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Review article

Bewildering Bs: an impression of the 1st B-Chromosome Conference

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Beginning

Ever since their first discovery B chromosomes have attracted attention. Why are they so appealing? The standard chromosomes of an organism are A chromosomes; B chromosomes are extra to this normal complement. In the B chromosome 'bible' (Jones & Rees, 1982) Bs are defined as dispensable supernumerary chromosomes that are not homologous and do not pair with A chromosomes. They have been further characterized as (1) morphologically different from As (usually smaller), (2) being inherited in a non-Mendelian fashion, (3) not (or only rarely) having nucleolus organisers, (4) often displaying nondisjunction at anaphase of mitosis resulting in frequencies varying between organs in the same individual, (5) reducing fertility and growth when present in high numbers, and (6) carrying no genes with major effects. These features of Bs were recently discussed at an international conference and the main ideas presented by the participants are reported here.

From 21 to 25 September 1993 the 1st B-Chromosome Conference was held in Spain. Over 50 scientists representing 25 laboratories in 12 countries met at Residencia 'La Cristalera' in Miraflores de la Sierra. The aim of the meeting was to bring together scientists interested in B chromosomes and to discuss all aspects of their research. Quietly located amid pinewoods in the Guadarrama mountains some 50 km north of Madrid, this conference centre of the Universidad Autónoma de Madrid served this purpose excellently. Lively discussions and social contacts were generated in a pleasant Spanish atmosphere. It is not surprising that Spain was chosen for this meeting as it is the world centre of B chromosome research, with laboratories at the Universidad Autónoma de Madrid, the Universidad Complutense de Madrid and the Universidad de Granada.

In the past, much effort has been put into describing B chromosomes in a variety of organisms. Indeed, Bs are now known to occur in about 15 per cent of described living species and new ones are continually being found. Nevertheless, they have been studied in detail in few organisms, such as the economically important grasses maize (*Zea mays*) and rye (*Secale cereale*), the grasshopper *Myrmeleotettix maculatus* and the mealybug *Pseudococcus affinis*. These studies have traditionally shaped our knowledge of Bs. However, other systems have recently been described in detail (e.g. the plant *Allium schoenoprasum*, the grasshopper *Eyprepocnemis plorans* and the parasitic wasp *Nasonia vitripennis*) that challenge some of our notions about Bs. With this in mind, some B-loving scientists (J. L. Bella, C. García de la Vega, J. Gosálvez, R. N. Jones, C. López-Fernández and J. de la Torre) organized an international conference on B chromosomes.

The meeting had an unorthodox structure; only poster contributions were accepted from the participants, there were no plenary lectures and no oral presentations. Instead, six 3-h discussion sessions, each on a separate topic, were moderated by a chairperson: (1) Polymorphisms and geographical distribution (J. P. M. Camacho, Universidad de Granada), (2) Transmission: non-Mendelian heredity (W. R. Carlson, University of Iowa), (3) Genetic structure and organization (J. S. Parker, University of Reading), (4) Phenotypic effects (S. M. Bougourd, University of York), (5) Population dynamics (G. M. Hewitt, University of East Anglia), and (6) Summary (R. N. Jones, University of Wales). On the first day, the sessions were prepared by the participants together with the chairperson. On the following days, the chairperson started each session with a short introduction laying out the questions, and plenary discussions followed.

In this review citations without dates refer to posters presented at the meeting and are not listed in the references; those placed in square brackets refer to a chairperson's introduction or to a comment made during discussion.

*B*est conditions?

The first session aptly dealt with polymorphisms and the geographical distribution of Bs. Questions addressed were why are Bs so widespread and do they occur at particular places or under particular conditions? B chromosomes have been found most commonly among the species of certain groups (e.g. grasses and grasshoppers) but it became very clear at the meeting that this distribution, at least in part, reflects the distribution of researchers and the ease of cytological techniques across different taxa. However, they are now being discovered in groups where they were previously unknown. For example, because of new karyological methods, Bs are now often found in rodents (S. Kasahara, V. Fagundes, M. J. J. Silva, M. F. L. Assis & Y. Yonenaga-Yassuda; R. M. S. Barros & M. F. L. Assis) and neotropical fishes (A. S. Fenocchio, L. A. C. Bertollo & C. S. Takahashi; F. Foresti, C. Oliveira, L. F. Almeida-Toledo & E. L. Maistro). It was concluded that Bs probably occur in all living taxa and in all parts of the world.

