man heterozygous for a paracentric inversion of chromosome 7 (q11q22). *Hum Genet* 1986;**73**:97–100.
[207] Watt JL, Ward K, Couzin DA, Stephen GS, Hill A. A paracentric inversion of 7q illustrating a possible interchromosomal effect. *J Med Genet* 1986;**23**:341–4.
[208] Spence JE, Perciaccante RG, Greig GM, *et al.* Uniparental disomy as a mechanism for human genetic disease. Submitted.
[209] Rowley JD, Golomb HM, Vardiman JW. Nonrandom chromosome abnormalities in acute leukemia and dysmyelopoietic syndromes in patients with previously treated malignant disease. *Blood* 1981;**58**:759–67.
[210] Whang-Peng J, Knutsen T, O'Donell JF, Brereton HD. Acute nonlymphocytic leukemia and acute myeloproliferative syndrome following radiation therapy for non-Hodgkin's lymphoma and chronic lymphocytic leukemia. *Cancer* 1979;**44**:1592–600.
[211] Zaccaria A, Rosti G, Testoni N, Mazza P, Cantora N, Tura S. Acute nonlymphocytic leukemias and dysmyelopoietic syndromes in patients treated for Hodgkin's lymphoma. *Cancer Genet Cytogenet* 1983;**9**:217–27.
[212] Pedersen-Bjergaard J, Philip P, Mortensen B, *et al.* Acute nonlymphocytic leukemia, preleukemia and acute myeloproliferative syndrome secondary to treatment of other malignant diseases. Clinical and cytogenetic results of in vitro culture of bone marrow and HLA typing. *Blood* 1981;**57**:712–23.
[213] Pedersen-Bjergaard J, Philip P, Tinggaard Pedersen N, *et al.* Acute nonlymphocytic leukemia, preleukemia and acute myeloproliferative syndrome secondary to treatment of other malignant diseases. II. Bone marrow cytology, cytogenetics, results of HLA typing, response to antileukemic chemotherapy, and survival in a total series of 55 patients. *Cancer* 1984;**54**:452–62.
[214] Arthur DC, Bloomfield CD. Banded chromosome analysis in patients with treatment associated acute nonlymphocytic leukemia. *Cancer Genet Cytogenet* 1984;**12**:189–212.
[215] Cyger M, Forest L, Vuong TE, *et al.* Therapy-induced preleukaemia in patients treated for Hodgkin's lymphoma: clinical and therapeutic relevance of sequential chromosome banding studies. *Br J Haematol* 1984;**58**:61–9.
[216] Groupe Français de Cytogénétique Hématologique. Chromosome analysis of 63 cases of secondary nonlymphoid blood disorders. A cooperative study. *Cancer Genet Cytogenet* 1984;**12**:95–105.
[217] Van den Berghe H, Vermaelen K, Mecucci C, Barbieri D, Tricot G. The 5q– anomaly. *Cancer Genet Cytogenet* 1985;**17**:189–255.
[218] Sandberg AA, Morgan R, Hecht BK, Hecht F. Translocation (1;7) (p11;p11): a new myeloproliferative hematologic entity. *Cancer Genet Cytogenet* 1985;**18**:199–206.
[219] Levin M, Le Coniat M, Bernheim A, Berger R. Complex chromosomal abnormalities in acute nonlymphocytic leukemia. *Cancer Genet Cytogenet* 1986;**22**:113–9.
[220] Benitez J, Carbonell F, Ferro T, Prieto F, Fayos JS. Cytogenetic studies in 18 patients with secondary blood disorders. *Cancer Genet Cytogenet* 1986;**22**:309–17.
[221] Mitelman F, Nilsson PG, Brandt L, Alimena G, Gastaldi R, Dallapiccola B. Chromosome pattern, occupation, and clinical features in patients with acute nonlymphocytic leukemia. *Cancer Genet Cytogenet* 1981;**4**:197–214.
[222] Golomb H, Alimena G, Rowley J, Vardiman J, Testa J, Sovik C. Correlation of occupation and karotype in adults with acute nonlymphocytic leukemia. *Blood* 1982;**60**:404–11.
[223] Fourth International Workshop on Chromosomes in Leukemia, 1982. Correlation of karyotype and occupational exposure to potential mutagenic and carcinogenic agents in acute nonlymphocytic leukemia. *Cancer Genet Cytogenet* 1984;**11**:326–31.
[224] Scheres JM, Hustinx TW, Holdrinet RS, Geraedts JP, Hagemeijer A, van der Blij-Philipsen M. Translocation 1;7 in dyshematopoiesis: possibly induced with a nonrandom geographic distribution. *Cancer Genet Cytogenet* 1984;**12**:283–94.
[225] Rowley JD. Chromosome abnormalities in human leukemia as indicators of mutagenic exposure. *Carcinog Compr Surv* 1985;**10**:409–18.
[226] Fourth International Workshop on Chromsomes in Leukemia, 1982. Abnormalities of chromosome 7 resulting in monosomy or in deletion of the long arm (7q–): review of translocations, breakpoints, and associated abnormalities. *Cancer Genet Cytogenet* 1984;**11**:300–3.
[227] Woods WG, Nesbit ME, Buckley J, *et al.* Correlation of chromosome abnormalities with patient characteristics, histologic subtype, and induction success in children with acute nonlymphocytic leukemia. *J Clin Oncol* 1985;**3**:3–11.
[228] Dewald GW, Kyle RA, Hicks GA, Greipp PR. The clinical significance of cytogenetic studies in 100 patients with multiple myeloma, plasma cell leukemia, or amyloidosis. *Blood* 1985;**66**:380–90.
[229] Yunis JJ, Rydell RE, Oken MM, Arnesen MA, Mayer MG, Lobell M. Refined chromosome analysis as an independent prognostic indicator in de novo myelodysplastic syndromes. *Blood* 1986;**67**:1721–30.
[230] Aurias A, Dutrillaux B, Buriot D, Lejeune J. High frequencies of inversions and translocation of chromosome 7 and 14 in ataxia telangiectasia. *Mutat Res* 1980;**69**:369–74.
[231] Sheres JM, Hustinx TW, Weemaes CM. Chromosome 7 in ataxia-telangiectasia. *J Pediatr* 1980;**97**:440–1.
[232] Weemaes CM, Taalman RD, Van Munster PJ, Hustinx TW, Bakkeren JA, Scheres JM. Immunologische en cytogenetische aspecten van ataxia telangiectasia. *Tijdschr Kindergeneeskd* 1984;**52**:164–9.

