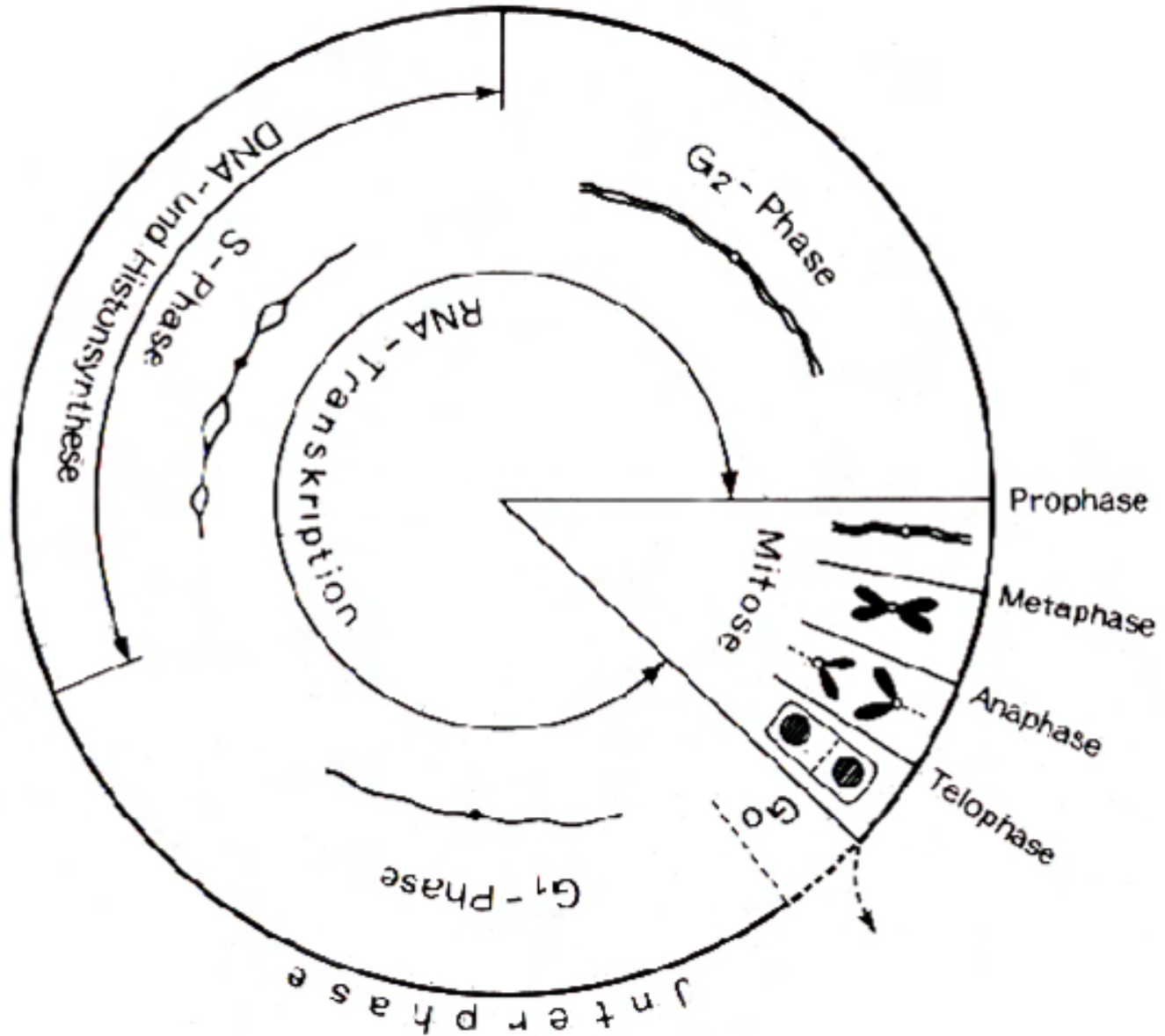


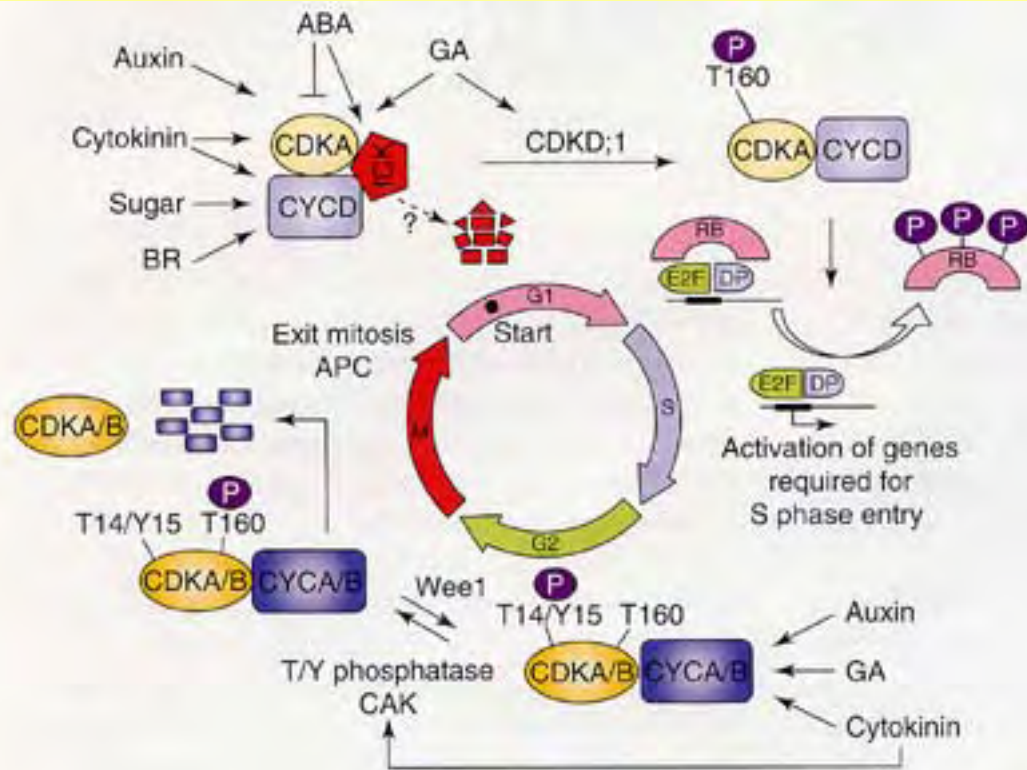
Classical & Molecular Cytogenetics – Selected Topics

1. Cytogenetics; mitotic cell cycle; chromosome structure & morphology (*V. faba*, standard +/- colchicine)
2. Meiosis (cereals: aneuploids, B chromosomes; *V. faba*)
3. Chromosome mutagenesis (incl. apoptosis in interspecific hybrids and gametocidal effects)
4. Karyotype analysis by chromosomal length differentiation *via* banding techniques (G, R, C, T, N, AgNO₃, RE, Q..., mobile NORs, nucleolar dominance)
5. Differential labelling of chromosomes by base analogues (cell cycle, replication patterns; SCE)
6. Mechanisms of karyotype evolution (ploidy level; primary & secondary rearrangements; chromosome number alterations, incl. fusion/fission and B-B-F-cycles; too long/too short chromosomes)
7. In situ hybridisation & immunostaining (FISH, GISH, PRINS, Painting & applications)
8. Microdissection, flow-sorting & applications
9. Telomeres & Centromeres - prerequisites for eukaryotic chromosomes
10. Heterochromatin & Euchromatin, features and marks
11. Interphase chromosome organisation (homologous pairing; sister chromatid cohesion, 'Rabl' orientation - mirror image orientation; meristematic vs. differentiated tissues; evolutionary conservation)

Cell cycle, chromosome structure and chromosome morphology

Mitotic cell cycle

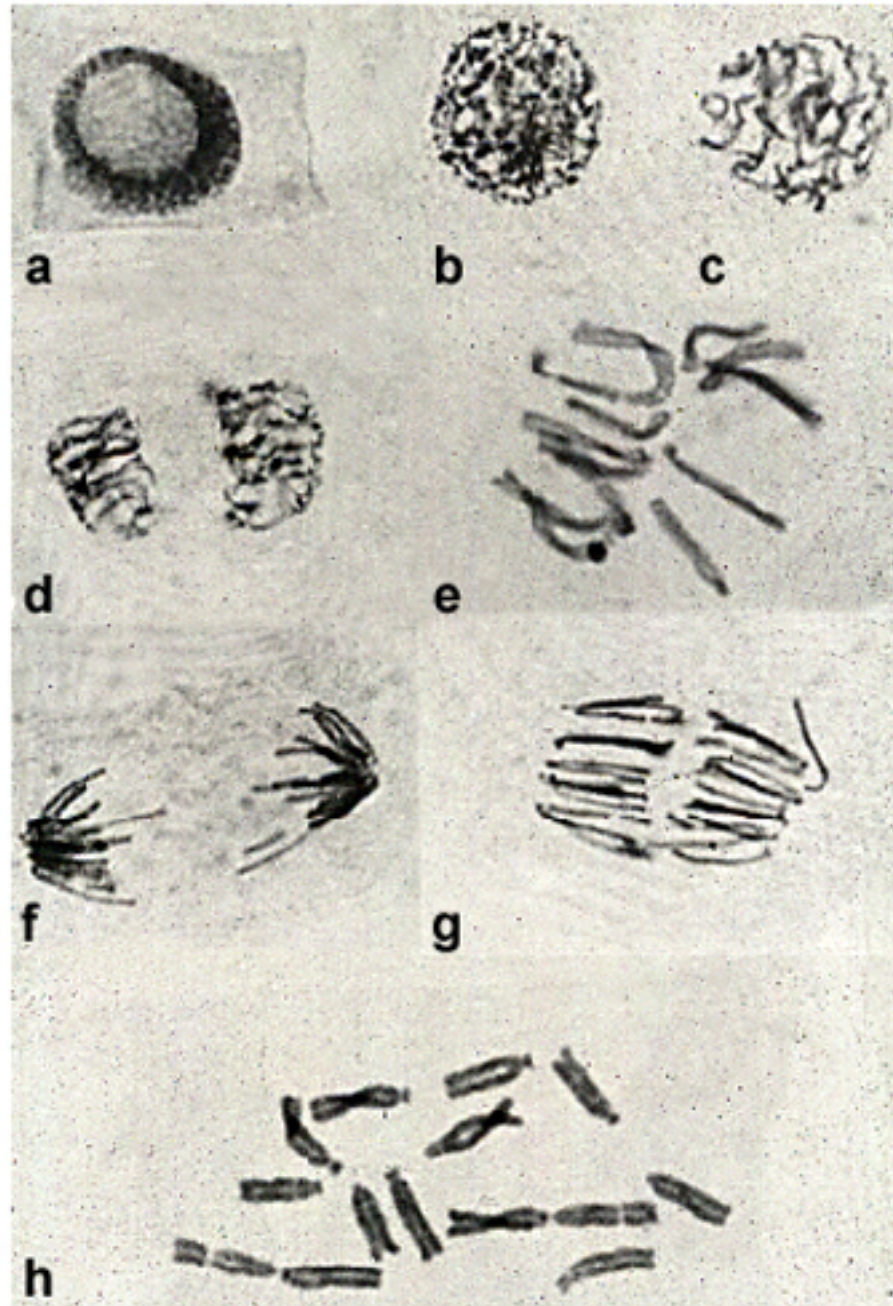




TRENDS in Plant Science

Fig. 1. Model for G1-S and G2-M transitions in plants based on results obtained in plants and on parallels with the mammalian cell-cycle control. During G1, several growth factors, such as auxin, cytokinin, abscisic acid (ABA), gibberellin (GA), brassinosteroids (BR) and sugar regulate the expression of D-type cyclins (CycD) and their catalytic subunit, cyclin-dependent kinase A (CDKA). Activation of the CDKA-CycD complex requires dissociation of the CDK inhibitory protein (ICK), the transcription of which is induced by the stress-responsive hormone ABA and phosphorylation of the Thr160 residue of CDKA by the CDK-activating kinase, CDKD;1, which is upregulated by GA. The active CDKA-CycD complex initiates the phosphorylation of retinoblastoma protein (RB) during late G1 phase, thereby releasing the E2F- Δ P complex that promotes the transcription necessary for progression into S phase. As mitotic activators auxin, cytokinin and GA also regulate the kinase activity of A- and B-type CDKs by activating the transcription of CDKs and of A- and B-type cyclins. The G2-M transition is associated with an activating Thr160 phosphorylation of CDK by a CDK-activating kinase (CAK) and by dephosphorylation of the inhibitory Tyr phosphorylation that is induced by cytokinin. A ubiquitin-dependent degradation pathway targets B-type cyclins for proteolysis by the anaphase-promoting complex (APC) at the metaphase-anaphase transition, thereby activating the exit from mitosis.

Cell cycle stages in field bean meristem





Metacentric



Sub-metacentric

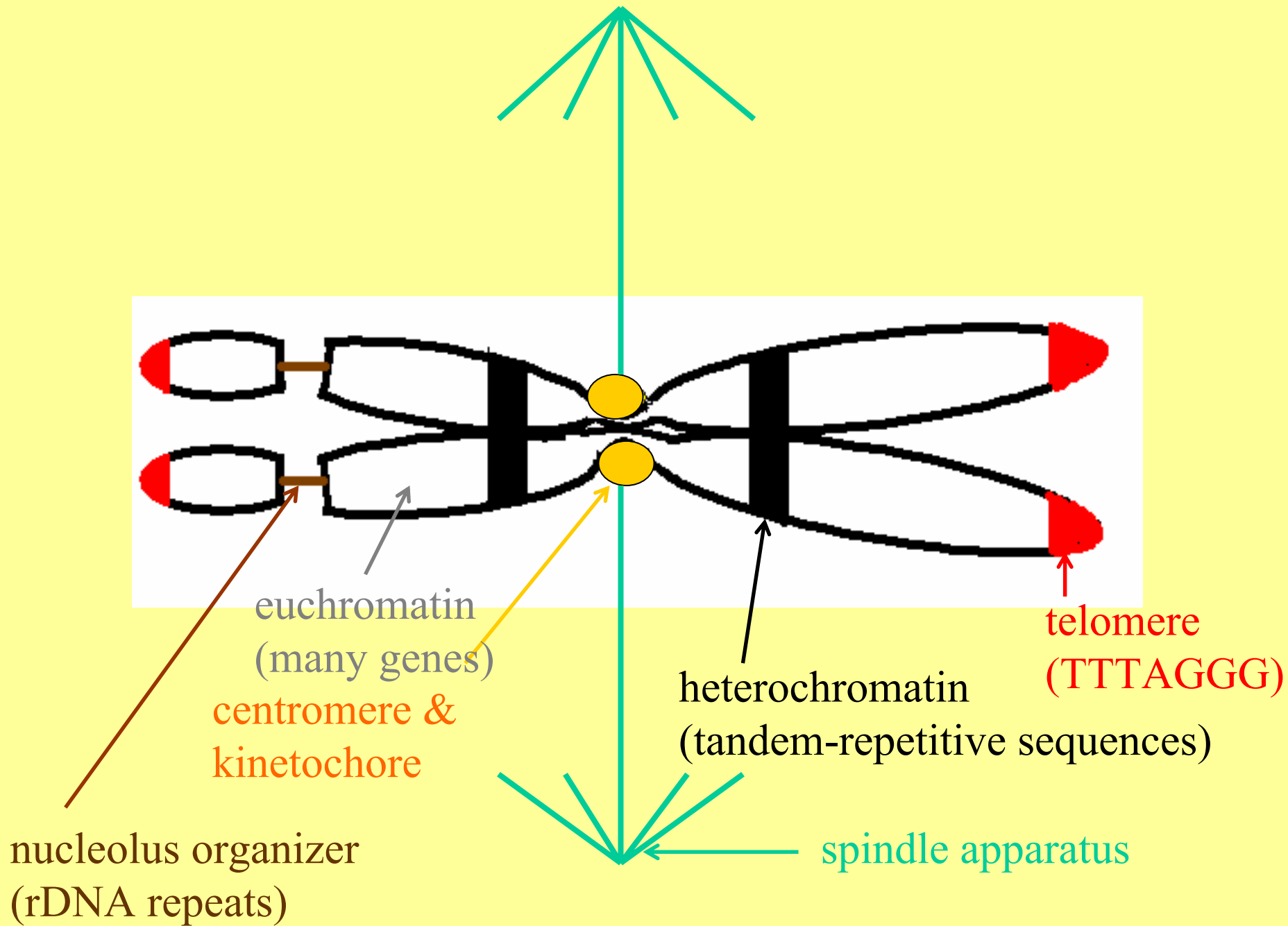


Acrocentric satellited

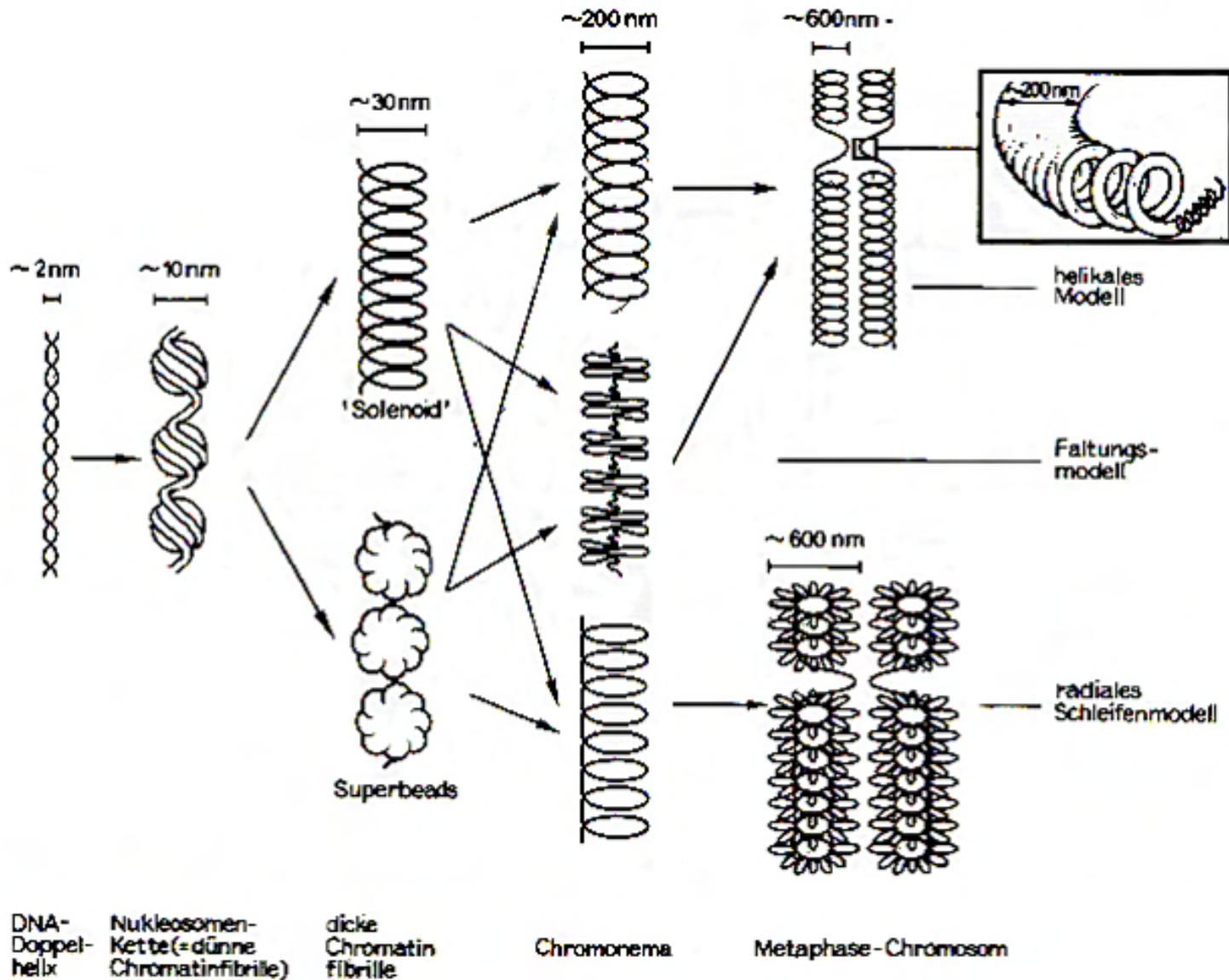


Telocentric

Chromosomes with centromeres at different positions



Hierarchic levels of chromosome structure



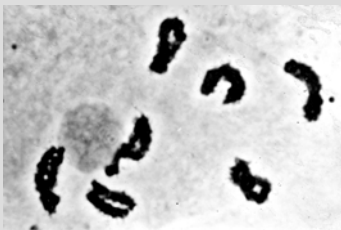
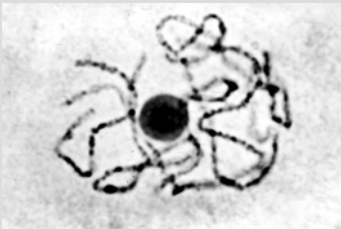
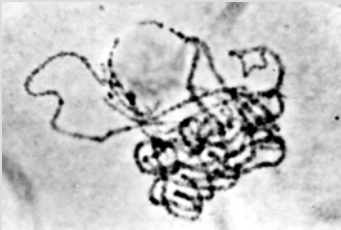
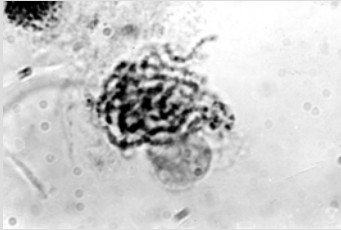
Vicia faba Standard karyotype



ACB



Microsporogenesis in barley (7 chromosome pairs)



MEIOTIC NUCLEAR DIVISIONS

Scheme for 2 chromosome pairs

Leptotene

Individual chromosomes become visible as thin structures.



Zygotene

Homologous chromosomes start pairing from their ends (and centromeres?).



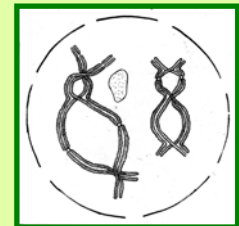
Pachytene

Chromosome pairing is complete. Recombination between chromatids of homologous chromosomes takes place (crossing over), resulting in exchange of paternal and maternal genes.



Diplotene

Chromosomes of paired bivalents shorten and begin to separate. Homologues are still connected due to preceding recombination (chiasmata).





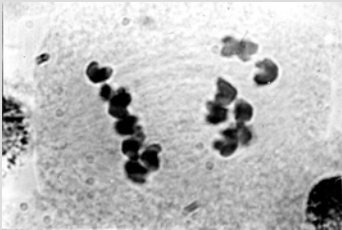
Diakinesis

Chromosomes show highest contraction. Chiasmata ,terminalize‘.



Metaphase I

The spindle is developed; bivalents congregate in equatorial plain between the spindle poles.



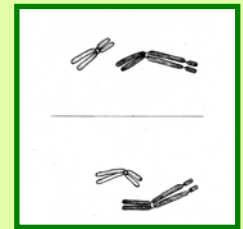
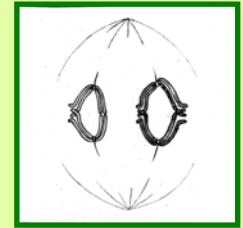
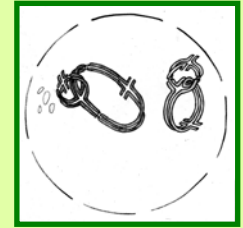
Anaphase I

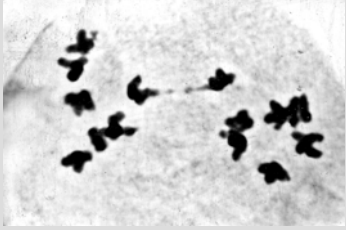
Homologous chromosomes segregate to opposite spindle poles, mediating reduction of chromosome number. The diploid chromosome complement gets haploid.



Telophase I

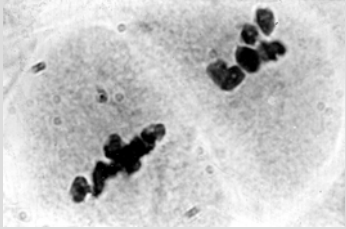
Homologous chromosomes arrive at the poles.





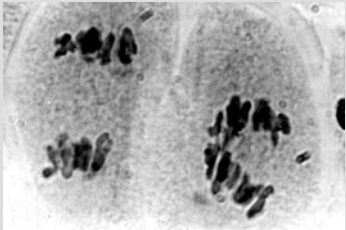
Interkinesis

Cohesion of sister chromatids is restricted to centromeres.



Metaphase II

A new spindle appears. Chromosomes arrange in equatorial plate.



Anaphase II

Sister centromeres separate, chromatids segregate toward the poles.



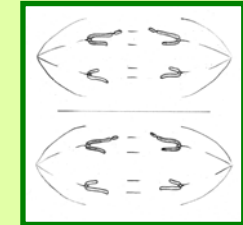
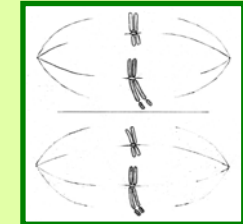
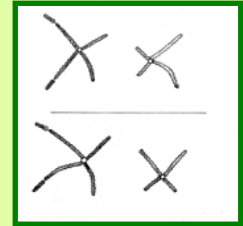
Telophase II

Chromosomes arrive at poles and decondense. The nuclear membrane is formed.



Pollen tetrade

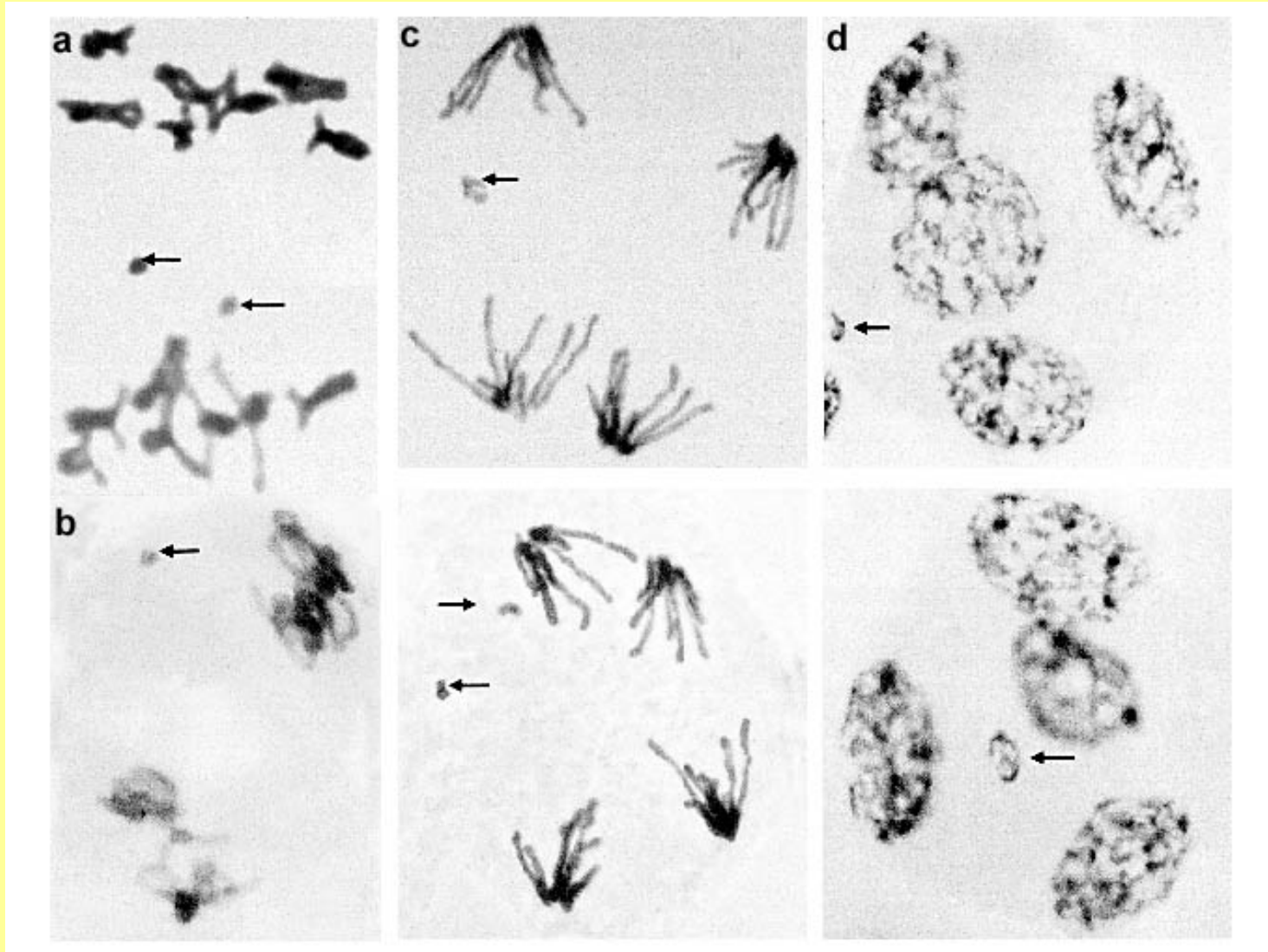
Four pollen cells with haploid chromosome number.



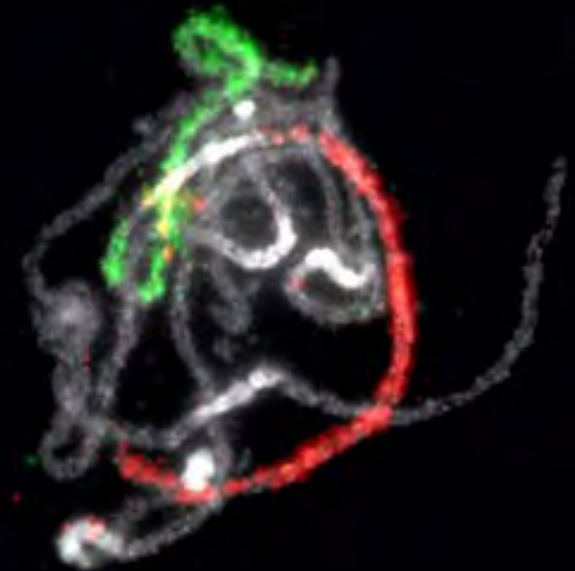
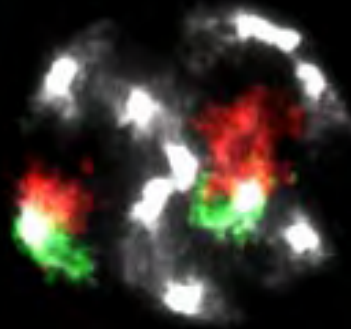
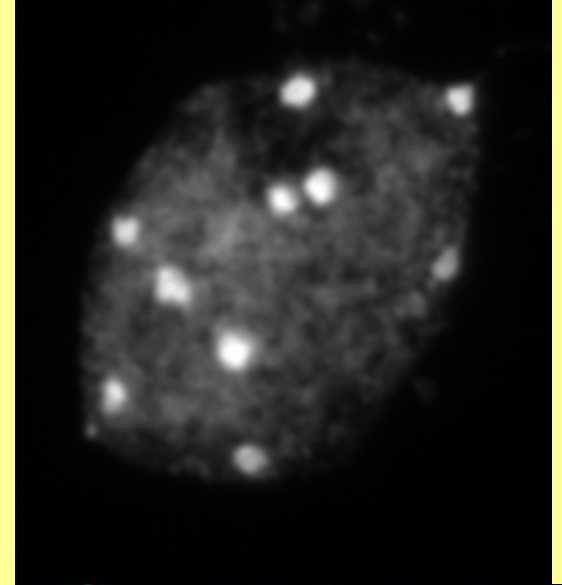
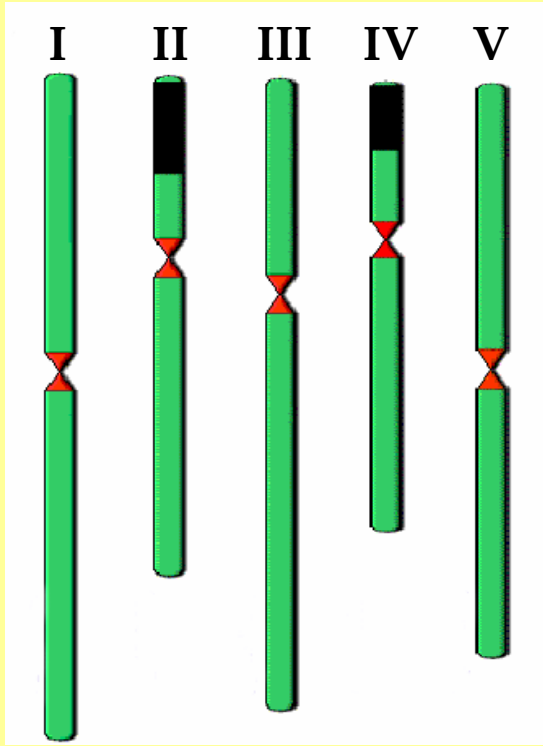
Metaphase I in field bean showing six bivalents



A minichromosome of field bean (comprising <math><5\%</math> of duplicated genomic sequences) does not segregate properly at meiosis



Arabidopsis thaliana ($2n=5$; 125 Mbp/C; ~15% repet. DNA)



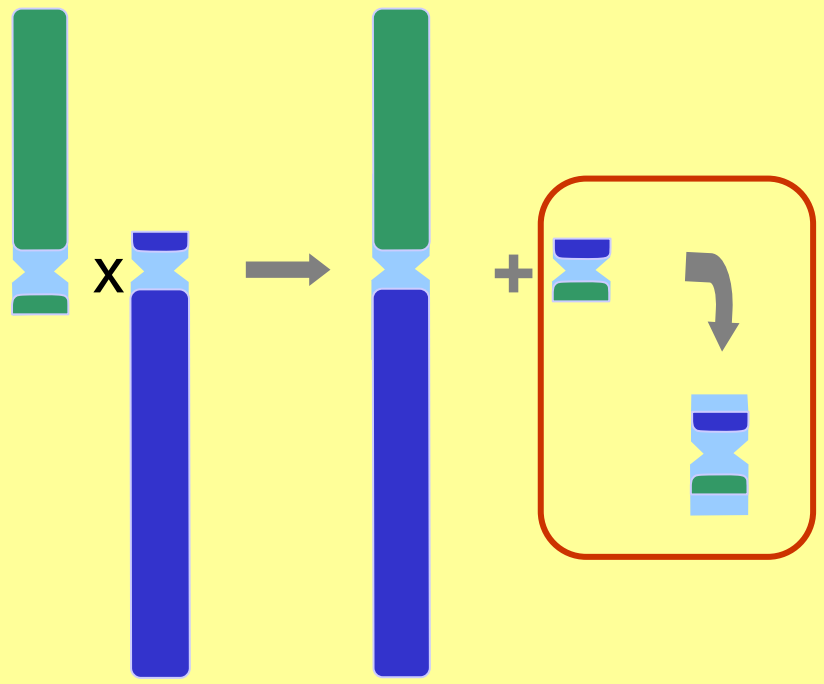
B chromosomes occur in many species in addition to A and sex chromosomes

They are:

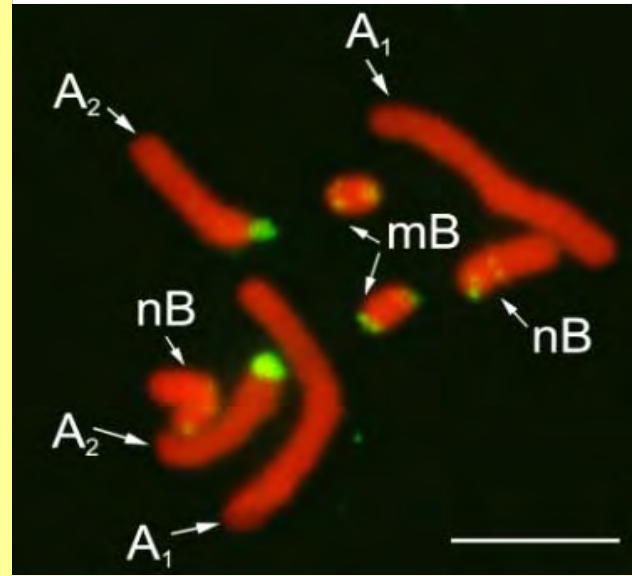
dispensable, generally transcriptionally inactive and display irregular and non-Mendelian modes of inheritance

They might have originated as:

- As remnant of by-product of interspecific hybridisation
- or derived from A and/or sex chromosomes, e.g., as one product of Robertsonian translocation that extends subsequently by sequence amplification and/or reshuffling



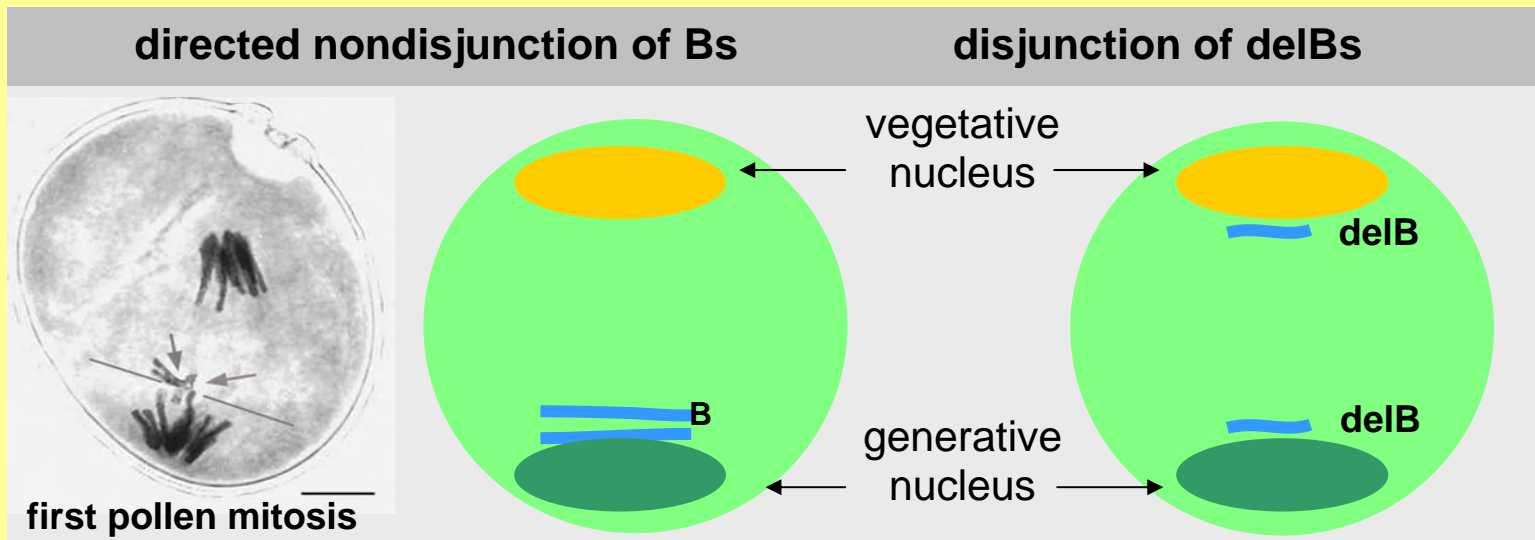
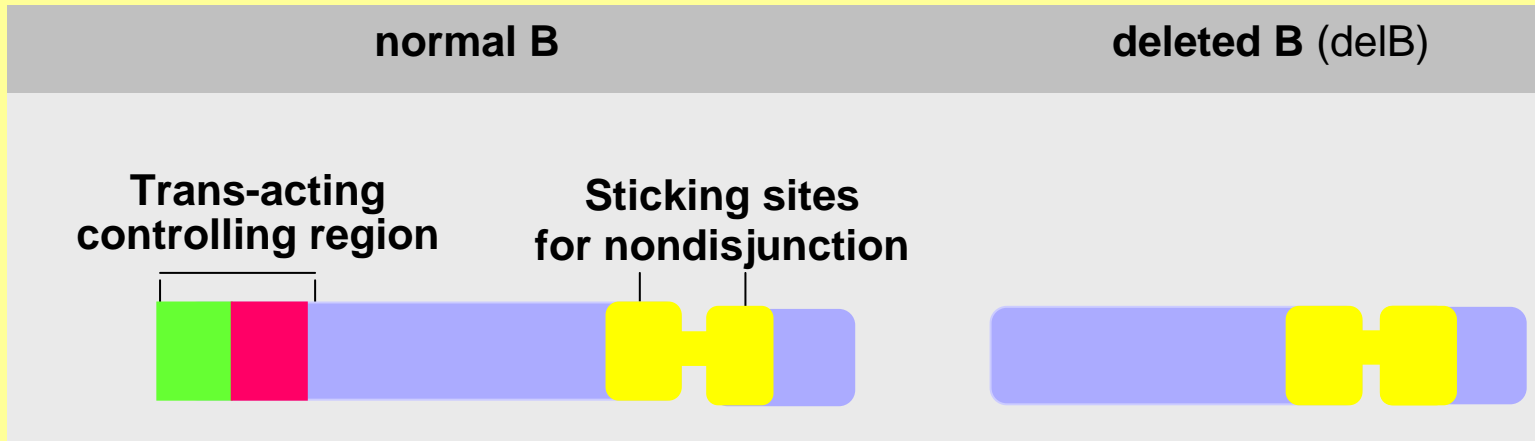
Brachycome dichromosomatica 2n=4+4B



Andreas Houben et al., IPK)

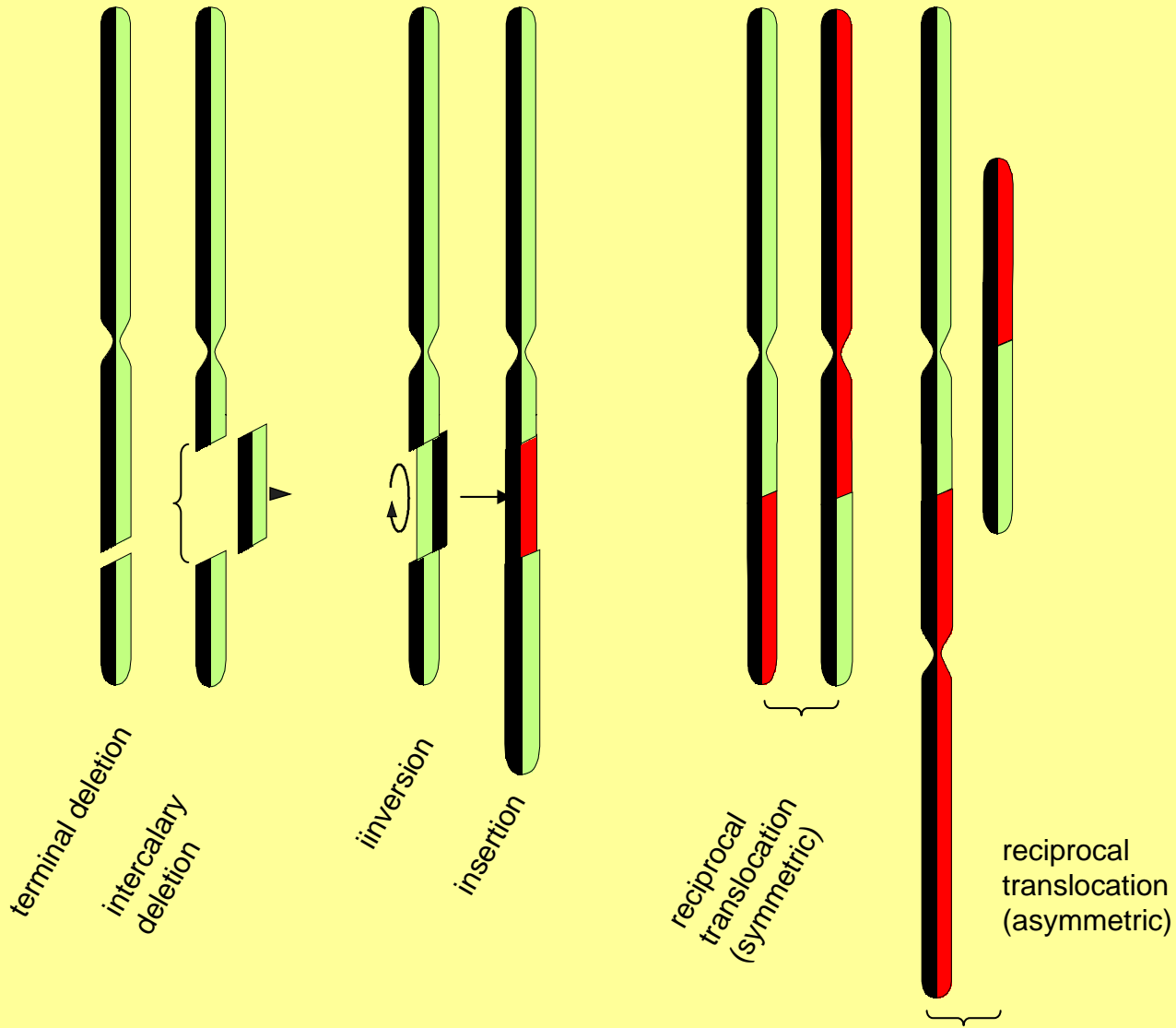
Nondisjunction of B chromosomes in rye

- █ D1100 B-specific repeat *transcriptionally active*
- █ E3900 B-specific repeat *transcriptionally active*