An important distinction has to be made when talking about B frequencies between the mean number of individuals with Bs and the mean number of Bs per individual. The first is more relevant in a geographical context and the second when considering transmission rates and individual variation. Bs may be so widespread in nature because they are prone to drive and/or because they are a by-product of general processes of karyotypical evolution. There is some evidence from plants and humans that spontaneous chromosomal breaks leading to new chromosomal variants occur frequently. Many such novelties may disappear early on and not be discovered.

Given that Bs are cosmopolitan, can certain regularities be detected in their distributions? There have been several reports stating that Bs only flourish in areas where the ecological conditions are optimal for the existence of the species in which they occur (Jones & Rees, 1982). These conclusions have been supported by the discovery of clines in the frequencies of Bs (e.g. Hewitt & Brown, 1970; Shaw, 1983). Bs have often been found to correlate negatively with altitude and rainfall and positively with temperature (Jones & Rees, 1982). However, it became clear that generalizing about such correlations is dangerous. For example, Bs in the South American grasshoppers *Dichroplus elongatus* were found to increase with altitude (M. I. Remis, J. C. Vilardi, V. A. Confalonieri & A. Sequeira). Similarly, Bs were more frequent at higher altitudes in some South African grasses but less frequent in others (J. J. Spies & H. Du Plessis).

An important point in this context is how one determines favourable ecological conditions. Most participants would use the abundance of the species as their criterion. Reproductive success of a population would be a better standard but would be harder to measure. An alternative approach would be to test individuals with and without Bs under experimental regimes which simulate the physical conditions that have been found to correlate with B frequency. Only very few attempts have been made in this direction and most have been unsuccessful. Most organisms are difficult to raise under laboratory conditions and it is difficult to simulate different natural selection pressures. A second way of testing the favourable ecological conditions theory would be to transplant populations from one site in a cline to another and monitor B frequencies in transplanted and autochthonous populations. This approach has some conceptual difficulties [G. M. Hewitt; J. S. Parker], such as how neighbouring populations are prevented from invading. Furthermore, A genomes may be genetically adapted to the local environment and less fit in the area of transplantation and would therefore obscure selection on Bs. Finally, adaptations between the A and B complements may also confound measurements of environmental effects. Not until more people have pursued transplantation experiments may insight be gained into these problems.

There are other difficulties with the generalization that Bs are more common when conditions are favourable for the species in which they occur. Firstly, if Bs are beneficial to a species such a correlation would not be expected. Secondly, the presence of Bs may be determined historically. They could have originated in a particular population and not have spread from it yet, as has been suggested for *A. schoenoprasum* (Bougourd & Parker, 1979). Thirdly, the possibility of genetic drift effects should not be neglected. Long-term monitoring of the frequencies and the geographical distribution of Bs, such as the classical studies on *M. maculatus* by Hewitt and coworkers (e.g. Hewitt, 1973a; Shaw, 1984) may shed light on the importance of each of these processes. In summary, Bs may be absent from a certain population because it is beyond the limit of the species' ecological tolerance for B chromosomes and/or because Bs have not reached this locality from their centre of origin.

Another intriguing point that was raised is why are there so few organisms with many different types of Bs? This does not seem to result solely from a lack of study. From a selective point of view, it may be that there is a narrow niche for a B to exist in a species and hence strong selection for a particular type of B. Alternatively, because Bs differ in their effects and transmission rates, the 'best' B, in terms of its own maintenance, may outcompete the weaker ones, as appears to be the case in *A. schoenprasum* (Holmes & Bougourd, 1989). Very little is known about niche widths for the exist-

ence of Bs or competition between types of B chromosomes. The study of the B polymorphism in the grasshopper *E. plorans* in Spain in an exciting step in this direction (J. Gosálvez, C. López-Fernández, M. Dye & C. García de la Vega; Henriques-Gil & Arana, 1990; López-León, *et al.* 1993).

Two *B* or not two *B*?

Many B chromosomes possess accumulation mechanisms; these commonly involve preferential segregation at meiosis or nondisjunction at gametophyte mitosis followed by preferential inclusion in the functional gametes. In either case, this directed migration may simply be a passive process; the spindle may often be asymmetrical causing Bs to end up at the generative pole simply by chance (e.g. *M. maculatus*, Hewitt, 1976). (Has anybody ever observed directed migration in cells with symmetrical spindles?) Less common accumulation mechanisms include premeiotic accumulation caused by mitotic instability, preferential fertilization (e.g. in maize) and paternal genome elimination (e.g. in *Nasonia*). Why do Bs so often undergo nondisjunction? This is probably because Bs have no drastic aneuploid effects whereas nondisjunction of A chromosomes would be instantly detrimental and therefore strongly selected against. It was even suggested [W. R. Carlson] that all chromosomes have an inherent tendency to drive and would do so in the absence of harmful consequences.