[233] Trent JM, Durie BG, Daviea JR, Veomett I. Cytogenetic and clinical assessment of six patients with erythroleukemia. *Anticancer Res* 1983;**3**:111–6.

[234] Werner-Favre E, Cabrol C, Beris P, Engel E. Isochromosome 21 and other chromosomal abnormalities in a patient with erythroleukemia. *Ann Genet (Paris)* 1983;**26**:240–2.

[235] O'Conor GT Jr, Wyandt HE, Innes DJ, Normansell DE, Hess CE. Gamma heavy chain disease: report of a case associated with trisomy of chromosome 7. *Cancer Genet Cytogenet* 1985;**15**:1–5.

[236] Tayssier JR, Sadrin R, Nou JM, *et al.* Trisomy 7 in a lung carcinoid tumor: precocious index of malignant transformation? *Cancer Genet Cytogenet* 1985;**15**:277–82.

[237] Atkin NB, Baker MC. Chromosome study of five cancers of the prostate. *Hum Genet* 1985;**70**:359–64.

[238] Sandberg AA, Berger CS, Haddad FS, Kerr D, Hecht F. Chromosome change in transitional cell carcinoma of ureter. *Cancer Genet Cytogenet* 1986;**19**:335–40.

[239] Psihramis KE, Althausen AF, Yoshida MA, Prout GR Jr, Sandberg AA. Chromosome abnormalities suggestive of malignant transformation in bilateral renal oncocytoma. *J Urol* 1986;**136**:892–5.

[240] McCulloch PB, Dent PB, Hayes PR, Liao SK. Common and individually specific chromosomal characteristics of cultured human melanoma. *Cancer Res* 1976;**36**:398–404.

[241] Bertrand S, Leftheriotis E, Dore JF. Anomalies chromosomiques numeriques et structurales dans 36 lignees de melanome malin humain. *C R Acad Sci [D] (Paris)* 1982;**294**:459–62.

