Published by Associazione Teriologica Italiana
Hystrix, the Italian Journal of Mammalogy
Volume 33 (2): 102–106, 2022
OPEN ACCESS
Available online at:
http://www.italian-journal-of-mammalogy.it
doi:10.4404/hystrix-00509-2021

Commentary

Adaptive nature of chromosome variation in placental mammals and applicability to domestication and invasiveness

Mazin B. Qumsiyeh*, Elias N. Handal

Palestine Institute for Biodiversity and Sustainability, Bethlehem University, Bethlehem, Palestine

Keywords:
chromosome number
meiosis
genetic variation

Article history:
Received: 03 December 2021
Accepted: 28 June 2022

Acknowledgements
We thank two anonymous reviewers who helped edit and made suggestions to this manuscript.
Funding for the Biodiversity center was from the EUPI project "Unity and Diversity in Nature and Society" (fund number ENI/2019/162124/83).

Abstract

Research on chromosome evolution accumulated in the past three decades that seems to validate the hypothesis postulated that chromosome changes that increase or decrease of chromosome number are adaptive partly because they increase or decrease in variation via effects on recombination and segregation in meiosis. The paper reviews some of the new data relevant to this question and especially focus on studies on interspecific and intraspecific chromosome variation in placental mammals. We find data in support of that hypothesis coming from many areas and for the first time suggest applications in some domesticated and invasive species of mammals. This hypothesis does not explains all chromosome number variation in mammals but it does advance our understanding and opens future avenues of research into mammalian variation and adaptability.

Introduction

The range of chromosome numbers in mammals is between 2n=6 to 7 (Wurster and Benirschke, 1970) to 2n=102 (a South American rodent though maybe polyploid, Gallardo et al., 1999). Chromosome evolution patterns and rates are variable with many lineage specific differences; some of it associated with higher speciation rates (Bush et al., 1977; Bengtsson, 1980; Bourque et al., 2004; Romanenko et al., 2012; Castiglia, 2014; Martinez et al., 2016). Older literature supposed fixation of even deleterious chromosome rearrangements because of genetic drift and population bottlenecks without chromosome rearrangements having adaptive value (Wilson et al., 1975; Bush et al., 1977). The fact that this variation is not random and certainly not seen only in populations of vertebrates with possibilities of bottlenecks (Ruiz-Herrera et al., 2012), forced many authors to start thinking of alternatives and the field of examining adaptive nature of rearrangements has expanded significantly (see Dobigny et al., 2017 for a review). Thus, recent data challenged the model of bottlenecks or extended them. This was necessitated to cover areas including step-wise adaptation and the drive towards fixation even in situation of monobrachial homology (Bickham and Baker, 1979; and see discussion and references below).

If one adopts the idea that chromosomal changes are adaptive and not merely because they allow reproductive isolation then it is possible to begin to understand patterns of chromosomal variation within a species and also between species and higher categories (see review in Qumsiyeh, 1994). Some authors postulated that Eukaryotic genome evolution proceeds directionally by fissions increasing chromosome numbers (Imai et al., 2002; Fontana and Rubini, 1990; Todd, 1975) while others postulate a "fusion´´ drive resulting in decrease in chromosome number (Fontana and Rubini, 1990). But there is now a significant body of evidence accumulated and a consensus that different lineages may acquire fusions or fissions and thus some lineages increase chromosome number and some decrease it (King, 1982; McClintock, 1993; Qumsiyeh, 1994; Phillips and Rab, 2001; Ferguson-Smith and Trifonov, 2007). There is also evidence that higher categories of classification that are clearly monophyletic (e.g. a monophyletic family like Rhinolophidae) have chromosome constitution that may not change much after the formation of the taxon (this is termed the canalization model of chromosomal change Bickham and Baker, 1979). The latter authors did not deal with mechanism of adaptability via chromosomal changes though they did mention the various possibilities without discussing them (position effects, supergenes, linkage groups, centromere position, and regulatory functions). For Robertsonian translocations (ROBs) and whole arm translocations that increase or decrease chromosome number without causing hardly any regulatory or position effect, the explanation has been more difficult. This is also compounded by the fact that ROBs are the most common rearrangements in mammals including for intraspecific variation (see below). Yet, there is some evidence that such translocations may indeed alter the nuclear architecture and thus have consequences beyond their effect in meiosis (Qumsiyeh, 1995; Garagna et al., 2001).