An intriguing question is at what level is the transmission of Bs controlled? Do Bs contain specific accumulation regions causing nondisjunction, or do both A and B chromosomes contain such regions, which are differently controlled in both types? There is evidence for genes on Bs that control nondisjunction in maize [W. R. Carlson] and rye (W. Lee, H. Kwon & J. Lee). Genes that affect the transmission of Bs may also be found on A chromosomes, as was shown in *M. maculatus* (Shaw & Hewitt, 1985), *P. affinis* (Nur & Brett, 1988) and rye (Romera *et al.*, 1991). Other control factors could be maternal effects (e.g. Puertas *et al.*, 1990), cytotype (Beukeboom & Werren, 1992) or male ejaculate (J. P. M. Camacho, personal communication). Genetic control of B chromosome transmission is an exciting field of research where substantial contributions could be made. Can these controlling regions be isolated? Are they active genes with open reading frames, DNA repeats or merely heterochromatic domains?

Of special interest are Bs that do not have accumulation mechanisms. Although the appropriate statistics have not been performed, a preliminary literature survey (A. B. Plowman, unpublished data) suggests that this is the case for 20–30 per cent of Bs. Two such systems are currently studied in detail. In *A. schoenoprasum* (S. M. Bougourd, A. B. Plowman & M. L. Elias) Bs have average transmission rates of 0.4 and tend to be lost during meiosis. They are maintained in populations because of their beneficial effects on seed germination (Plowman & Bougourd, 1994). In *E. plorans* (López-León *et al.*, 1992) there is no drive (transmission rate is 0.5) and no selective elimination through harmful effects. Thus, this B may be regarded as being inherited at a Mendelian rate (i.e. 0.5).

*B*rain *b*oggling effects

A long-established feature of Bs is their effect on chiasma frequency of the A chromosomes. They have been found to increase, decrease or alter the location of crossover events. A chromosomes, which are synaptic in the absence of Bs, may become completely asynaptic in the presence of Bs, as is the case in *Lolium* hybrids (G. Jenkins & G. Jiménez). A heterochromatic region of the B in maize causes an increase in crossing-over between As (W. R. Carlson). Has this phenomenon any functional importance, e.g. is it good for the As, the Bs or both? Increased chiasma frequency has long been considered from an adaptive point of view. It may be a defence mechanism of the As to get rid of the Bs by creating new resistant genotypes through recombination (Red Queen hypothesis, Bell & Burt, 1990). There are several problems with this idea. As mentioned, the effect is neither universal nor unidirectional. Moreover, no one has ever shown that increased chiasma frequency results in any recombinants in which transmission rates of Bs are reduced. Until evidence for adaptive explanations is available, the ramifications of the crossover phenomenon remain unclear. It may very well be nonadaptive and merely a by-product of competition between As and Bs for the cell's replication machinery. Alternatively, increased cell cycle time as a result of the presence of Bs may permit formation of more chiasmata [J. P. M. Camacho].

There is a long-standing notion that Bs cannot survive in inbred lines, suggesting that they do better in a heterogeneous background (see Shaw & Hewitt, 1990). However, Benito *et al.* (1992) recently found the opposite, i.e. a negative correlation between the frequency of rye plants with Bs and the mean heterozygosity for isozyme loci. This relationship has never been empirically tested, but could be by introducing identical Bs into genetic backgrounds with varying degrees of homozygosity (inbreeding). Such experiments require organisms that are easily bred in the laboratory.

Another peculiar feature of Bs is their so-called 'odds and evens' effect. All sorts of effects of Bs (e.g. on chiasma frequency, fertility and growth rate) are more pronounced when the Bs are present in odd numbers than when present in even numbers. This effect has been found in diploid and polyploid organisms and its underlying mechanism remains a complete mystery, although some ideas were raised under the guarantee that they would not be published.

Birth

The first question that comes to mind when considering the genetic structure of Bs is their origin. The predominant view has been that Bs lack (sufficient) homology with the A chromosomes (to pair at meiosis). They have also been regarded as having orginated from the A complement. Until recently, little evidence was available to resolve these seemingly conflicting views. Some progress in this area was presented at the meeting and more will be presented in the near future. Table 1 lists the information currently available about sequence homology between A and B chromosomes (excepting rDNA). Some B chromosomes share DNA sequences with the A chromosomes (mostly located at centromeric and distal regions) and some have B-specific DNA repeats. It remains to be seen what fraction of each B is comprised of repetitive DNA. It is tempting to analyse the B-specific sequences but an analysis of sequences shared with the A chromosomes will be more revealing about the origin of a B. For example, do Bs contain one or a few regions of one A chromosome or do they share DNA with many members of the A complement? It is worth mentioning that if Bs arose from As, they will become less homologous with As over time because of accumulation of mutations (action of Muller's ratchet, Green, 1990). To test this hypothesis, it may be worthwhile to look for degenerated A chromosome genes in B DNA.