Chromosome mutagenesis

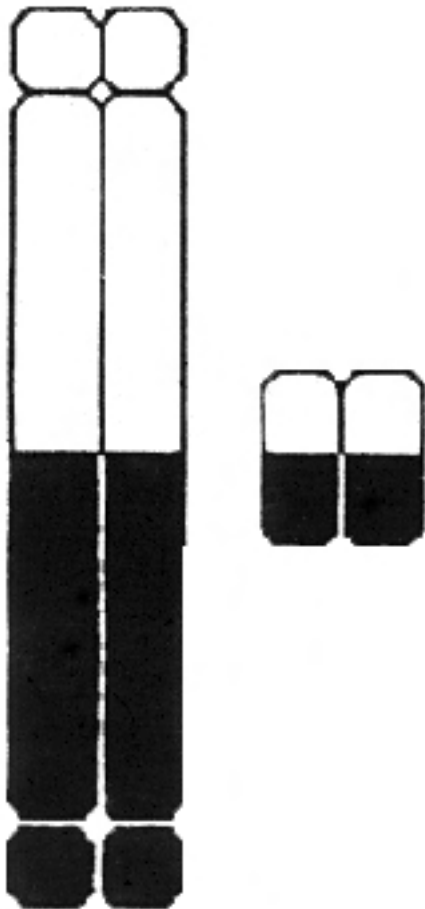
Structural chromosome aberrations (deletions & rearrangements)



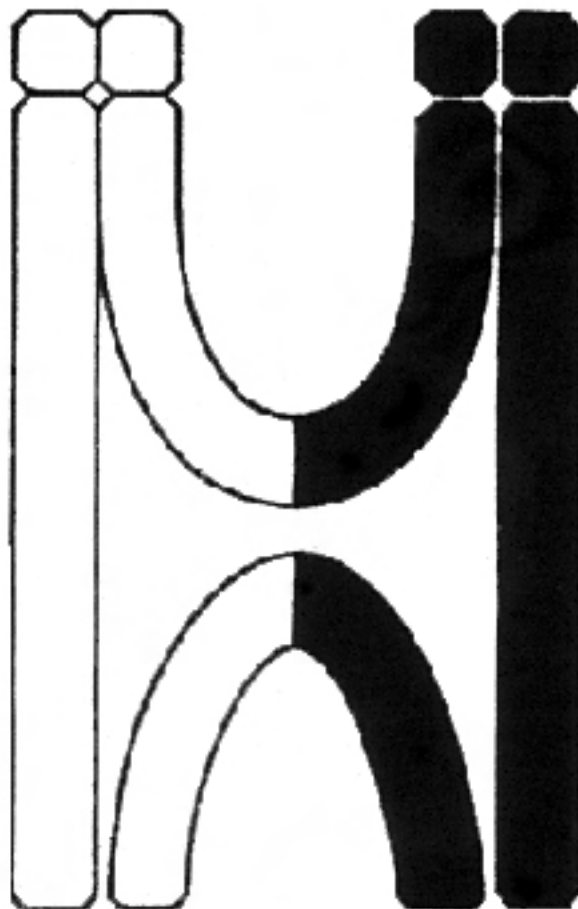
G1

S

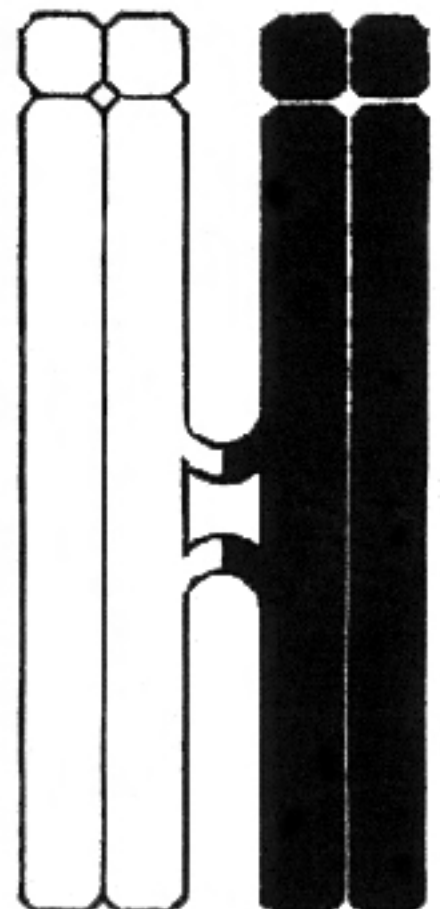
G2



a



b



c

Chromosome-type
translocation

Chromatid-type translocation

Subchromatid-type
translocation

Chromatid-type aberrations



Reciprocal
translocation

Interkalary
deletion

Duplication
deletion

Isochromatid
break

Chromatid
break

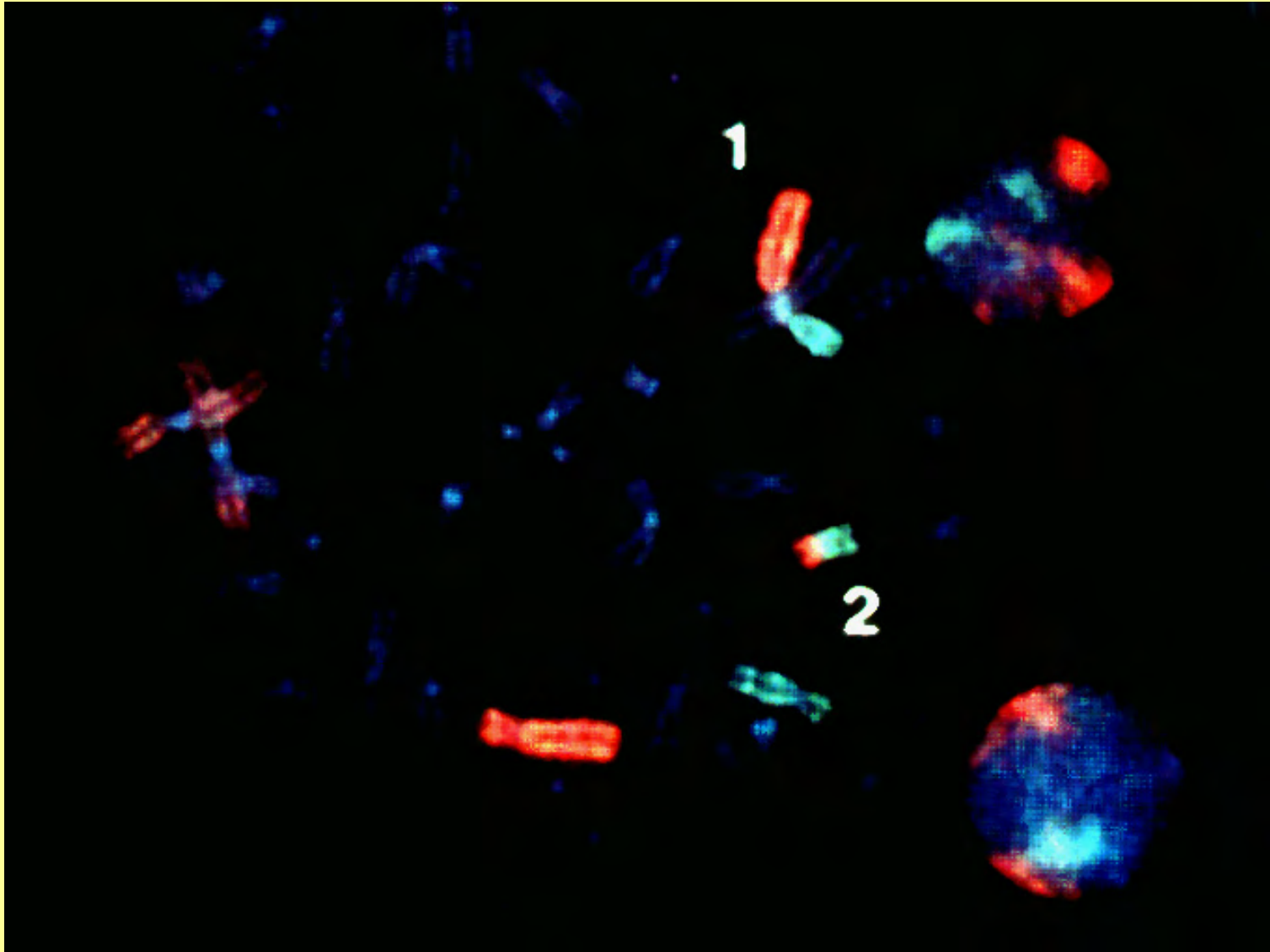
Multiple chromatid translocation (*Vicia faba*)

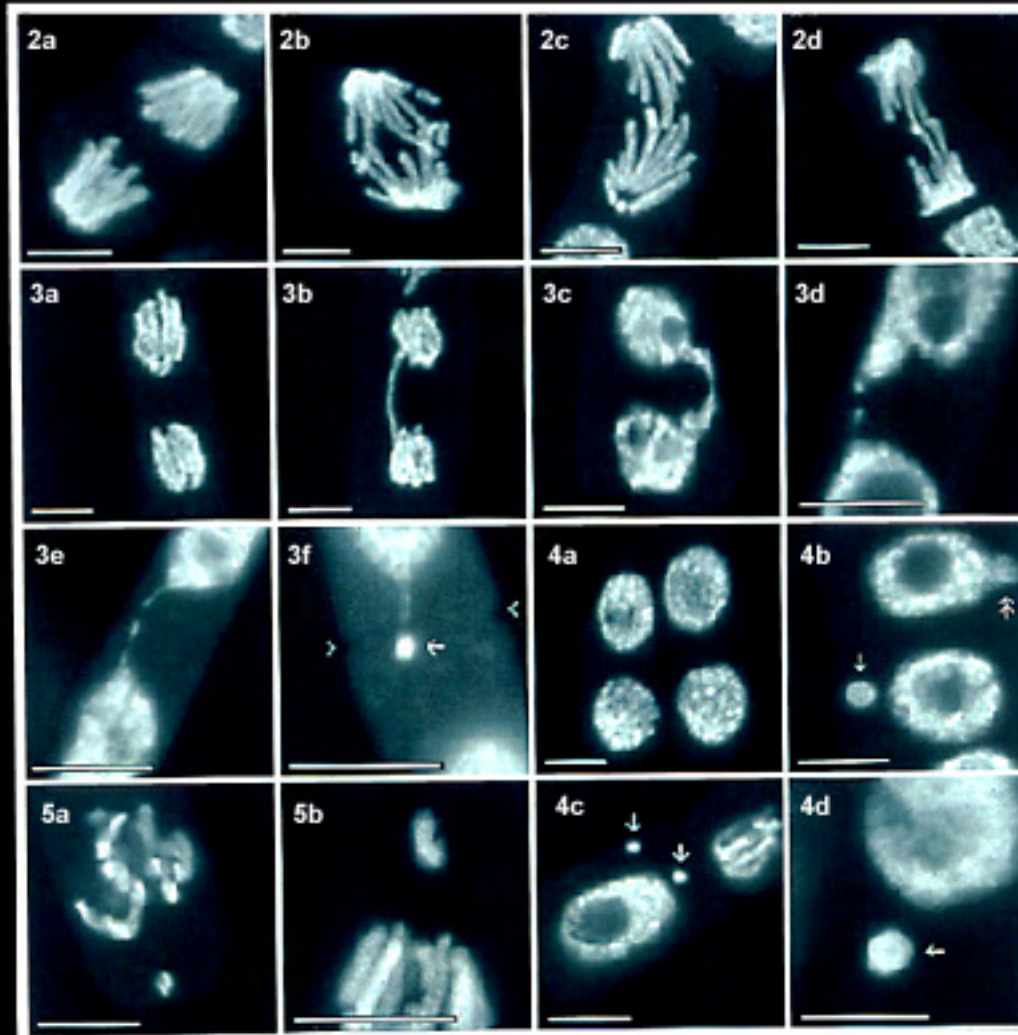


Anaphase bridge and acentric fragment as a consequence of an asymmetric translocation



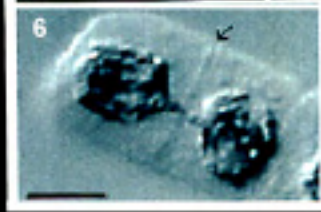
Translocation between human chromosomes 1 and 2 visualized by chromosome painting



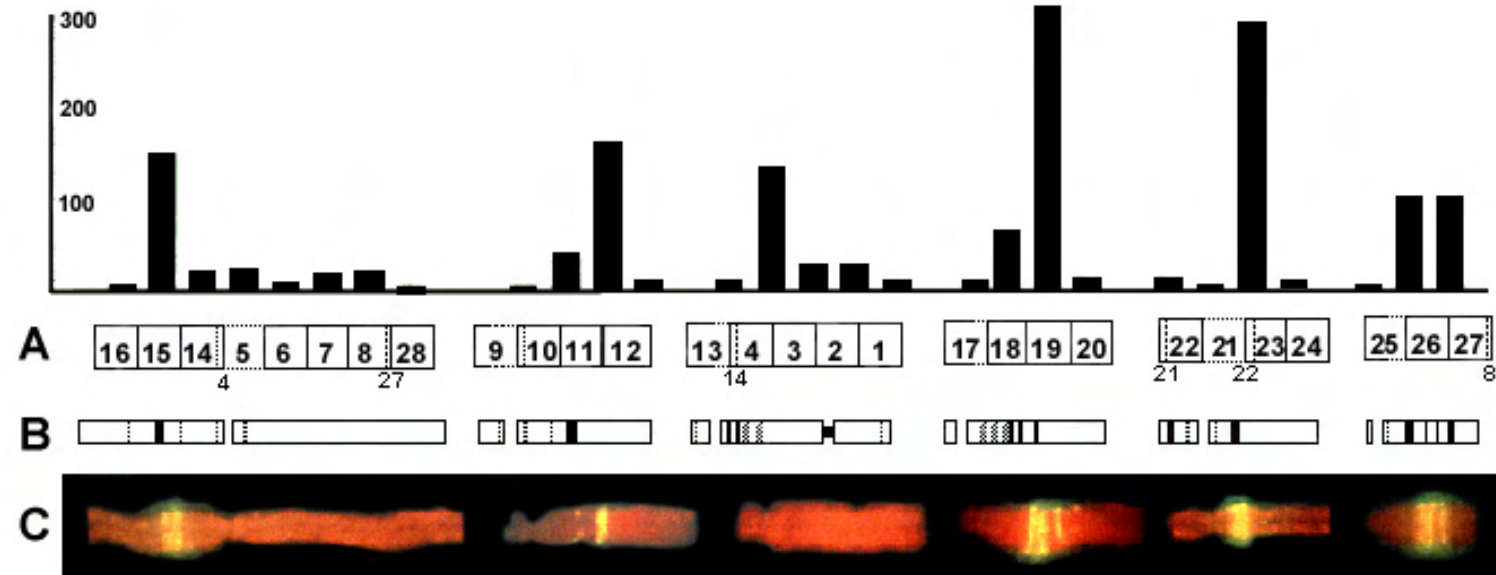


Telophase bridges

Micronuclei



Distribution of aberration breakpoints along field bean chromosomes after MNU treatment



Most preclastogenic lesions are correctly repaired (*Vicia faba*)

MNU (10^{-3} M, 1h) → ~30% metaphases with chromatid aberrations (12 h RT)
>2/3 within heterochromatin (~10% of the genome)

O⁶-MeG

~ linear dose relationship in all cell cycle stages
uniformly distributed and removed (immuno-slot-blot)

SSB

~ linear dose relationship in all cell cycle stages
uniformly distributed in eu-/heterochromatin (comet assay)

Under adaptive conditions (pre-treatment with MNU 10^{-4} M, 1h): ≥50% reduction of CA, O⁶-MeG, SSB

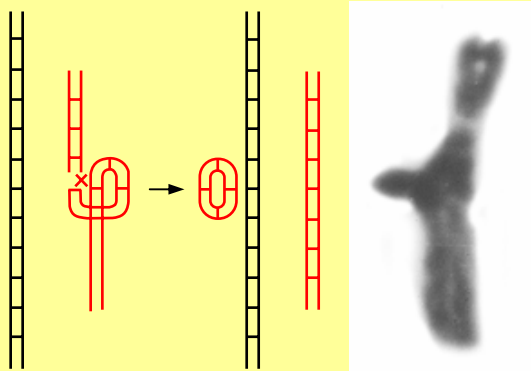
| | |
|--|---|
| 1 SCE per $>4 \times 10^4$ O ⁶ -MeG | 1 CA per $\sim 2.5 \times 10^7$ O ⁶ -MeG |
| ~600 : 1 | |

Hypothesis:

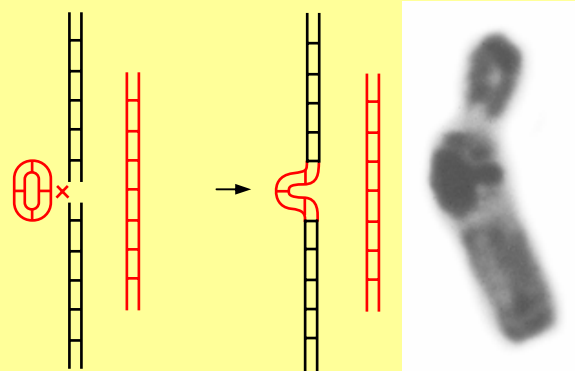
Most (non-DSB) preclastogenic lesions are correctly repaired (dealkylation; excision repair; PRR/SCE)
only a minority may lead by coincidence of repair intermediates (SSB)
with replication discontinuities to DSBs
that cause CA via erroneous recombination between damaged homologous repeats
(heterochromatin)

Mechanistic interpretation of the origin of isochromatid breaks, intercalary deletions and duplication-deletions via recombinative repair of DNA breaks in tandemly arranged sequence repeats

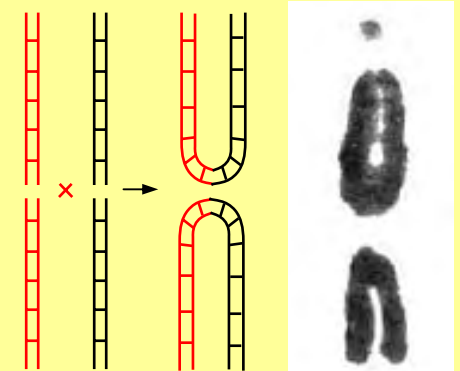
a) intercalary deletion



b) duplication-deletion



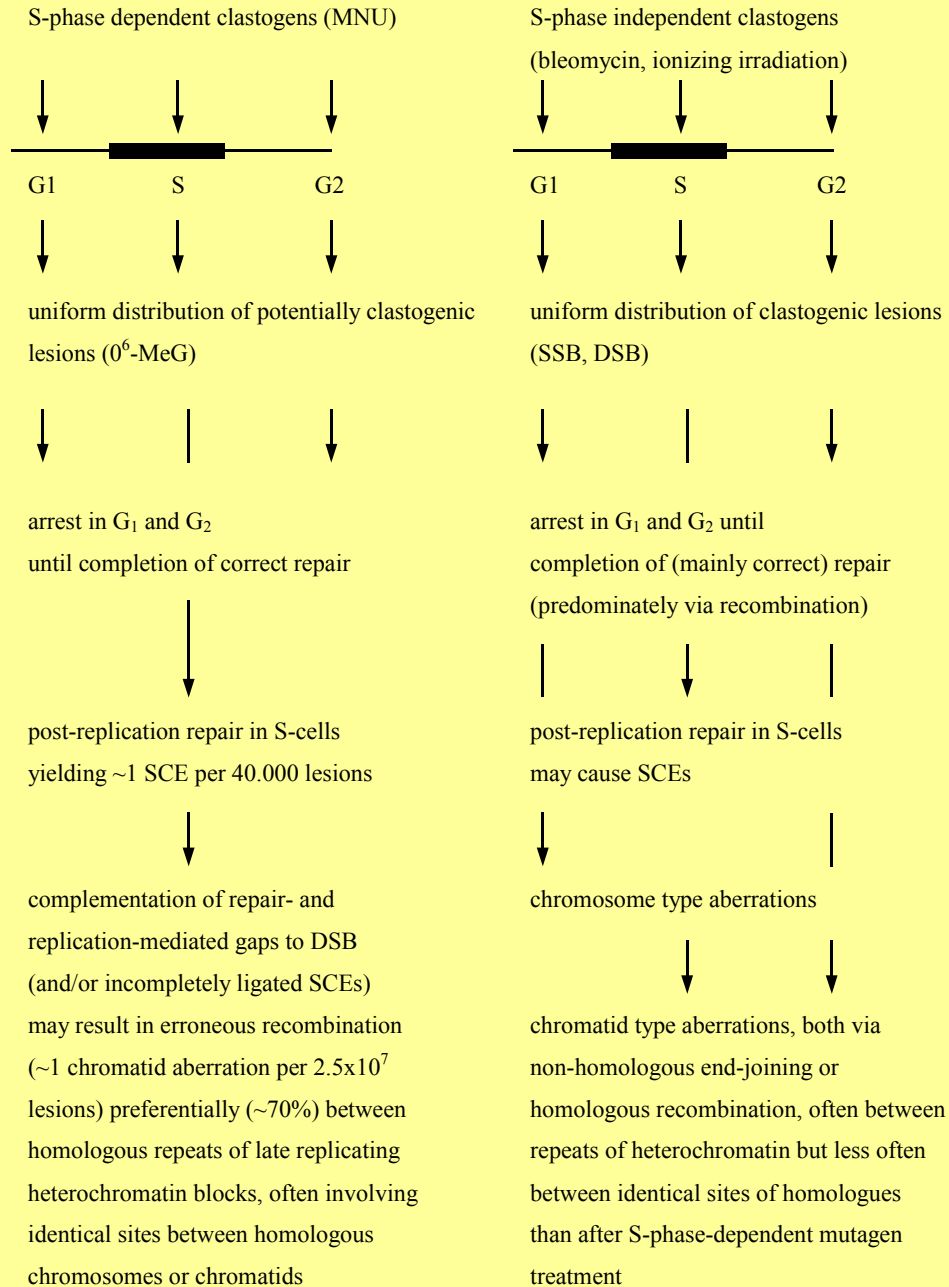
c) isochromatid break



HR or NHEJ?

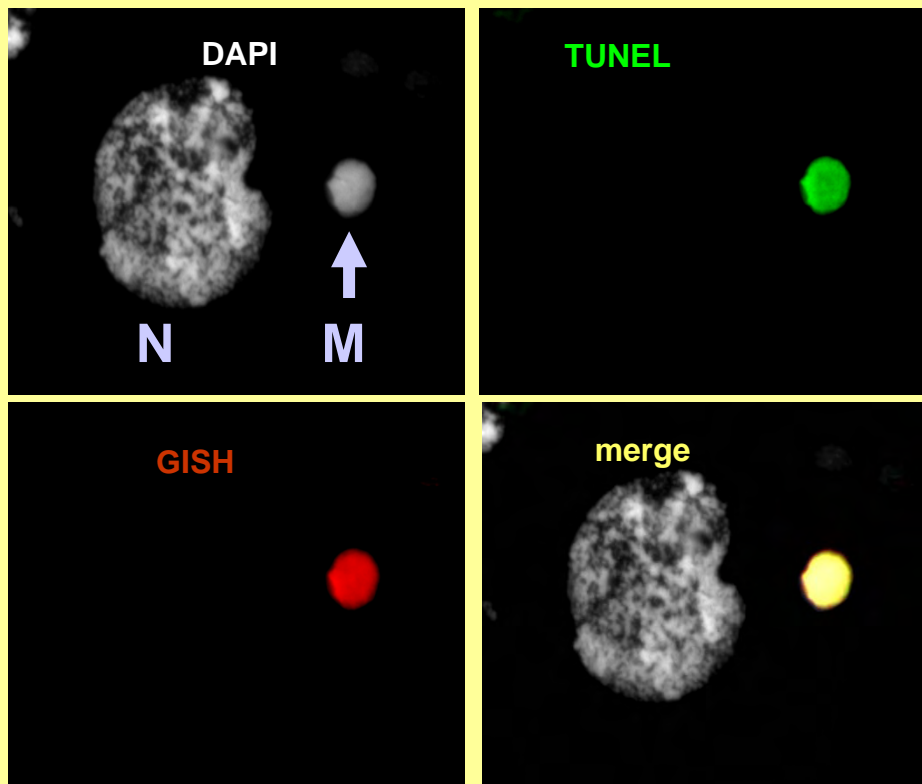
Scheme of DNA damage processing during aberration formation

treatment with:



Early chromatin elimination via apoptosis and micronucleus formation of the paternal genome in interspecific hybrids

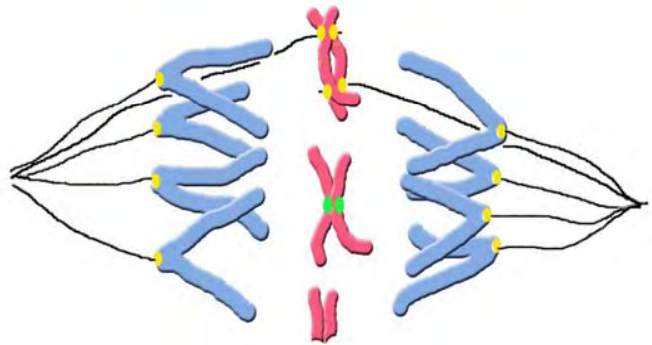
Wheat × pearl millet



Two modes of early uniparental genome elimination in interspecific hybrids

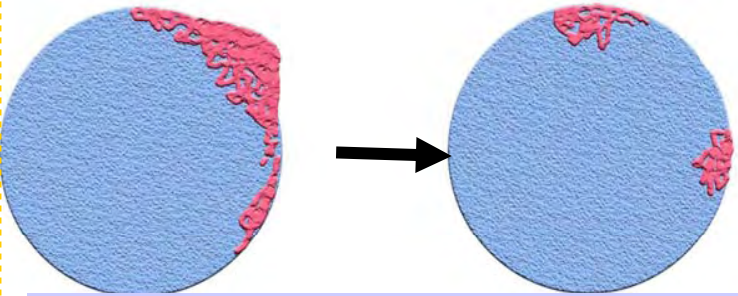
1) Mitosis-dependent

By imperfect segregation, or parent-specific CENH3 inactivation?



2) Mitosis-independent

By spatial separation of parental genomes



+ extrusion (,budding') of the apoptotic (paternal) genome

+ heterochromatinization and disintegration of micronuclei

A. Houben group:
Plant Cell 17: 2431 (2005)
Chromosoma 116: 275 (2007)
CGR 114: 169 (2006)

Karyotype analysis and chromosome length differentiation *via*
banding techniques

The first chromosome banding remained unnoticed

Chromosoma, Bd. 7, S. 620—626 (1956)

Aus dem Biologischen Institut der Keiō-Universität zu Yokohama
DIFFERENTIELLE FÄRBUNG DER SOMATISCHEN
METAPHASECHROMOSOMEN VON CYPRIPIEDIUM DEBILE *

I. Mitteilung

Von

NORIKO YAMASAKI



1

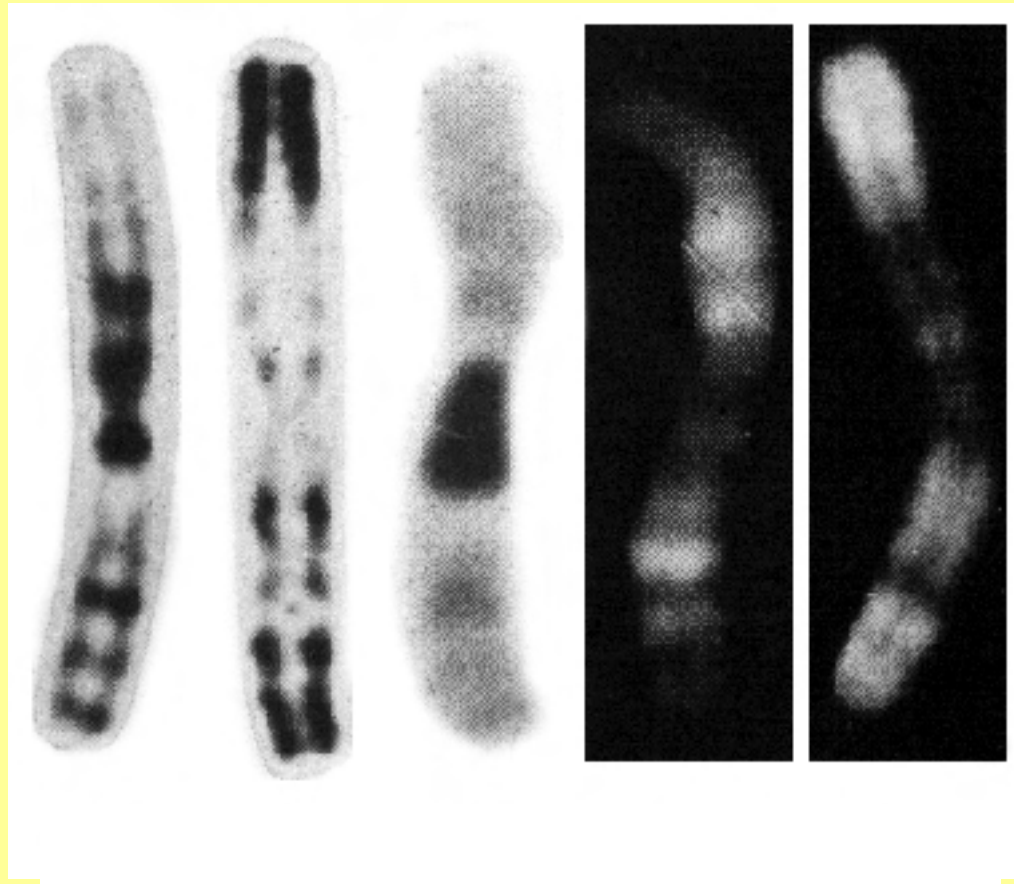


2

Abb. 1. Orceinssigsäure-Salzsäure-Färbung ohne Vorbehandlung. Vergr. 1215mal

Abb. 2. Vorbehandlung mit 8-Oxychinolin. Die Pfeile bezeichnen ein an den Querstreifen erkennbares homologes Chromosomenpaar. Der Kern ist durch den Druck auf den Objektträger beschädigt worden; einige Chromosomen sind infolgedessen zerbrochen oder aus dem Kern herausgepreßt worden. Vergr. 1215mal

Various banding patterns of human chromosome 1



G

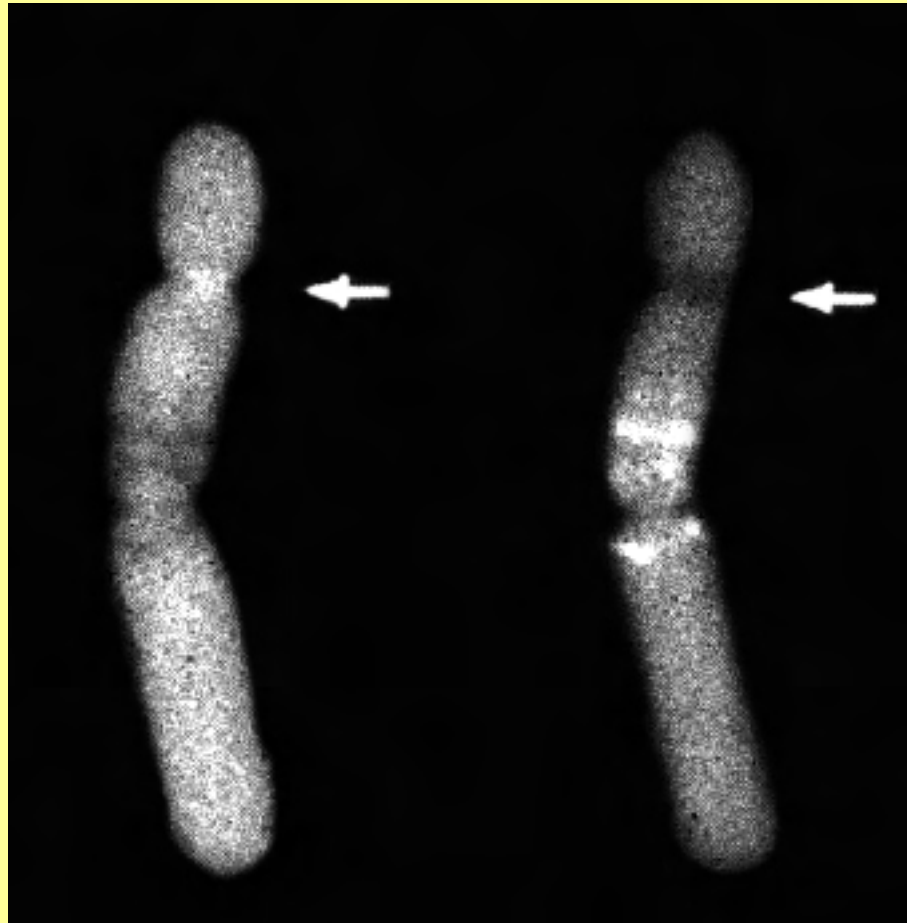
R

C

Q

AO

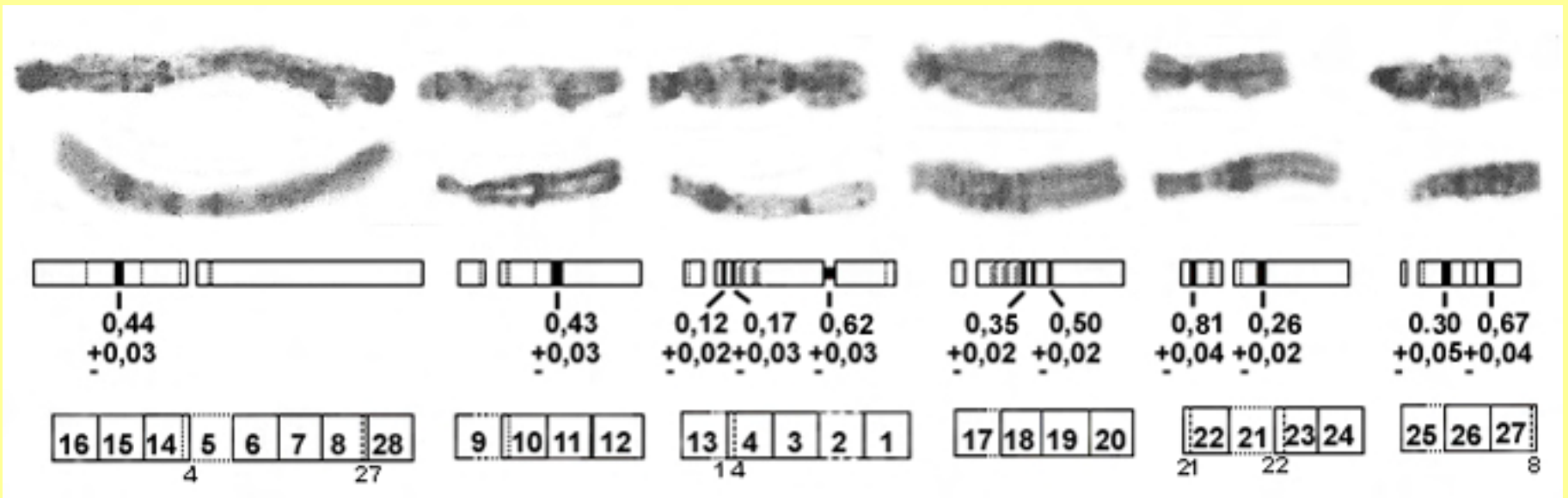
Complementary fluorescence banding on *Vicia faba*
chromosome I



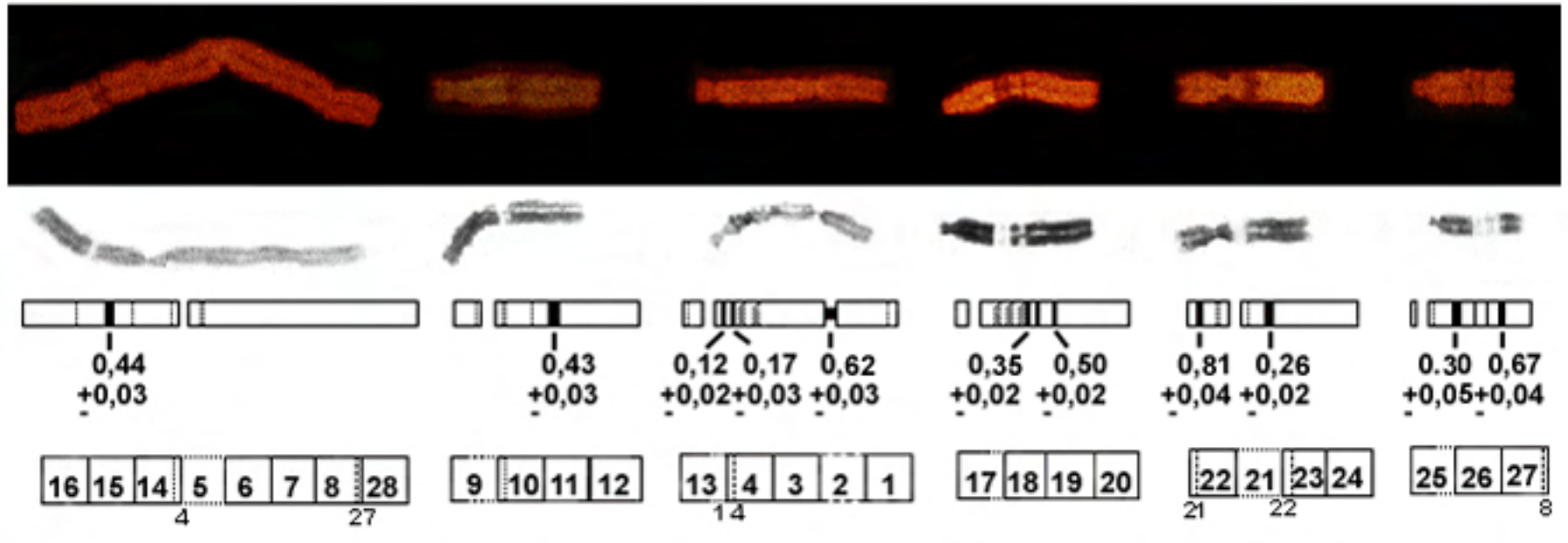
CMA-DA

DAPI-AD

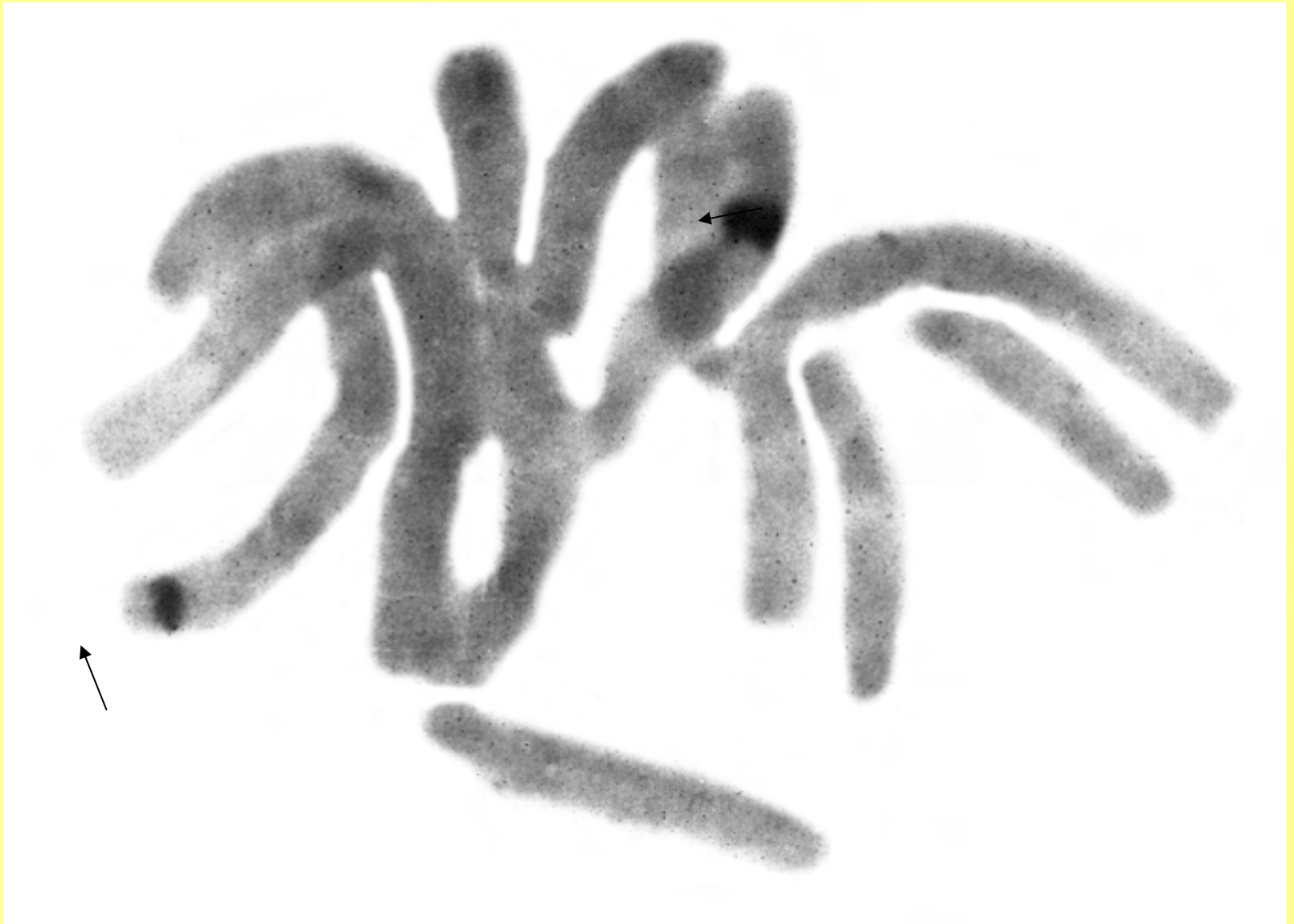
Interstitial and centromeric Giemsa bands in *Vicia faba* (karyotype ACB)