A model that attempted to explain the widespread presence of fissions and fusions in mammals was proposed more than 20 years ago (Qumsiyeh, 1994) that challenges the earlier notions of deme size models and fixation of random events. The model is based on the simple idea that increase in chromosome number in itself allow more variation in progeny due to two factors: a) random segregation in meiosis where number of possible outcomes is 2^n, and b) due to the fact that higher chromosome numbers are also associated with increased chiasma frequency. The model thus argues that the drive to increase or decrease chromosome numbers in different lineages creates more or less variation that is then advantageous depending on the environmental situation. According to this model, there could be selection for increased chromosome numbers in lineages which increase variation in progeny thus allowing response to stressed or heterogeneous environments. The reverse is true with species subjected to stable environments undergo-

*Corresponding author
Email address: mazin@qumsiyeh.org (Mazin B. Qumsiyeh)

ing reduction in chromosome numbers once they reached the optimum karyotype. This is not a challenge to the canalization model (Bickham and Baker, 1979) but an explanation of it.

Since that notion of linkage of segregation and recombination with variation was developed in 1994 as an explanation of possible adaptive nature of the karyotype after fissions/fusions (though obviously not the only effect of such rearrangements), many authors published data on chromosome number in heterogeneous or stressed environments that fit well within this model and the authors commented on its applicability. This paper reviews accumulated data and explain potential applicability of the hypothesis to recent on issues ranging from interspecific variation in wild species to invasive and domestic species to mammalian intraspecific variation. The paper reviews accumulated data, both for interspecific variation and intraspecific variation with special emphasis on decrease and increase in chromosome number by fusion and fission (and not other chromosome rearrangements) in association with organisms' adaptability. In the case of inversions, the effect on recombination is found in the selection for suppression of recombination both within the inversion loop in heterozygotes and elsewhere in the genome (Trickett and Butlin, 1994; Hoffmann and Rieseberg, 2008).

Recombination and genetic variation

The hypothesis of Qumsiyeh (1994) basically can be summarized as stating that these effects on recombination provide a "drive" towards higher chromosome number (via fissions) in unstable heterogenous environments and towards lower chromosome numbers (via fusion) in stable environments when the species is highly adapted to that environment. This is due to both impact in increase on probabilities of gametes by random segregation of chromosomes (probabilities 2^n where n is haploid chromsoome number) *and* on recombination rates. Qumsiyeh (1994) provided numerous examples of how this can increase variation (genetic and hence also phenotypic) and discussed the issue of recombination relating to increase and decrease in chromosome number and effect on recombination. More recent data on recombination confirmes these trends.

Chromosome numbers and FN (number of autosomal arms) are directly proportional to recombination which also impacts variation (Qumsiyeh, 1994). This requires some more research but note for example the correlation between the sex averaged genetic map in mammals and number of chromosomal arms (Coop and Przeworski, 2007). There are data that show positive relationship of FN to recombination in rodents, marsupials and primates (De Villena and Sapienza, 2001a and references therein). The chromosome size in and of itself (outside the issue of random segregation) affects recombination (Gazave et al., 2003; Kaback and Guacci, 1992; Kaback, 1996; Qumsiyeh, 1994; Kong et al., 2002). Further, patterns of recombination are affected not just by length of chromosome but also by interference and centromere and telomere effects (Borodin et al., 2008).

If the environment is constantly shifting, one would expect natural selection to favor increased variation in progeny (Burt and Bell, 1987). Increased recombination can also drive genome size reduction in mammals leading to stabilization (Nam and Ellegren, 2012). There are still many questions about genetic controls of recombination but there is no question now about evolutionary significance associated with variation in chromosome number and structure (see reviews in Coop and Przeworski, 2007; Paigen and Petkov, 2010; Baudat et al., 2013). New mechanisms for regulation of recombination hotspots are also being discovered and related to potential selective forces operating at the molecular level which could explain meiotic drive (e.g. Odenthal-Hesse, 2014). Recombination rate and hotspots of recombination are controlled by few genes such as PRDM9 (Paigen and Petkov, 2018; Úbeda et al., 2019) and these can be subject to environmental selection that suits ecological and habitat needs (see Burt, 2000; Stapley et al., 2017 and references therein). The relationship of recombination to chromosome number is reviewed by Stapley et al. (2017) and shows a clear relationship between chromosome number and recombination in fungi, plants, and animals though for animals the relationship was quadratic. Certainly, more work is needed in this area. Indeed recombination changes due to FN changes or other mutations that increase recombination can help explain issue of phenotypic variation not related to chromosome number. The cat with 2n=38 has a very high recombination rate (4370 cM, Menotti-Raymond et al., 2009) while the dog 2n=78 is lower (1978 cM, Campbell et al., 2016). It would be difficult to say that dogs have either higher or lower variation than cats based on these data.