Recently, ribosomal DNA cistrons, mostly in the form of nucleolar organizer regions (NORs), have been found on many B chromosomes (reviewed by Green, 1990). Why do B chromosomes contain ribosomal DNA? Are the rDNA cistrons more likely to reside on the B than on any of the As? Do these rDNA clusters on Bs serve a function? There are several possible scenarios that can be tested. Their presence may be random and simply the result of the transposing capacity of rDNA sequences. The correct statistics have not been performed to test this idea. It may be that being part of a NOR confers a selective advantage to a B, e.g. in stabilizing its behaviour during meiosis ('cohesiveness'). Alternatively, rDNA may serve to attract certain cell products necessary for replication or it may affect nondisjunction. Finally, having a B with active rDNA cistrons may be advantageous for an organism through an increase in ribosomes that may indirectly benefit the B. These possibilities are open to experimental study. The B variants that have been found in *E. plorans* may be particularly useful for this purpose.

These ideas relate to the question of B chromosome DNA activity in general. The heterochromatic nature of Bs has often been linked with genetic inertness. Although the presence of major active genes on Bs seems to be exceptional, they clearly have genetic effects. Few people have investigated whether B rDNA is transcriptionally active. In the frog *Leiopelma hochstetteri* (Green, 1988) and the fly *Simulium juxtacrenobium* (Brockhouse *et al.*, 1989) active rDNA has been reported but in *E. plorans* the rDNA is inactivated by methylation (López-León *et al.*, 1991; M. Dye, J. M. Rubio, C. García de la Vega, C. Juan, J. Gosálvez & C. Lópes-Fernández). In rye, the rDNA is active in pollen cells, but inactive in somatic cells (M. Delgado, N. Neves, L. Morais-Cecílio, A. Barão, R. N. Jones & W. S. Viegas). More experimental work such as that of Delgado and coworkers, using methylation-sensitive restriction enzymes and artificial activation by incorporation of 5-azacytidine (5-AC), may reveal more about possible selective advantages of having rDNA. Obviously, the question of genetical activity of Bs is not restricted to rDNA but applies to the whole chromosome. One way to look for transcribed B sequences is to use subtractive methods with cDNA libraries [S. M. Bougourd]. Indeed, B chromosomes may turn out to be suitable model systems for studying the regulation of chromosomal activity.

Ribosomal DNA may play a role in the origin of Bs, e.g. by generating chromosomal breaks while transposing. This may lead to new B variants for which some evidence has recently been found in *E. plorans* (López-León *et al.*, 1993). The very fact that A and B chromosomes contain rDNA enables one to study their origin and phylogeny. For the first time, the age of a B may be determined by comparing the sequences of rDNA spacer regions between As and Bs. Of course, one should control for intra- and interchromosomal homogenising processes, such as gene conversion, that may occur between rDNA cistrons.

In grasshoppers, B chromosomes often resemble X chromosomes in their state of heterochromatinization. However, the X is usually facultatively heterochromatic whereas Bs are often obligately heterochromatic. Therefore, different mechanisms for the regulation of heterochromatin seem to be present in the cells. Particularly illustrative of this is the finding in mealybugs of a protein that specifically binds to heterochromatinized chromosomes in males but not to co-occuring heterochromatic Bs (Epstein *et al.*, 1992). Thus, Bs may be used to study the question as to how the structure of chromatin is regulated. How strict is the relation

Table 1 Current data on DNA homologies between A and B chromosomes (excepting rDNA sequences)

Species	B-specific sequences	Present in A genome of related species?	Sequences shared with As	Method†	Reference
Secale cereale (rye)	Two tandem repeats (1.1 and 3.9 kb)	Unknown		6	Sandery *et al.* (1990) Blunden *et al.* (1993)
Secale cereale (rye)	12%	Unknown	88%	4	R. Schlegel & A. Houben; A. Houben (unpublished data)
Crepis capillaris (plant)	None	—	Telomeres	1,2	Jamilena *et al.* (1994) J. Maluszynska & D. Schweizer
Brachycome dichromosomatica (plant)	One tandem repeat (176 bp), 10% of B	Yes*	Yes*	3,5	John *et al.* (1991) C. R. Leach, S. S. Spiniello, T. K. Franks & J. N. Timmis
Glossina spp. (tse-tse fly)	None	Yes	Telomeres, centromeres	2	Amos & Dover (1981)
Eypreprocnemis plorans (grasshopper)	None	—	One tandem repeat (180 bp)	2,6	López-León *et al.* (1994)
Nasonia vitripennis (wasp)	Three families of tandem repeats (171–214 bp), ⩾ 30% of B	Yes	One tandem repeat (94 bp)	1,6	Eickbush *et al.* (1992)

*About 85% homologous.