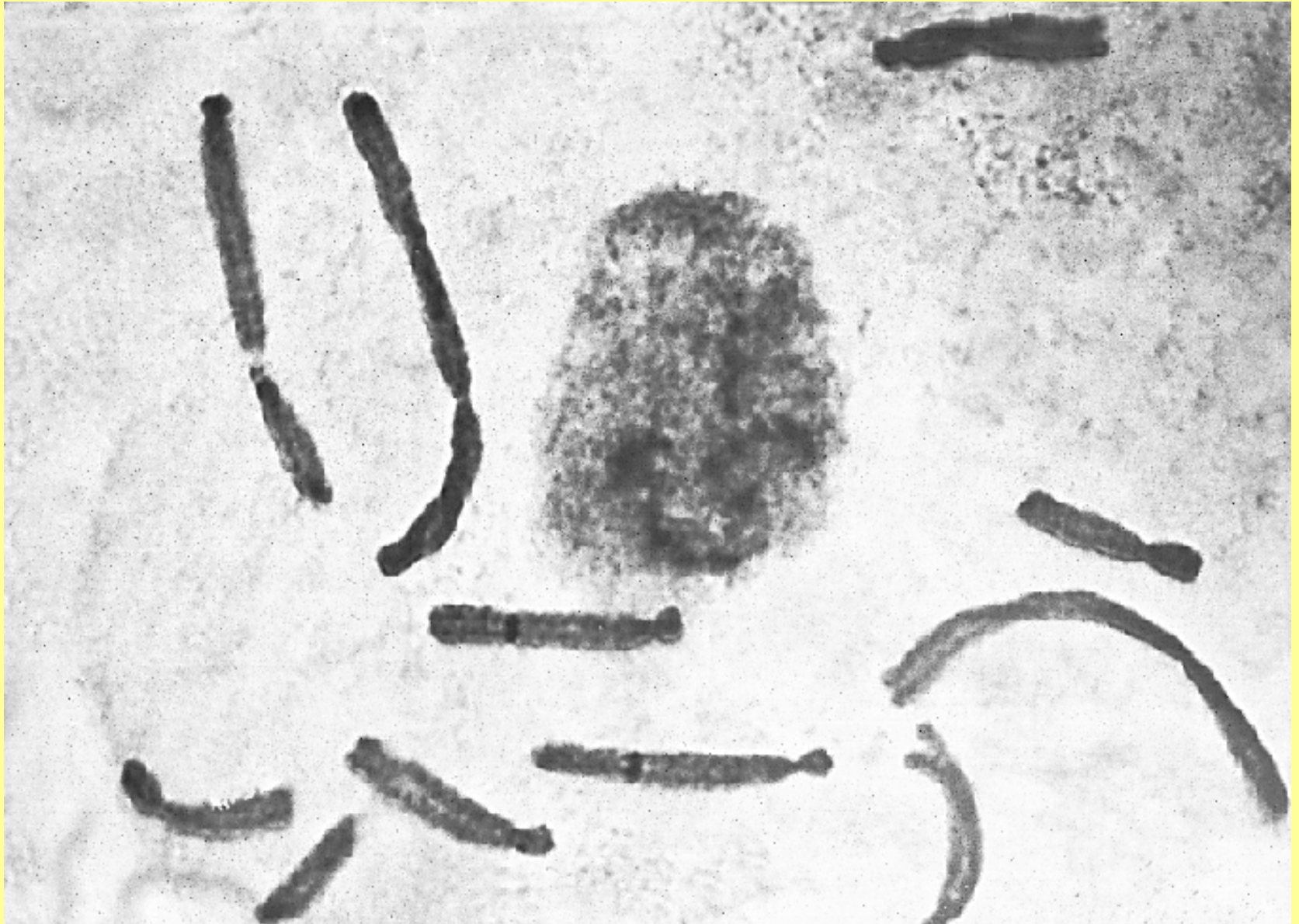
Giemsa- and PI-banding pattern after *FokI* digestion of field bean chromosomes



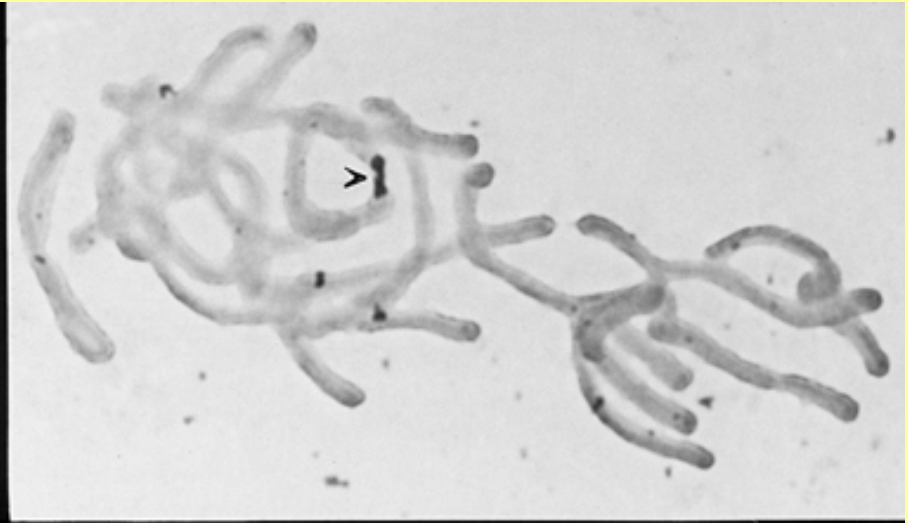
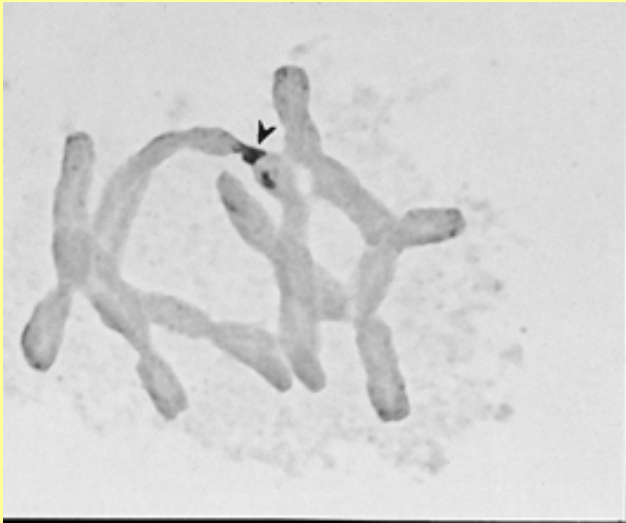
Giemsa banding of the NOR (N banding)



Silver staining of *Vicia faba* NORs



Terminal bridges between silver-stained NORs of onion chromosomes



Mobile nucleolus organisers in Allium

A. cepa

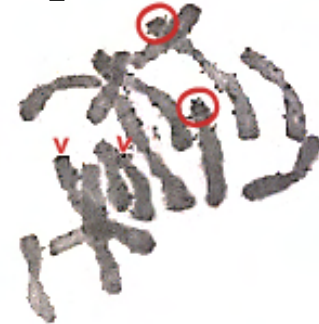
A. cepa x *A. fistulosum*

After loss of satellites



2 NORs

4 NORs

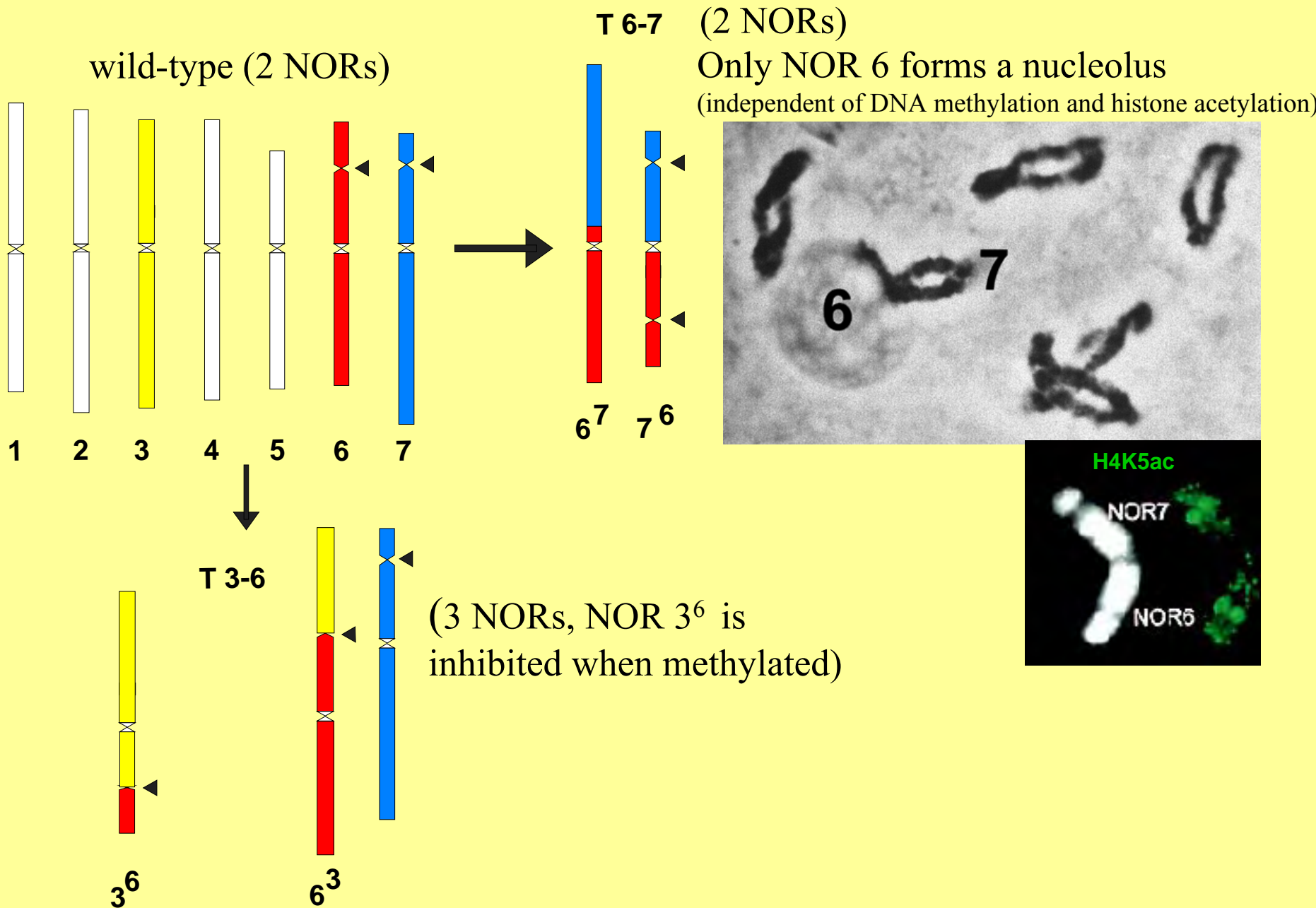


4 NORs

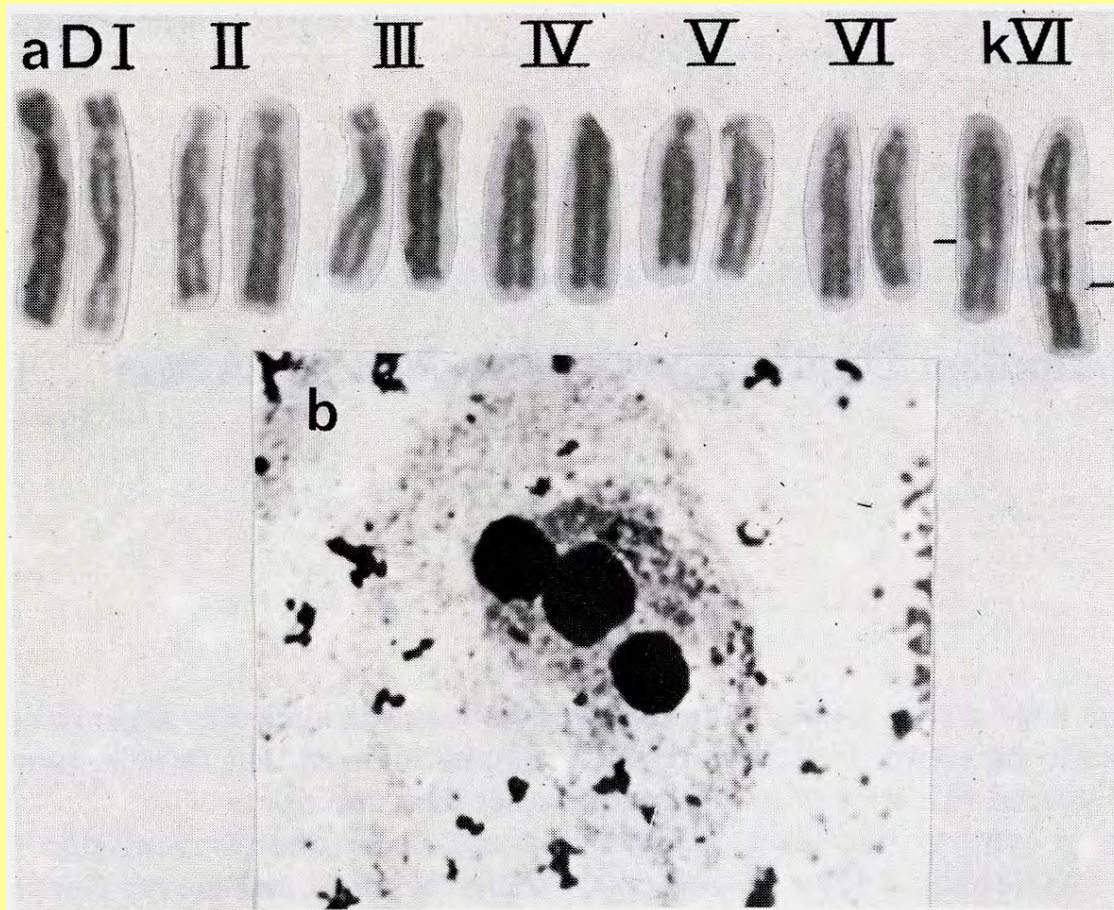


Chromosomes with terminal NORs

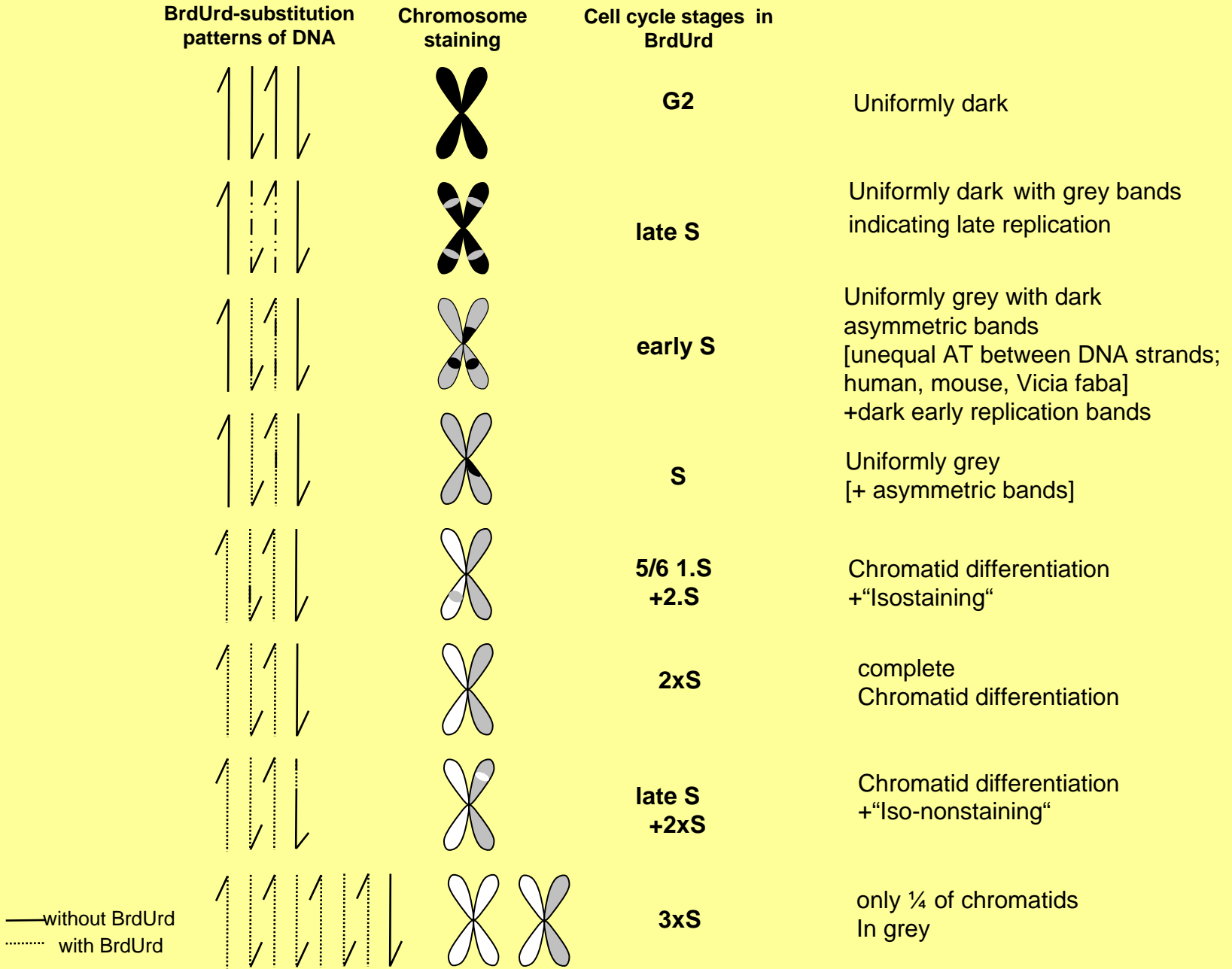
Intraspecific nucleolar dominance in barley



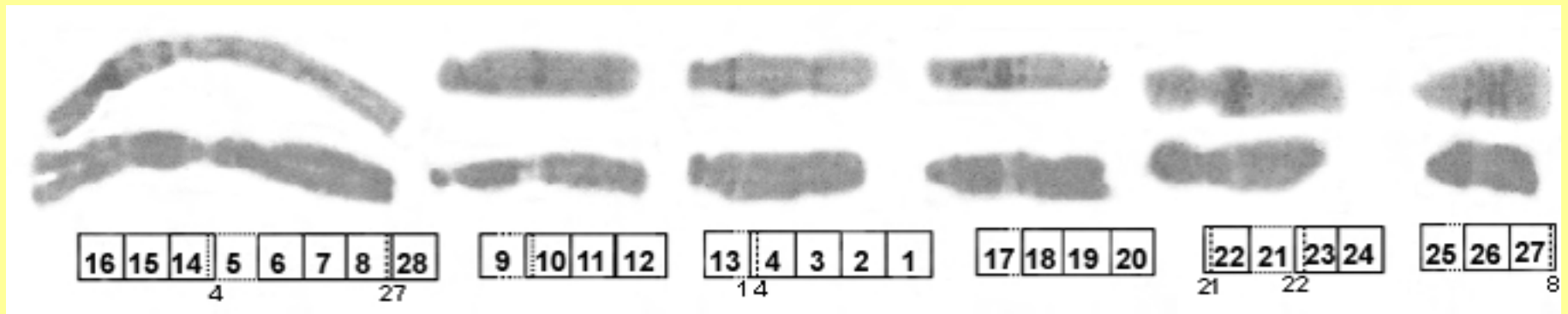
A duplicated NOR forms a nucleolus
within the reconstructed field bean karyotype DKP14



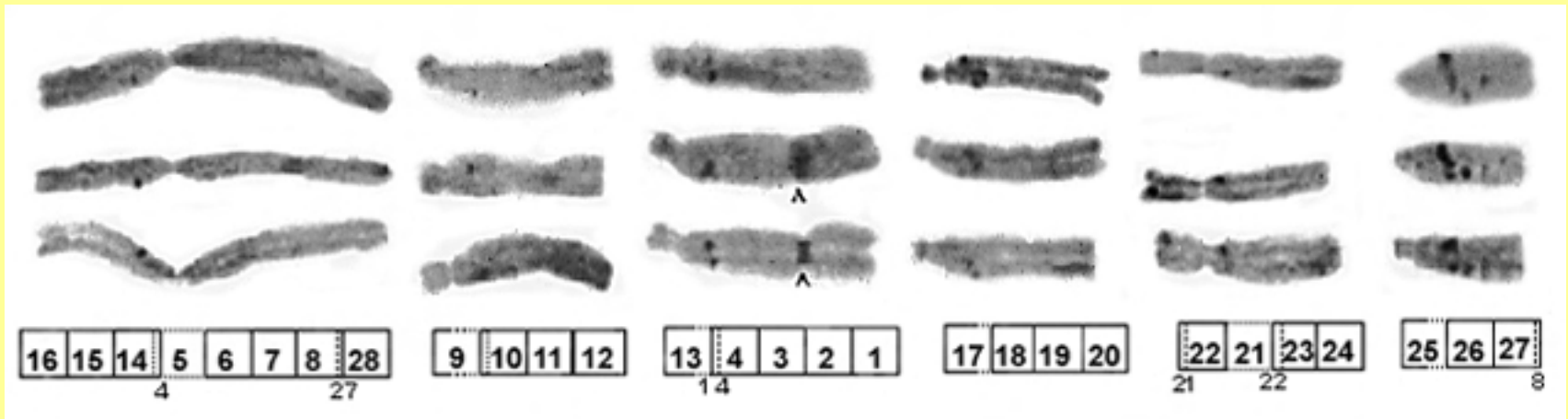
Differential labeling of chromosomes by incorporation of the base analogue BrdUrd and fluorescence-plus-Giemsa (FPG) staining



Replication patterns after BrdUrd[±] exposure (5 h) & FPG-staining



Asymmetric bands after BrdUrd-incorporation (17 h) and FPG-staining

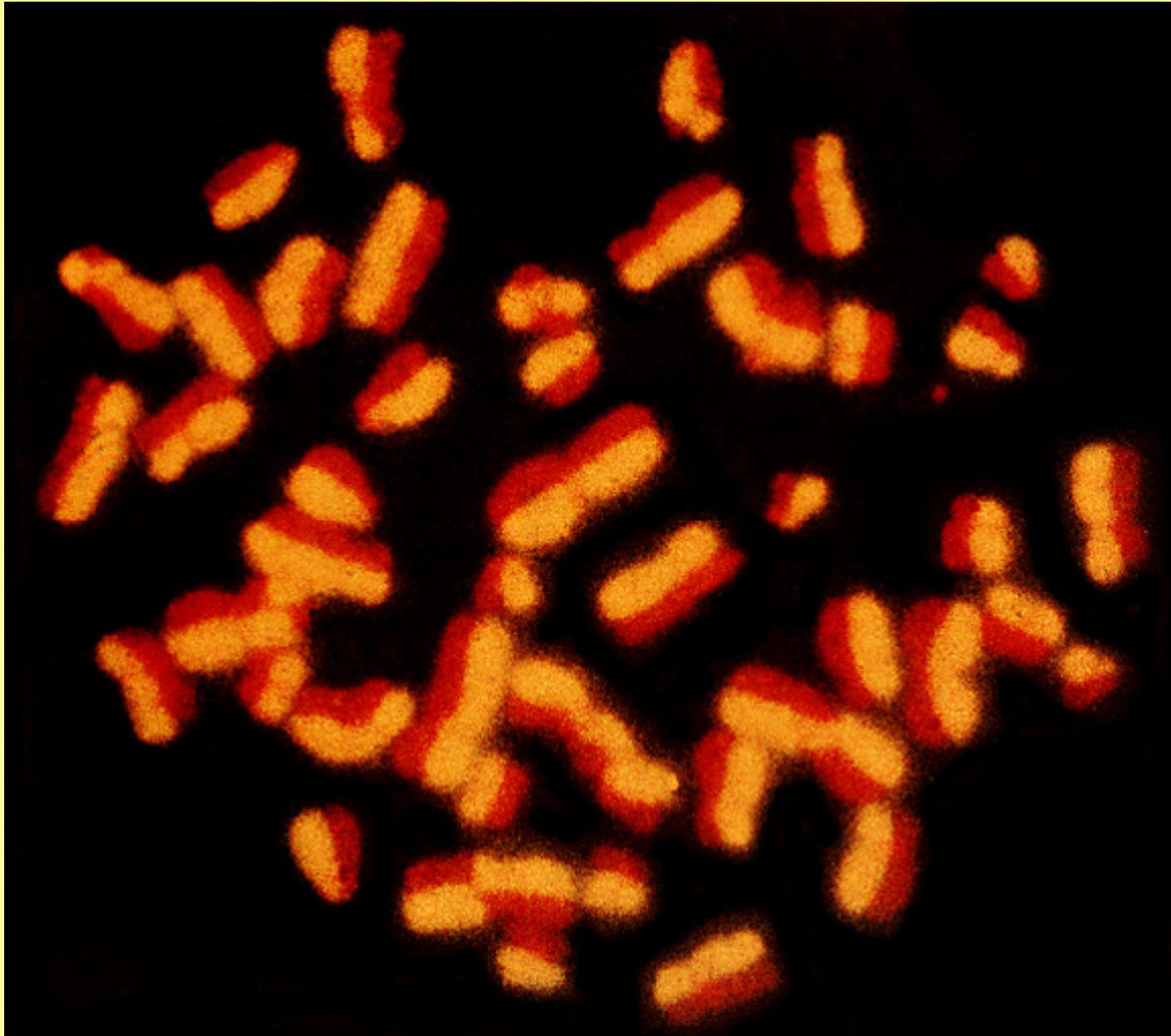


Vicia faba chromosomes:

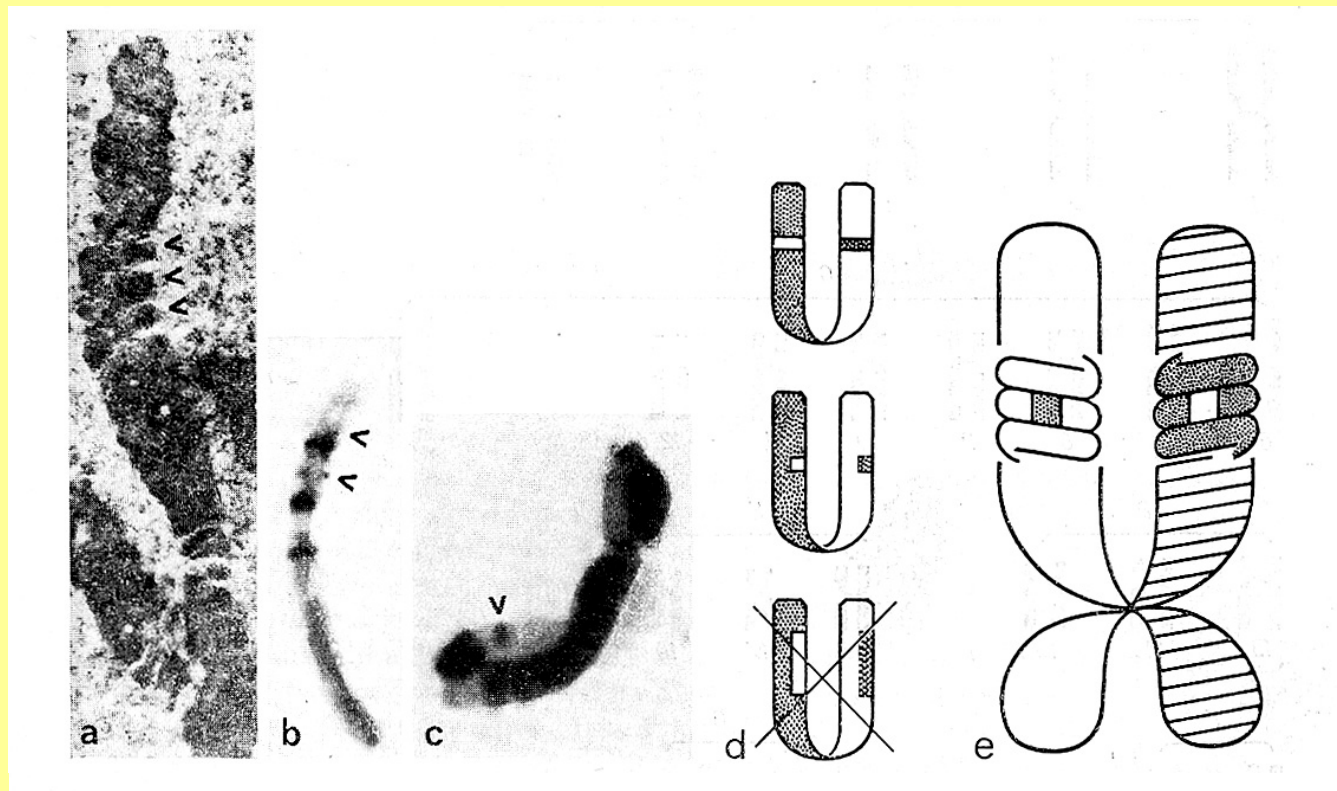
SCEs without and after mutagen exposure



SCEs on human chromosomes detected by anti-BrdUrd/PI



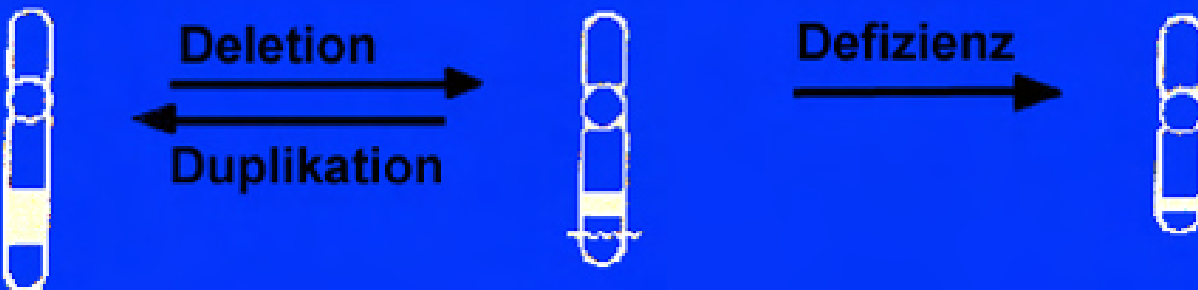
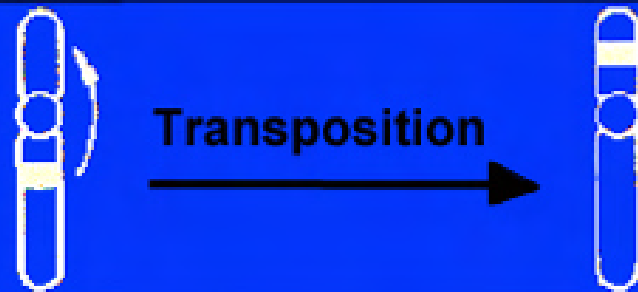
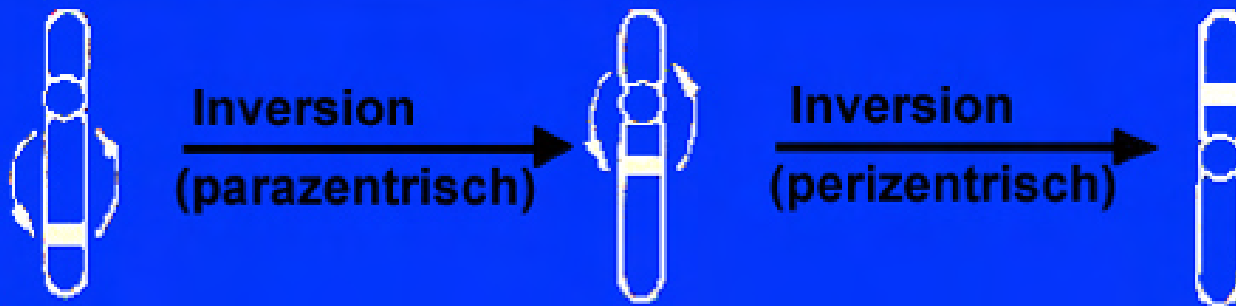
The longitudinal extension of bands and SCEs which do not comprise the entire chromatid diameter does never exceed that of the smallest ones covering the whole diameter.