Intraspecific variation

Intraspecific variations/chromosome polymorphims are common in mammals including marsupials, insectivora, primates, carnivores, perissodactyla, and rodents (Qumsiyeh, 1994; Dobigny et al., 2017). It is also not surprising then that there are many species with intraspecific variation of chromosome numbers especially in small mammals because of the ease of getting karyotypes on good sample sizes (Dobigny et al., 2017). In the mole rats of the genus *Spalax* there is strong evidence that the chromosomal species with the highest chromosome number occur in the most stressed habitats (Nevo, 1991, 1998; Nevo et al., 1994).

One of the most polymorphic species studied intensively is *Blarina carolinensis* and the data is suggestive of rapid polymorphism via fissions that lead to increase in chromosome number, which is correlated to the tectonic instability of the area (Qumsiyeh et al., 1999, 1997). The fact that some species with highly polymorphic chromosomes in Robertsonian translocations seem to fit the Hardy-Weinberg expectations indicate that there is little strong negative consequences for heterozygotes (Nachman and Myers, 1989; Qumsiyeh et al., 1997). ROBs could have impact on volume or positions of chromosomes in interphase nucleus which is critical in gene expression (Qumsiyeh, 1999, 1995; Garagna et al., 2001) but in some cases they do impact structure of the nucleus (Acloque et al., 2013). While some authors postulated that we would see more centric fusions than centric fission in ROBs, clearly this view is an oversimplification and there are many mammalian lineages with centric fissions (Perry et al., 2004).

The house mouse is a good model of significant variation in chromosome number due to Robertsonian translocations with 2n ranging from 22 to 40 occurring relatively recently and associated with little genic variation (Britton-Davidian et al., 1989). While controversial, there are data that suggest habitat segregation for mice with low and high chromosome number (Chatti et al., 1999; Castiglia and Capporioni, 2005). Mice with Robertsonian translocation were found to have a decrease and a re-patterning in chiasma frequency (Dumas and Britton-Davidian, 2002) which substantiates the hypothesis of link between recombination and chromosome speciation (see discussion in Giménez et al., 2016). Further there is an apparent non-randomness in terms of chromosomes involved in fusion suggesting selective forces operate in the mouse model (Gazave et al., 2003) different than the human model (De Villena and Sapienza, 2001b). In such cases and in the case of human carriers of balanced translocations, the gametes produced are not random in terms of segregation but their appears to be a drive in female meiosis towards increased gametes with either higher number of centromeres or lower number of centromeres in other words non-random preferential segregation in female meiosis (De Villena and Sapienza, 2001a). This may actually explain mechanistically the way the drive is selected for to increase or decrease chromosome numbers via female meiosis (i.e. drive to fix after starting via heterozygous condition, see Chmátal et al., 2014). The West African *Gerbillus nigeriae* also shows significan variation with Robertsonian translocation and excess heterozygocity which suggested that this helps the species in its rather unstable environment (Hima et al., 2011).

Interspecific variation

There has been an accumulation of data since the initial publication in 1994 that show that interspecific chromosome variation in wild mammals is associated with degree of environmental heterogeneity (i.e. unstable environments have taxa with higher chromosome numbers). A quick scan of the literature reveals that the hypothesis relating to independent assortment, recomibination, and variation is not only a applied

to taxa listed above but include other mammals (Peppers, 1998; Ropiquet et al., 2008; Nash et al., 1999) and is even noted in fish (Symonova, 2013; Nirchio and Oliveira, 2006; Phillips and Rab, 2001; Danzmann et al., 2005; Azevedo et al., 2007), reptiles (Olmo et al., 2002), beetles (Proença et al., 2002), scorpions (Qumsiyeh et al., 2014), lice (Shao et al., 2009), ants and wasps (Imai et al., 2001), and cereal plants (Devos and Gale, 2000). By contrast, few authors questioned whether the models fit their group of interest such as data on deer chromosomes (e.g. Slate et al., 2002).