†1, Southern hybridization; 2, *in situ* hybridization; 3, subtractive hybridization; 4, cross-hybridization of microclones; 5, PCR amplification; 6, sequencing.

between repetitive DNA and heterochromatin? For example, the maize B chromosome is largely heterochromatic but does not seem to contain a lot of repetitive DNA (W. R. Carlson, personal communication). Are A and B heterochromatin identical in make-up and regulation?

*B*right future

During the meeting several new molecular techniques were mentioned or displayed on posters. A special section of the 'Genetic structure and organization' session was devoted to discussing these. In the past, many techniques have been used to analyse the DNA contents of Bs (e.g caesium chloride gradient centrifugation, renaturation kinetics, thermal denaturation, etc.). Better techniques are now available. One straightforward method to look for B-chromosome-specific DNA is to restriction digest 0B and +B individuals and score novel bands in the latter (RFLP analysis). Other methods that are now used to find B-specific sequences are subtractive hybridization and PCR using random primers (RAPD). *In situ* hybridization has become a popular technique now that, for instance, rDNA and telomeric probes are readily obtainable. However, the most exciting new technique is undoubtedly microdissection followed by microcloning of the B chromosomes (D. S. Holmes, S. Taylor, M. L. Elias & S. M. Bougourd; E. A. Robson, A. Houben, S. T. Bennett, J. W. Forster & J. S. Parker; R. Schlegel & A. Houben). In this technique, single Bs are taken off a microscope slide with a tiny needle and subsequently used to construct a B-specific genomic library. Microclones can then be analysed by cross-hybridization with A-specific microclones. This method opens up many new possibilities. One could determine for each B sequence whether it occurs on any of the As. Comparing sequences of shared DNA may shed light on karyological processes leading to the origin of Bs (translocations, fusions, etc.) and their subsequent evolution.

The most imaginative topic of the meeting was whether B chromosomes could be used for transformation (so called supernumerary chromosome vectors). Bs have some obvious advantages for this purpose: they are tolerated by the host, they are inherited, they can vary in number (which enables introduction of variable dosages of a particular insert) and there is potential for interspecies transfer. However, at present many technical aspects are unclear. To mention one, how are genes inserted and activated on Bs? Yet another reason to study DNA regulation in Bs!

*B*aleful, *b*ad, *b*enign or *b*eneficial?

By definition, B chromosomes cannot have dramatic effects on the phenotypic fitness of their host because of their dispensability. Is this therefore an irrelevant topic, as suggested by some? Or do the observed fitness effects have an evolutionary relevance? It will be clear from the above discussion that Bs do have effects, although many may be subtle in the context of fitness. Some drastic fitness effects have been reported, such as the Paternal Sex Ratio chromosome in *Nasonia* that changes males into females by destroying the paternal chromosomes (Werren, 1991) and the B of the fungus *Nectria haematococca* that confers toxin resistance (Miao *et al.*, 1991).

For decades it has been debated whether having B chromosomes is good or bad for one's health. Two different views have prevailed: the heterotic and the parasitic. The heterotic model maintains that Bs are beneficial to an organism, at least at low frequency. Frequencies are reduced by meiotic or mitotic loss and/or through reduced fitness of the hosts when present at high numbers. The parasitic model regards Bs as genomic parasites that are maintained by a balance between their drive mechanism and their negative effects on the host's fitness, even at low numbers. Surprisingly, the parasitic view seemed to dominate among the participants, confirming the wide acceptance of the selfish DNA theory. Peculiarly, being raised at a selfish DNA school, I was struck by the clear evidence for the beneficial effects of some Bs (e.g. the case of *A. schoenoprasum*, Plowman & Bougourd, 1994). We may now have reached a stage where both models are considered valid. A thorough consideration of all transmission rates of Bs in the literature will help to resolve further the issue. A third view that Bs would evolve from being parasitic to neutral to heterotic and vice versa [J. P. M. Camacho] may very well gain more support.