[242] Becher R, Gibas Z, Karakousis C, Sandberg AA. Nonrandom chromosome changes in malignant melanoma. *Cancer Res* 1983;**43**:5010–6.

[243] Balaban GB, Herlyn M, Clarke WH Jr, Nowell PC. Karyotypic evolution in human malignant melanoma. *Cancer Genet Cytogenet* 1986;**19**:113–22.

[244] Lee CL, Kamra OP. The pattern of radiation induced transmissible aberrations in a human cell culture. *Hum Genet* 1981;**57**:380–4.

[245] Wallace C, Bernstein R, Pinto MR. Non-random in vitro 7;14 translocations detected in a routine cytogenetic series: 12 samples and their possible significance. *Hum Genet* 1984;**66**:157–61.

[246] Diehl V, Pfreundschuh M, Fonatsch C, *et al.* Phenotypic and genotypic analysis of Hodgkin's disease derived cell lines: histological and clinical implications. *Cancer Surv* 1985;**4**: 339–419.

[247] de la Chapelle A, Benger R. Report of the committee on chromosome rearrangements in neoplasia and on fragile sites. (HGM7) *Cytogenet Cell Genet* 1984;**37**:274–311.

[248] Sutherland GR, Hecht F. *Fragile sites on human chromosomes.* New York: Oxford University Press, 1985.

[249] Yunis JJ, Soreng AL. Constitutive fragile sites and cancer. *Science* 1984;**226**:1199–204.

[250] Glover TW, Berger C, Coyle J, Echo B. DNA polymerase alpha inhibition by aphidicolin induces gaps and breaks at common fragile sites in human chromosomes. *Hum Genet* 1984;**67**:136–42.

[251] Barbi G, Steinbach P, Vogel W. Non-random distribution of methotrexate-induced aberrations on human chromosomes. Detection of further folic acid sensitive fragile sites. *Hum Genet* 1984;**68**:290–4.

[252] Hecht F. Rare, polymorphic, and common fragile sites: a classification. *Hum Genet* 1986;**74**:207–8.

[253] Koprowski H, Herlyn M, Balaban G, Parmiter A, Ross A, Nowell P. Expression of the receptor for epidermal growth factor correlates with increase dosage of chromosome 7 in malignant melanoma. *Somat Cell Mol Genet* 1985;**11**:297–302.

[254] Shimizu N, Kondo I, Gamou S, Behzadian MA, Shimizu Y. Genetic analysis of hyperproduction of epidermal growth factor receptors in human epidermoid carcinoma A431 cells. *Somat Cell Mol Genet* 1984;**10**:45–53.

[255] Merlino GT, Ishii S, Whang-Peng J, *et al.* Structure and localization of genes encoding aberrant and normal epidermal growth factor receptor RNAs from A431 human carcinoma cells. *Mol Cell Biol* 1985;**5**:1722–34.

[256] Gill GN, Weber W, Thompson DM, *et al.* Relationship between production of epidermal growth factor receptors, gene amplification, and chromosome 7 translocation in variant A431 cells. *Somat Cell Mol Genet* 1985;**11**:309–18.

[257] Hunts JH, Shimizu N, Yamamoto T, *et al.* Translocation chromosome 7 of A431 cells contains amplification and rearrangement of EGF receptor gene responsible for production of variant mRNA. *Somat Cell Mol Genet* 1985;**11**:477–84.

[258] Korc M, Meltzer P, Trent J. Enhanced expression of epidermal growth factor receptor correlates with alterations of chromosome 7 in human pancreatic cancer. *Proc Natl Acad Sci USA* 1986;**83**:5141–4.

[259] Henn W, Blin N, Zang KD. Polysomy of chromosome 7 is correlated with overexpression of the erbB oncogene in human glioblastoma cell lines. *Hum Genet* 1986;**74**:104–6.

[260] Brito-Babapulle V, Matutes E, Parreira L, Catovsky D. Abnormalities of chromosome 7q and Tac expression in T cell leukemias. *Blood* 1986;**67**:516–21.

[261] Zech L, Godal T, Hammaratrom L, *et al.* Specific chromosome markers involved with chronic T lymphocyte tumors. *Cancer Genet Cytogenet* 1986;**21**:67–77.