Mechanisms of chromosome and karyotype evolution



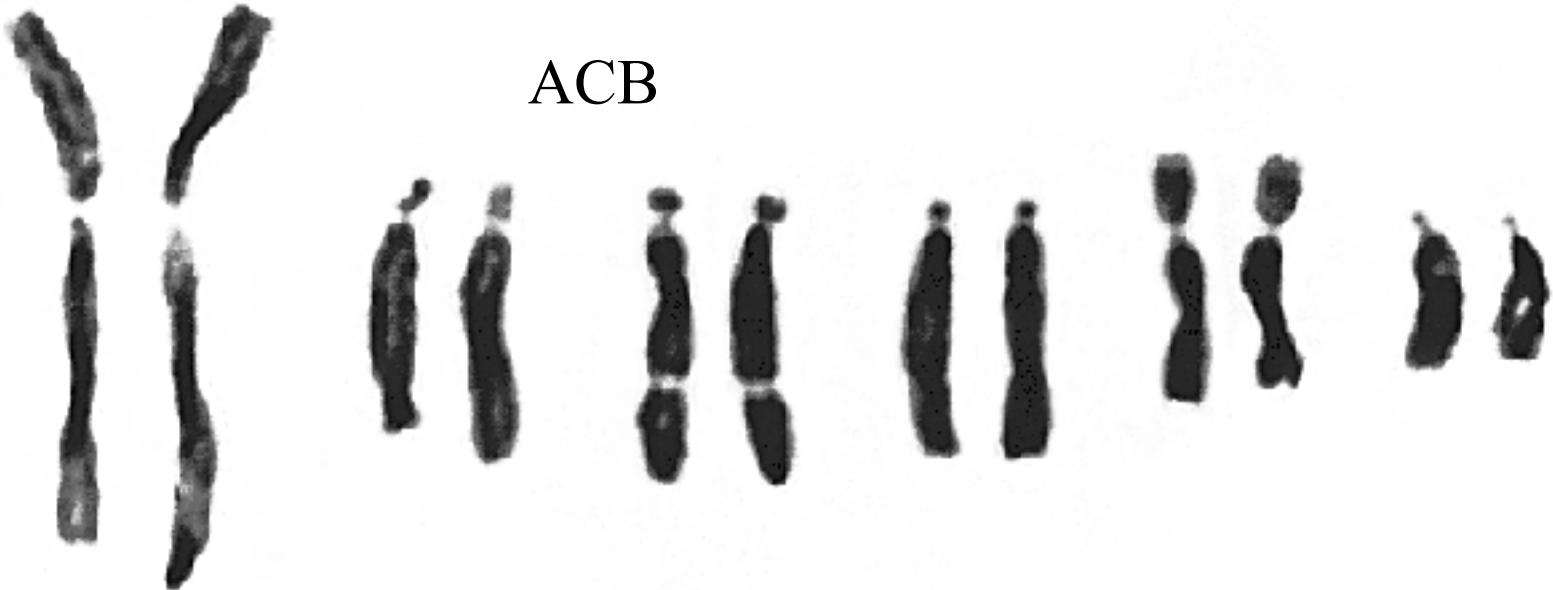
Primary karyotype rearrangements



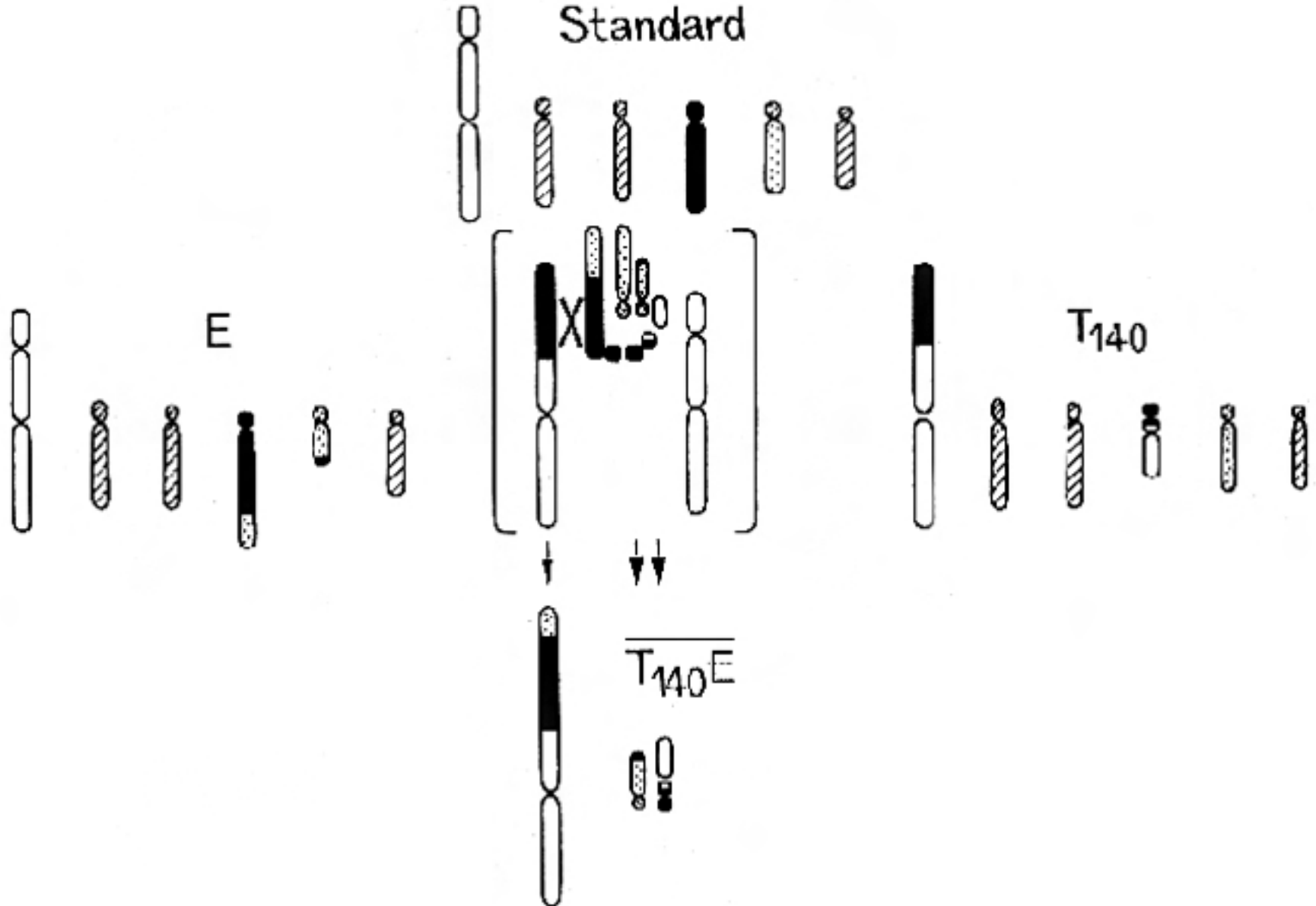
Vicia faba standard karyotype

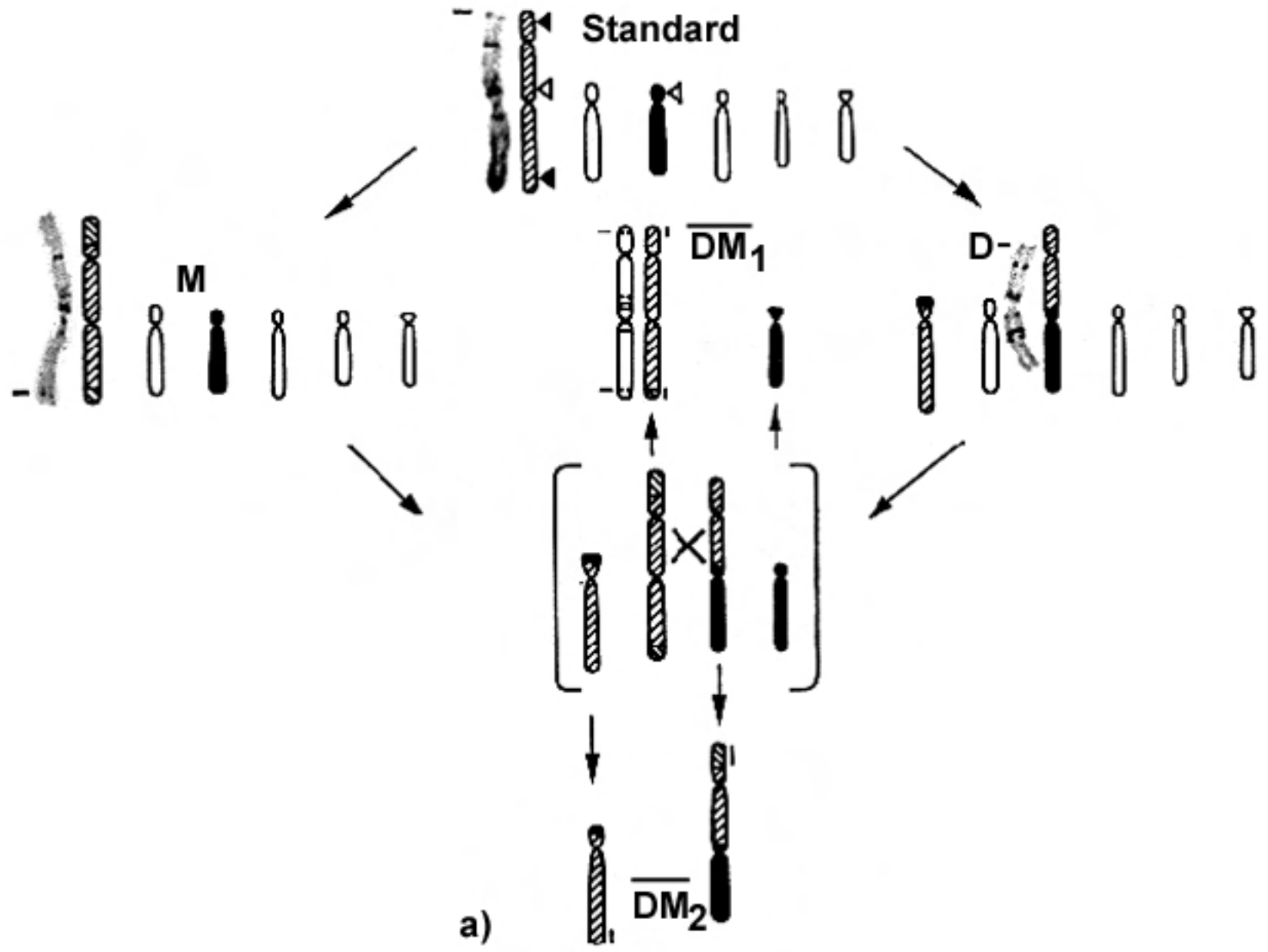


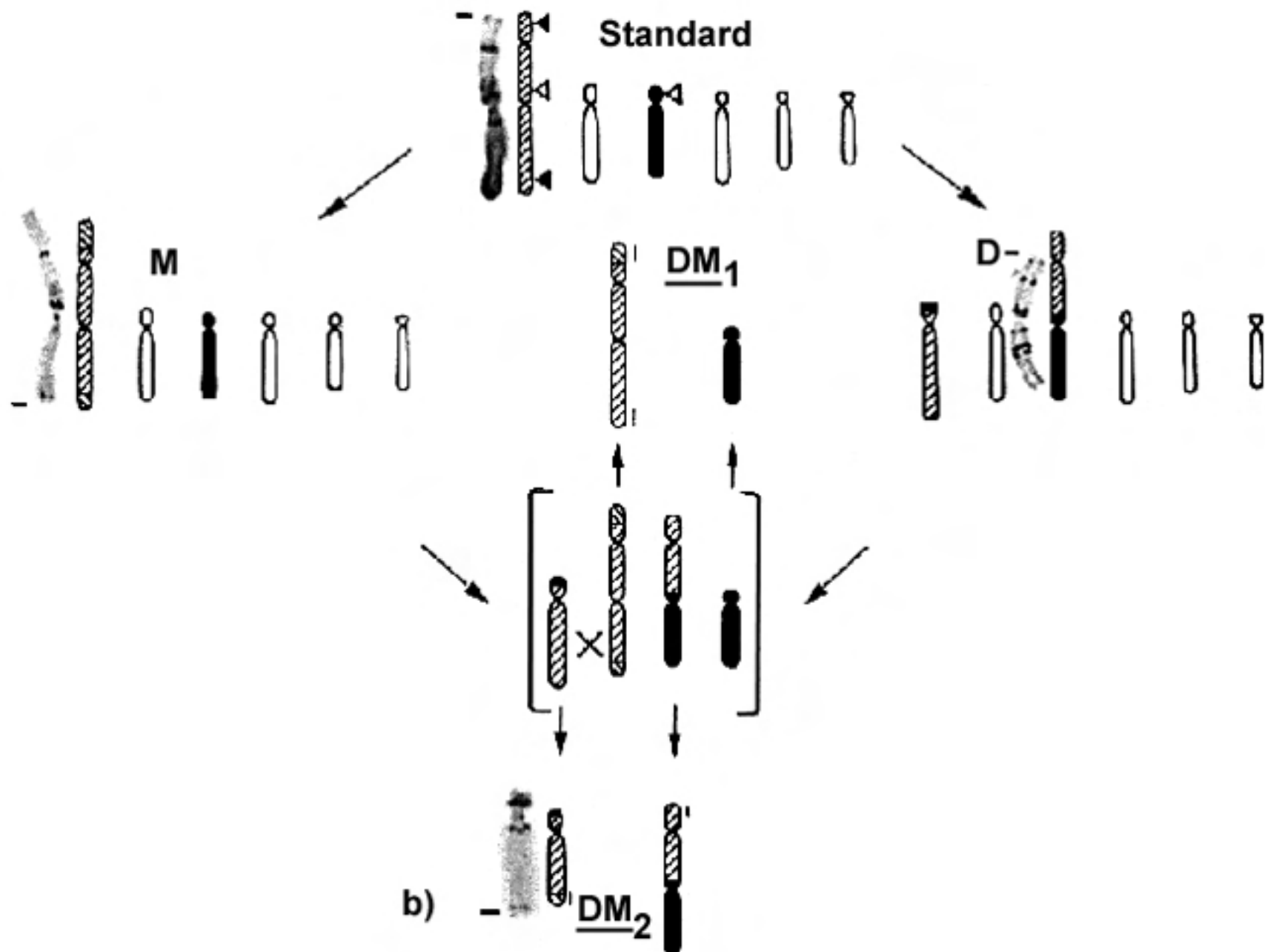
ACB

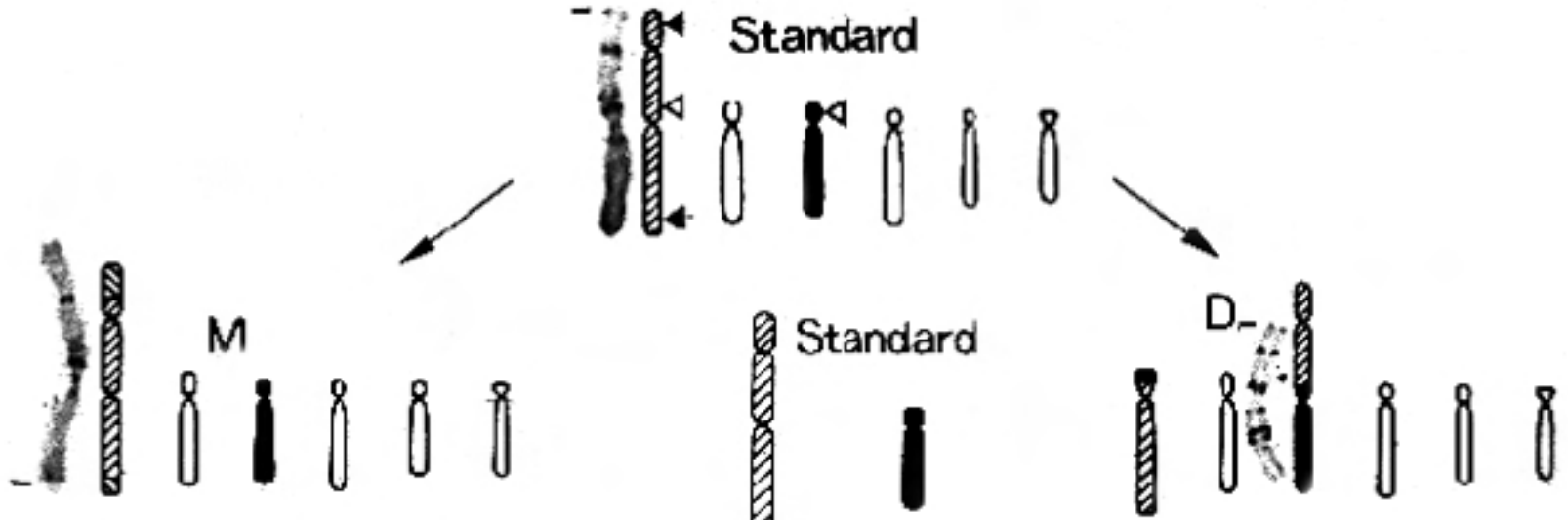


Secondary karyotype rearrangement through meiotic recombination



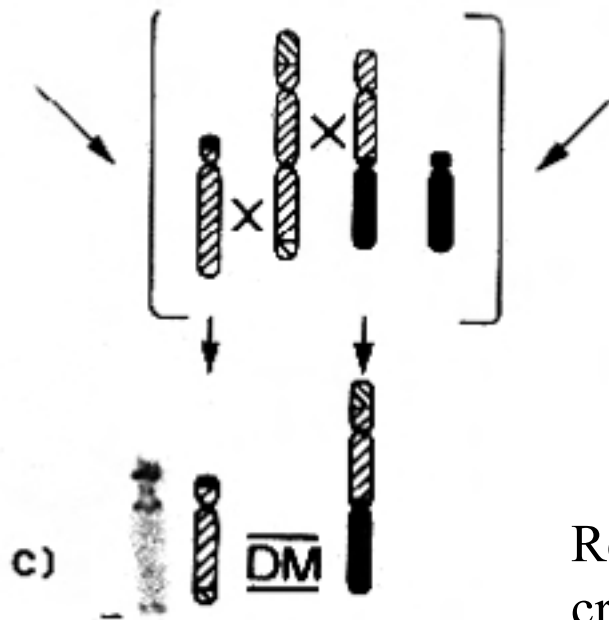






Pericentric inversion

Translocation



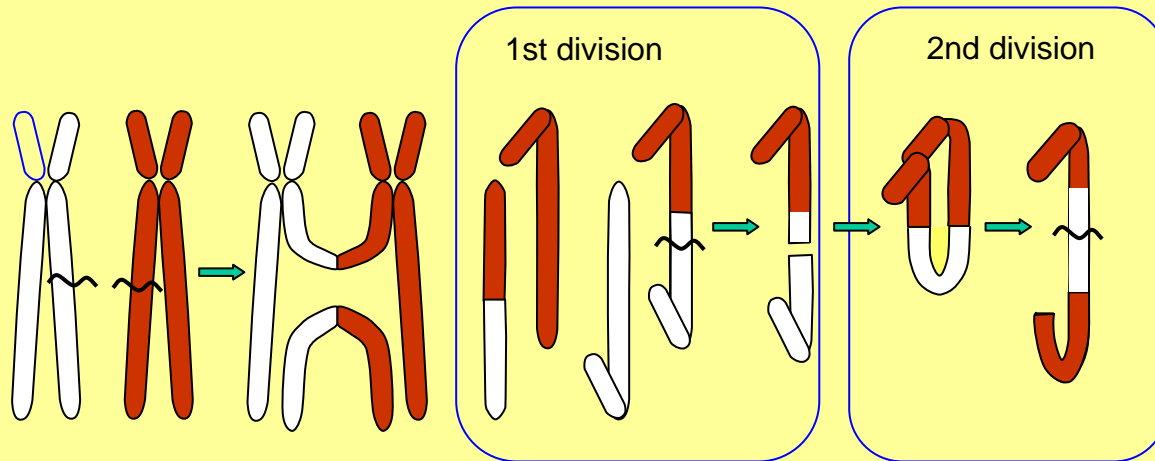
Rearrangement *via* double crossover

c)

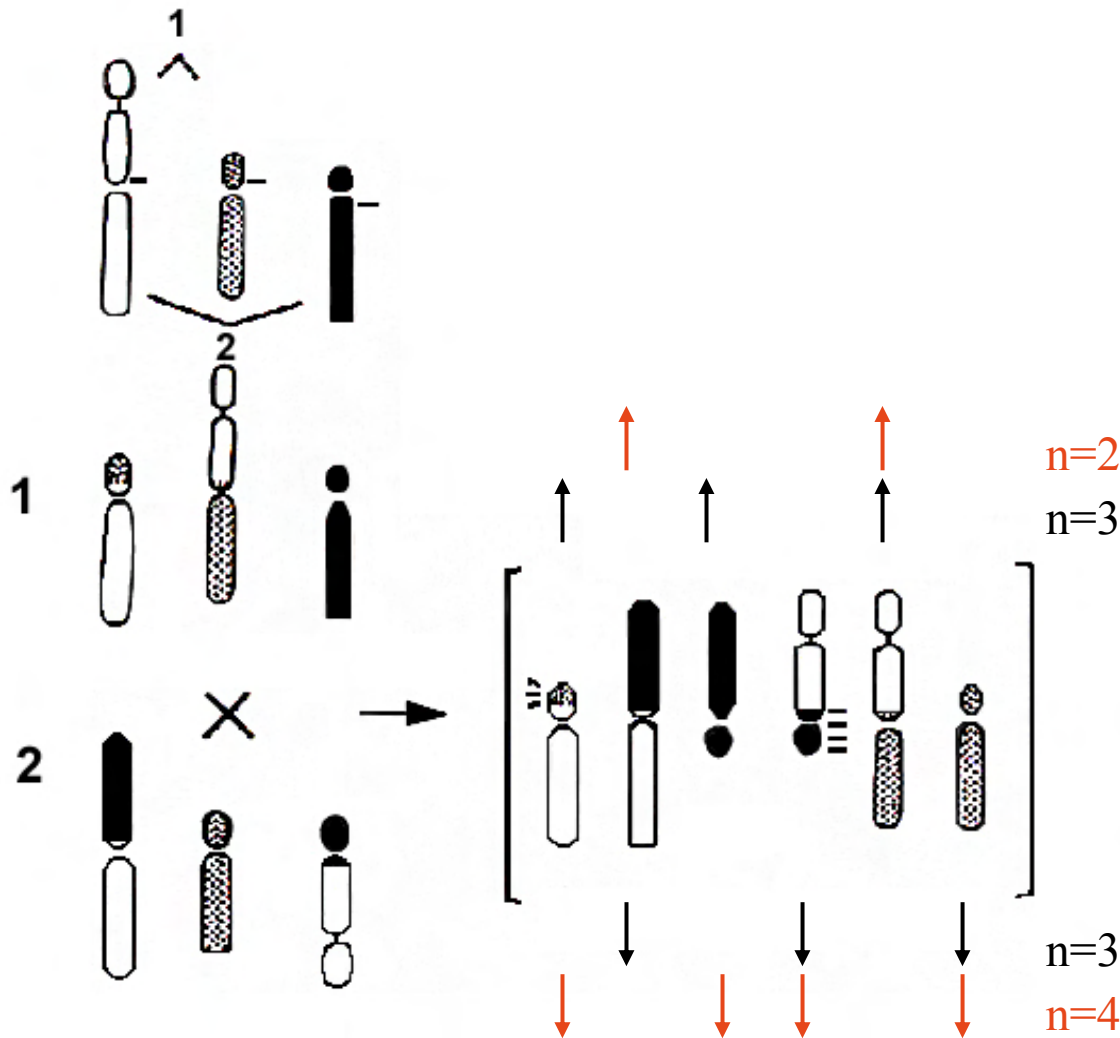
DM

Bridge.breakage-fusion cycles

lead to chromosome rearrangements
(duplications/inversions/deletions)



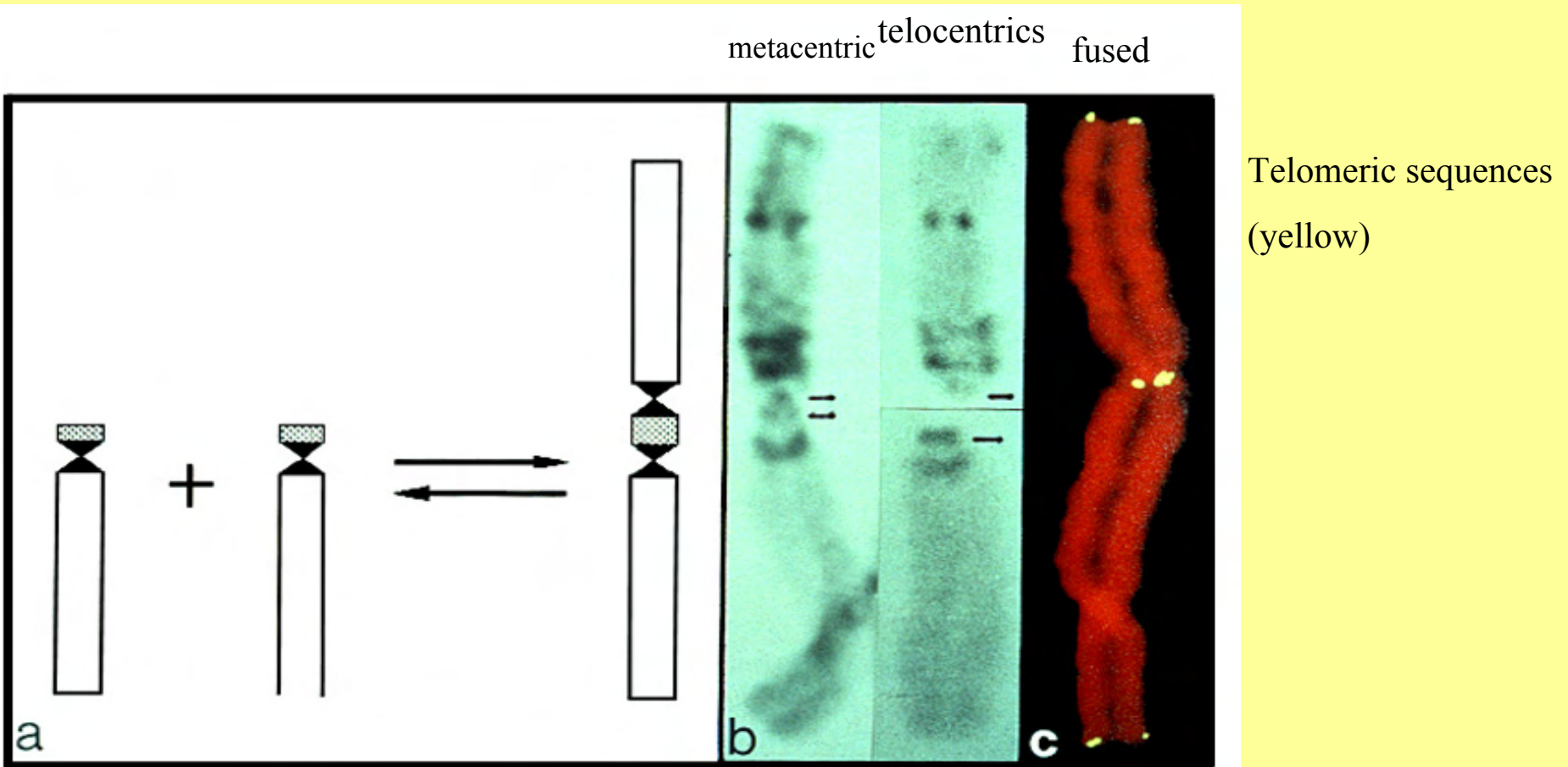
Alterations of chromosome numbers via mis-segregation from meiotic multivalents in individuals heterozygous for 2 translocations, with one chromosome involved in both translocations, and breakpoints close to centromeres

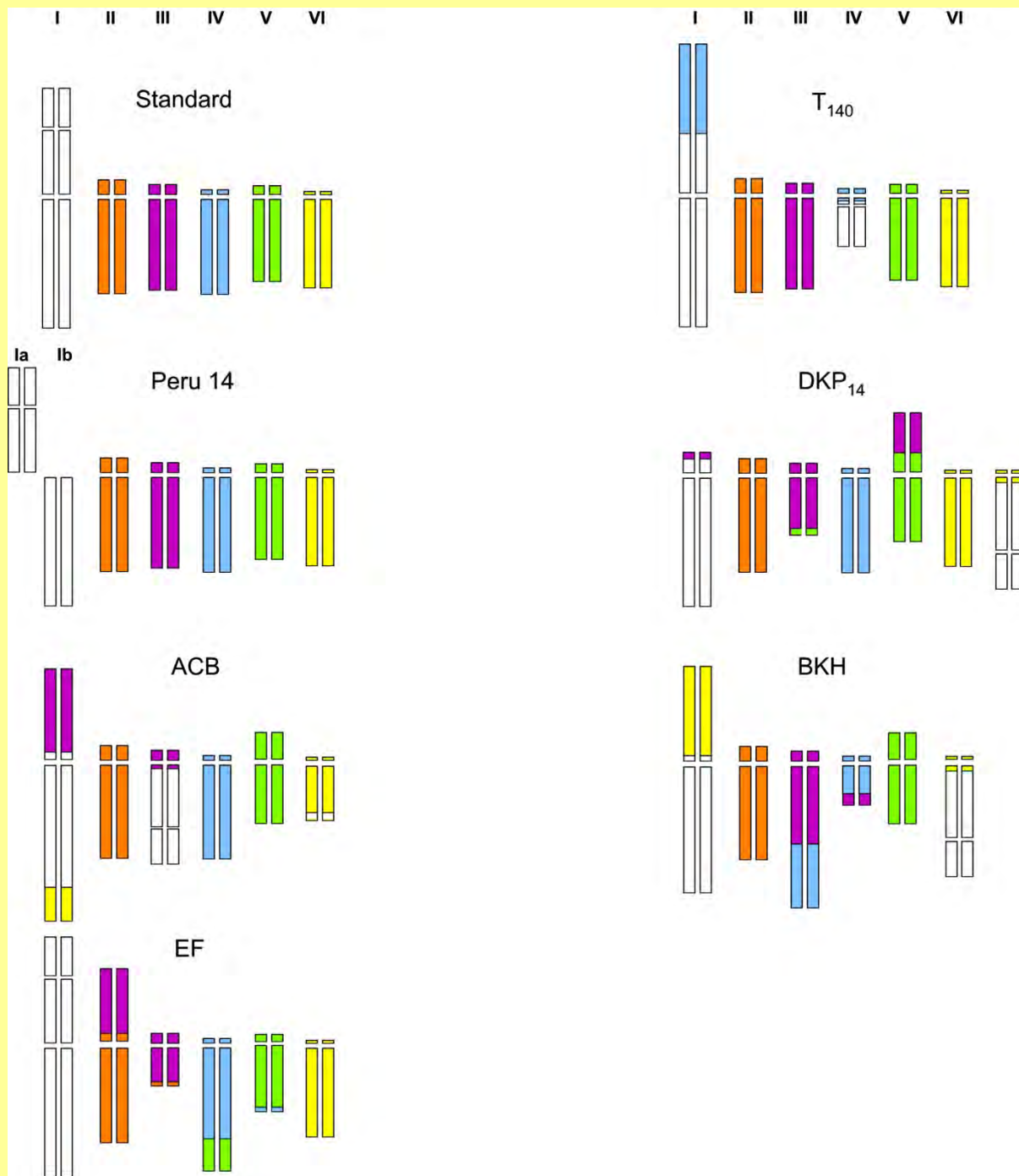


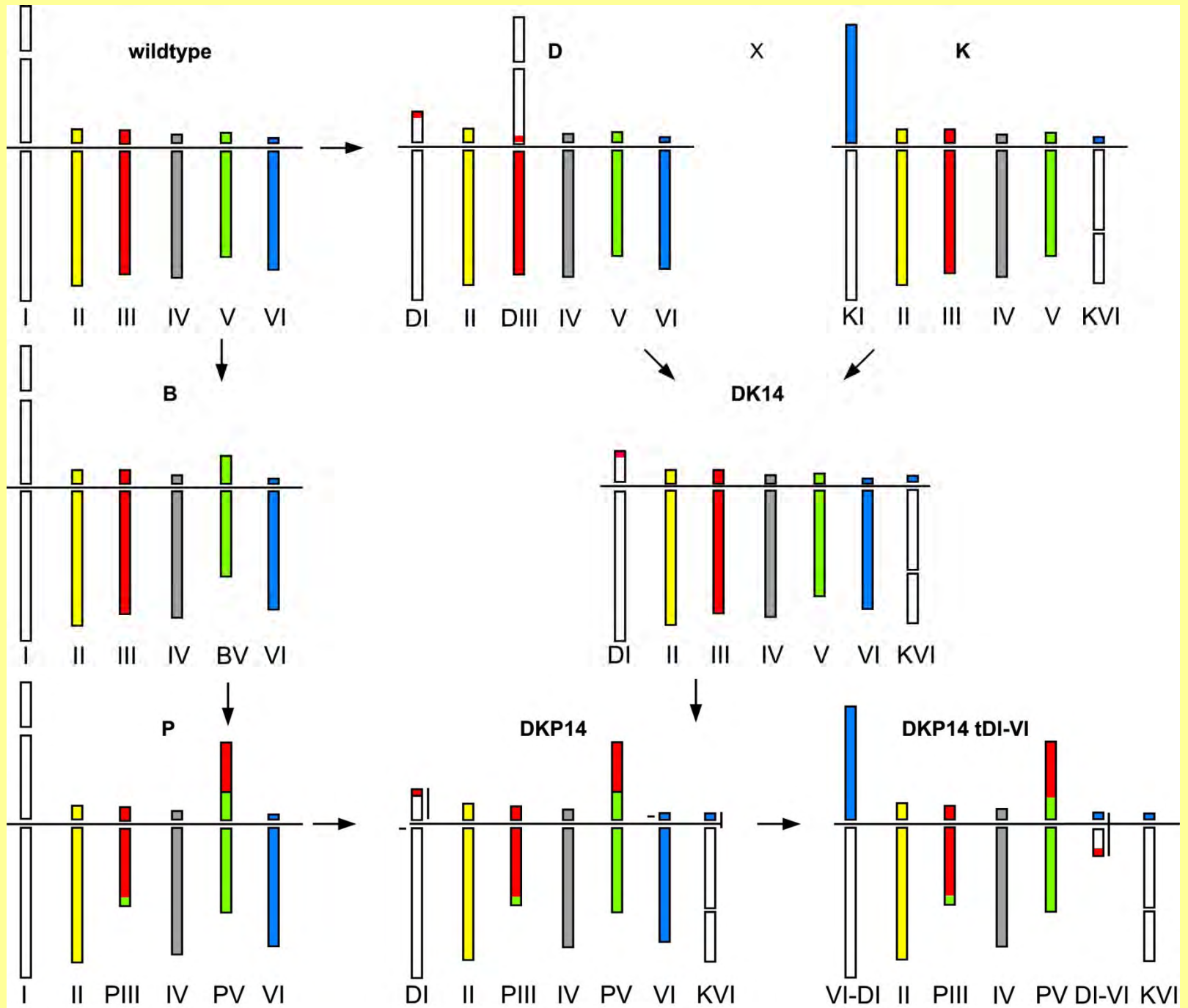
AJ 14



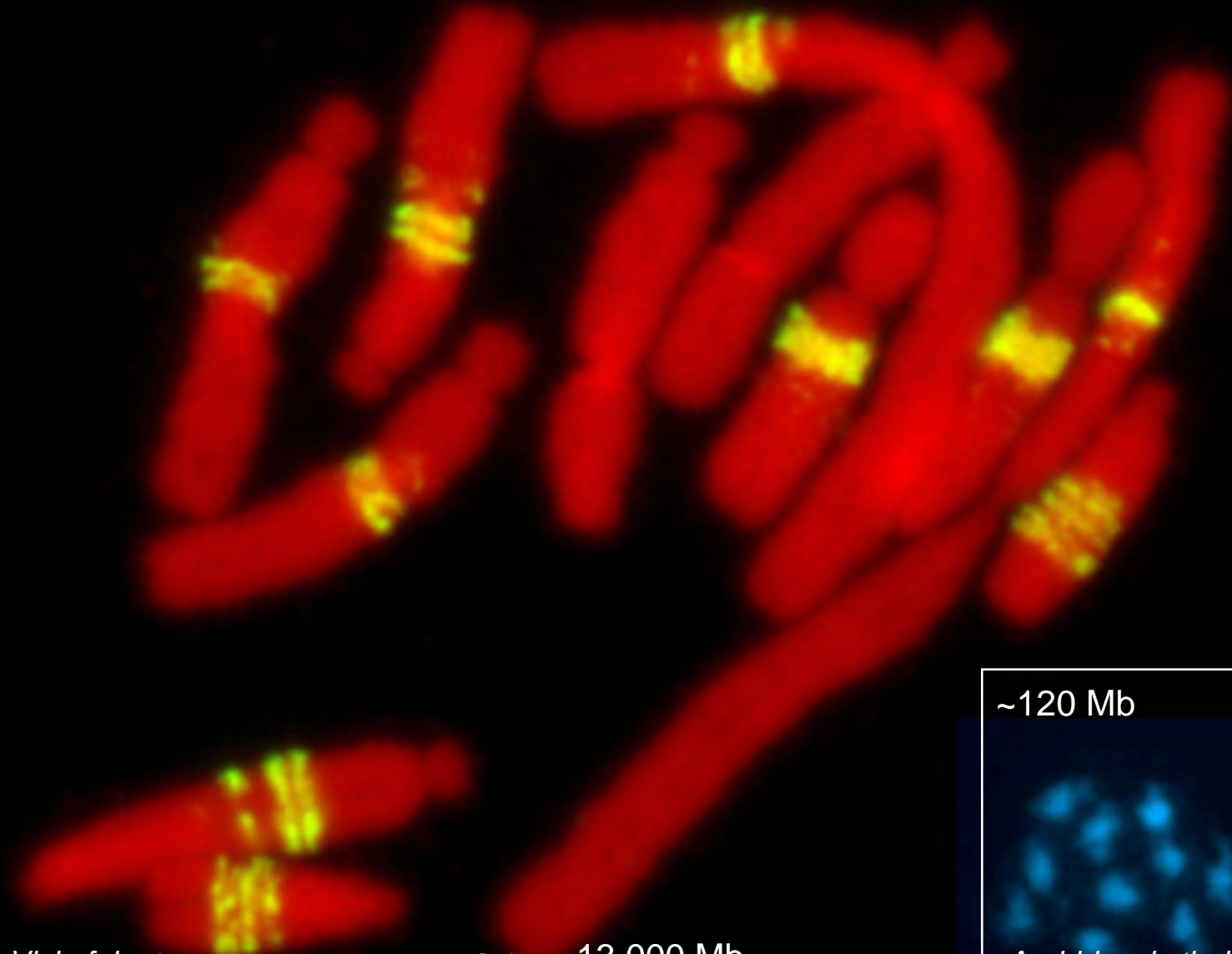
Alterations of chromosome number via reversible fusion of telocentric and fission of meta(di)centric chromosomes







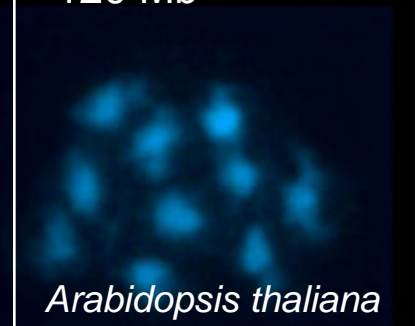
Evolution of chromosome size



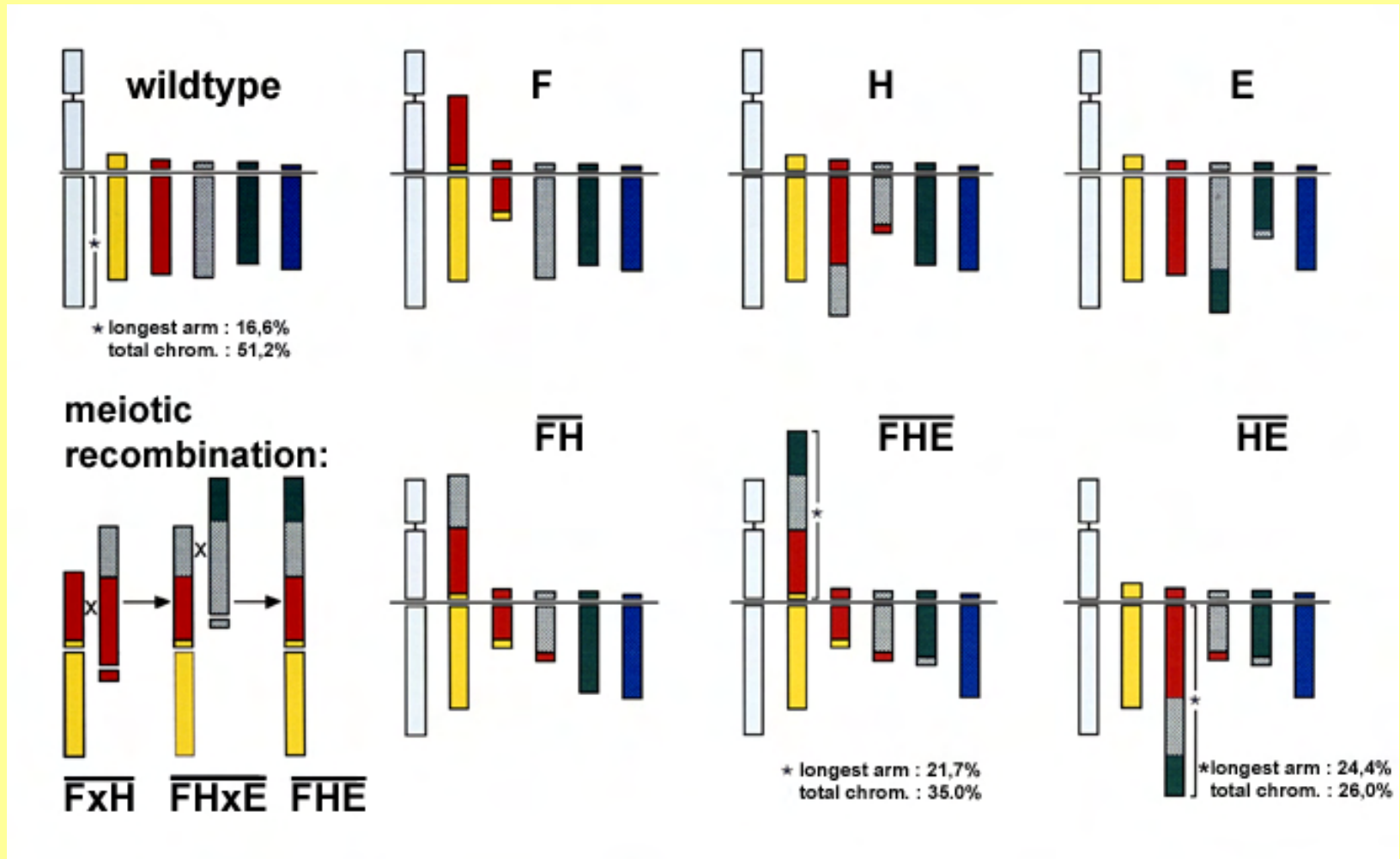
Vicia faba (reconstructed karyotype ACB) ~13,000 Mb

~120 Mb

Arabidopsis thaliana



Elongation of chromosome arms *via* meiotic recombination between partially homologous translocation chromosomes



Incomplete separation of ,over-long‘ chromosome arms during mitosis results in micronuclei and chromatin deletions

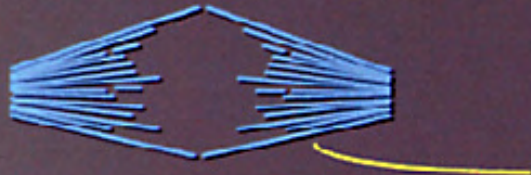


Chromosome arms $> 1/2$ of the average spindle axis length may disturb mitosis and organismic development

When chromosome separation causes problems



Wildtype
longest arm: $10.6 \mu\text{m}$



Hypothetical (stable) karyotype
longest arm: $13.5 \mu\text{m}$
arm = half spindle size at telophase



separation problems when:
longest arm $>$ telophase half spindle

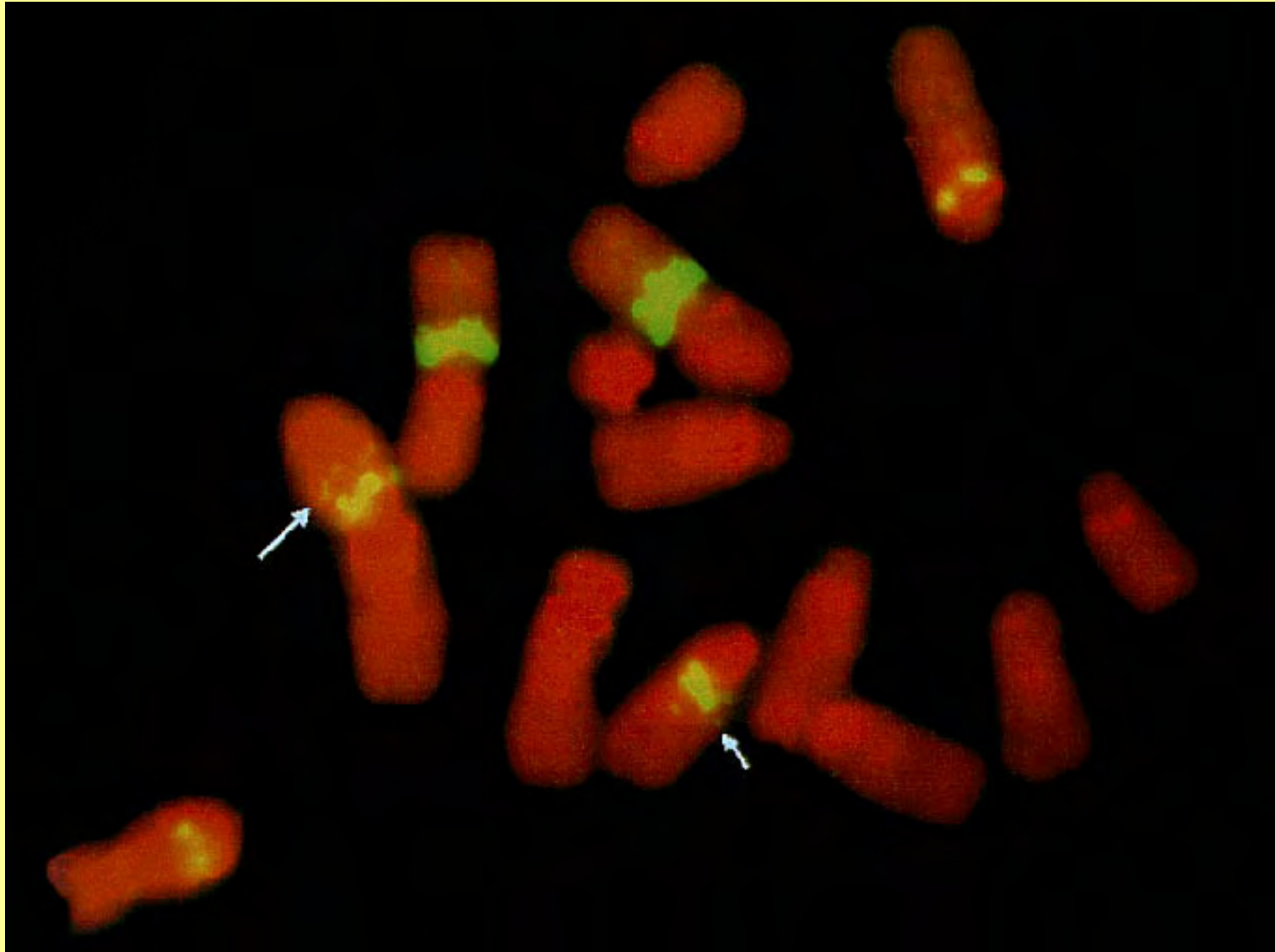
FHE
longest arm: $14.1 \mu\text{m}$

Mitotic/meiotic transmission of artificial chromosomes of different size

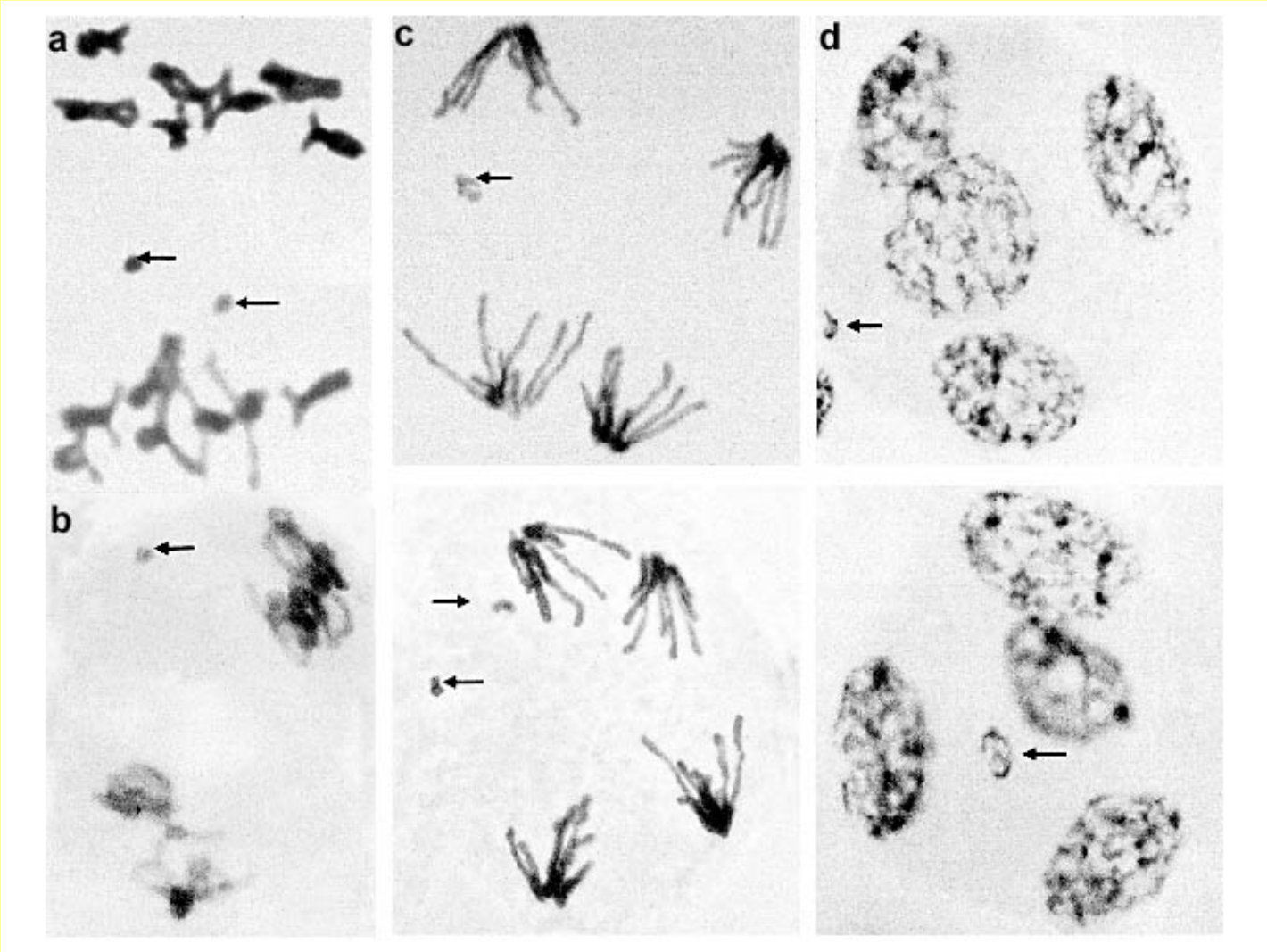
| Species | Minichromosome size | Mitosis | Meiosis | DNA content (1C) | References |
|------------|---|--|--|------------------|--|
| Yeast | ≥150 kb <55 kb ^a | 100% <100% | 100% <100% | 10 Mb | Murray et al., 1986 Murray and Szostack, 1983 |
| Drosophila | 1.3 Mb 0.2 Mb ^b | 100% ~87% | ~50% 0.3-14% | 150 Mb | Williams et al., 1998 |
| Mouse | ~60 Mb ^c ~30 Mb ^d 4.5 Mb ^e | ~99% >99% in vitro 30-80% in vivo | <1-33% <20% | 3.300 Mb | Telenius et al., 1999 Tomizuka et al., 1997 Shen et al., 1997, 2000 |
| Human | ~50 Mb ~8 Mb ~2.4 Mb ~5 Mb >10 Mb 5-20 Mb 5-10 Mb | 100% ^f 95% ^f 88-92% ^g ~99% ^h 99.8% ⁱ >90% 99% | 32-38% ^j 31-35% ^k | 3.500 Mb | Mills et al., 1999 Ikeno et al., 1998 Harrington et al., 1997 Heller et al., 1996 Kuroiwa et al., 2000 Shinohara et al., 2000 Tomizuka et al., 2000 Voet et al., 2001 |
| Field bean | ≤700 Mb | 100% | 17.0% ^l 66.7% ^m | 13.500 Mb | Schubert, 2001 |

- a Linear centric plasmid.
b Neocentric fragment.
c sat-DNA-based artificial chromosome.
d Human Y-derived artificial chromosome.
e Human artificial chromosome with mouse sat-DNA.
f In hamster cells.
g In chicken DT 40 cells.
h In human HT 1080 or CHO cells.
i In chicken DT40, mouse ES cells and chimeric mouse.
j Up to F3 in mice.
k F1 in mice.
l Hemizygous.
m Homozygous.