An intriguing question that remains unanswered is how Bs exert negative effects on host fitness. One suggestion is that a host has to deal with more DNA which confers a cost, for example, by increasing the cell cycle. Another idea concerns the intracell competition for the replication machinery. If Bs compete with As for certain enzymes, then having too many Bs may hamper proper replication of the As. One may therefore expect that polyploids would tolerate larger numbers of Bs, assuming they have larger amounts of enzymes. However, no such correlation is known.

*B*alance through *b*attle?

Two parameters must be known to understand the dynamics of any B chromosome system: (1) how they are inherited, and (2) what effects they have on the fitness of their carrier. Many Bs have an accumulation mechanism (drive); they may be transmitted disproportionately during particular stages of cell division in the germline of only one or both sexes. The majority of Bs

also have negative fitness effects, especially when present in large numbers. In these instances, population frequencies of Bs are believed to result from a balance between accumulation by non-Mendelian transmission and elimination by reduced reproductive success of their carriers. However, some Bs have no drive; they increase in populations because they have advantageous effects on host fitness.

Besides selective elimination of individuals with many Bs, B frequencies in populations can be reduced by genetic factors located on A chromosomes (Shaw, 1984; Shaw & Hewitt, 1990). Genotypes may evolve that counteract the accumulation of Bs. Such transmission-reducing genotypes (TRGs, also referred to as modifiers or suppressors) have been found in several organisms. It is worth considering this arms race between As and Bs in more detail (Red Queen hypothesis). How wide is the niche for the A complement to evolve a modifier of B chromosome transmission? Obviously, a successful modifier must specifically affect transmission of the B (and not the As) and not have severe fitness reducing effects at the same time. A number of variables may play a role, such as the developmental stage at which accumulation occurs, the mechanism of accumulation and potentially the nature of the B. Suppressing effects may be overcome if the accumulation mechanisms of Bs are changed or if their deleterious consequences are decreased. Unfortunately, few data are available to verify the reality of this potential arms race. How often do variants with respect to transmission rates arise among Bs? By which processes can transmission rates be affected? Recent studies of B variants substitution and the rate of formation of new variants in *E. plorans* (Henriques-Gil & Arana, 1990; López-León *et al.*, 1993) are an exciting step forward. Another particularly well documented case is the mealy bug *P. affinis* (Nur & Brett, 1988), where genotypes have been found that affect the heterochromatinization of the Bs but not of the As. Maybe more such data can be obtained in laboratory cultures using organisms with short generation times and high numbers of offspring.

Currently, very few B systems are studied from a population biology perspective. Such studies require proper measurement of transmission rates, fitness effects, dispersal rates, population size and structure, etc. General models of population dynamics may be developed to serve as a guide for measuring the correct variables. Such models may also be useful to determine the feasibility of obtaining the appropriate data from a particular system. One could imagine a model that includes all possible life cycle steps at which Bs may exert an effect, i.e. accumulate, be lost or have fitness effects. Processes at the population level should also be included, such as genetic drift, mating structure and sexual selection. Finally, one may model the effects of suppressor genotypes at different stages. Kimura & Kayano (1961) made the first attempt of such a model. A similar approach was recently undertaken for the B of *A. schoenoprasum* (A. B. Plowman & S. M. Bougourd).

During the meeting the need for more long-term geographical studies was questioned. Several arguments were raised in favour of such studies. Firstly, as discussed, to distinguish between historical (e.g. place of origin) and selective effects in explaining the distribution of a particular B, extensive geographical data are necessary. Another argument in support is the sheer lack of knowledge about distribution and dynamics of suppressor genotypes and/or cytotypes. In *Nasonia*, a particular cytotype was found to increase the B frequency and predictions were made about the co-occurrence of both under natural conditions (Werren & Beukeboom, 1993). Detailed field studies that monitor the distributions and frequencies of Bs and TRGs, in combination with measuring fitness variables, may add to our understanding of B dynamics. A final argument is that data obtained from one population may not be valid for another, as demonstrated in *M. maculatus* (Hewitt, 1973b; Shaw & Hewitt, 1985).

Beautiful outlook?

One of the key conclusions of the meeting was that a new definition of a B chromosome is necessary. Several of the 'defining features' of Bs are not tenable anymore, such as their lack of homology with the As and their absence of nucleolus organizers. In the last session no participant objected to the new definition that was developed by J. P. M. Camacho and J. S. Parker prior to the meeting and proposed by J. P. M. Camacho during the first session: the modern B is defined as '*a dispensable supernumerary chromosome that does not recombine with the A chromosomes and follows its own evolutionary pathway*'.