Literature does not include a discussion of why some mammal species were prone to domestication and invasion in relation to genetic variation due to higher chromosome numbers as would be predicted by the model. Natural selection leading to invasiveness would be expected in species with higher chromosome numbers/higher FN/higher recombination which would allow them to adapt to various conditions in different invaded habitats (different than the one they evolved in). Domestication is in essence similar to invasiveness but in a selective breeding program by human mediated artificial selection rather than natural selection. In other words, species with high variation would be easier to domesticate than species with lower variation (easy to find enough variation in each generation). Domesticated animals today would be expected to have on average higher chromosome number than wild undomesticated and related species. The consensus is that the ancestral chromosome number in placental mammals ranges from 44 to 50 with the most likely being 46 which happens to also be around the mean number (Ferguson-Smith and Trifonov, 2007; Ruiz-Herrera et al., 2012).

In a quick examination of chromosome numbers of invasive, domestic and some commensal mammalian species were noted to have high chromosome numbers but that there are exceptions. Examples of high chromosome numbers include dogs (2n=78), reindeer (2n=70), donkeys (2n=62), horses (2n=64), goats (2n=60), yak (2n=60), guinea pig (2n=64), common cattle/cow (2n=60), Cambodian cattle (2n=56), Asian elephant (2n=56), llama (2n=74), and camels (2n=74). But there are some species that have intermediate range chromosome numbers: mink (2n=30), fox (2n=34), pig (2n=36), cat (2n=38), black rat (2n=38), ferret (2n=40), coypu (2n=42), Norwegian rat (2n=42), pig (2n=38), nutria (2n=42), hamster (2n=44), and rabbit (2n=44), domesticated hedgehog (2n=48). The reasons might be lineage specific meiotic drive (see discussion below). But let us take all these 25 species and compare their average and standard deviation to those of mammals with known chromosome numbers compiled by O'Brien et al. (2006). The data for 854 mammals (all orders) have a mean diploid number of 42.671 (standard deviation 14.6882, maximum 102, minimum 7). The data for the 25 species listed above have a mean diploid number of 51.76 (standard deviation 14.17, maximum 78, minimum 30). The 25 species have diploid numbers with the mean number of chromosomes (51.76) significantly higher than the population mean of 42.671 (2-tailed t-test, p=0.004).

Yet, even though they are significantly higher in mean 2n, invasive and domesticated species fall in two categories: one with diploid numbers of 60 to 78 and one group with 38–44. The latter though have higher FN (more arms and hence more recombination) than similar species with similar numbers that are not domesticated or invasive.The discussion in Driscoll et al. (2009) about the modes of domestication of cat (2n=38) versus dog (2n=78) suggest the latter was under more artificial human section than the self-selection of the former. The human karyotype (2n=46) actually fits as a species that has self-selected as in Driscoll et al.'s 2009 cats! But this subject needs further research.

Nine inversions and one fusion distinguish humans from their closest evolutionary relatives, the chimpanzee (Szamalek et al., 2006). The average chiasmata in human and chimp are respectively 51 and 44 (Datta, 1972; Falek and Chiarelli, 1968). The difference in chiasmata would not be strictly attributable to increase in diploidy by one pair in human but by unique heterochromatin blocks in Chimpanzees that lowered recombination rate — or alternatively humans as having more recombination resulted in molecular plasticity (Ventura et al., 2012; Rogers and Gibbs, 2014). Evolutionary rearranged human chromosomes (compared to Chimpanzee) are reported to have higher variability in nucleotide sequences than those that are co-linear (un-rearranged) (Navarro and Barton, 2003). The evolution of the recombination map of human compared to chimpanzee has been indeed very rapid (Munch et al., 2014) and perhaps explains the phenotypic plasticity of humans due to a rich recombination around hotspots in both humans and chimpanzees (Auton et al., 2012; Stevison et al., 2016). Taken together, these data point to the need for understanding why the rearrangement in higher primates happened the way they did and their consequences for increased genomic variation and spread of human populations as well as recombination landscape (see Stapley et al., 2017) as a driver of phenotypic variation and ultimately selection and evolution.

There is strong evidence that invasive species are invasive because they were able to use molecular mechanisms such as genome reshuffling and polyploidy in plants to increase variation (Prentis et al., 2008). The increased number of progeny and higher variation due to increased chromosome number in invasive mammal species, might compensate for the bottleneck effect of initial invasion (Excoffier et al., 2009). Further studies especially on invasive mammal species are needed.