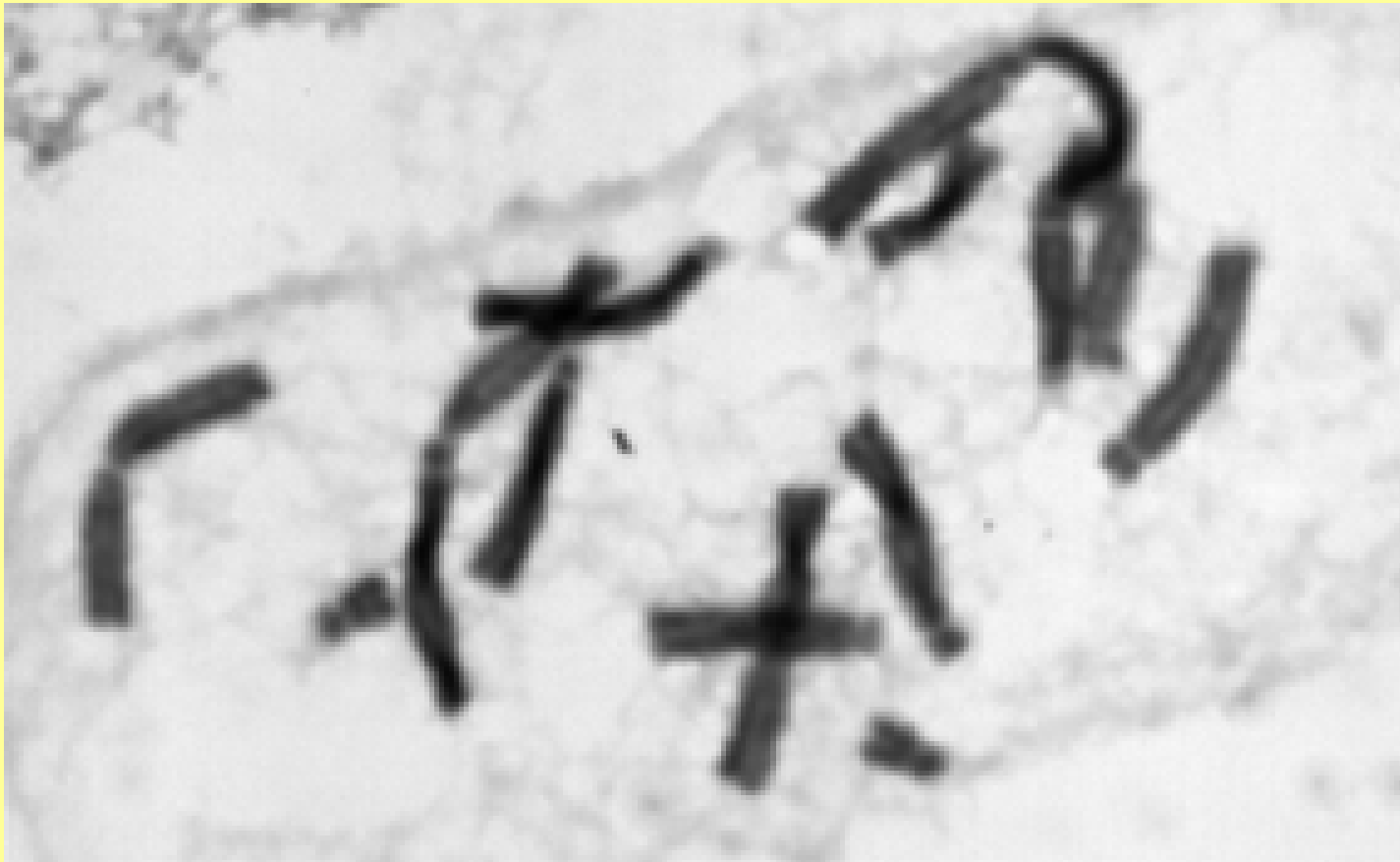
Minichromosome of the field bean carrying
<5% of the genome as duplication

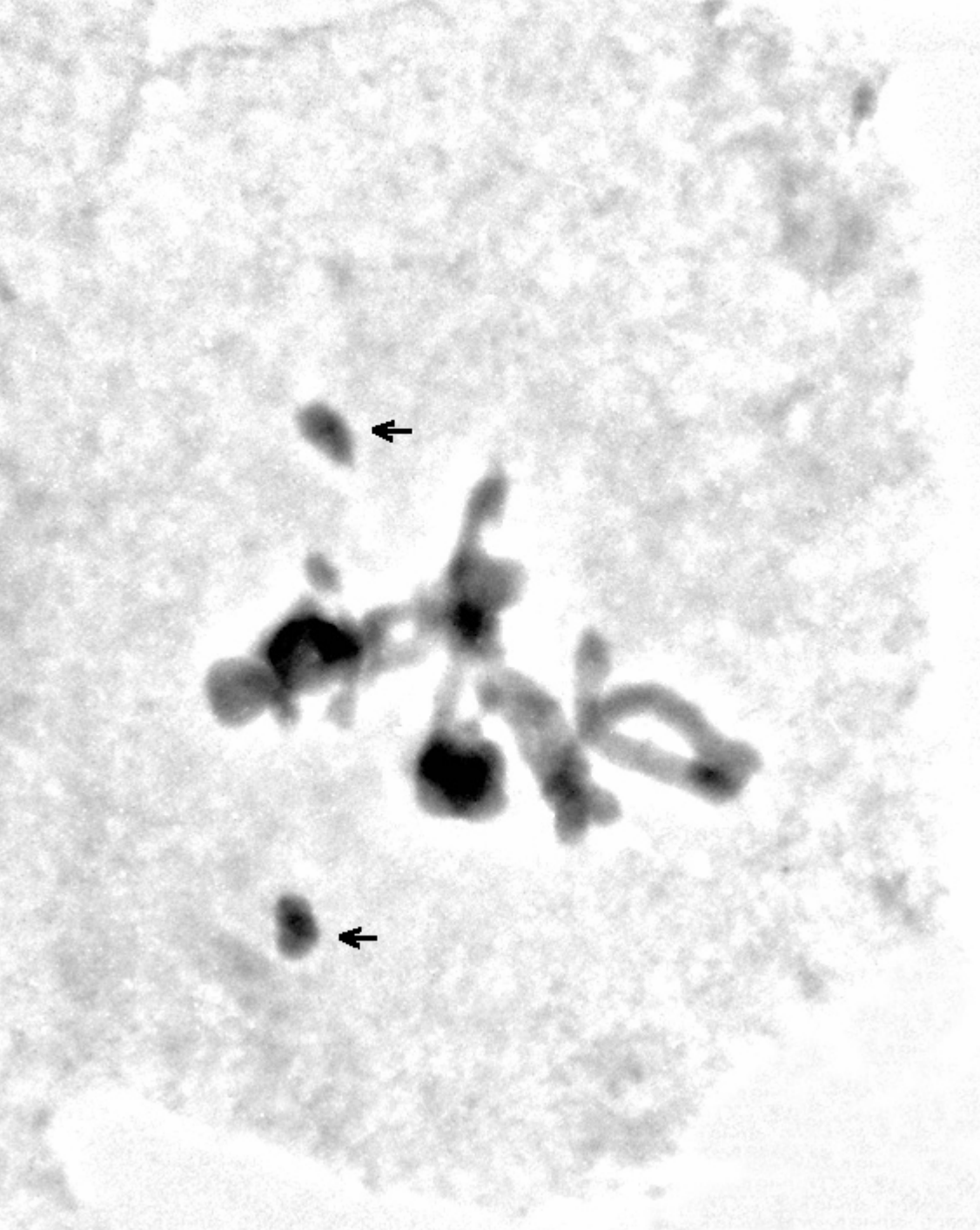


Frequently the minichromosome gets lost during meiosis



Field bean karyotype with homozygous
minichromosome (2n=14)





Failure of meiotic pairing
leads to loss of
minichromosomes in 33%
of germ cells

Sequence localiation by *in situ* hybridisation:

Fluorescence in situ hybridisation (FISH),

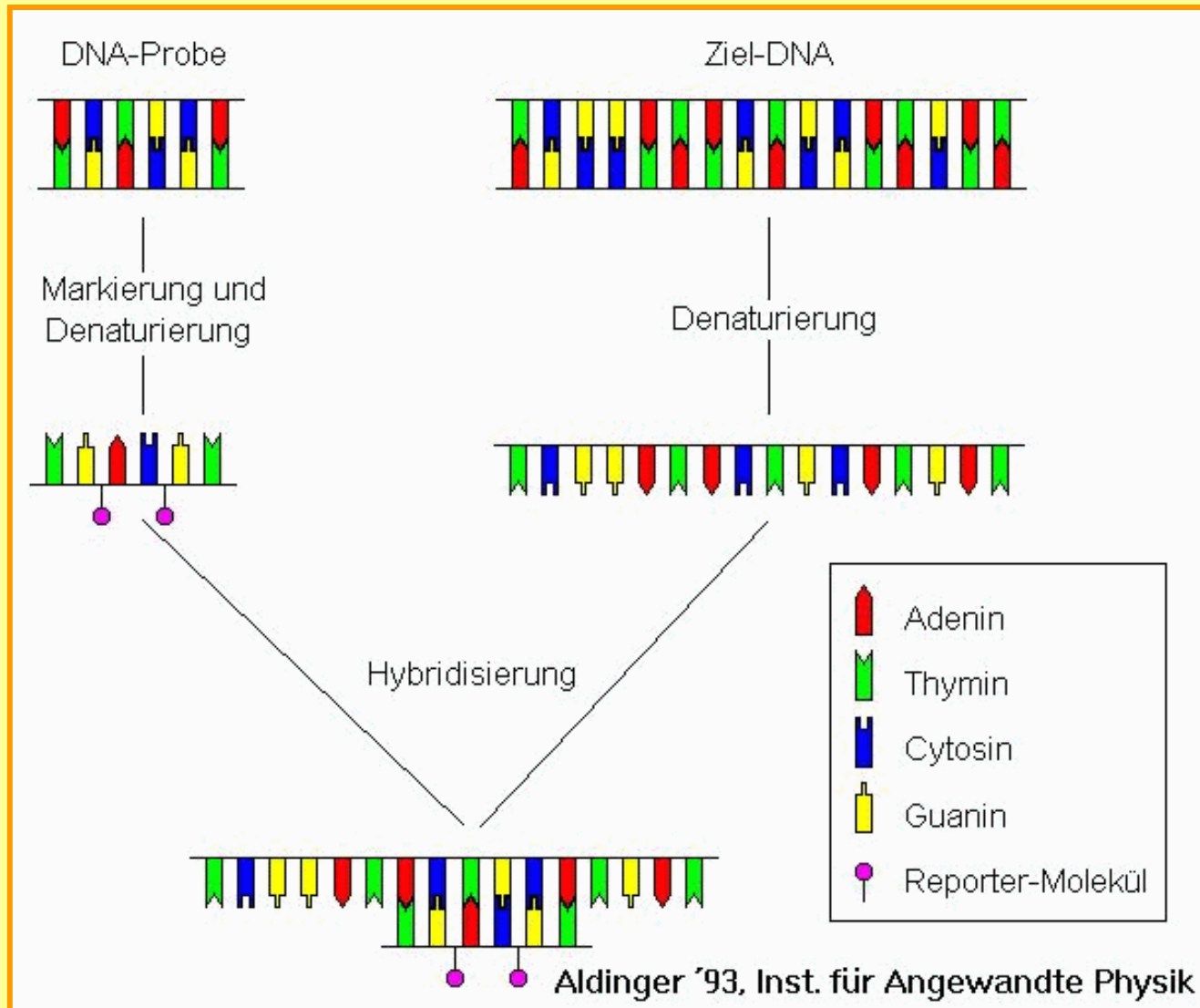
Genomic in situ hybridisation (GISH),

Primed in situ synthesis (PRINS),

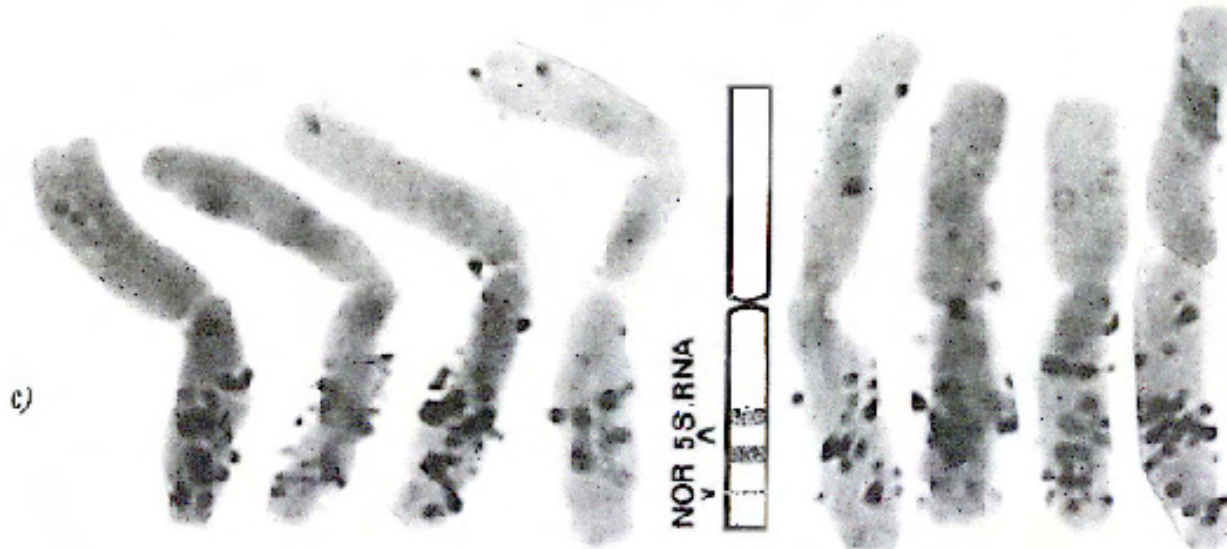
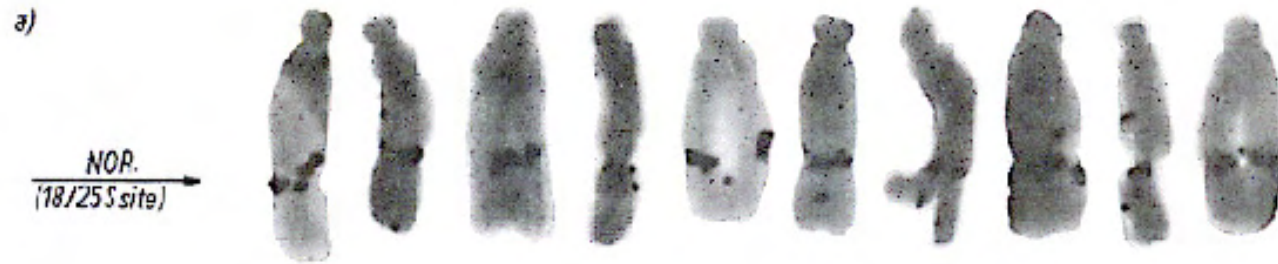
Chromosome painting

Immunostaining of chromosomal/nuclear antigens *in situ*

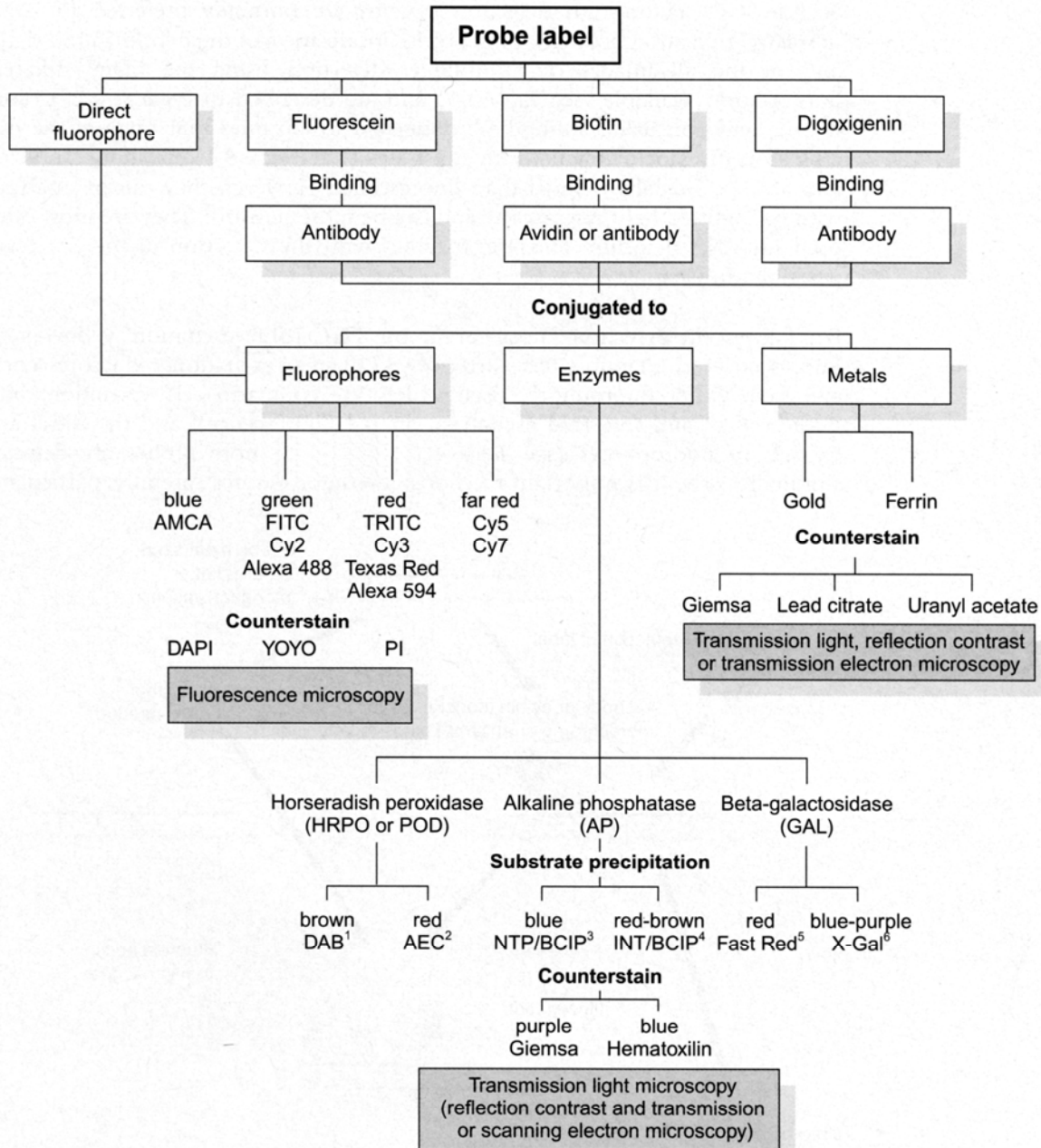
In situ Hybridisation



In situ hybridisation with radioactive (^3HT ; ^{125}J) rDNA probes

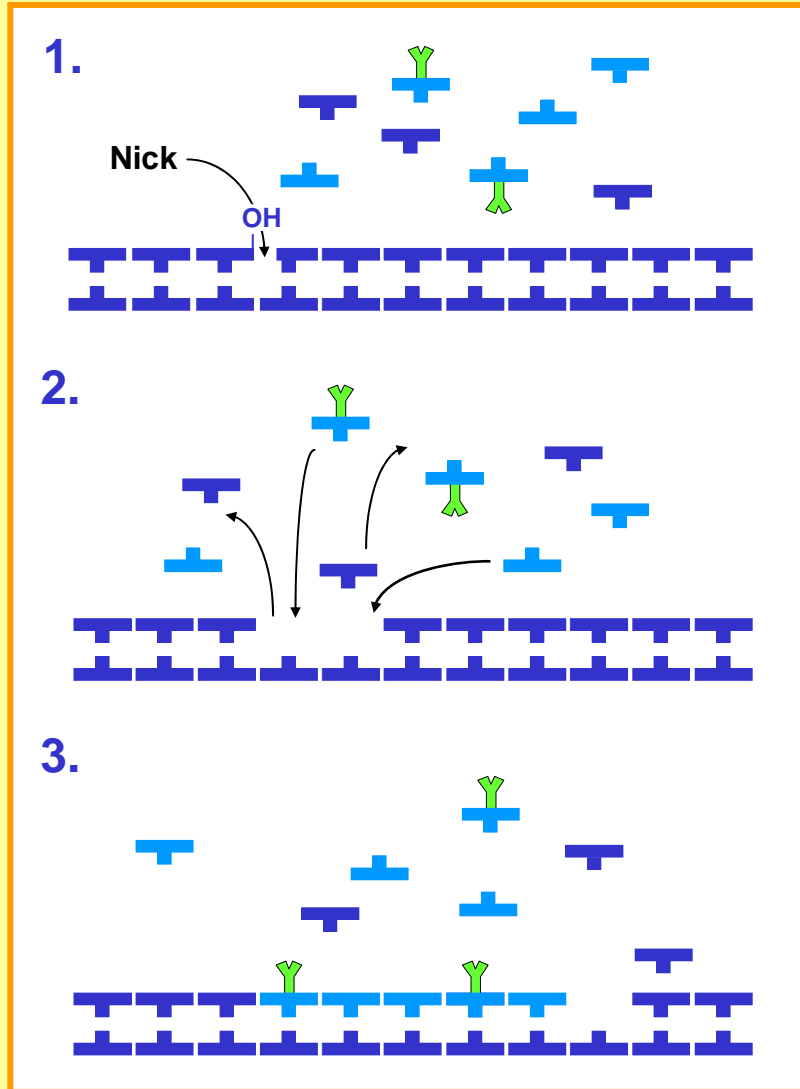


Probe Labels



Nick Translation

(Rigby et al. 1977)

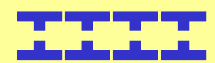


Induction of single strand nicks

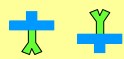
Enzyme: DNase I

Excision of nucleotids at the nick and
de novo synthesis using labeled nucleotids

Enzyme: DNA Polymerase I



Template-DNA



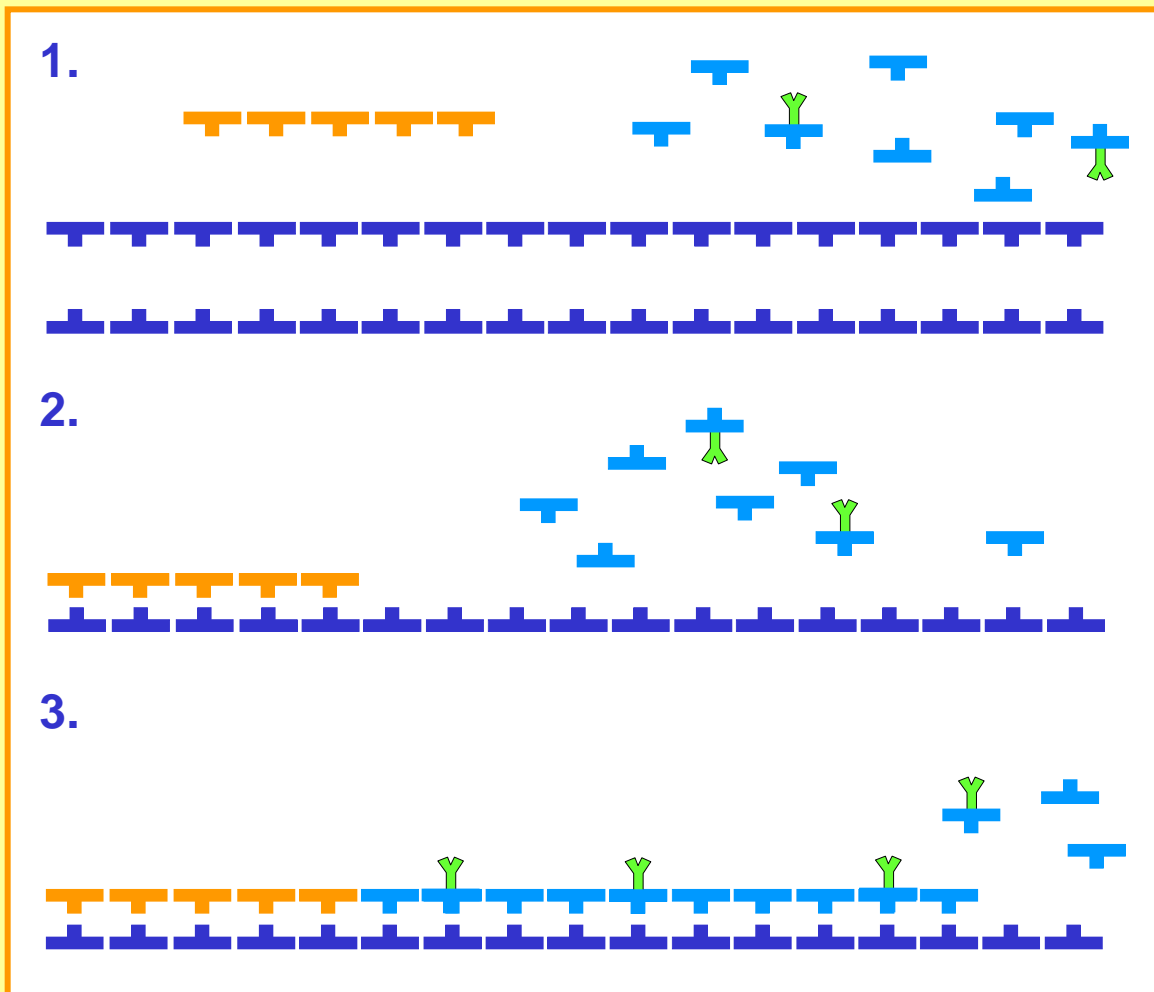
markierte Nukleotide



unmarkierte Nukleotide

Random Priming

(Feinberg and Vogelstein 1983)



Denaturation of probe DNA

Annealing of random primers

Extension of primers by incorporation of labeled nucleotids

Enzyme: Klenow fragment



Random primer



Template-DNA



unlabeled
nukleotid



labeled
nukleotid

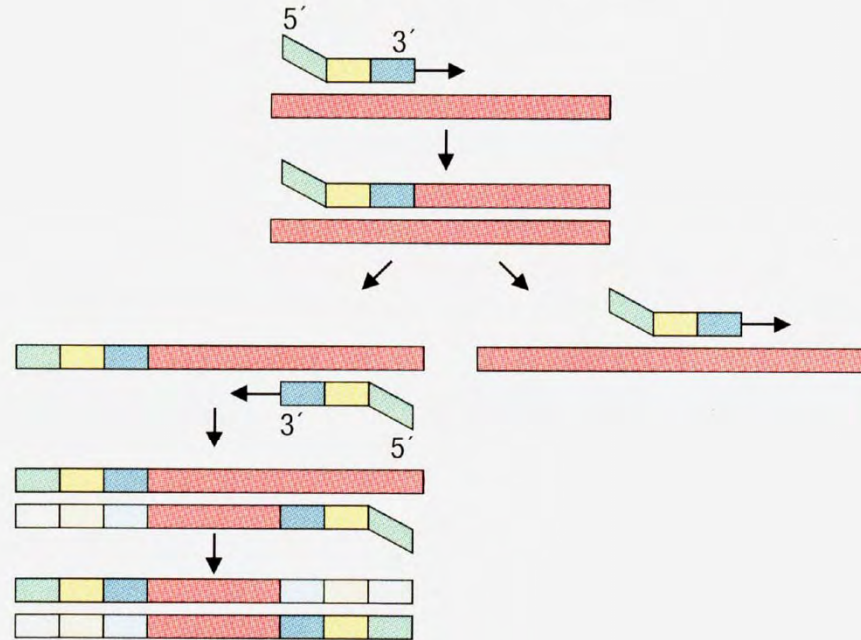
DOP-PCR

for preferential random
amplification of
transcribed sequences

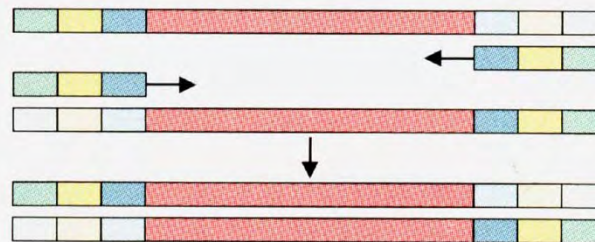
(Telenius et al. 1992)

5' CCGACTCGAG NNNNNN ATGTGG 3'

1. Low stringency PCR ($T_a = 30^\circ\text{C}$; 5 cycles)
→ frequent priming at multiple sites



2. Higher stringency PCR ($T_a = 62^\circ\text{C}$; 35 cycles)
→ specific priming of pre-amplified sequences



18/25S rRNA genes

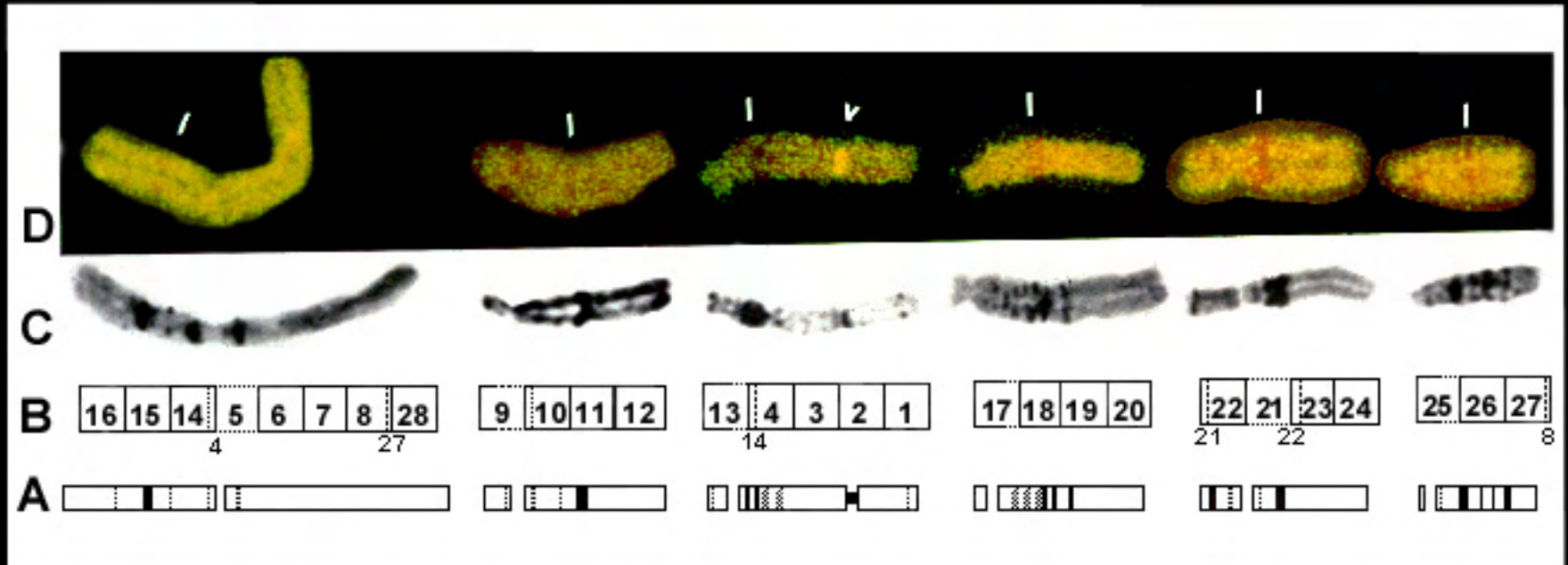
5S rRNA genes



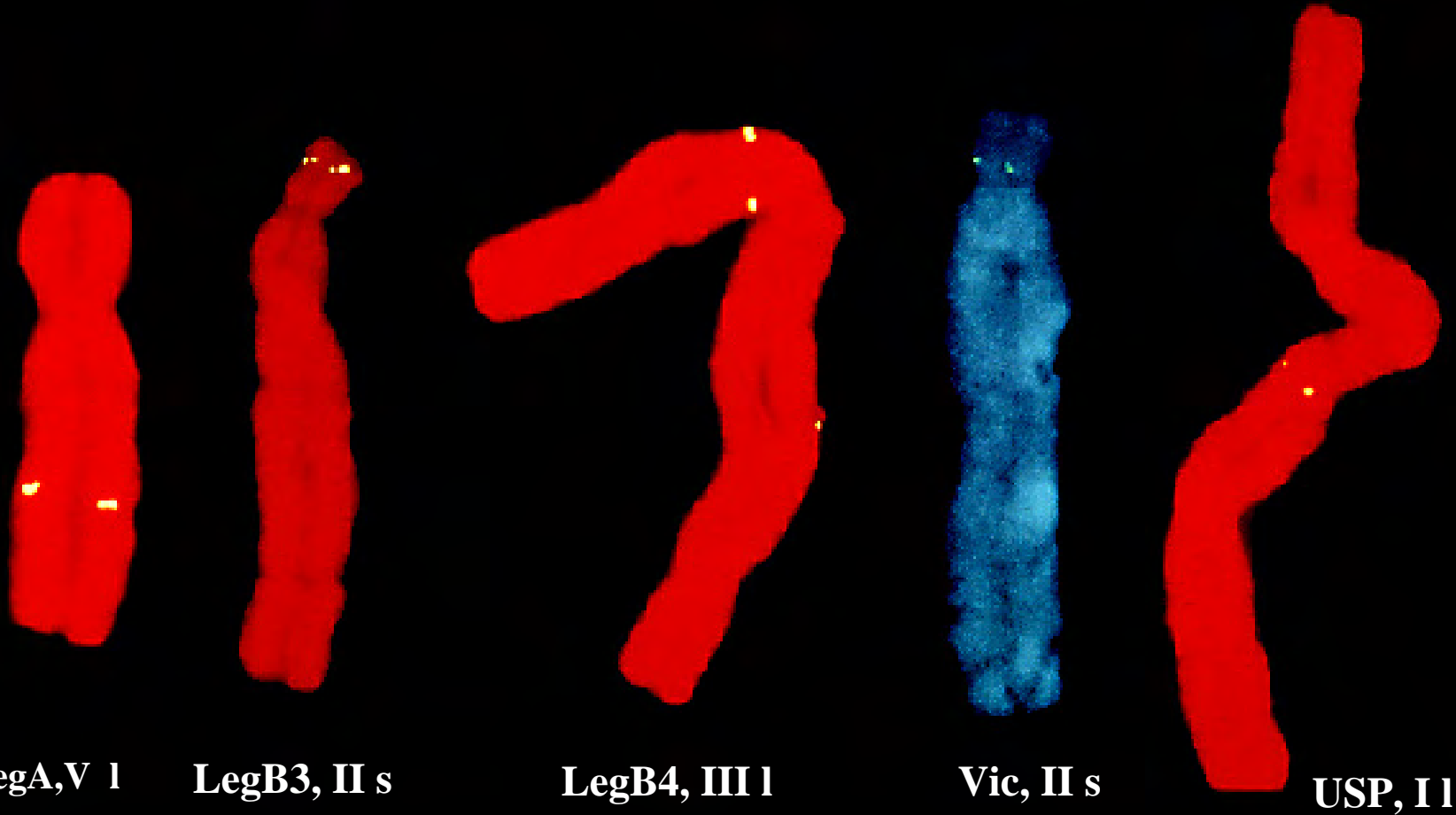
Helianthus annuus

5 μm

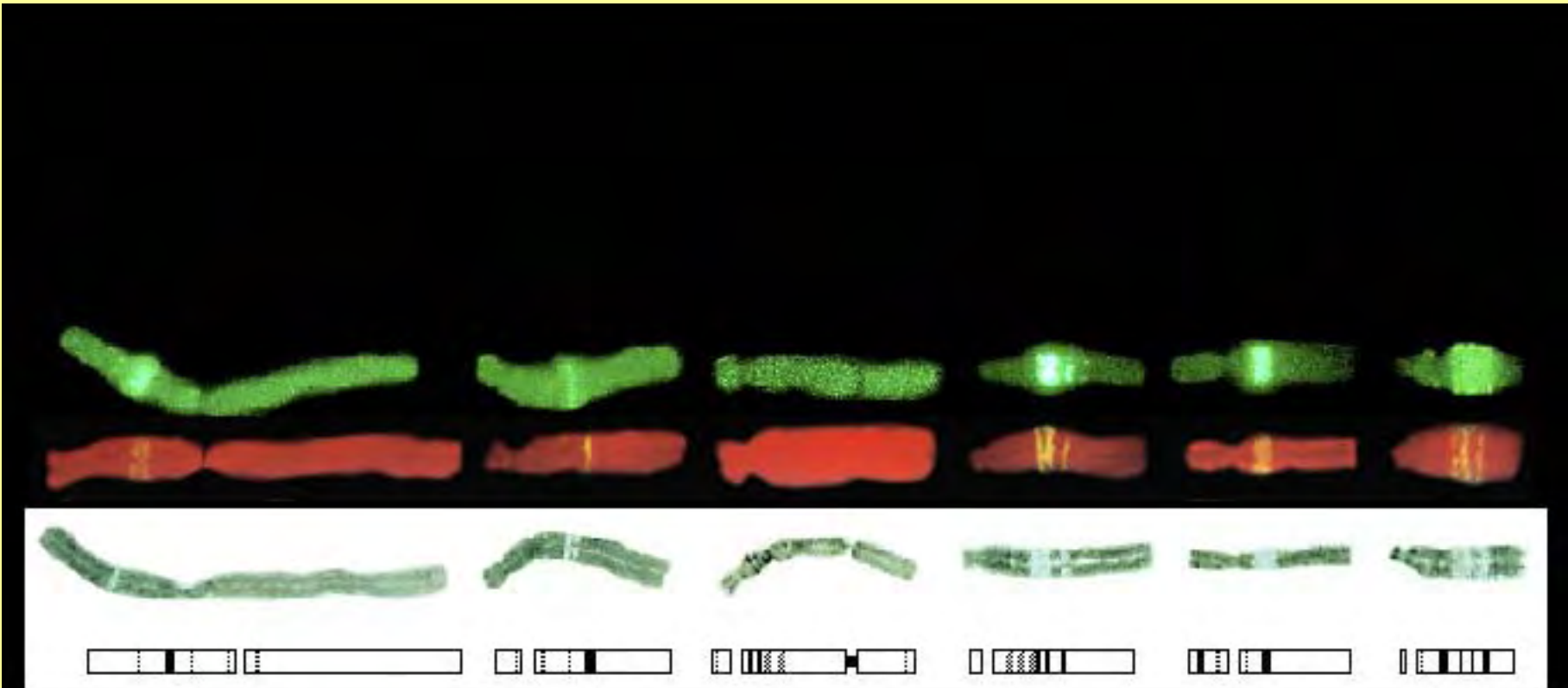
FISH with cDNA containing retroelement sequences on field bean chromosomes

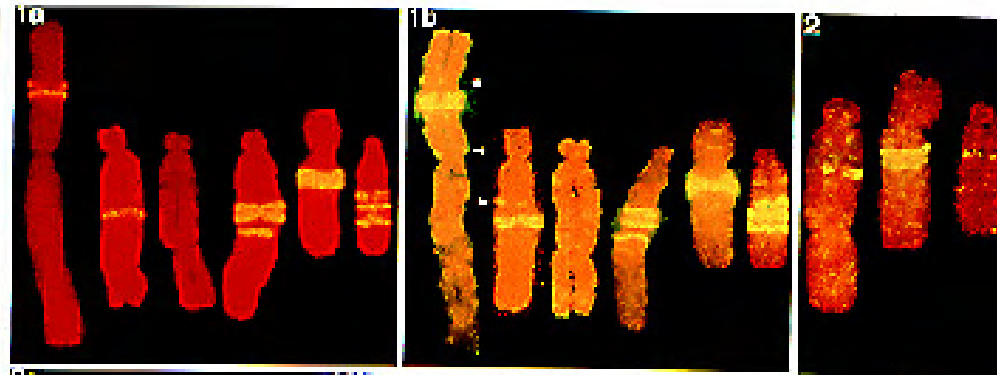


FISH with low-copy sequences on field bean chromosomes



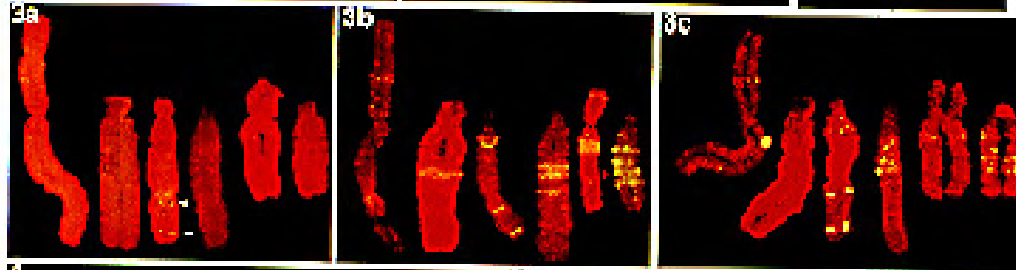
Field bean chromosomes after *FokI*-digestion or after
FISH and PRINS with *FokI*-elements (59bp)



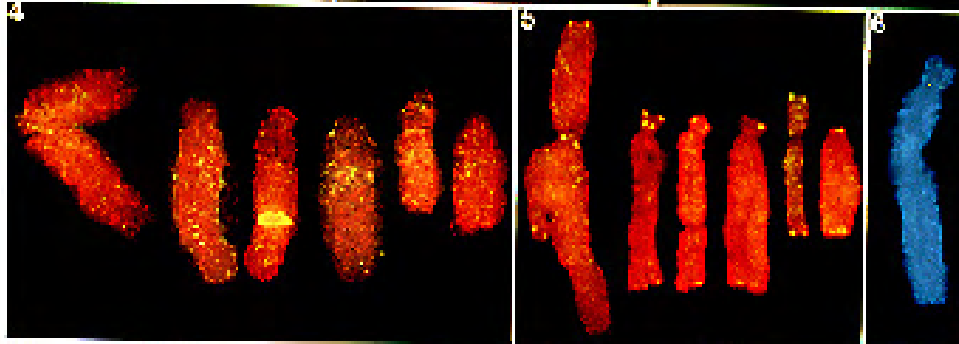


V. faba

Fok/pVf7
(PRINS)

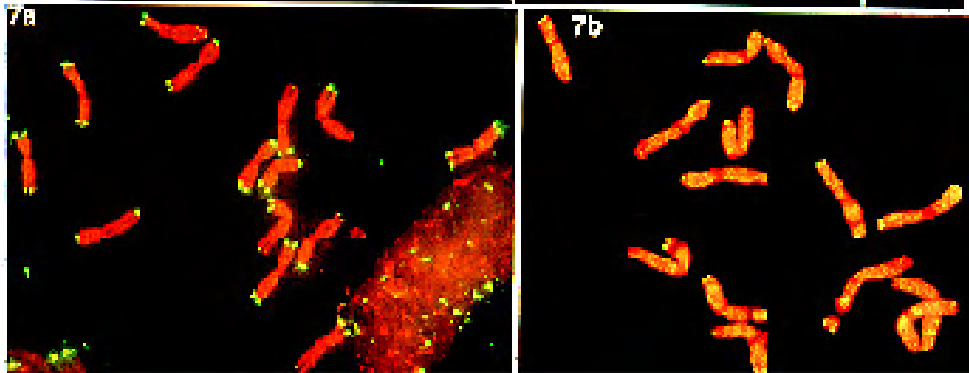


5S rDNA
(PRINS)



45S rDNA/telomeric
(PRINS)