A second message was the identification of major routes of research on Bs in the near future. A lot of knowledge may be gained from molecular characterization of Bs. This may reveal more about their origin, how old they are, how often new variants arise and how they become different from the A chromosomes. The latter relates to the intriguing question as to how the DNA of Bs is (in)activated. Another topic that needs further exploration is the types of interactions that occur between As and Bs (e.g. chiasma formation, transmission suppression, etc.). Many basic questions about B chromosomes still need further research. They are known to accumulate by nondisjunction but what is

the exact mechanism? How do they attain preferential fertilization? Finally, studies of population dynamics are still important. It was proclaimed that in 2 years all B chromosome work will have been carried out [R. N. Jones]. I hope that these comments have conveyed the contrary.

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References

AMOS, A. AND DOVER, G. 1981. The distribution of repetitive DNAs between regular and supernumerary chromosomes in species of *Glossina* (Tsetse): a two-step process in the origin of supernumeraries. *Chromosoma*, **81**, 673–690.

BELL, G. AND BURT, A. 1990. B-chromosomes: germ-line parasites which induce changes in host recombination. *Parasitology*, **100**, S19–S26.

BENITO, C., ROMERA, F., DÍEZ, M. AND FIGUEIRAS, A. M. 1992. Genic heterozygosity and fitness in rye populations with B chromosomes. *Heredity*, **69**, 406–411.

BEUKEBOOM, L. W. AND WERREN, J. H. 1992. Population genetics of a parasitic chromosome: experimental analysis of PSR in subdivided populations. *Evolution*, **46**, 1257–1268.

BLUNDEN, R., WILKES, T. J., FORSTER, J. W., JIMENEZ, M. M., SANDERY, M. J., KARP, A. AND JONES, R. N. 1993. Identification of the E3900 family, a second family of rye B chromosome specific repeated sequences. *Genome*, **36**, 706–711.

BOURGOURD, S. M. AND PARKER, J. S. 1979. The B-chromosome system of *Allium schoenoprasum* III. An abrupt change in B-frequency. *Chromosoma*, **75**, 385–392.

BROCKHOUSE, C., BAS, J. A. B., FERADAY, R. M. AND STRAUS, N. A. 1989. Supernumerary chromosomes evolution in the *Simulium vernum* group (Diptera: Simuliidae). *Genome*, **32**, 516–521.

EICKBUSH, D. G., EICKBUSH, T. H. AND WERREN, J. H. 1992. Molecular characterization of repetitive DNA sequences from a B chromosome. *Chromosoma*, **101**, 575–583.

EPSTEIN, H., JAMES, T. C. AND SINGH, P. B. 1992. Cloning and expression of *Drosophila* HP1 homologs from a mealybug *Planococcus citri*. *J. Cell Sci.*, **101**, 463–474.

GREEN, D. M. 1988. Cytogenetics of the endemic New Zealand frog *Leiopelma hochstetteri*: extraordinary supernumerary chromosome variation and a unique sex-chromosome system. *Chromosoma*, **97**, 55–70.

GREEN, D. M. 1990. Muller's ratchet and the evolution of supernumerary chromosomes. *Genome*, **33**, 818–824.

HARVEY, P. H. 1989. Parasitological teeth for evolutionary problems. *Nature*, **342**, 230.

HENRIQUES-GIL, N. AND ARANA, P. 1990. Origin and substitution of B chromosomes in the grasshopper *Eyprepocnemis plorans*. *Evolution*, **44**, 747–753.

HEWITT, G. M. 1973a. Evolution and maintenance of B chromosomes. *Chromosomes Today*, **4**, 351–369.

HEWITT, G. M. 1973b. Variable transmission rates of a B-chromosome in *Myrmeleotettix maculatus* (Thunb.) (Acrididae: Orthoptera). *Chromosoma*, **40**, 83–106.

HEWITT, G. M. 1976. Meiotic drive for B-chromosomes in the primary oocytes of *Myrmeleotettix maculatus* (Orthoptera: Acrididae). *Chromosoma*, **56**, 381–391.

HEWITT, G. M. AND BROWN, F. M. 1970. The B-chromosome system of *Myrmeleotettix maculatus* V. A steep cline in East Anglia. *Heredity*, **25**, 363–371.

HOLMES, D. S. AND BOUGOURD, S. M. 1989. B-chromosome selection in *Allium schoenoprasum*. I. Natural populations. *Heredity*, **63**, 83–87.

JAMILENA, M., RUIZ REJÓN, C. AND RUIZ REJÓN, M. (1994). A molecular analysis of the origin of the *Crepis capillaris* B. chromosome. *J. Cell Sci.*, **107**, 703–708.