Discussion

The data cited above from many authors over the past few decades about intraspecific and interspecific variation of chromosome rearrangement rates validated the adaptive (non-random) advantage of changes in diploid numbers and karyotypic structure including those caused by ROBs. The model of effect on recombination and segregation (Qumsiyeh, 1994) received significant support subsequently from cytogenetic studies of many groups of animals with variation in numbers correlated with habitats and adaptation (see numerous citations above for different groups of animals studied). Yet, there was little discussion in the literature on relevance of the model to intraspecific variation and other issues like domestication, and invasive species.

Some models of karyotypic evolution are based on fixation of chromosome rearrangements in parapatric population which then result in negative heterosis of progeny for example if the rearrangements are inversions in different chromosomes (Kirkpatrick and Barton, 2006). Many of the existing models for ROBs suffer from the same shortcoming mentioned for inversions both in explaining existing empirical data and in lacking molecular cytogenetic explanations (Faria and Navarro, 2010).

The developments of the fields of molecular biology in combination with cytogenetic studies allowed for a reexamination of the classical notions of mutations via step-wise events (classical Darwinian) with constraints and selection pressures acting to subdue potential dramatic evolutionary changes. One of the earliest of such data came from the drastic reorganization of the maize genome noted by Barbara McClintock (McClintock, 1993). Subsequent reevaluation of chromosome changes validated the importance of structural chromosome issues in chromosomal orthoselection and punctuated evolution (von Sternberg, 1996; Qumsiyeh, 1994, 1995, 1999; King, 1982; Parsons, 1987; Bickham and Baker, 1979).

For intraspecific variation, the model of ROBs impact on variation and adaptability appears to fit. This would explain the relatively recent "Robertsonian fans" in animals like *Mus*, *Blarina* and *Spalax* in unstable environments such as areas of volcanic activities (Capanna and Castiglia, 2004; Qumsiyeh, 1999; Qumsiyeh et al., 1997). After all, what other explanation for the intraspecific variation by Robertsonian translocation in such habitats exist when populations of the same species occur elsewhere in the range without chromosome variation?

One of the questions mechanistically that had to be dealt with for fixation of chromosomal rearrangements via ROBs is the effect in producing unbalanced offspring (negative heterosis). The effects are not uniform for different species. Non-disjunction rates in simple ROB heterozygotes of *Sorex araneus* were 1.2 to 7.4% depending on ROB involved (Fedyk and Chętnicki, 2007) while in house mice germ cell death can be produced from single ROBs from 19.5% to 30.2% due to unbalanced progeny (Sans-Fuentes et al., 2010). Humans fall in between but in all cases there seems to be deviation from expected frequencies

based on random segregation with a preponderance of balanced progeny for ROBs (e.g. Honda et al., 2000; Ogur et al., 2006; De Villena and Sapienza, 2001a).

What cytogenetic and molecular mechanisms explain selection forces leading to both increase or decrease in chromosome number in different lineages and selection for non-random segregation in heterozygous ROB carriers? One hint comes from comparing human and mouse genomes. Humans have a drive towards metacentric chromosomes (fusions) and mice towards acrocentrics (fissions). It turns out that heterozygous carriers of Robertsonian translocation differ in human vs mouse female meiosis in that the eggs preferentially get the acrocentric chromosomes in mice while they preferentially get the metacentric chromosome in humans (De Villena and Sapienza, 2001b). The latter paper cited references for human and mouse segregation distortion (noted in females but not males) and suggested there is (yet to be discovered) mechanism that involves meiotic spindle preference for acrocentrics (in mouse) or metacentrics (in human) meiosis. However it turns out that female meiotic drive has been well studied in numerous papers dealing with maize and is due to proteins associated with centromeres (Malik and Henikoff, 2002; Chmátal et al., 2014 and references therein). Further, there are mechanisms that can explain the non-random segregation in carriers of ROBs (avoiding negative heterosis) such as asymmetry in meiosis and functional heterozygosity at a locus that mediates spindle attachment (De Villena and Sapienza, 2001b).

Other taxa cited above as having decrease and increase in chromosome number might also be shown with additional studies to have a non-random distribution of chromosomes in heterozygous carriers between the egg and the polar body in female meiosis (see Chmátal et al., 2014). For example, female *Blarina carolinensis* carriers of Robertsonian translocations would be predicted to preferentially produce eggs carrying the acrocentric chromosomes. One caution to make is that independent acquisition of fissions might be missed in some lineages much more than independent acquisition of fusions (Qumsiyeh, 1989) and this could lead to bias in favor of documenting fusions via retrospectively examining current living individuals.