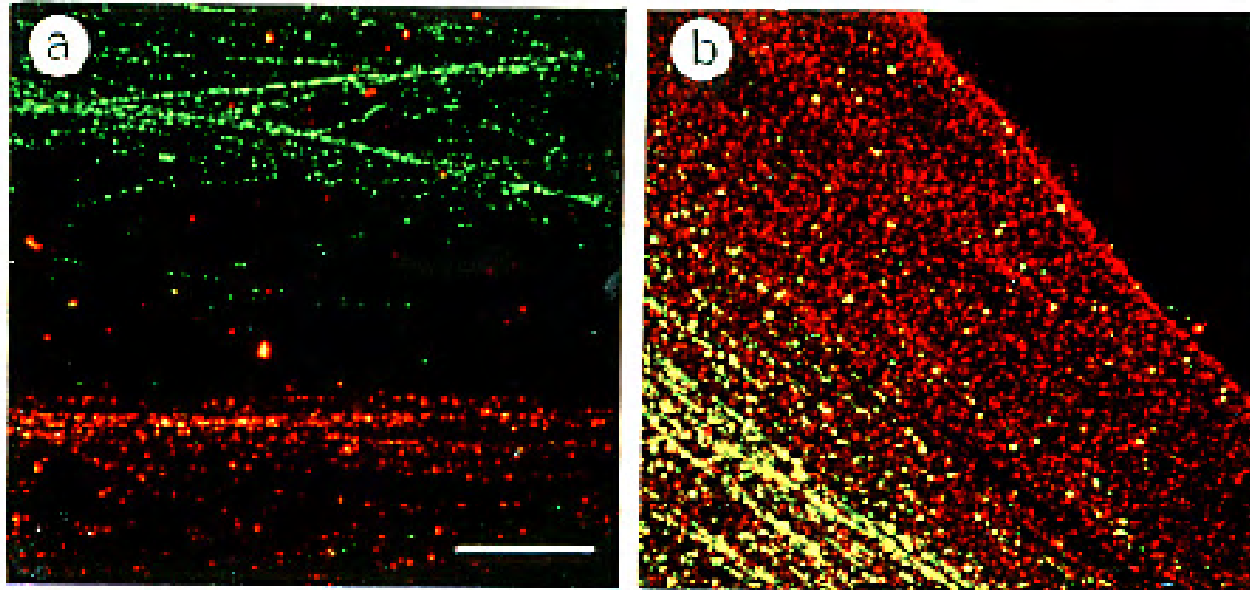
Vicilin (FISH)



barley

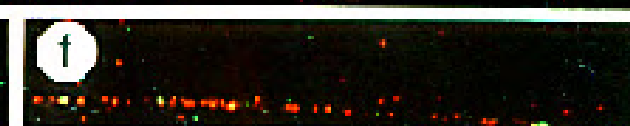
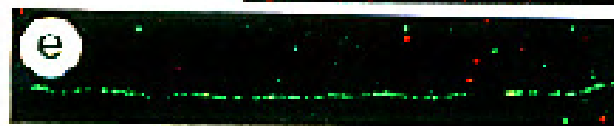
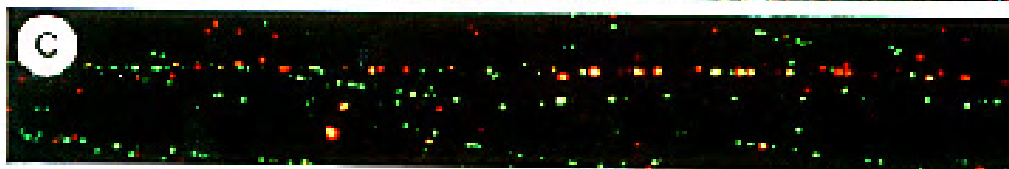
subtelo-repeat
(FISH/PRINS)

Extended fibre-FISH on *A. cepa* chromatin fibers

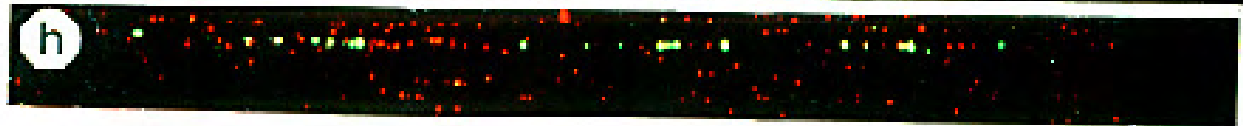


Sat-DNA (red),
rDNA (green)

Sat-DNA (red),
Copia (green)



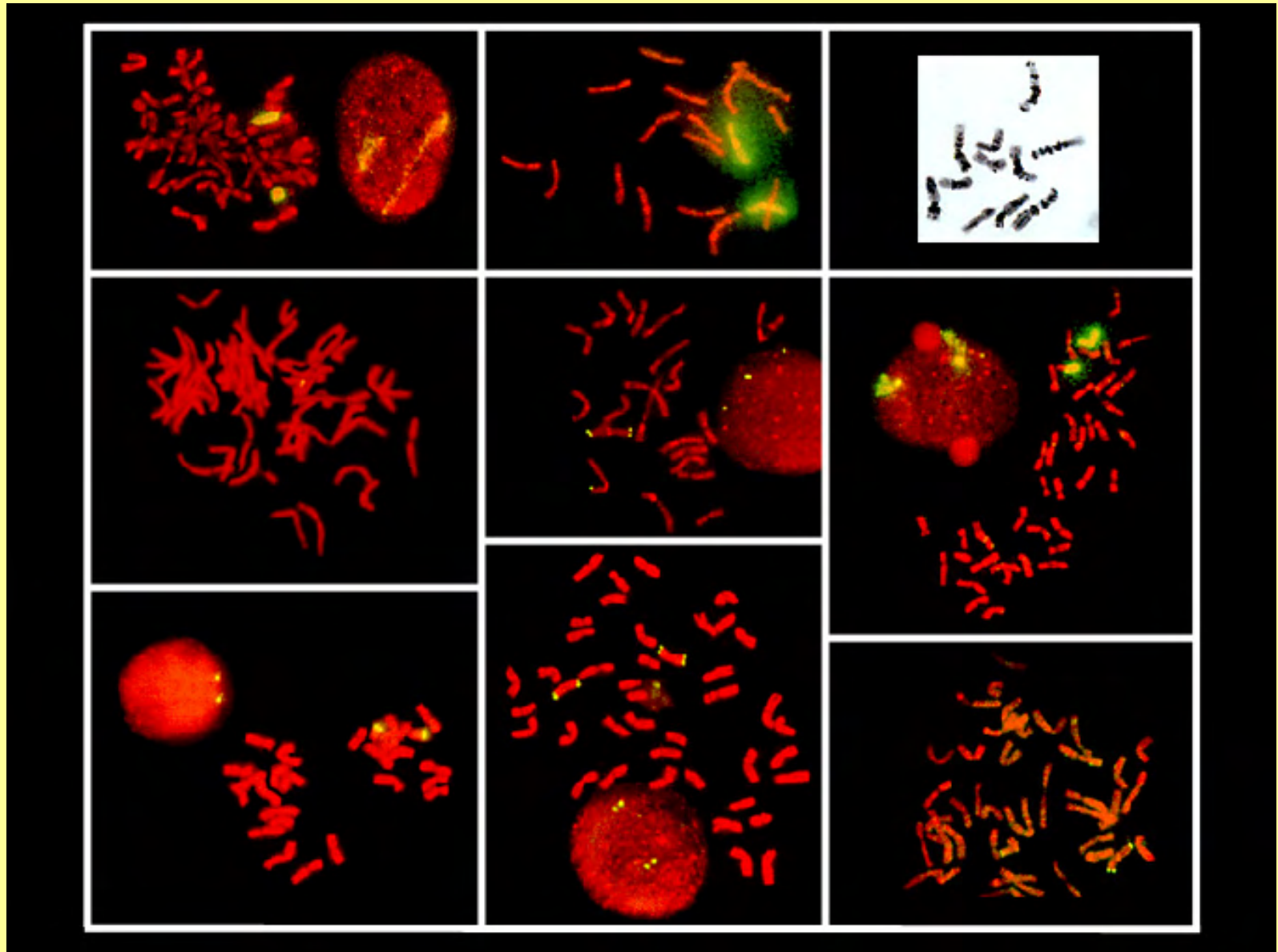
Sat-DNA (red),
Copia (green)



Sat-DNA (red),
En/Spm (green)

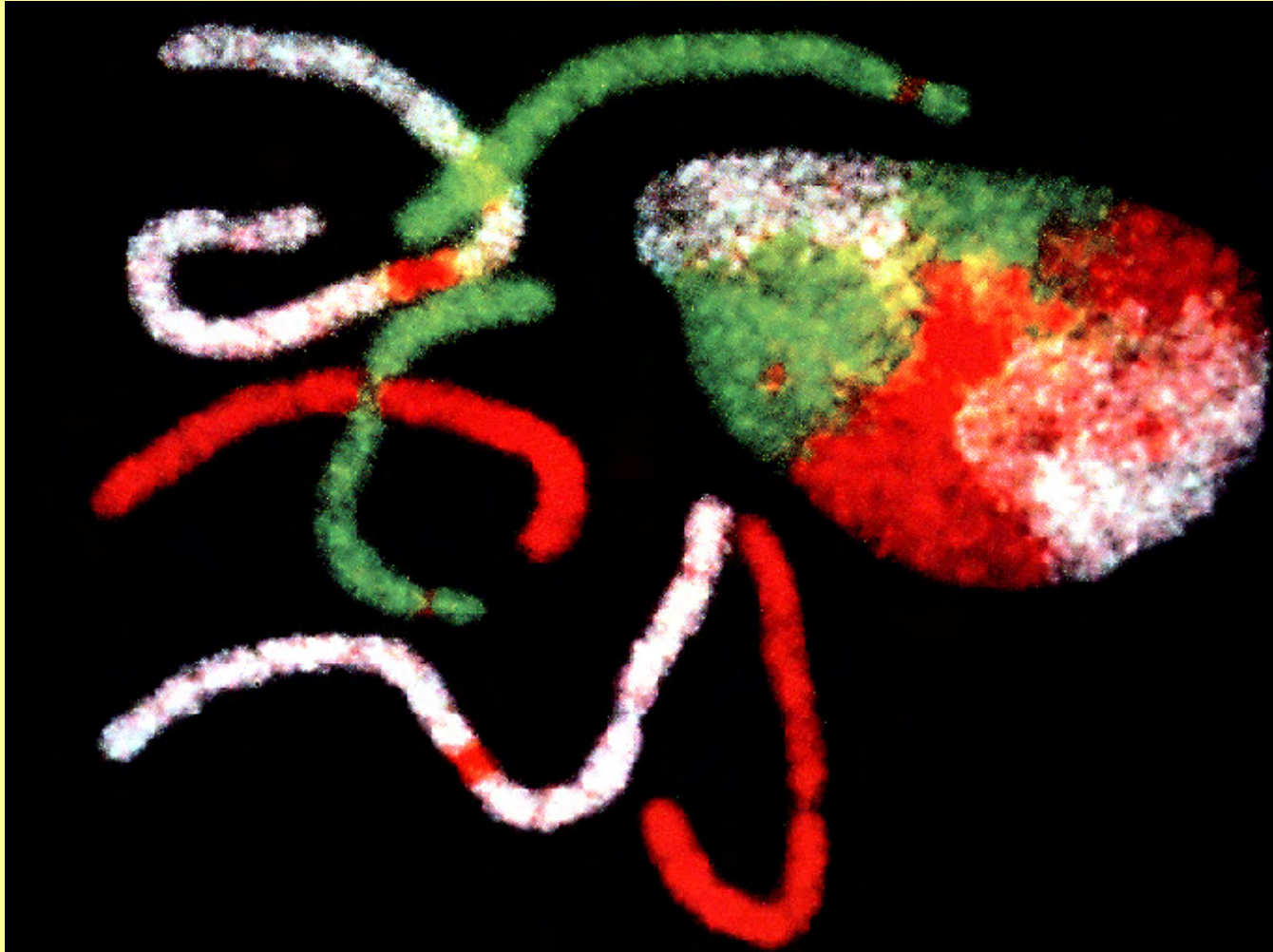
GISH of barley chromosomes 4HL and 7H in wheat background

(Schubert et al. 1998, Plant J. 14:489-495)

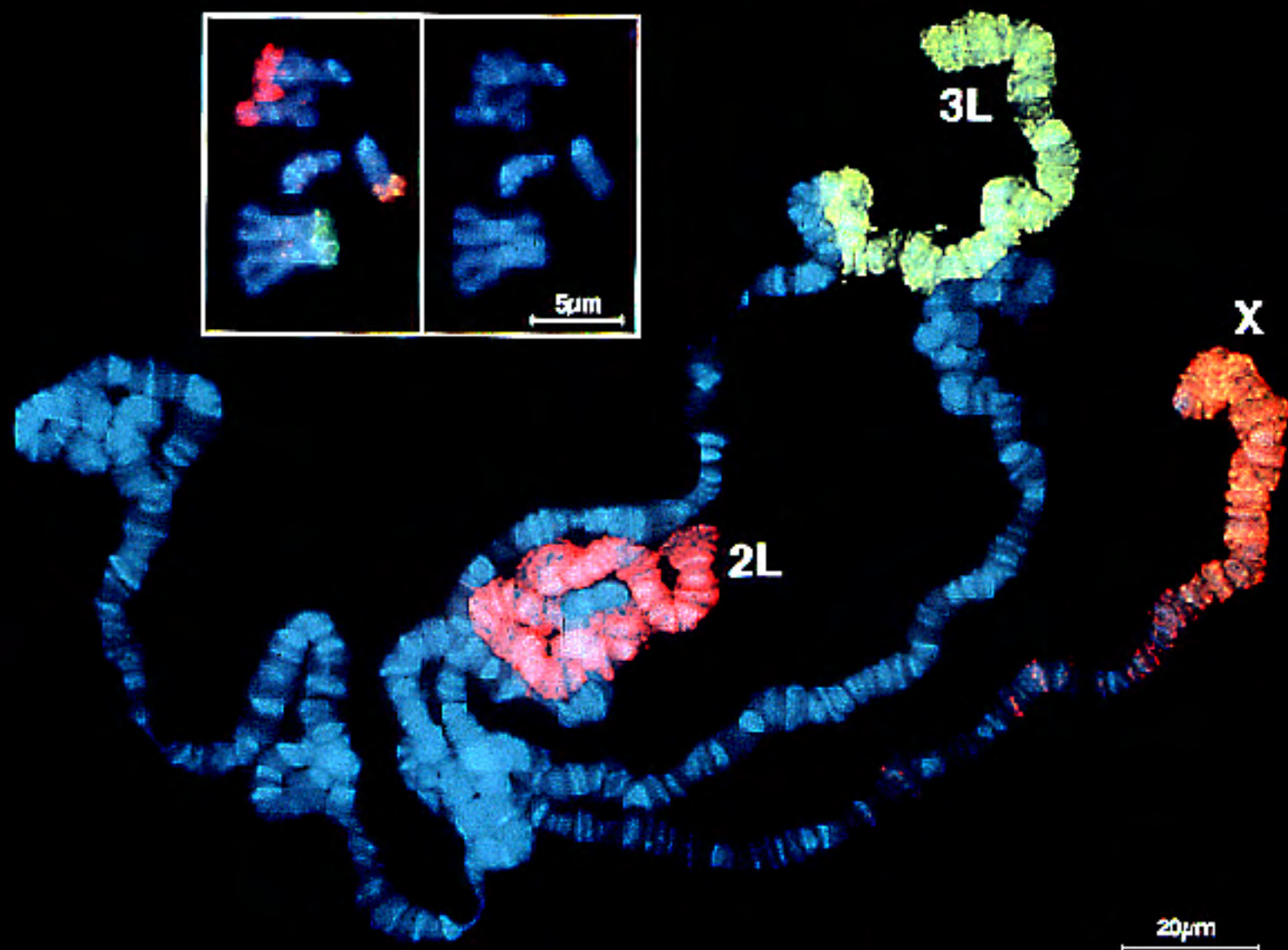


Painted Indian muntjak chromosomes

(F. Yang & M. Ferguson-Smith)

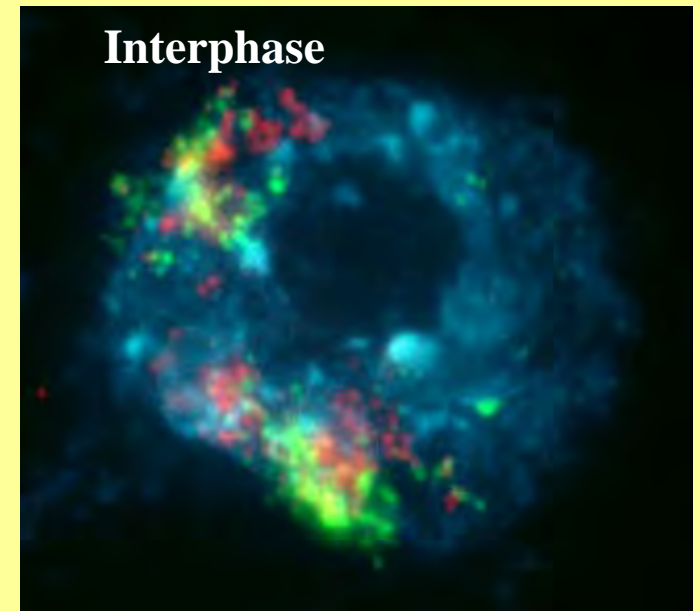
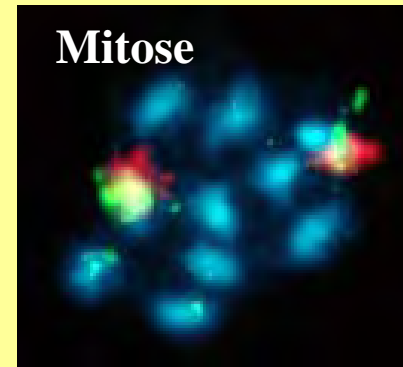
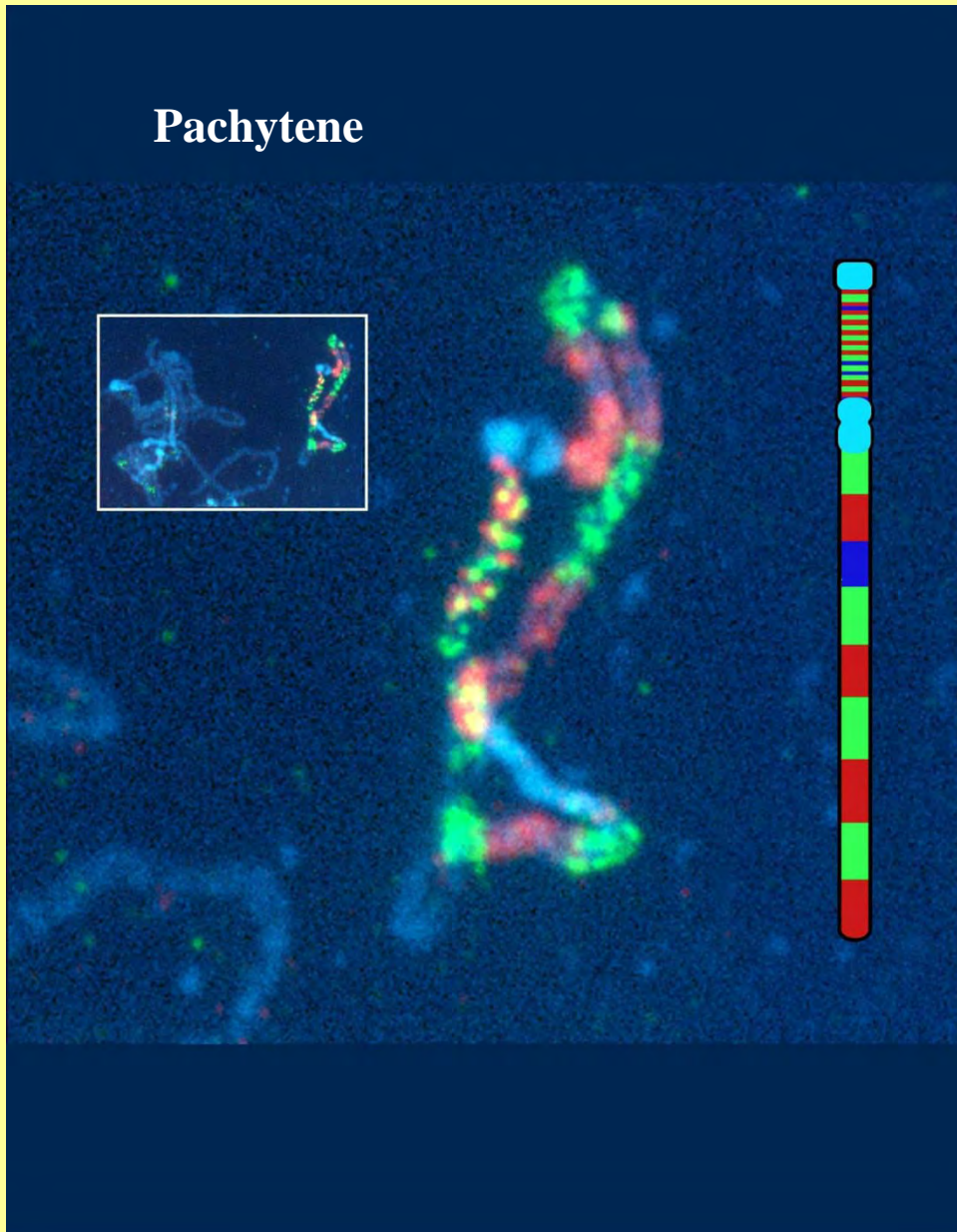


Drosophila melanogaster

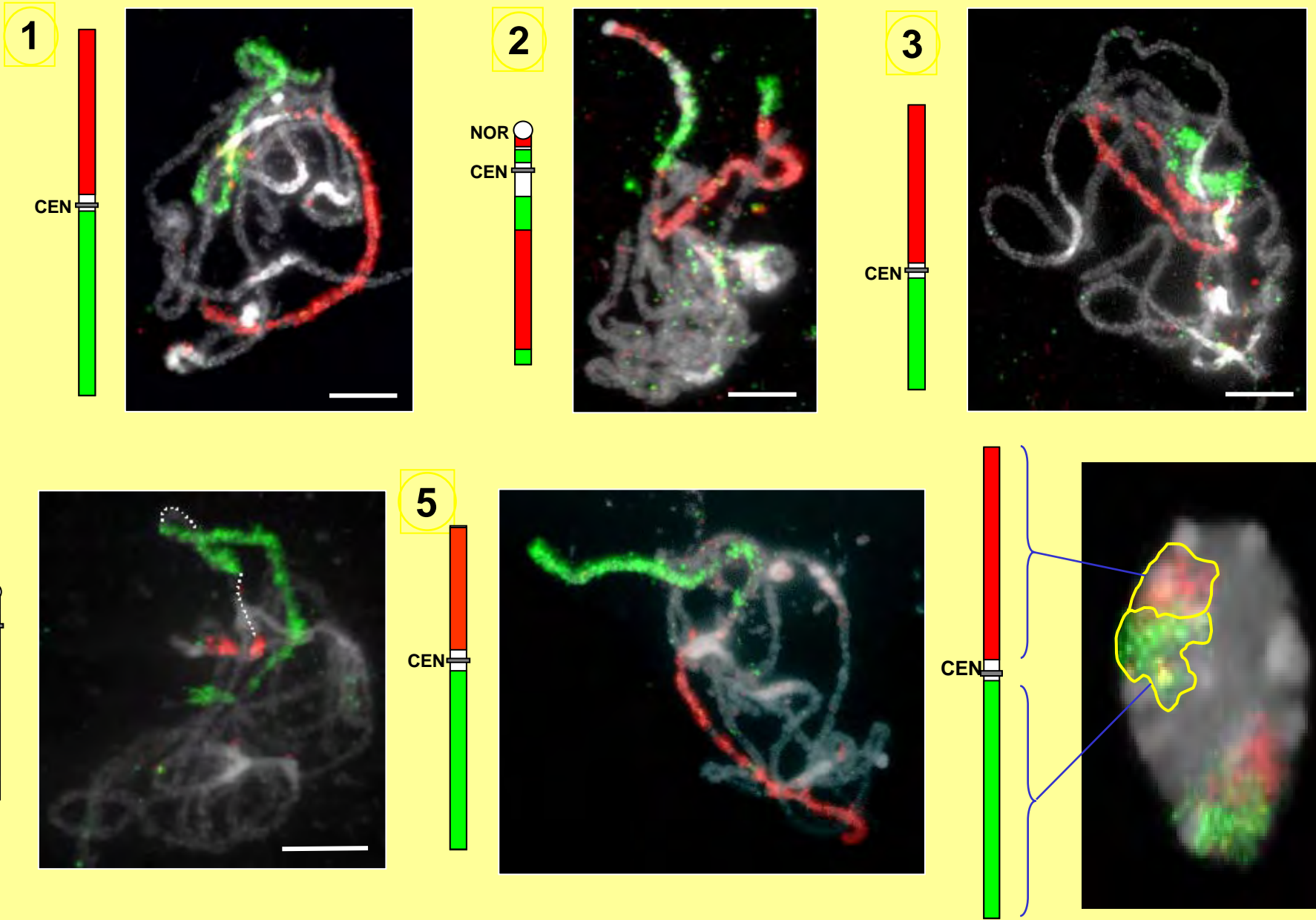


Arabidopsis thaliana

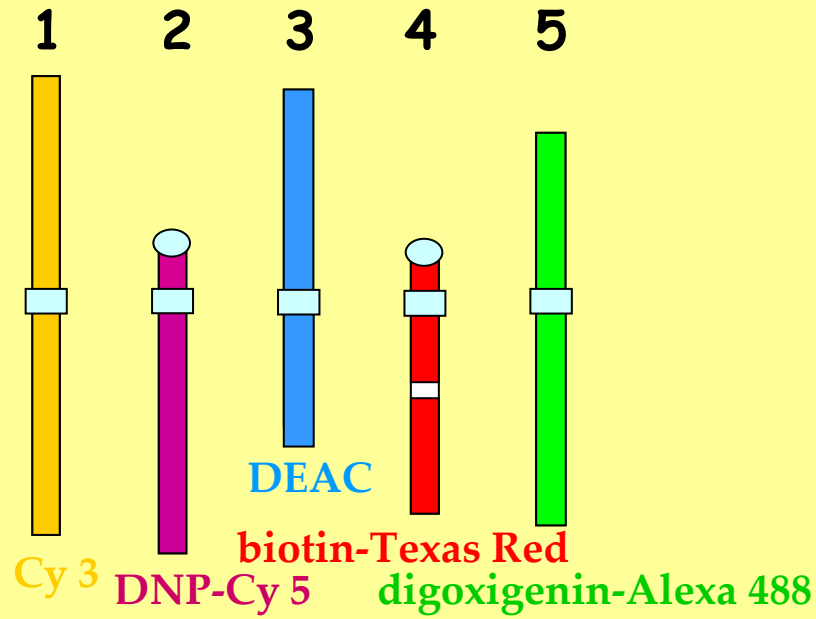
Chromosome 4 painting
(131 BACs)



Painting of Arabidopsis chromosomes 1 to 5

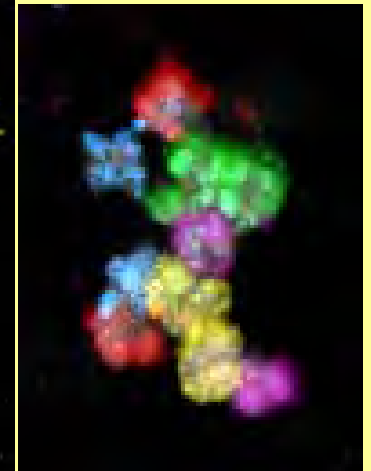


Simultaneous multicolour-painting of all 5 Arabidopsis chromosomes

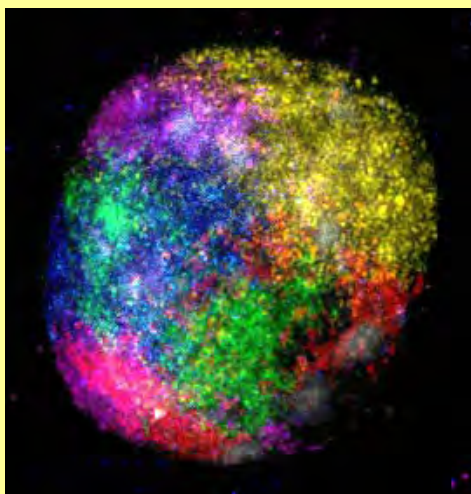


Pachytene

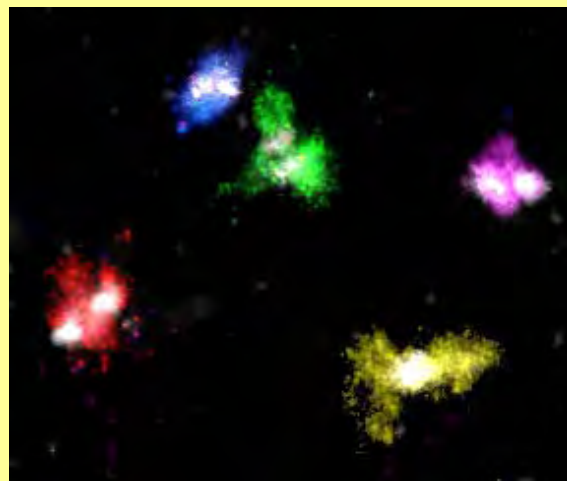
mitotic metaphase



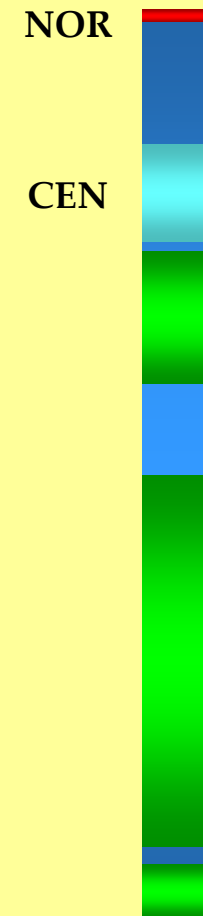
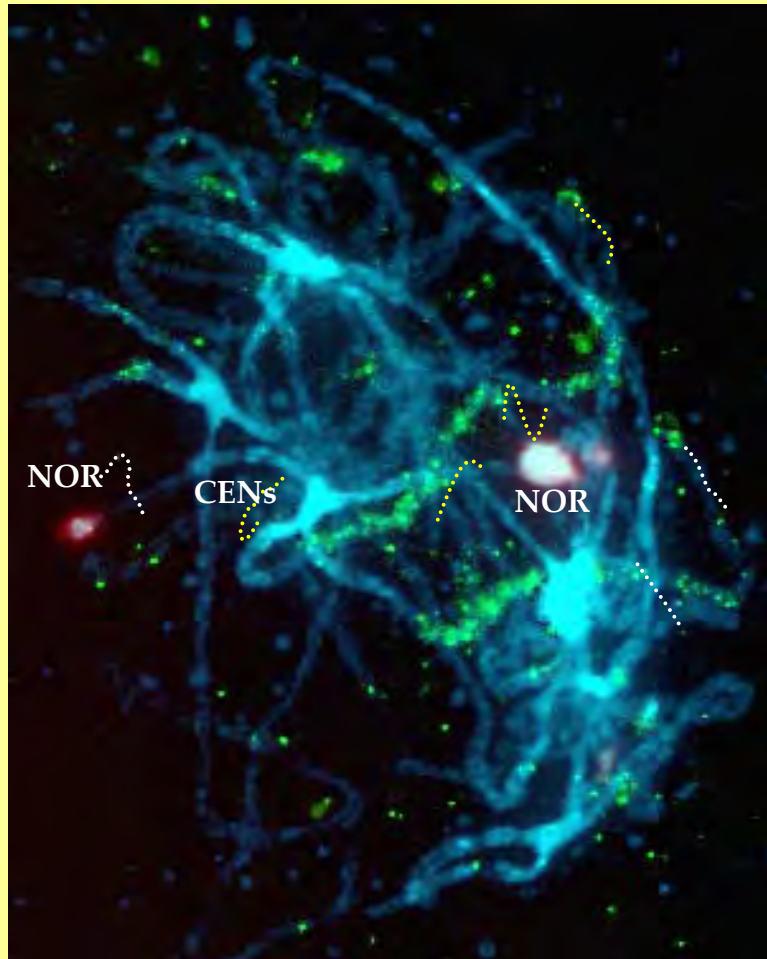
interphase



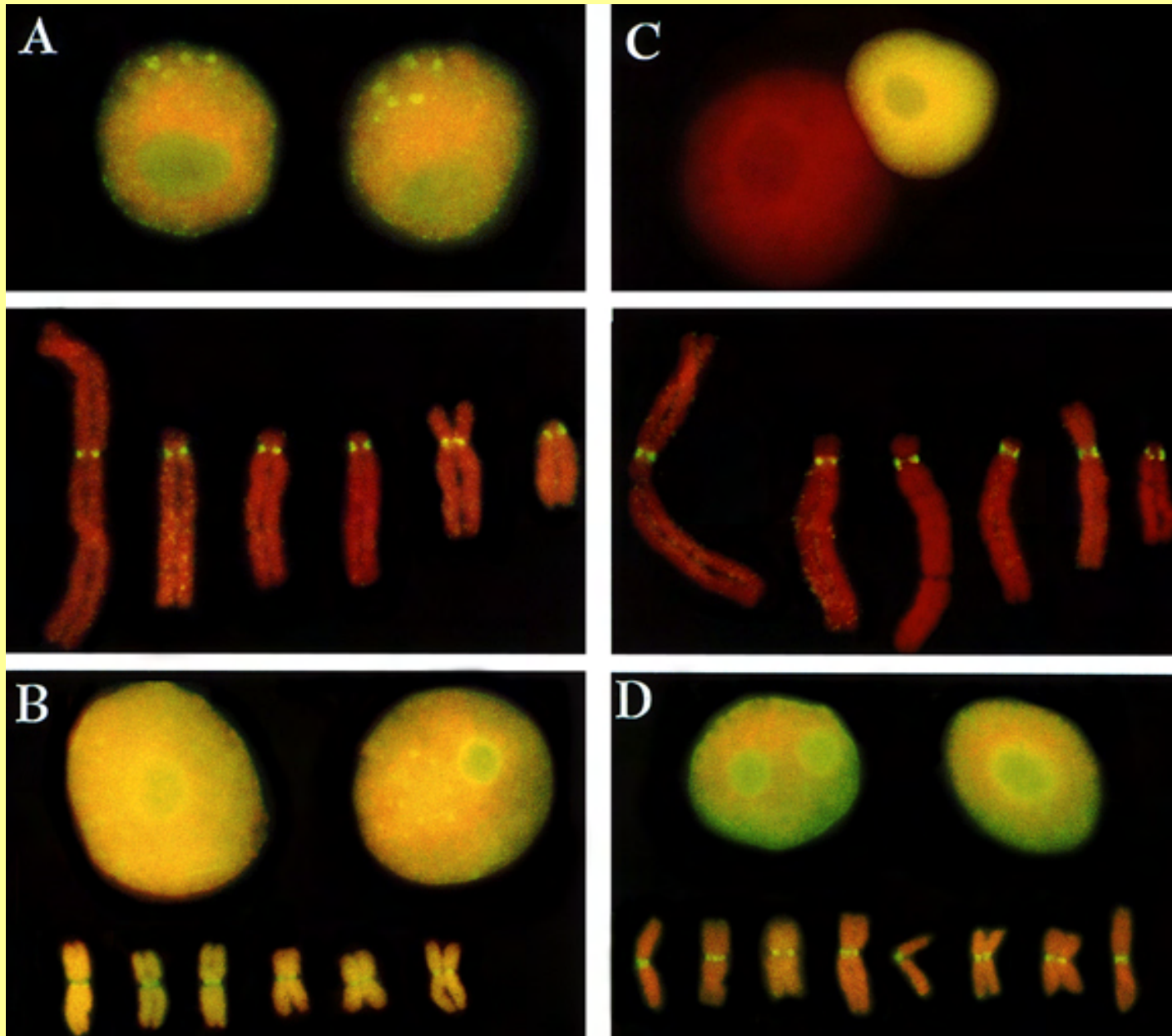
diakinesis



Two homeologues of chromosome 4 of *A. thaliana* revealed in *Arabidopsis arenosa* ($2n = 32$)