JOHN, U. P., LEACH, C. R. AND TIMMIS, J. N. 1991. A sequence specific to B chromosomes of *Brachycome dichromosomatica*. *Genome*, **34**, 739–744.

JONES, R. N. AND REES, H. 1982. *B Chromosomes*. Academic Press, London.

KIMURA, M. AND KAYANO, H. 1961. The maintenance of supernumerary chromosomes in wild populations of *Lilium callosum* by preferential segregation. *Genetics*, **46**, 1699–1712.

LÓPEZ-LEÓN, M. D., CABRERO, J. AND CAMACHO, J. P. M. 1991. A nucleolus organizer region in a B chromosome inactivated by DNA methylation. *Chromosoma*, **100**, 134–138.

LÓPEZ-LEÓN, M. D., CABRERO, J., CAMACHO, J. P. M., CANO, M. I. AND SANTOS, J. L. 1992. A widespread B chromosome polymorphism maintained without apparent drive. *Evolution*, **46**, 529–539.

LÓPEZ-LEÓN, M. D., CABRERO, J., PARDO, M. C., VISERAS, E., CAMACHO, J. P. M. AND SANTOS, J. L. 1993. Generating high variability of B chromosomes in *Eypreprocnemis plorans* (grasshopper). *Heredity*, **71**, 352–362.

LÓPEZ-LEÓN, M. D., NEVES, N., SCHWARZACHER, T., HESLOP-HARRISON, T. S., HEWITT, G. AND CAMACHO, J. P. M. 1994. Possible origin of a B chromosome deduced from its DNA composition using double FISH technique. *Chromosome Res.*, **2**, 87–92.

MIAO, V. P. W., MATTHEWS, D. E. AND VANETTEN, H. D. 1991. Identification and chromosomal locations of a family of cytochrome P-450 genes for pisatin detoxification in the

fungus *Nectria haematococca*. *Mol. Gen. Genet.*, **226**, 214–223.

NUR, U. 1977. Maintenance of a "parasitic" B chromosome in the grasshopper *Melanoplus femur-rubrum*. *Genetics*, **87**, 499–512.

NUR, U. AND BRETT, B. L. H. 1988. Genotypes affecting the condensation and transmission of heterochromatic B chromosomes in the mealybug *Pseudococcus affinis*. *Chromosoma*, **96**, 205–212.

PLOWMAN, A. B. AND BOUGOURD, S. M. 1994. Selectively advantageous effects of B chromosomes on germination behaviour in *Allium schoenoprasum* L. *Heredity*, **72**, 587–593.

PUERTAS, M. J., JIMÉNEZ, M. M., ROMERA, F., VEGA, J. M. AND DÍEZ, M. 1990. Maternal imprinting effect on B chromosome transmission in rye. *Heredity*, **64**, 197–204.

ROMERA, F., JIMÉNEZ, M. M. AND PUERTAS, M. J. 1991. Genetic control of the rate of transmission of rye B chromosomes. I. Effects in 2B × 0B crosses. *Heredity*, **66**, 61–65.

SANDERY, M. J., FORSTER, J. W., BLUNDEN, R. AND JONES, R. N. 1990. Identification of a family of repeated sequences on the rye B chromosome. *Genome*, **33**, 908–913.

SHAW, M. W. 1983. Rapid movement of a B-chromosome frequency cline in *Myrmeleotettix maculatus* (Orthoptera: Acrididae). *Heredity*, **50**, 1–14.

SHAW, M. W. 1984. The population genetics of the B-chromosome of *Myrmeleotettix maculatus* (Thunb.) (Orthoptera: Acrididae). *Biol. J. Linn. Soc.*, **23**, 77–100.

SHAW, M. W. AND HEWITT, G. M. 1985. The genetic control of meiotic drive acting on the B-chromosome of *Myrmeleotettix maculatus* (Orthoptera: Acrididae). *Heredity*, **54**, 187–194.

SHAW, M. W. AND HEWITT, G. M. 1990. B chromosomes, selfish DNA and theoretical models: where next? In: Futuyma, D. and Antonovics, J. (eds) *Oxford Surveys in Evolutionary Biology*, vol. 7, pp. 197–223. Oxford University Press, Oxford.

WERREN, J. H. 1991. The paternal-sex-ratio chromosome of *Nasonia*. *Am. Nat.*, **137**, 392–402.

WERREN, J. H. AND BEUKEBOOM, L. W. 1993. Population genetics of a parasitic chromosome: theoretical analysis of PSR in subdivided populations. *Am. Nat.*, **142**, 224–241.