Recent molecular tools such as chromosome painting have allowed for better reconstruction of ancestral mammalian karyotypes confirming many elements of the adaptive nature of the changes during evolution (Ferguson-Smith and Trifonov, 2007).

It does not escape our attention that the variation in chromosome number seen in population of the same species and potentially partly explainable by recombination and variation would be a prelude for understanding variation between species (i.e. chromosome evolution and speciation). The difference between the karyotype of the Indian and Chinese muntjacs are very good examples of two closely related species with different specializations/niches that is associated with different chromosome numbers (2n=6/7 and 2n=46) (Qumsiyeh, 1994). Another example of this might be the variation seen in the genus *Acomys* whereby there are two groups of extant species with high and low chromosome numbers (Denys et al., 1994). Yet a third example is the genus *Gerbillurus* from South Africa (Qumsiyeh et al., 1991). In the latter cases, there are indeed habitat differences. For example, the *Acomys* species with high diploid numbers are the diurnal ones while the lower diploid numbers are found to be nocturnal (hence more protected from predation) (Qumsiyeh, 1996).

Nothing in what was said implies that chromosome number variation is the sole explanation or even the main one in population variation but that it is a contributing factor and may help shed some light on some biological phenomena. Having said that, this field can use many more studies especially focusing on meiotic drive, asymmetrical spindles, and segregation controlling genes. Many of the examples cited above for intraspecific variation could act as tests of the model by examining meiotic behavior and level of genic variation in populations with different karyotypes as was partially done for *Mus* and *Spalax*. It is now also possible to test our hypothesis using chromosome painting FISH techniques (Murphy et al., 2005; Graphodatsky et al., 2011). Another area of exciting research that could add data to support (or reject) the hypothesis relating to adaptive role of chromosome rearrangements in evolution is to apply molecular studies of recombination such as those now available in yeast and human (e.g. see Baudat et al., 2013; Odenthal-Hesse, 2014) to interspecific and intraspecific variations noted in the model organisms above. Yet another idea is to collect data on recombination in domestic animals that are self-domesticated (e.g. cat with 2n=38) and intentionally domesticated (dog with 2n=78) (see discussion of mode of domestication in Driscoll et al., 2009).

References

Acloque H., Bonnet-Garnier A., Mompart F., Pinton A., Yerle-Bouissou M., 2013. Sperm nuclear architecture is locally modified in presence of a robertsonian translocation t (13; 17). PloS ONE 8(10): e78005.

Auton A., Fledel-Alon A., Pfeifer S., Venn O., Ségurel L., Street T., Leffler E.M., Bowden R., Aneas I., Broxholme J., Humburg P., Iqbal Z., Lunter G., Maller J., Hernandez R.D., Melton C., Venkat A., Nobrega M.A., Bontrop R., Myers S., Donnelly P., Przeworski M., Mcvean G., 2012. A fine-scale chimpanzee genetic map from population sequencing. Science. 336: 193–198.

Azevedo M., Oliveira C., Pardo B., Martinez P., Foresti F., 2007. Cytogenetic characterization of six species of flatfishes with comments to karyotype differentiation patterns in pleuronectiformes (teleostei). J. Fish. Biol. 70: 1–15.

Baudat F., Imai Y., De Massy B., 2013. Meiotic recombination in mammals: localization and regulation. Nature Rev. Genet. 14: 794–806.

Bengtsson B.O., 1980. Rates of karyotype evolution in placental mammals. Hereditas 92: 37–47.

Bickham J.W., Baker R.J., 1979. Canalization model of chromosomal evolution. Bull. Carnegie Mus. Nat. Hist. 13: 84.

Borodin P.M., Karamysheva T.V., Belonogova N.M., Torgasheva A.A., Rubtsov N.B., Searle J.B., 2008. Recombination map of the common shrew, *Sorex araneus* (Eulipotyphla, Mammalia). Genetics. 178: 621–632.

Bourque G., Pevzner A., Tesler G., 2004. Reconstructing the genomic architecture of ancestral mammals: lessons from human, mouse, and rat genomes. Genome Res. 14: 507–516.

Britton-Davidian J., Nadeau J.H., Croset H., Thaler L., 1989. Genic differentiation and origin of Robertsonian populations of the house mouse (*Mus musculus domesticus* Rutty). Genetical Res. 53: 29–44.