[262] Cooper CS, Blair DG, Oskarsson MK, Eader LA, Vande Woude GF. Characterization of human transforming genes from chemically-transformed, teratocarcinoma, and pancreatic carcinoma cell lines. *Cancer Res* 1984;**44**:1–10.

[263] Park M, Dean MN, Cooper CS, *et al.* Mechanism of met oncogene activation. *Cell* 1986;**45**:895–904.

[264] Tempest PR, Reeves BR, Spurr NK, Rance AJ, Chan AM, Brookes P. Activation of the met oncogene in the human MNNG-HOS cell line involves a chromosomal rearrangement. *Carcinogenesis* 1986;**7**:2051–7.

[265] Croce CM, Huebner K, Girardi AJ, Koprowski H. Genetics of cell transformation by simian virus 40. *Cold Spring Harbor Symp Quant Biol* 1975;**39**:335–43.

[266] Rabin M, Uhlenbeck OC, Steffensen DM, Mangel WF. Chromosome sites of integration of simian virus 40 DNA sequences mapped by in situ hybridization in two transformed hybrid cell lines. *J Virol* 1984;**49**:445–51.

[267] de la Chapelle A, Vuopio P, Sanger R, Teesdale Ph. Monosomy-7 and the Colton blood group. *Lancet* 1975;**ii**:871.

[268] Mohr J, Eiberg H. Colton blood groups: indication of linkage with the Kidd (Jk) system as support for assignment to chromosome 7. *Clin Genet* 1977;**11**:372–4.

[269] Kedes LH. Histone genes and histone messengers. *Annu Rev Biochem* 1979;**48**:837–70.

[270] Heintz N, Zernik M, Roeder RG. The structure of the human histone genes: clustered but not tandemly repeated. *Cell* 1981;**24**:661–8.

[271] Sierra F, Lightler A, Marashi F, *et al.* Organization of the human histone genes. *Proc Natl Acad Sci USA* 1982;**79**:1795–9.

[272] Hereford LM, Fahrner K, Woodford J Jr, Rosbash M, Kaback DB. Isolation of yeast histone genes H2A and H2B. *Cell* 1979;**18**:1261–71.

[273] Sittman DB, Chiu IM, Pan CJ, Cohn RH, Kedes LH, Marzlouff WF. Isolation of two clusters of mouse histone genes. *Proc Natl Acad Sci USA* 1981;**78**:4078–82.

[274] Hentschel CC, Birnstiel ML. The organization and expression of histone gene families. *Cell* 1981;**25**:301–13.

[275] Harvey RP, Wells JRE. Isolation of a genomic clone containing chicken histone genes. *Nucleic Acids Res* 1979;**7**:1787–98.

[276] Engel JD, Dodgson JB. Histone genes are clustered but not tandemly repeated in the chicken genome. *Proc Natl Acad Sci USA* 1981;**78**:2856–60.

[277] Yu LC, Szabo P, Borun TW, Prensky W. The localization of the genes for histone H4 in human chromosomes. *Cold Spring Harbor Symp Quant Biol* 1977;**42**:1101–5.

[278] Chandler ME, Kedes LH, Cohn RH, Yunis JJ. Genes coding for histone proteins in man are located on the distal end of the long arm of chromosome 7. *Science* 1979;**205**:908–10.
[279] Green L, Van Antwerpen R, Stein J, *et al.* A major human histone gene cluster on the long arm of chromosome 1. *Science* 1984;**226**:838–40.
[280] Lathrop GM, Farrall M, O'Connell P, *et al.* Refined linkage map of chromosome 7 in the region of the cystic fibrosis gene. *Am J Hum Genet* (in press).
[281] Barker D, Green P, Knowlton R, *et al.* A genetic linkage map of 63 chromosome 7 markers. *Proc Natl Acad Sci USA* 1987;**84**:8006–10.
[282] Searle AG, Peters J, Lyon MF, Evans EP, Edwards JH, Buckle VJ. Chromosome maps of man and mouse III. *Genomics* 1987;**1**:3–18.
[283] Dean M, Kozak C, Robbins J, Callahan R, O'Brien S, Vande Woude GF. Chromosomal localization of the met proto-oncogene in the mouse and cat genome. *Genomics* 1987;**1**: 167–73.

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Genetic markers on chromosome 7.

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J Med Genet 1988 25: 294-306
doi: 10.1136/jmg.25.5.294