Monoclonal antibodies against plant kinetochores

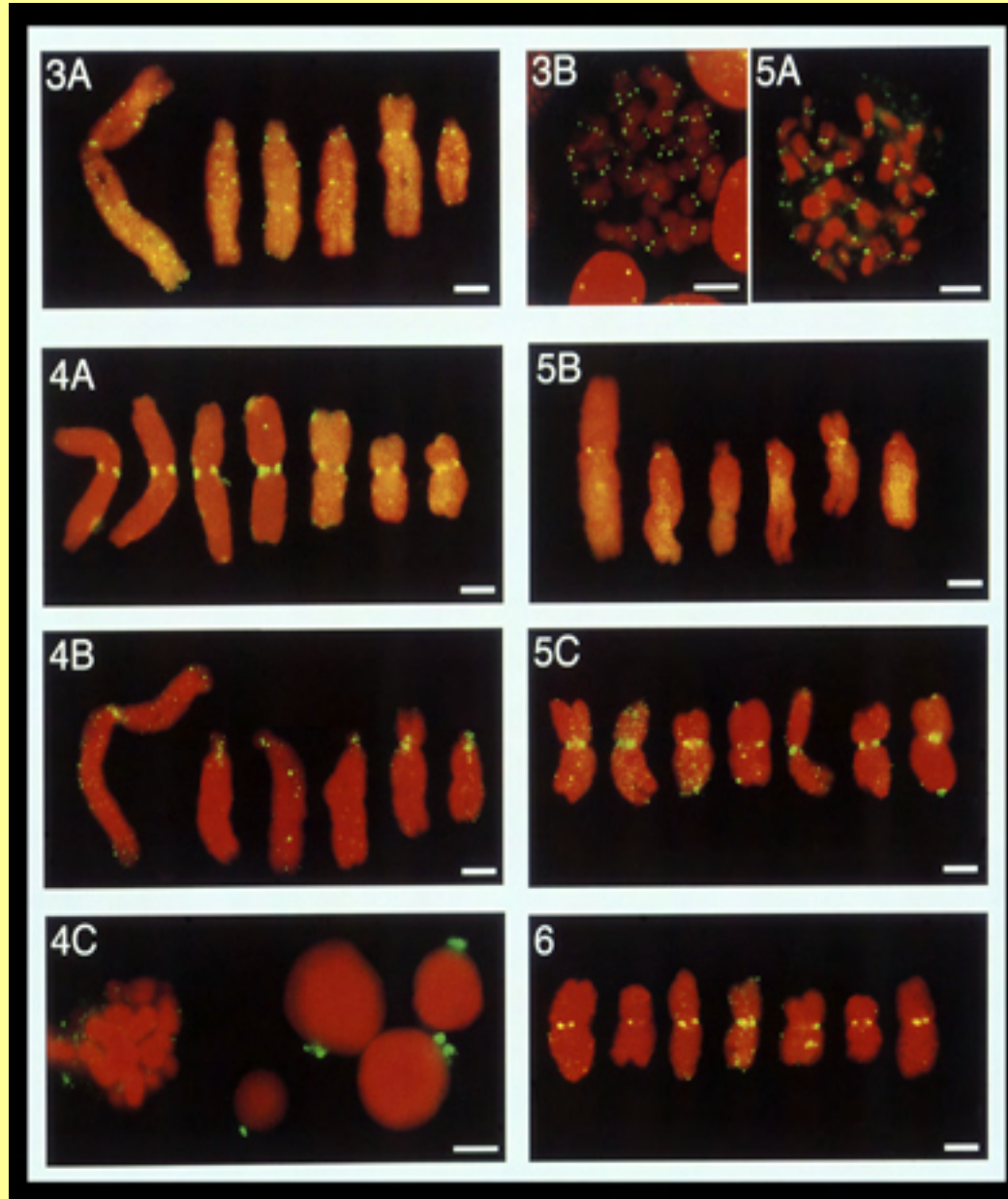


V. faba

Barley

Immunodetection of kinetochore-specific antigens across species borders

Anti-hCENP C

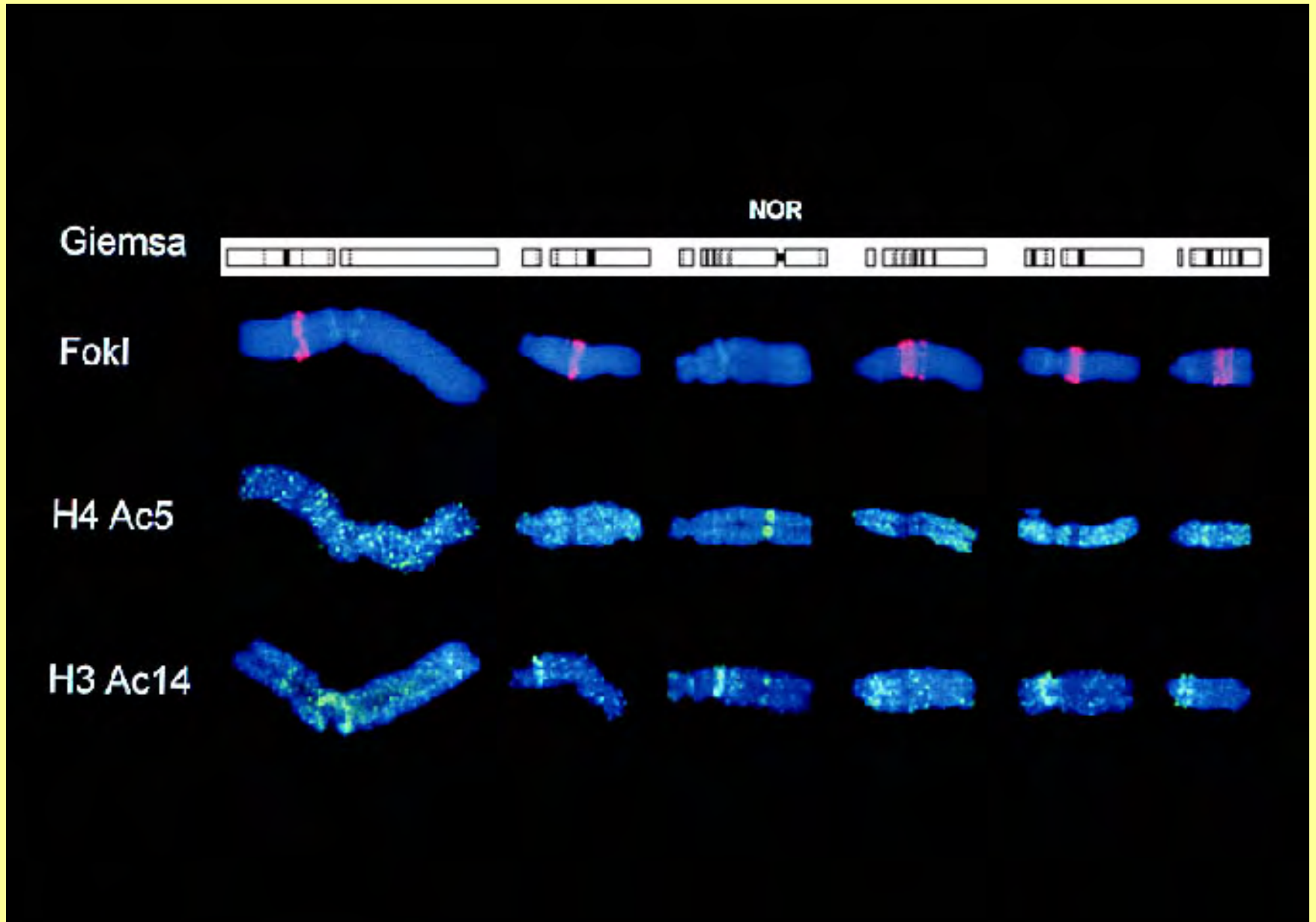


Anti-hCENP E

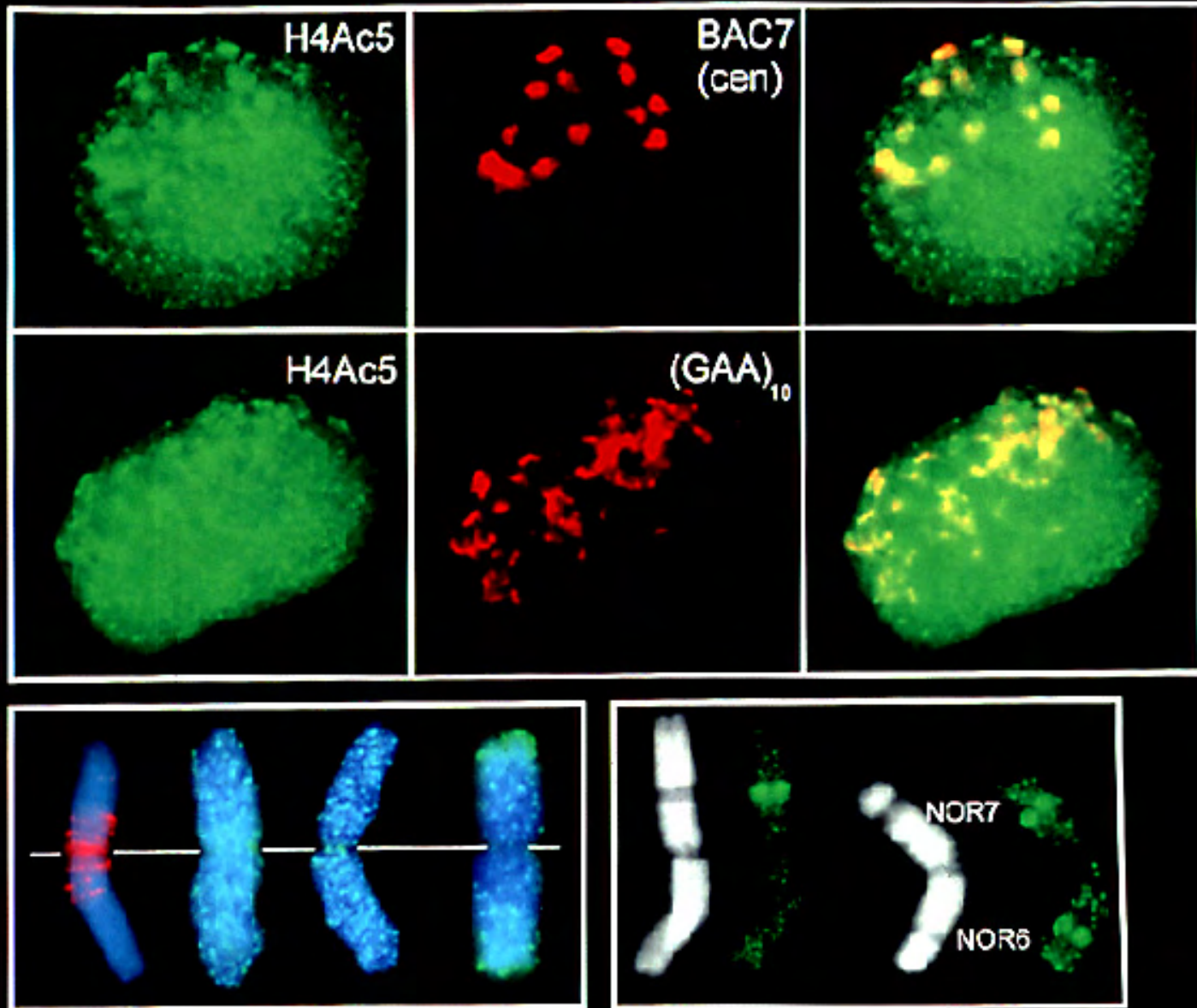
Anti-maize
CENP C

Anti-hCENP F

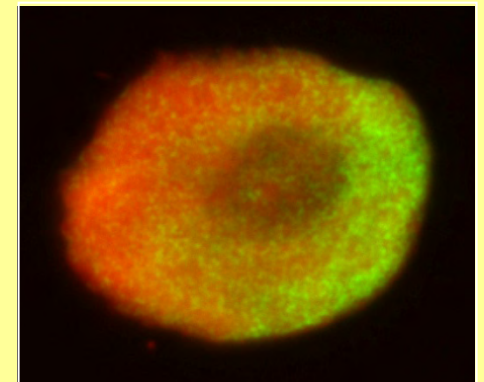
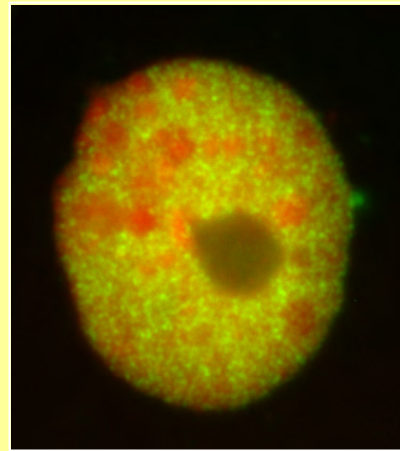
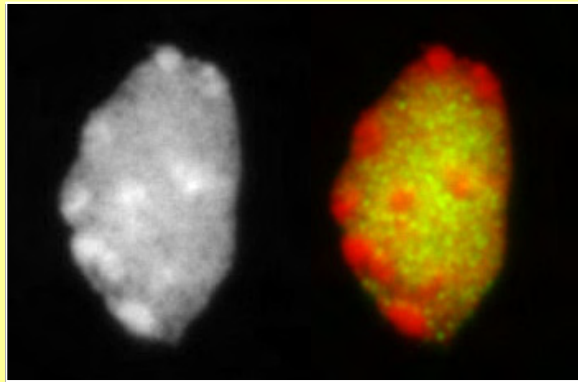
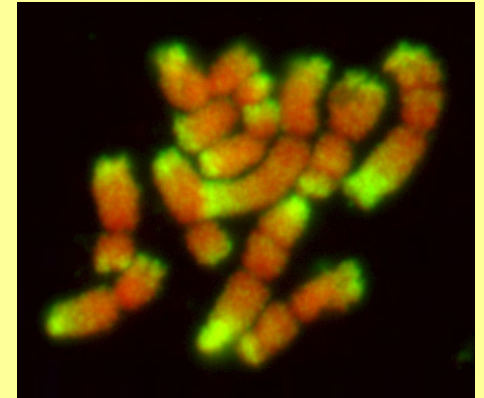
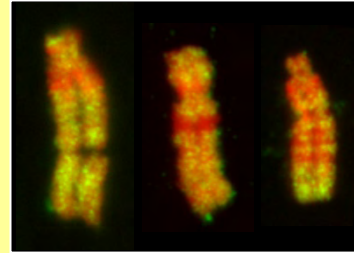
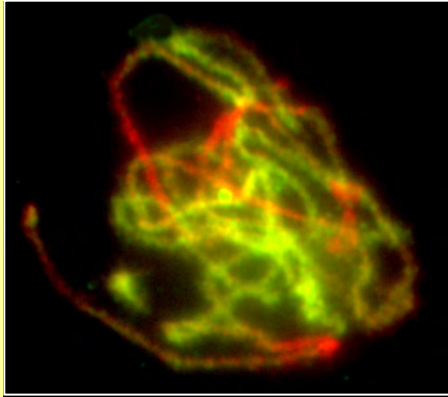
Field bean chromosomes after FISH/Immunostaining



Barley chromosomes and nuclei after FISH/Immunostaining



methyated H3 - lysine4 (detected at transcriptionally active regions and borders of transcriptionally silenced domains in fission yeast)

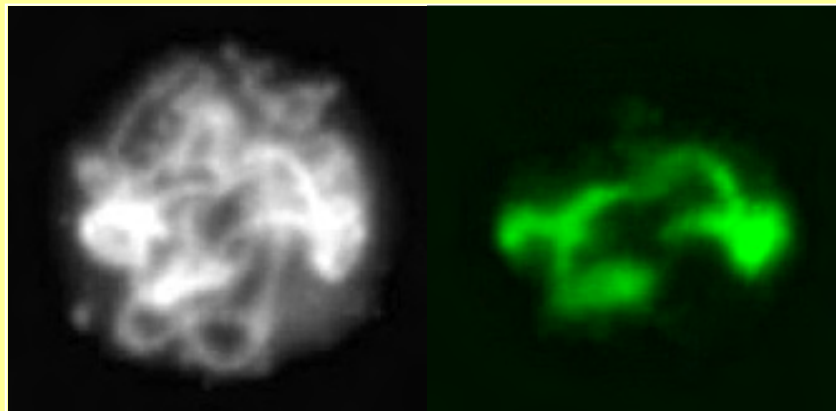
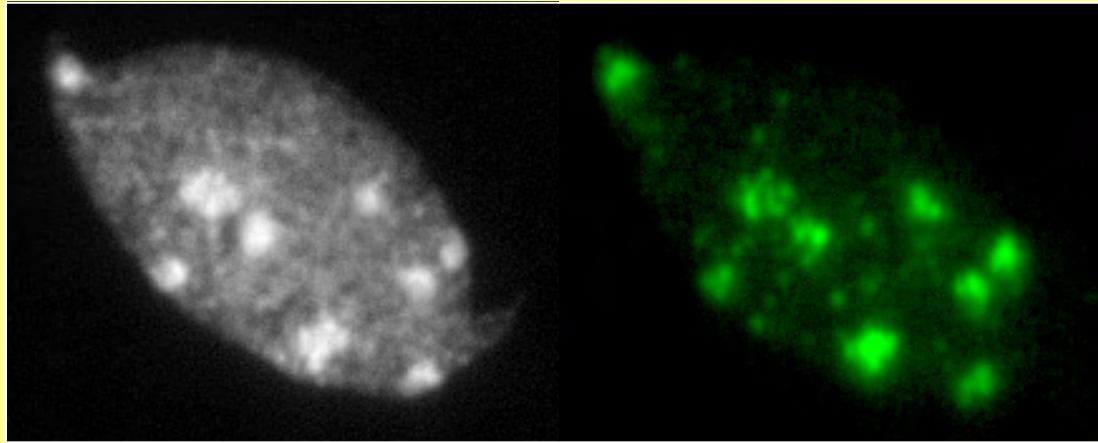


Arabidopsis

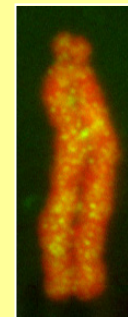
Vicia

Hordeum

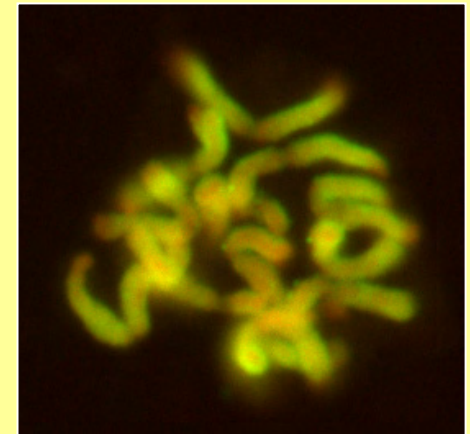
methyated H3 - lysine9 (involved in heterochromatin assembly in fission yeast, *Drosophila*, mouse)



Arabidopsis

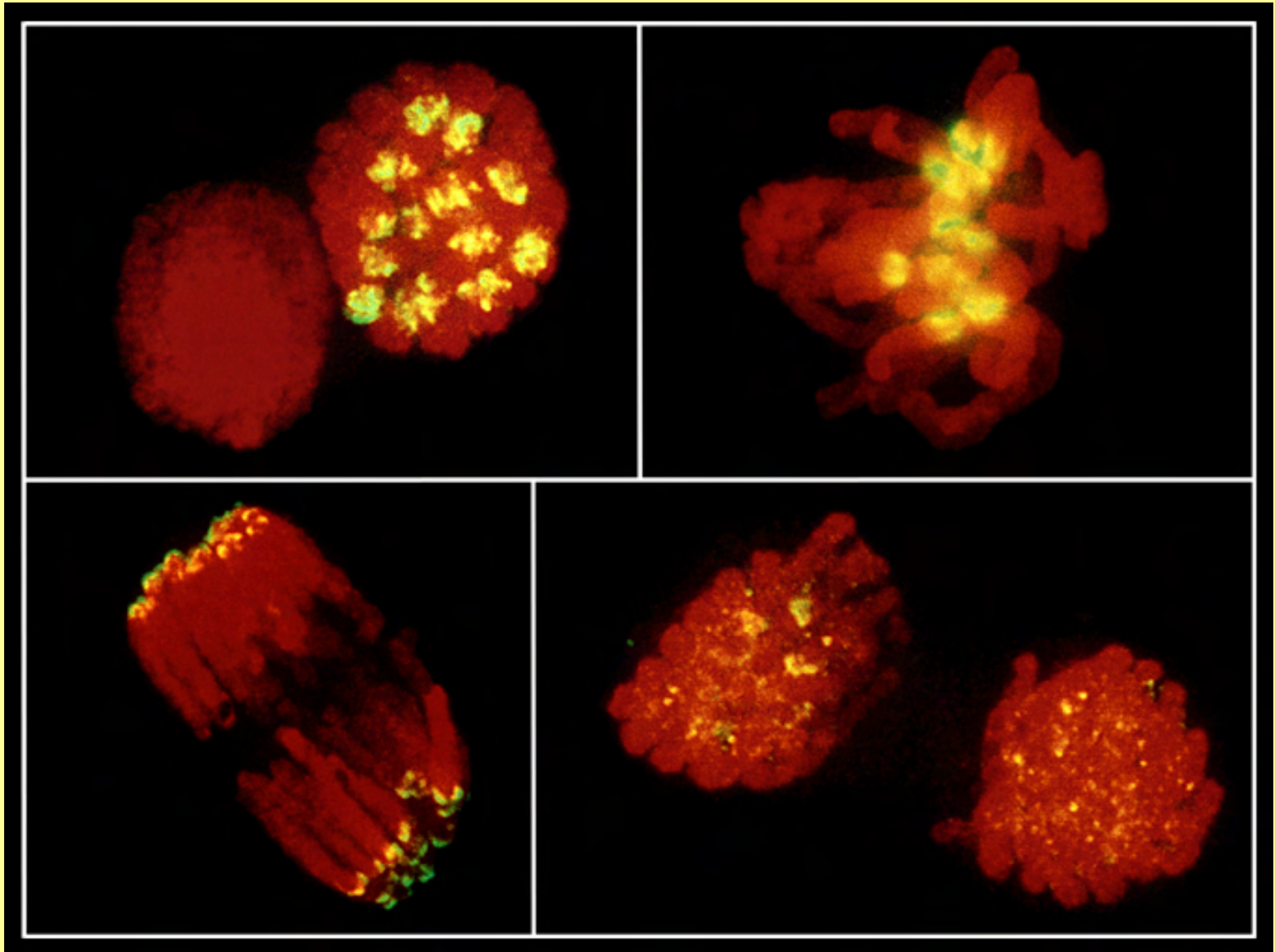


Vicia



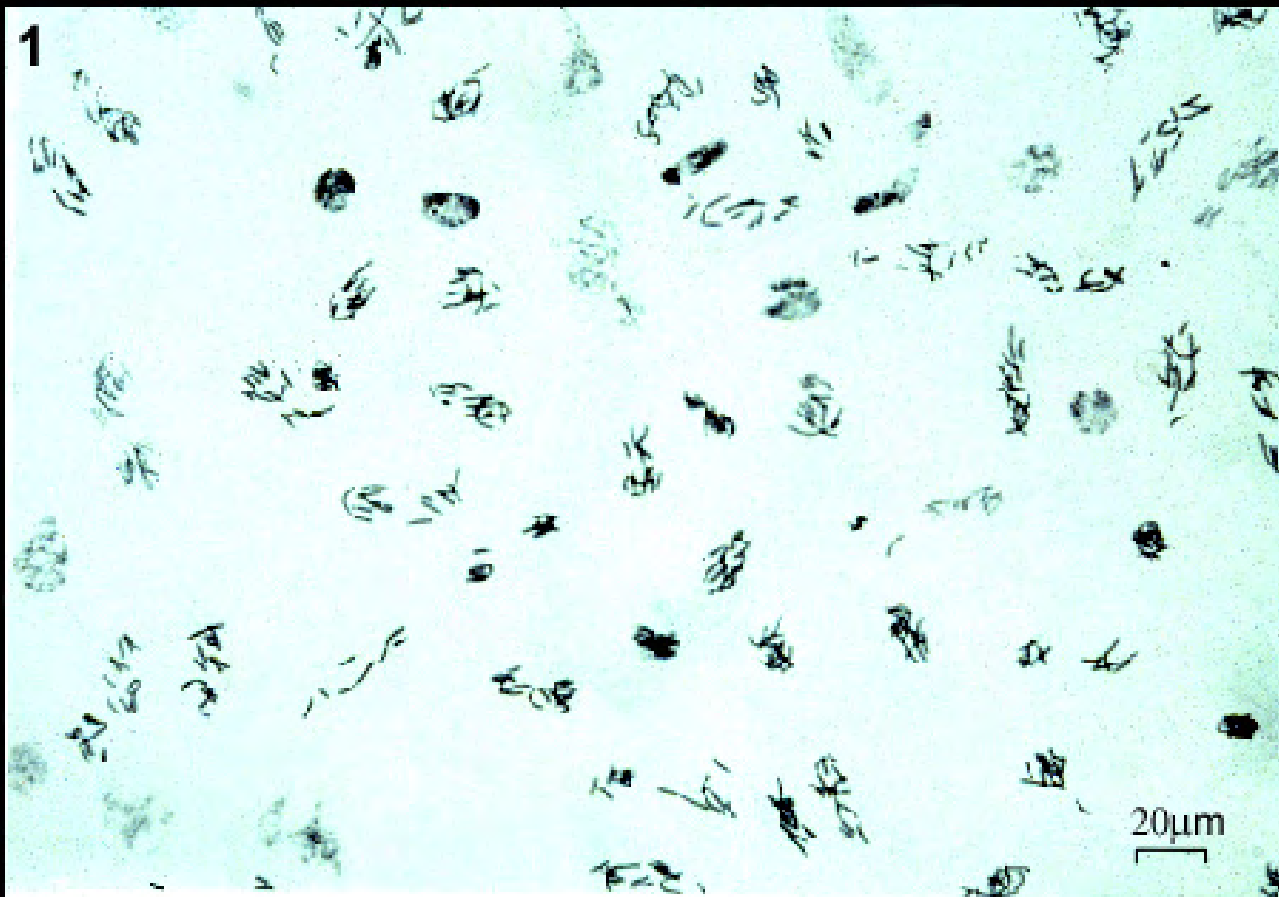
Hordeum

H3 Ser 10 phos, barley



Microdissection of chromosomes and chromosome regions

Isolation and flow-sorting of chromosomes and nuclei



**Synchronized
field bean
meristem**

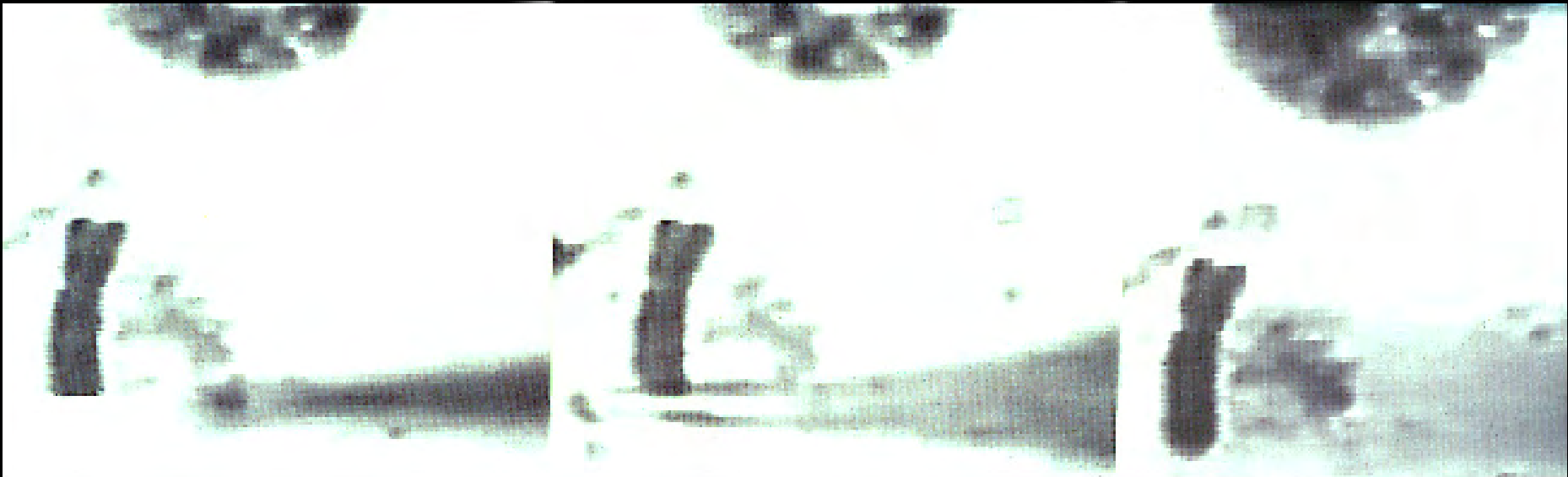


**Microdissected
chromosome I**

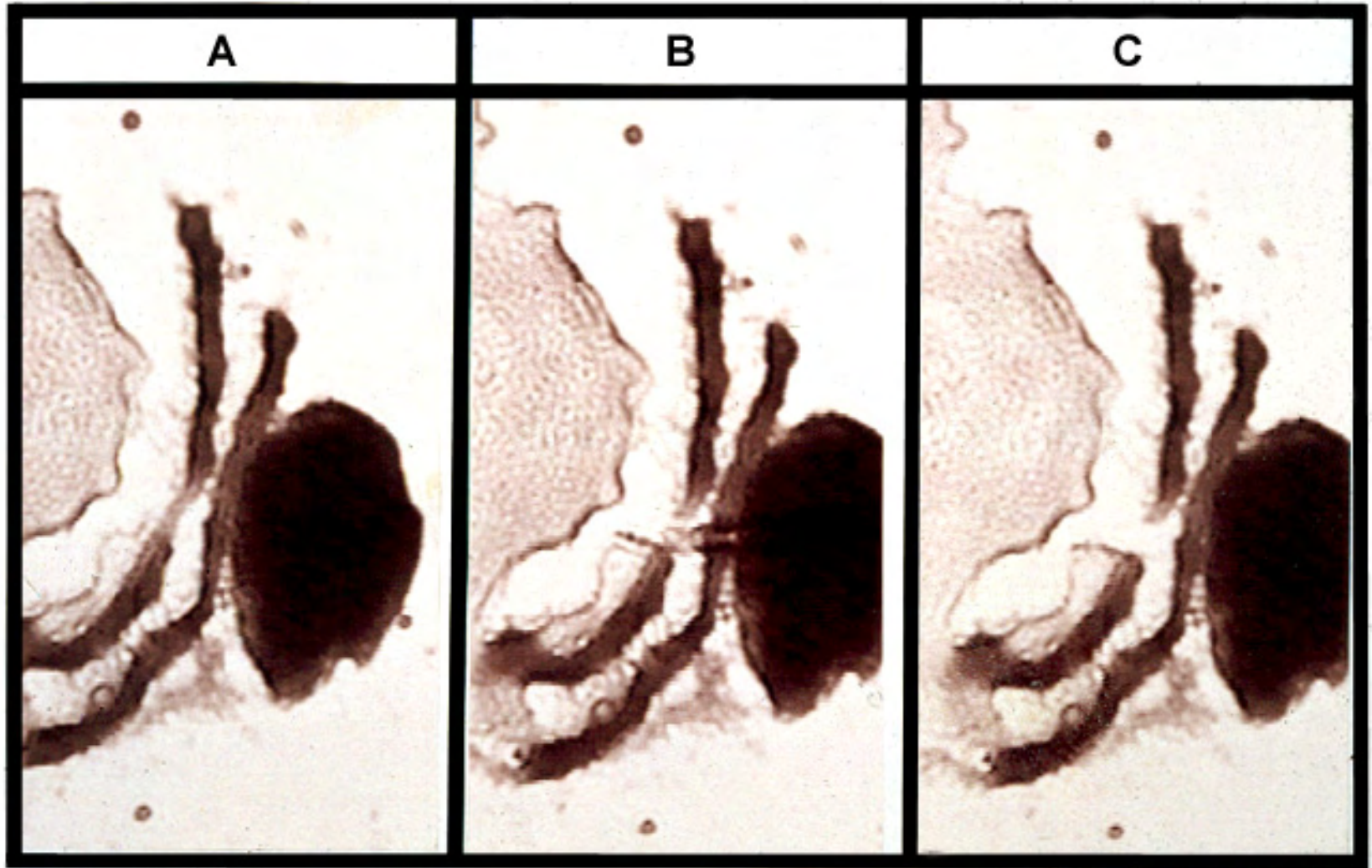


**Laser microdissection
of the segment 4
of the C-banded
Vicia faba chromosome 3
karyotype ACB**

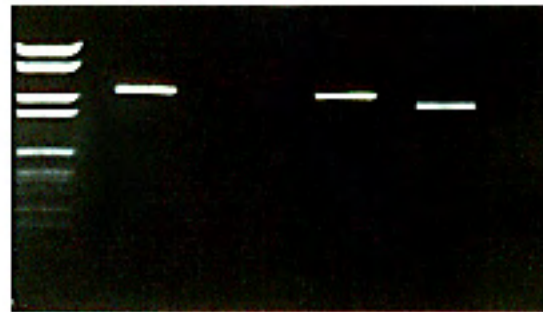
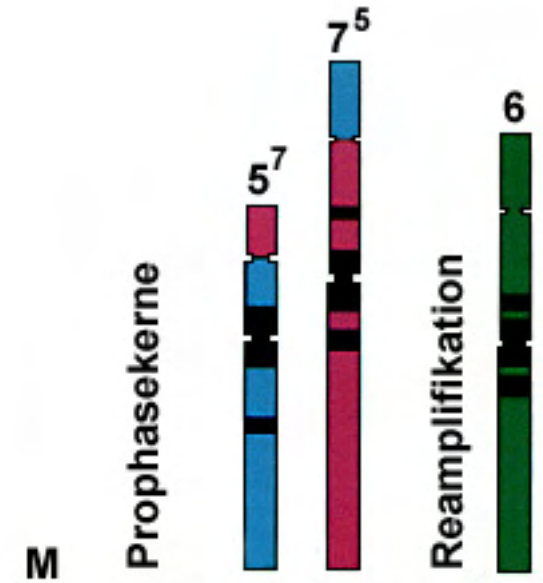
Microdissecting the centric end of a telocentric field bean chromosome



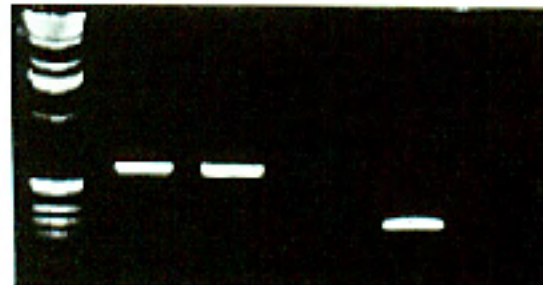
Microdissecting the centromere of a *Tradescantia* chromosome



PCR mapping on
microdissected
translocation
chromosomes of barley



Hor1 (C)



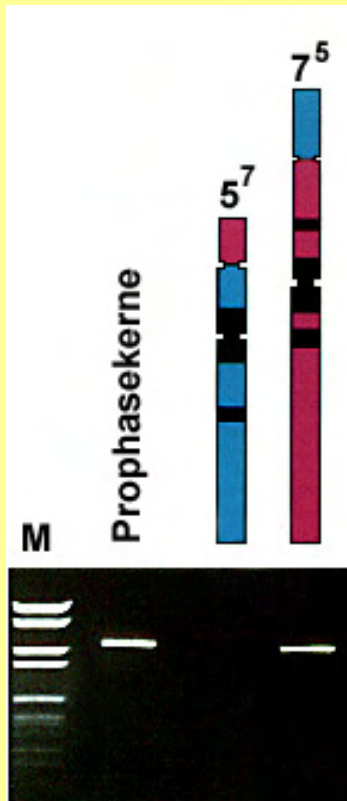
MWG 800

Physically and genetically integrated map of barley

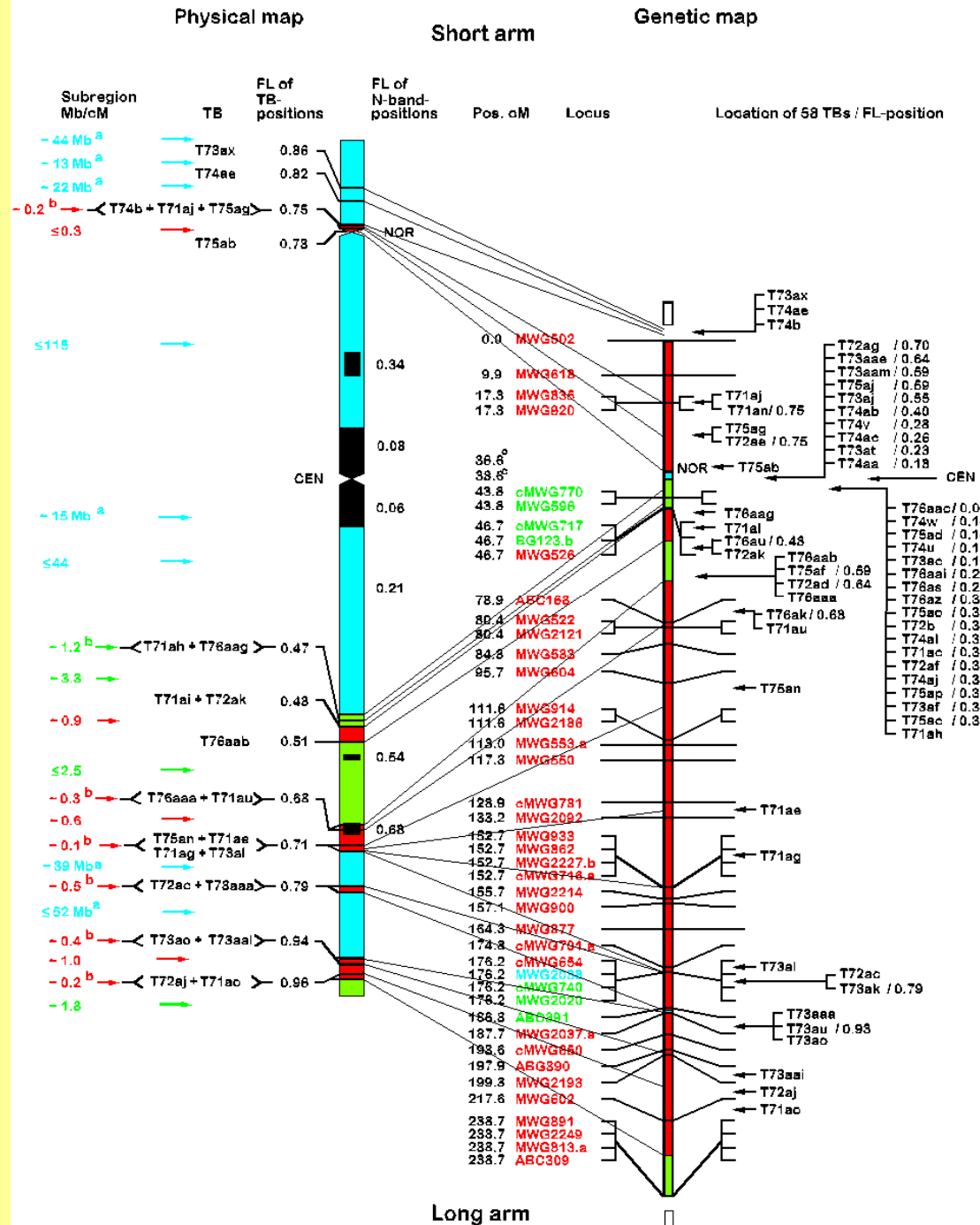
(G Künzel, L Korzun, A Meister, Genetics 2000)

429 markers; 240 TBP; 139 regions

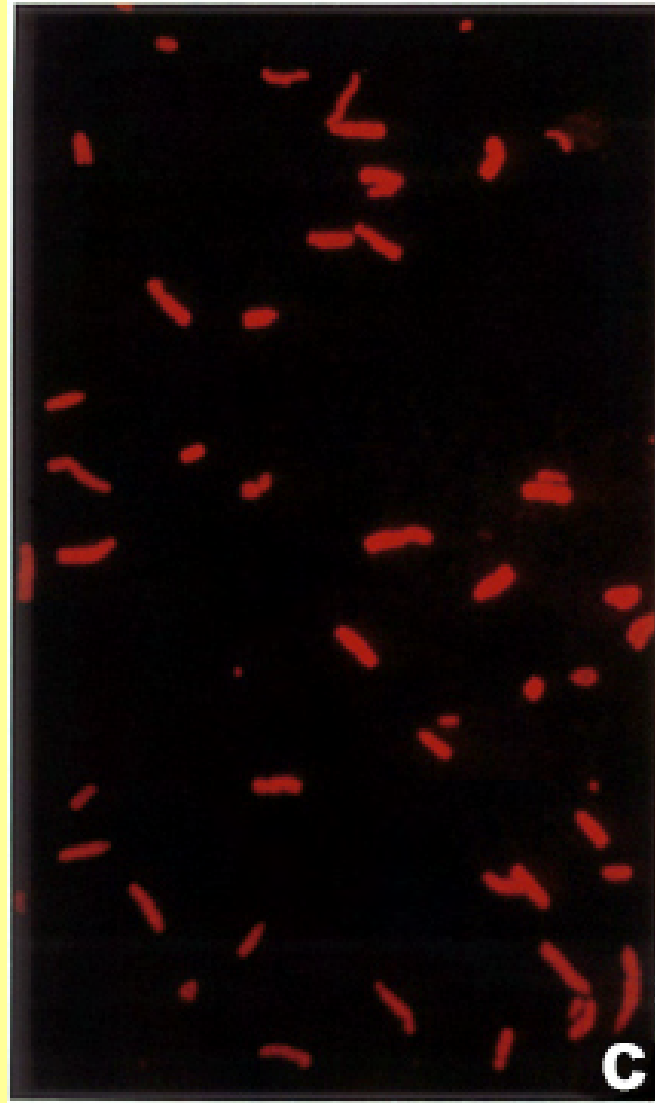
<1Mb/cM = 4.9% of genome = 47% of markers



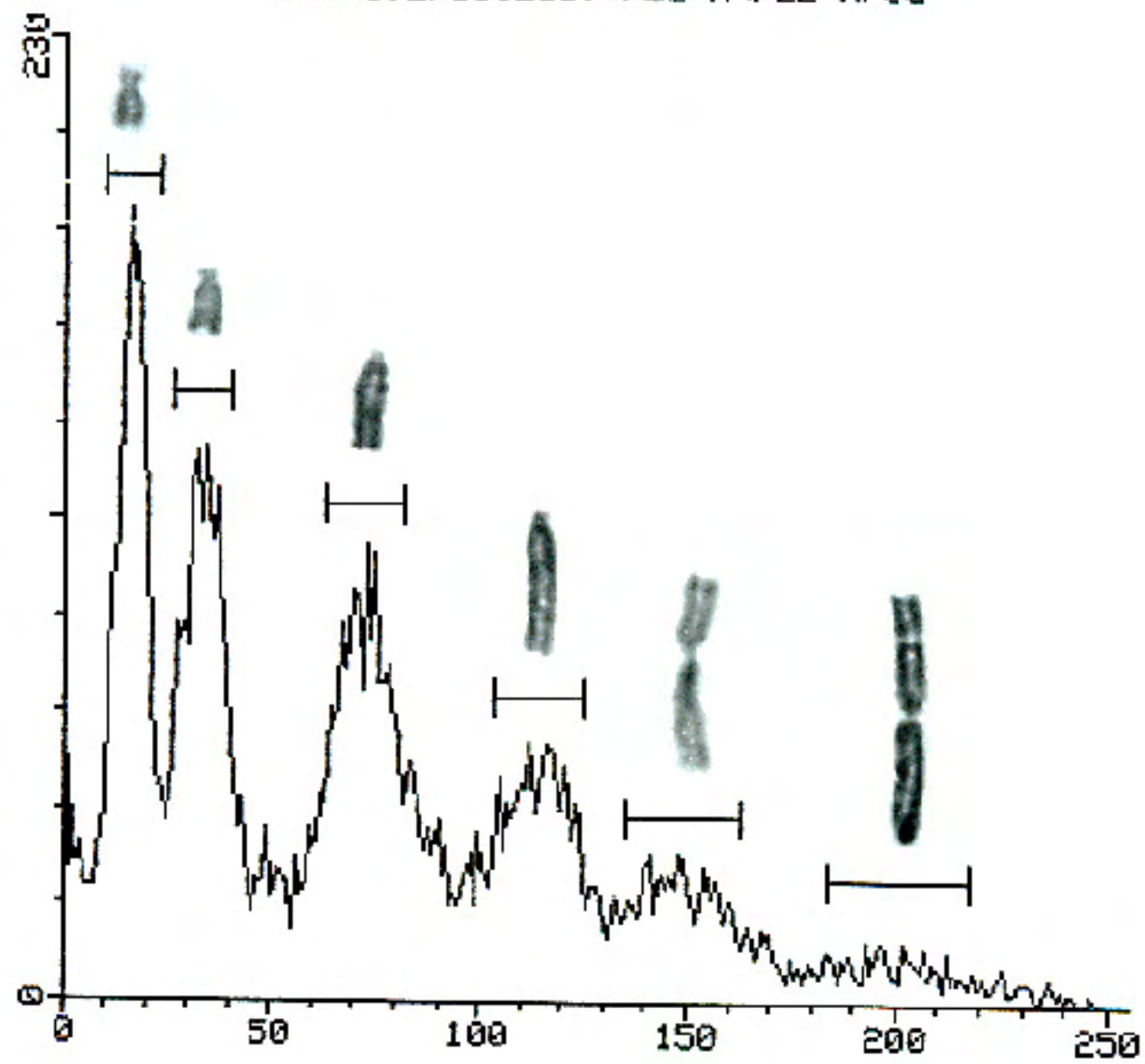
CHROMOSOME 5H(7)



Chromosome suspension of *V. faba*



FUCHS: EF0312009\FL2-A\FL2-Area



Flow-sorted chromosomes of the *Vicia faba* karyotype EF

FISH with
rDNA

a

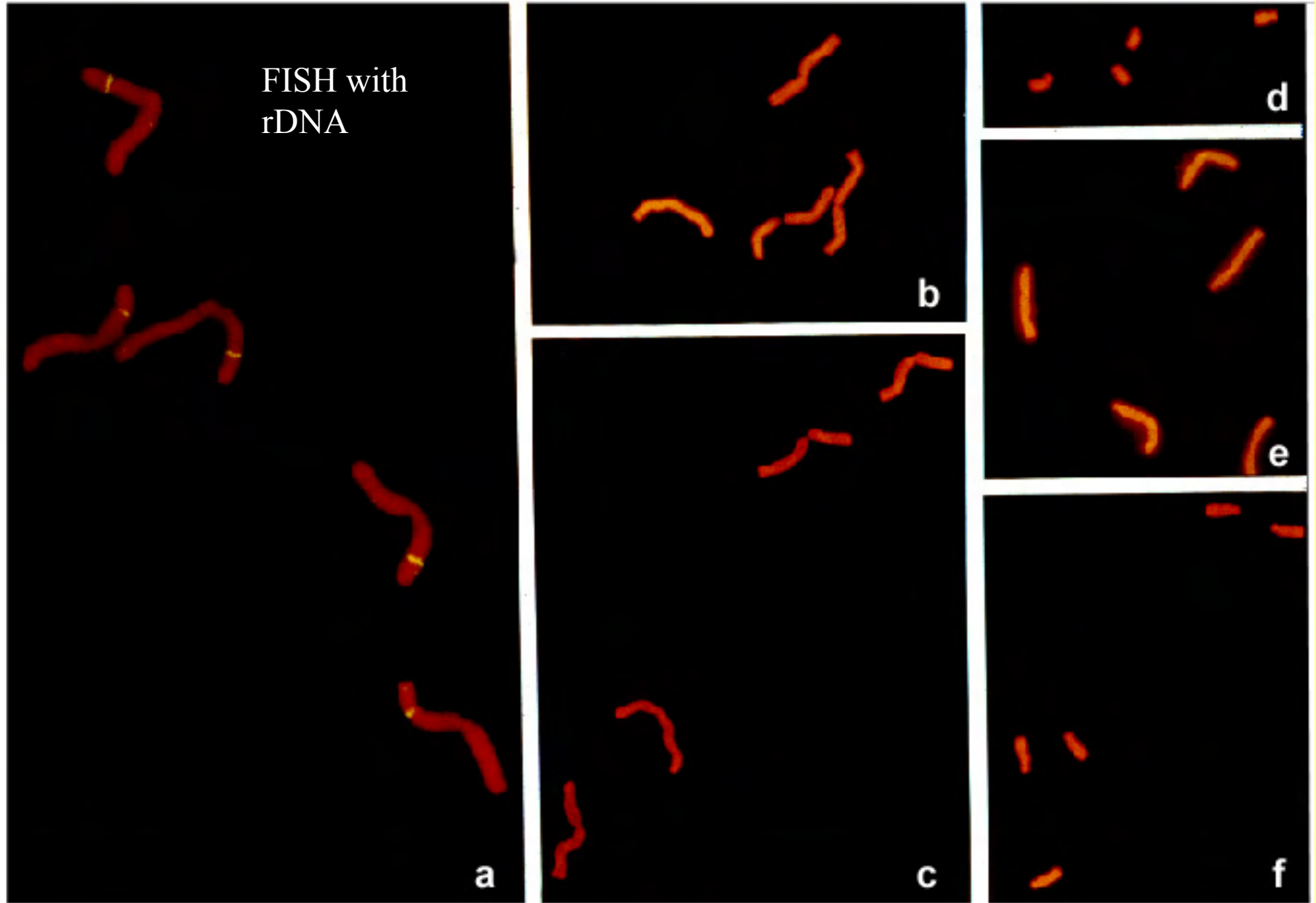
b

d

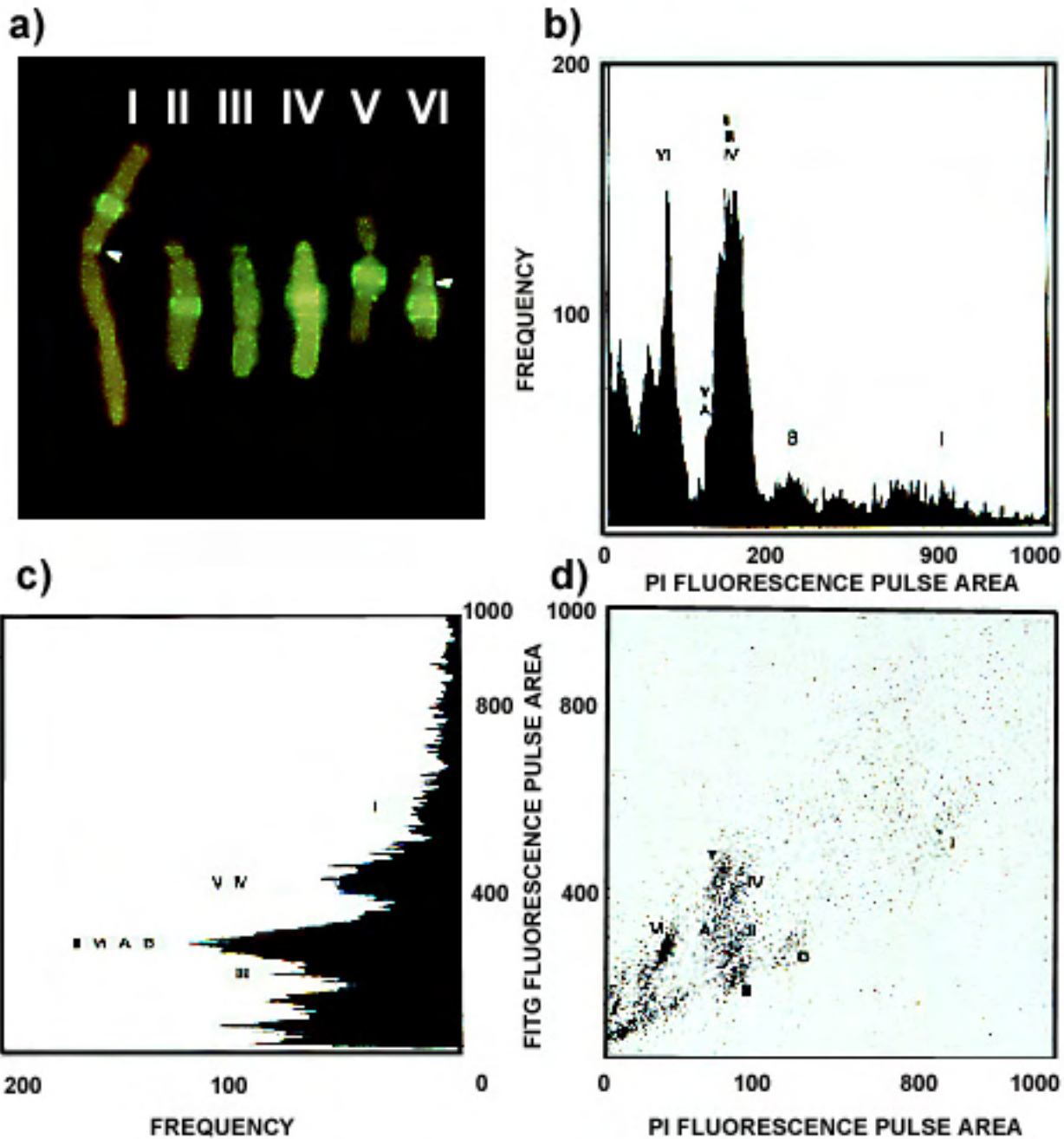
e

c

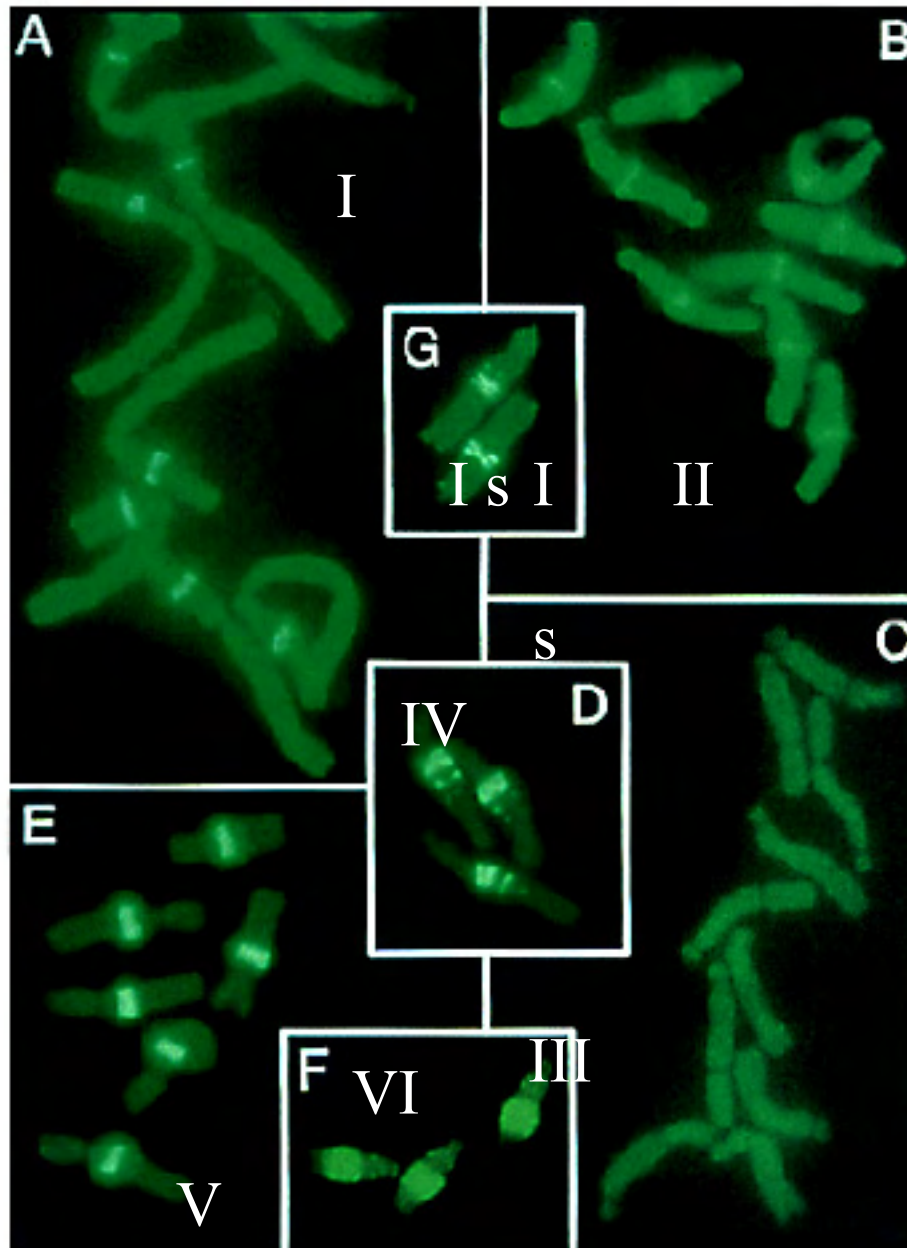
f



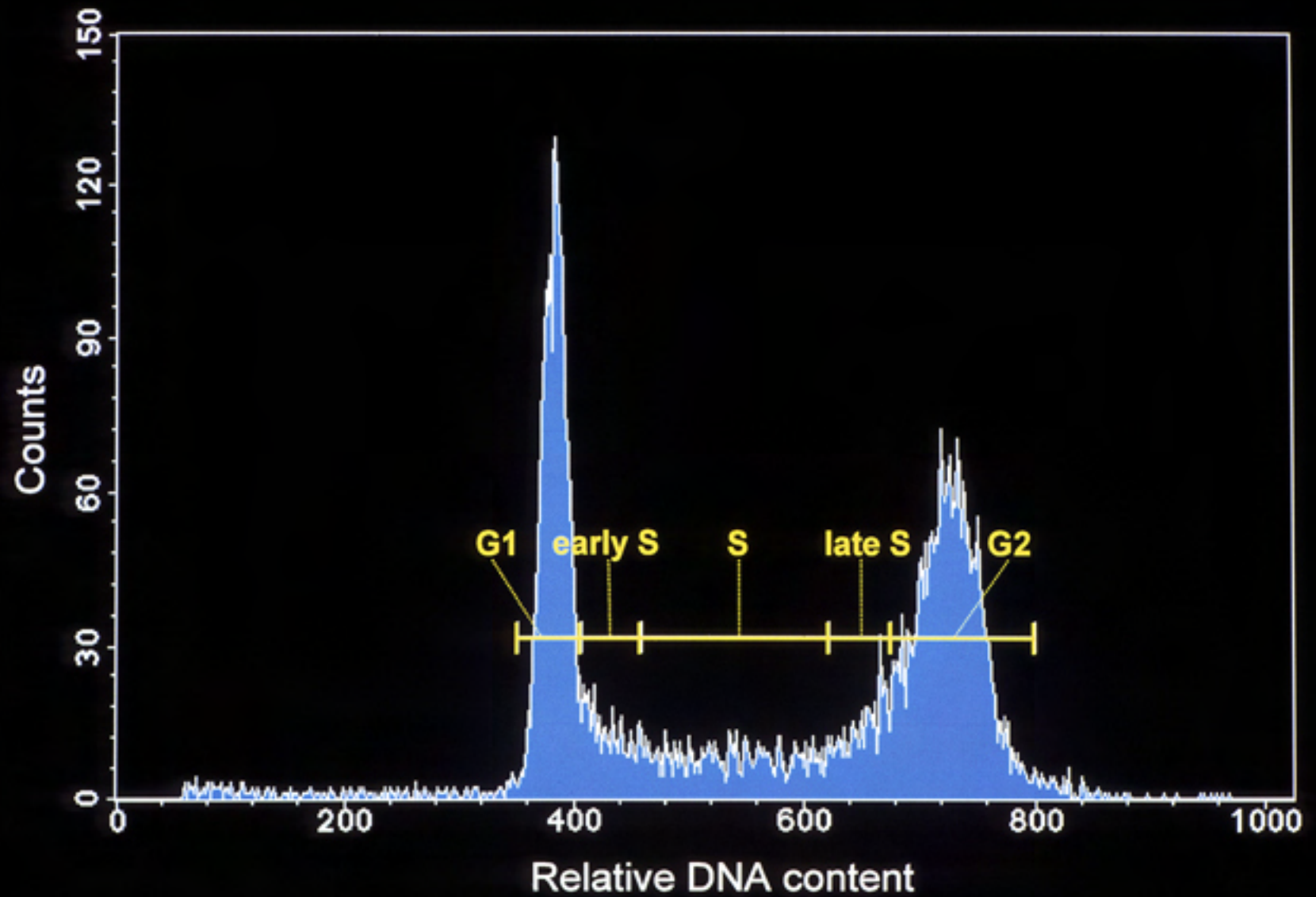
Vicia faba karyotype ACB after PRINS with *FokI* elements



Flow-sorted field bean chromosomes of karyotype ACB

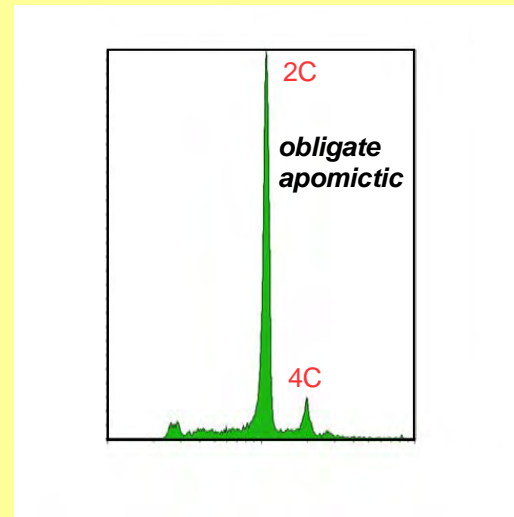
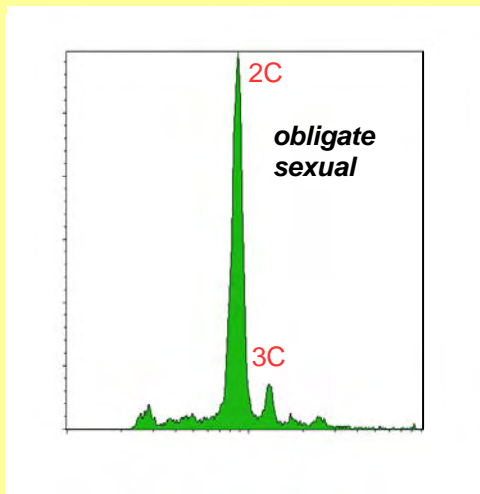
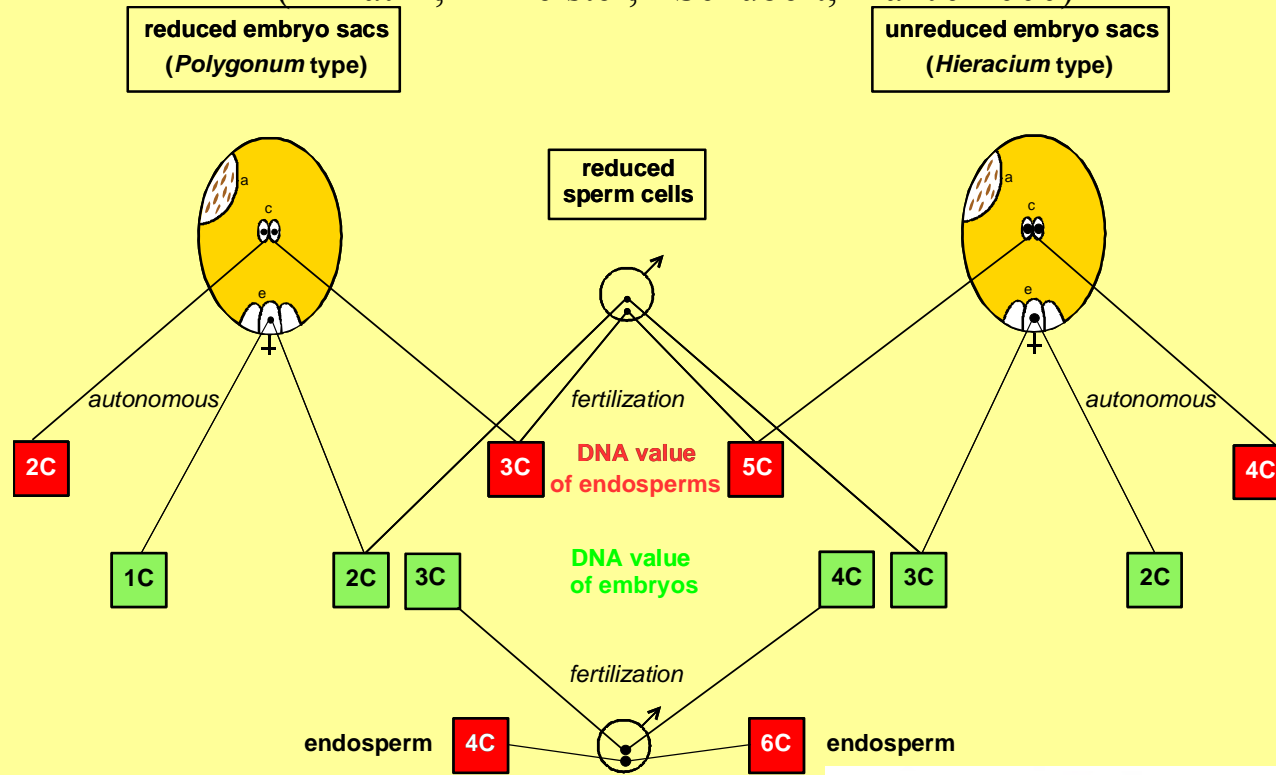


Flow-sorted field bean nuclei



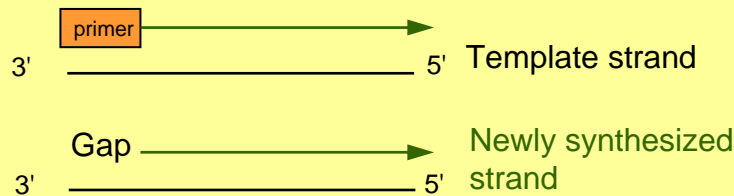
Flow-cytometric seed screen to elucidate the mode(s) of reproduction

(F Matzk, A Meister, I Schubert; Plant J 2000)

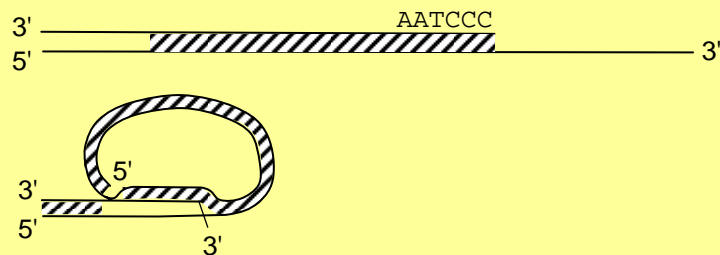


Short telomeric repeats stabilise the ends of linear eukaryotic chromosomes but lack in Dipteres and in Allium

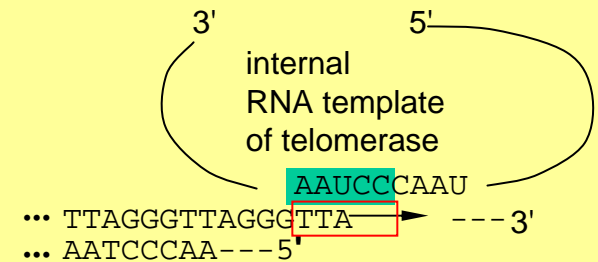
1) The 'end replication' problem



3) Telomere loop formation



2) Elongation of 3'-ends by telomerase

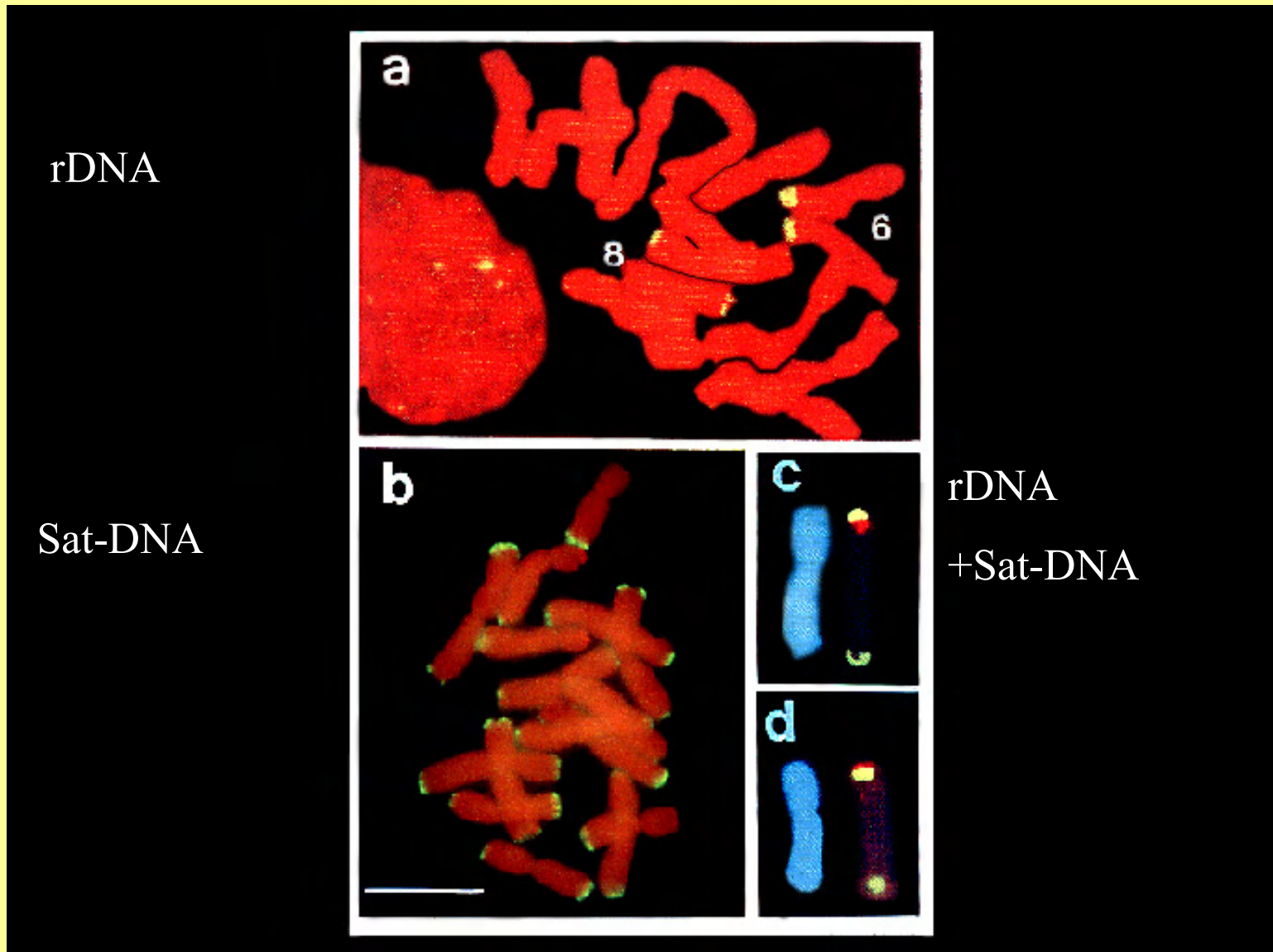


4) In Allium no TTTAGGG repeats detectable by FISH, Southern, asymmetric PCR or Bal 31 digestion (Pich, Fuchs & Schubert, 1996)

...no telomerase activity in TRAP-assay (Sykorova et al. ProcRSocLondB, 2003)

...instead recombinative ALT? at 375 bp repeats, rDNA and possibly En/Spm transposons

rDNA, Sat-DNA (and transposons?) apparently substitute original telomeric sequences at the termini of onion chromosomes



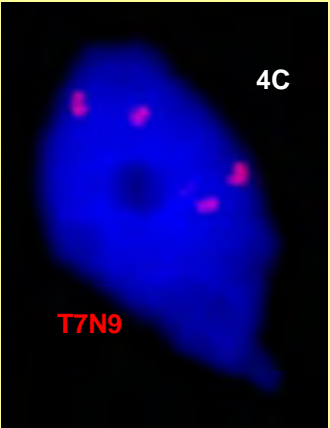
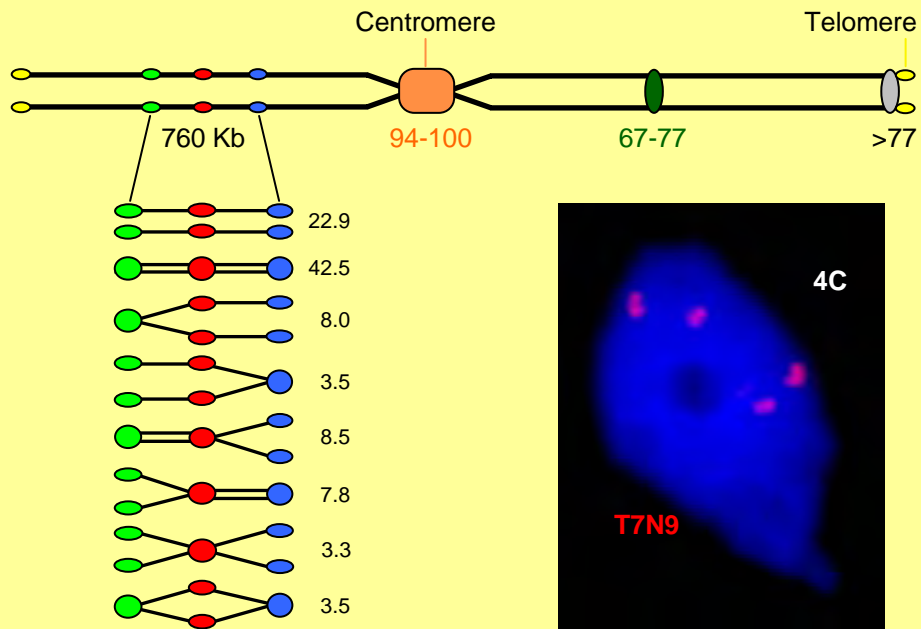
Heterochromatin features:

- Allocycl
- Special staining behaviour
- Late replication/underreplication
- Low transcriptional activity, few if any genes, ‚position effect variegation‘
- (Tandem-) repetitive sequences
- Deviating base composition
- Intense DNA methylation
- Specific histone modifications/protein composition (HP1)
- No meiotic recombination, frequent mis-repair -> chromosome aberrations
- Fast evolution

We distinguish:

Constitutive, facultative (sex), functional heterochromatin





Leibniz-Institut für Pflanzengenetik und Kulturpflanzenforschung (IPK)

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06466 Gatersleben
Telefon: 03 94 82/5-0
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Email: info@ipk-gatersleben.de
Internet: <http://www.ipk-gatersleben.de>

Stand: 1. Januar 2007



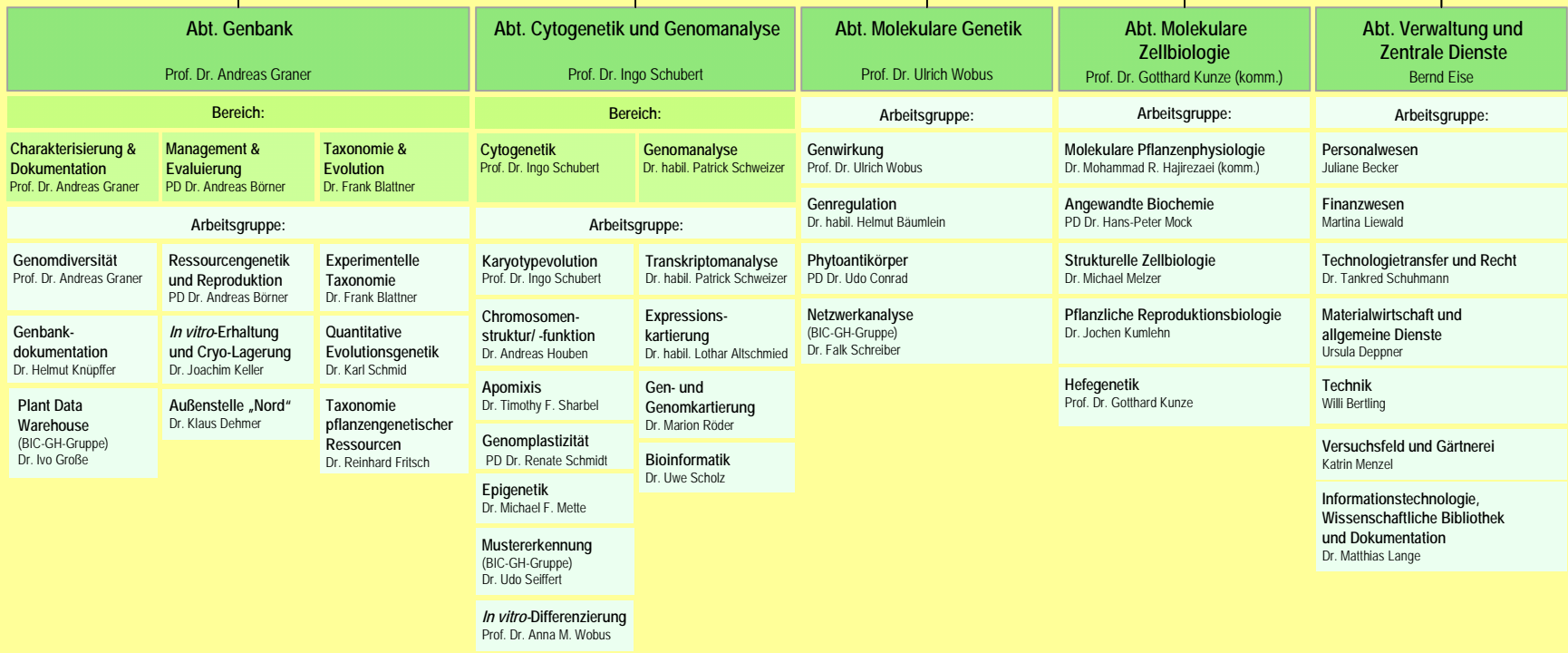
Stiftungsrat
Vorsitzender:
MinDirig. Dr. Joachim Welz
Stellv. Vorsitzender:
MinRat Dr. Jürgen Roemer-Mähler

Direktorium
Prof. Dr. Ulrich Wobus ¹⁾
Geschäftsführender Direktor
Bernd Eise ¹⁾
Administrativer Leiter
Prof. Dr. Andreas Graner
Prof. Dr. Ingo Schubert
Prof. Dr. Gotthard Kunze (komm.)

Wissenschaftlicher Beirat
Vorsitzender:
Prof. Dr. Eberhard Schäfer
Genbank-Beirat
Vorsitzender: Dr. Reinhard von Broock

Geschäftsstelle
Wissenschaftsorganisation
und Öffentlichkeitsarbeit
Waltraud Mühlenberg

Personalrat
Vorsitzender: Bernhard Claus



| | |
|---|--|
| Pflanzengenom-Ressourcen-Centrum (PGRC) | Koordinator: Dr. habil. Patrick Schweizer |
| Bioinformatik-Centrum Gatersleben-Halle (BIC-GH) | Koordinator: Prof. Dr. Stefan Posch/Universität Halle-Wittenberg |

¹⁾ Geschäftsführung

Institut für Pflanzengenetik und Kulturpflanzenforschung (IPK)

Gatersleben
Leibniz-Institut



Vorgeschichte des IPK

- 1943** Gründung des Instituts für Kulturpflanzenforschung der Kaiser-Wilhelm-Gesellschaft zur Förderung der Wissenschaften am 1. 4. 1943 in Tuttenhof bei Wien. Ernennung von Hans Stubbe zum Direktor.
- 1945** Januar bis April: Verlagerung der Sammlungen in das Dorf Stecklenberg/ Harz. Oktober: Die Domäne Gatersleben (damals Kreis Quedlinburg) in Sachsen-Anhalt wird für die Ansiedlung des Instituts zur Verfügung gestellt.
- 1946** Angliederung des Instituts an die Universität Halle-Wittenberg unter Beibehaltung seines Namens.
- 1948** Eingliederung in den Kreis der Forschungsinstitute der Deutschen Akademie der Wissenschaften zu Berlin (Nachfolgerin der Preußischen Akademie der Wissenschaften).
- 1948-64** Aufbau der Institutsgebäude nach einem Generalebauungsplan.
- 1969** Prof. Dr. Dr. hc. mult. Hans Stubbe gibt die Leitung des Instituts aus Altersgründen ab. Nachfolger wird sein Schüler Prof. Dr. Helmut Böhme.
- 1969** Umbenennung in „Zentralinstitut für Genetik und Kulturpflanzenforschung.“
- 1969-72** Reform der Institutsstruktur; Bildung von wissenschaftlichen Bereichen und von Querschnittsbereichen.
- 1983** Prof. Dr. Helmut Böhme übergibt die Leitung an Prof. Dr. Dieter Mettin (früher Universität Halle-Wittenberg).
- 1989** Bildung eines demokratisch gewählten Wissenschaftlichen Rates, der den Rücktritt der Institutsleitung durchsetzt.
- 1990** Berufung von Prof. Dr. Klaus Müntz zum Direktor, der das Institut in einer komplizierten Übergangsphase leitet. Umbenennung des Instituts in „Institut für Genetik und Kulturpflanzenforschung“.
- 1991** Schließung des Instituts am 31. 12. gemäß Artikel 38 des Einigungsvertrages.
- 1992** Neugründung als „Institut für Pflanzengenetik und Kulturpflanzenforschung“ gemäß Empfehlung des Wissenschaftsrates. Zum Geschäftsführenden Direktor wird Prof. Dr. Ulrich Wobus und zum Administrativen Leiter Bernd Eise berufen.

Institut für Pflanzengenetik und Kulturpflanzenforschung (IPK)

Gatersleben Leibniz-Institut



Entwicklung des IPK

1. Januar 1992

Gründung des Instituts für Pflanzengenetik und Kulturpflanzenforschung (IPK).

9. Juni 1993

Festveranstaltung anlässlich der Neugründung des IPK, und wissenschaftliches Symposium anlässlich der Gründung des Vorgängerinstituts vor 50 Jahren am 10. Juni.

29. November bis 1. Dezember 1993

Durchführung der ersten Institutstage; 38 Vorträge und 61 Posterbeiträge vermitteln ein umfassendes Bild von der wissenschaftlichen Arbeit des IPK.

Herbst 1993

Berufung und Konstituierung der Stiftungsorgane Stiftungsrat (Vorsitzender Dr. Christoph Helm/Magdeburg) und Wissenschaftlicher Beirat (Vorsitzender Prof. Dr. Diter von Wettstein/Kopenhagen).

13. / 14. April 1994

Eine Flutkatastrophe führt zu erheblichen Schäden an und in den Gebäuden des Instituts und zu Verlusten von Forschungsmaterial. Land und Bund ermöglichen mit einer Soforthilfe den schnellen Beginn von kurz- und mittelfristigen Sanierungsarbeiten.

31. Dezember 1995

Dr. habil. Peter Hanelt beendet aus Altersgründen seine Tätigkeit als Leiter der Abteilung Taxonomie. Nach kommissarischer Leitung durch Dr. Reinhard Fritsch übernimmt am 1. März 1996 Prof. Dr. Konrad Bachmann die Leitung der Abteilung.

29. April 1996

Konstituierende Sitzung des Genbank-Beirates (Vorsitzender Prof. Dr. Dr. Gerhard Fischbeck).

6. bis 10. Juni 1996

Erstmalige Durchführung der „Gatersleben Research Conference“ zum Thema „Molecular Markers in Plant Genome Analysis and Crop Plant Improvement“.

2. September 1996

Erstmalige Verleihung des gemeinsam vom Institut und der Gemeinschaft zur Förderung der Kulturpflanzenforschung Gatersleben e. V. (gegründet am 1. März 1993) gestifteten Gaterslebener Forschungspreises zur Förderung des wissenschaftlichen Nachwuchses.

1. Januar 1997

Wiederberufung von Prof. Dr. Ulrich Wobus als Geschäftsführenden Direktor und Bernd Eise als Administrativen Leiter. Prof. Dr. Konrad Bachmann übernimmt die geschäftsführende Leitung der Genbank.

1. April 1997

Beginn des Aufbaus des Pflanzengenom-Ressourcen-Centrums (PGRC); Berufung von Dr. habil. Andreas Graner zum Koordinator.

31. Juli 1997

Altersbedingt gibt Prof. Dr. Klaus Müntz die Leitung der Abteilung Molekulare Zellbiologie an Priv.-Doz. Dr. Gotthard Kunze, der die Abteilung bis zum 15. Januar 1998 kommissarisch leitet.

16. Januar 1998

Prof. Dr. Uwe Sonnwald wird als Leiter der Abteilung Molekulare Zellbiologie berufen.

3./4. März 1998

Erneute Begutachtung des Instituts durch den Wissenschaftsrat.

10. Juli 1998

Veröffentlichung der Stellungnahme des Wissenschaftsrates zur Begutachtung des Instituts. Dem IPK wird eine sehr positive Entwicklung auf sehr hohem wissenschaftlichen Niveau bescheinigt.

24. Juli 1998

Ausgründung der ersten Biotechnologie-Firma - der Firma SunGene - am Standort Gatersleben; später folgen Novoplant (Geschäftsaufnahme 2000) und TraitGenetics (Geschäftsaufnahme 2001).

1. Januar 1999

Die Genbank des Instituts erhält den Abteilungsstatus. Priv.-Doz. Dr. Andreas Börner übernimmt die kommissarische Leitung.

30. April 1999

Grundsteinlegung für den Laborneubau Genomzentrum in Anwesenheit zahlreicher Gäste.

25. Oktober 1999

Beginn einer umfassenden Sanierung der Infrastruktur des IPK.

1. November 1999

Prof. Dr. Andreas Graner übernimmt die Leitung der Abteilung Genbank.

6. September 2000

Zur Verbesserung der Ausbildung der am IPK tätigen Doktoranden wird ein spezielles Weiterbildungsprogramm für Doktoranden begründet.

8. September 2000

Fierliche Eröffnung des IPK-Genomzentrums.

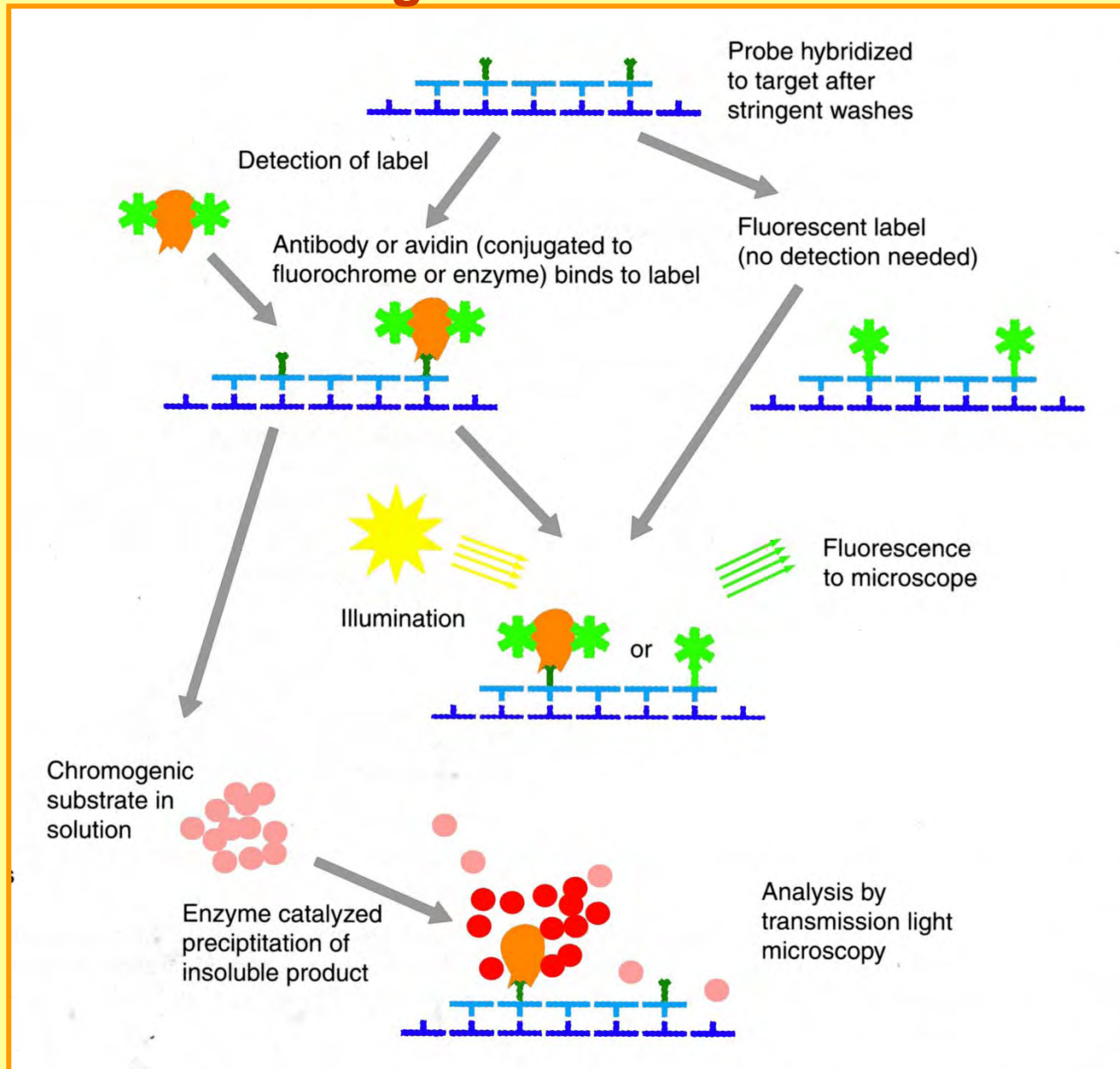
19. März 2001

Beginn der Rekonstruktion des Gebäudes Genetik.

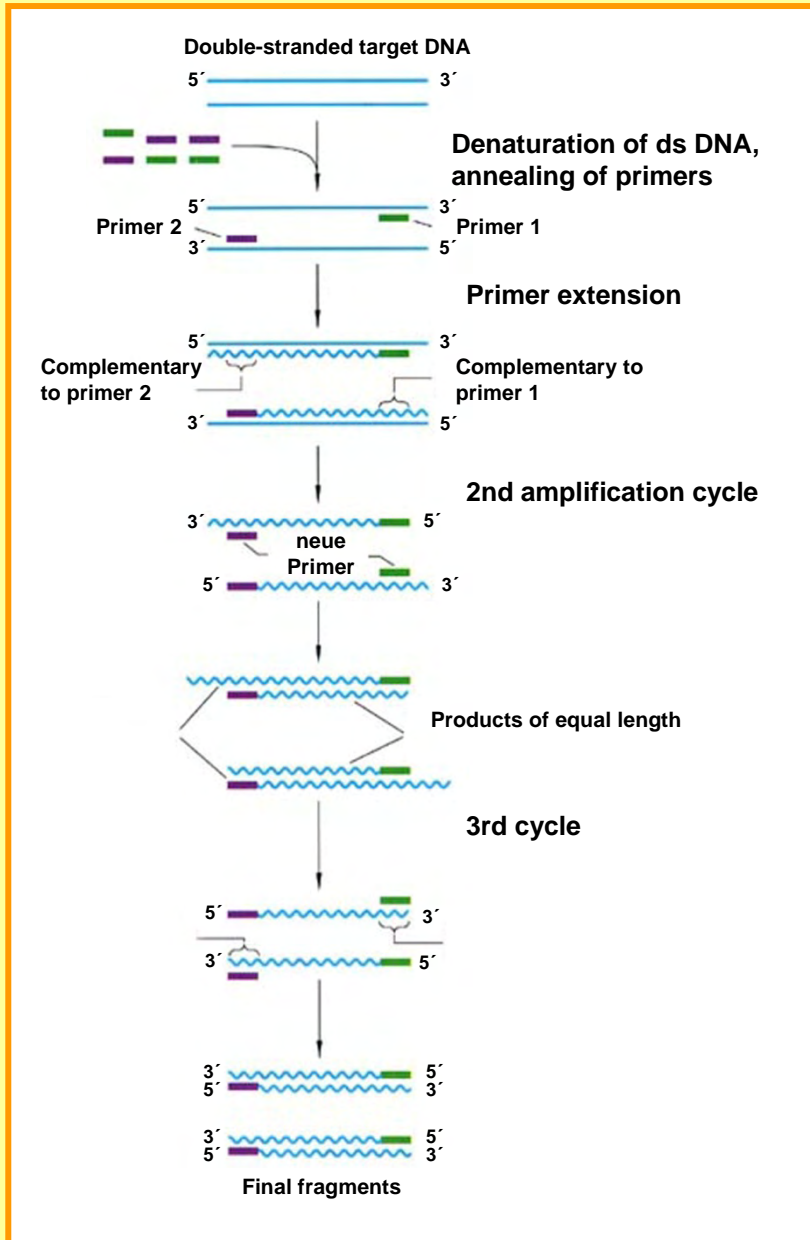
1. Januar 2002

Prof. Dr. Ulrich Wobus wird als Geschäftsführender Direktor und Bernd Eise als Administrativer Leiter wiederberufen.

Signal detection



PCR – Polymerase chain reaction (Saikai et al. 1985)



Cyclic amplification process comprising:

- i) Denaturation of template-DNA (90-95°C)
- ii) Annealing of sequence-specific primers (40-60°C)
- iii) Primer extension (70-75°C)

Enzyme: *Taq*-DNA-Polymerase

| Number of cycles | Number of equal-sized amplified ds fragments |
|------------------|--|
| 1 | 0 |
| 2 | 1 |
| 3 | 2 |
| 4 | 4 |
| 5 | 8 |
| 6 | 16 |
| 7 | 32 |
| 8 | 64 |
| 9 | 128 |
| 10 | 256 |
| 11 | 512 |
| 12 | 1 024 |

32 1 073 741 824