Burt A., 2000. Perspective: sex, recombination, and the efficacy of selectionwas Weismann right? Evolution 54: 337–351.

Burt A., Bell G., 1987. Mammalian chiasma frequencies as a test of two theories of recombination. Nature 326: 803–805.

Bush G.L., Case S., Wilson A., Patton J., 1977. Rapid speciation and chromosomal evolution in mammals. Proc. Natl. Acad. Sci. 74: 3942–3946.

Campbell C.L., Bhérer C., Morrow B.E., Boyko A.R., Auton A., 2016. A pedigree-based map of recombination in the domestic dog genome. G3: Genes, Genomes, Genetics 6(11): 3517–3524.

Capanna E., Castiglia R., 2004. Chromosomes and speciation in *Mus musculus domesticus*. Cytogenet. Genome Res. 105: 375–384.

Castiglia R., 2014. Sympatric sister species in rodents are more chromosomally differentiated than allopatric ones: implications for the role of chromosomal rearrangements in speciation. Mammal Rev. 44: 1–4.

Castiglia R., Caporioni M., 2005. Altitudinal distribution and outdoor occurrence in chromosomal races of the house mouse (*Mus musculus domesticus*) in central Italy. Folia Zoologica Praha 54(3): 225–229.

Chatti N., Ganem G., Benzekri K., Catalan J., Britton-Davidian J., Said K., 1999. Microgeographical distribution of two chromosomal races of house mice in Tunisia: pattern and origin of habitat partitioning. Proc. Royal Soc. London. Series B: Biol. Sci. 266(1428): 1561–1569.

Chmátal L., Gabriel S.I., Mitsainas G.P., Martínez-Vargas J., Ventura J., Searle J. B., Schultz R.M., Lampson M.A., 2014. Centromere strength provides the cell biological basis for meiotic drive and karyotype evolution in mice. Curr. Biol. 24(19): 2295–2300.

Coop G., Przeworski M., 2007. An evolutionary view of human recombination. Nat. Rev. Genet. 8: 23–34.

Danzmann R., Cairney M., Davidson W., Ferguson M., Gharbi K., Guyomard R., Holm L.-E., Leder E., Okamoto N., Ozaki A., 2005. A comparative analysis of the rainbow trout genome with 2 other species of fish (arctic charr and atlantic salmon) within the tetraploid derivative salmonidae family (subfamily: Salmoninae). Genome 48: 1037–1051.

Datta M., 1972. Study of Meiosis in the Chimpanzee, *Pan troglodytes troglodytes* (Blumenbach 1779). Cytologia 37: 207–216.

De Villena F.P.-M., Sapienza C., 2001a. Female meiosis drives karyotypic evolution in mammals. Genetics 159: 1179–1189.

De Villena F.P.-M., Sapienza C., 2001b. Nonrandom segregation during meiosis: the unfairness of females. Mammalian Genome 12: 331–339

Denys C., Gautun J.-C., Tranier M., Volobouev V., 1994. Evolution of the genus *Acomys* (Rodentia, Muridae) from dental and chromosomal patterns. Isr. J. Zool. 40: 215–246.

Devos K.M., Gale M.D., 2000. Genome relationships: the grass model in current research. Plant Cell 12: 637–646.

Dobigny G., Britton-Davidian J., Robinson T.J., 2017. Chromosomal polymorphism in mammals: an evolutionary perspective. Biol. Rev. 92(1): 1–21.

Driscoll C.A., Macdonald D.W., O'Brien S., 2009. From wild animals to domestic pets, an evolutionary view of domestication. Proc. Natl. Acad. Sci. 106(S1): 9971–9978.

Dumas D., Britton-Davidian J., 2002. Chromosomal rearrangements and evolution of recombination: comparison of chiasma distribution patterns in standard and robertsonian populations of the house mouse. Genetics 162: 1355–1366.

Excoffier L., Foll M., Petit R.J., 2009. Genetic consequences of range expansions. Ann. Rev. Ecol. Evol. System. 40: 481–501.

Falek A., Chiarelli B., 1968. Meiotic chromosomes of man. Am. J. Phys. Anthropol. 28: 351–354.

Faria R., Navarro A., 2010. Chromosomal speciation revisited: rearranging theory with pieces of evidence. Trends Ecol. Evol. 25: 660–669.

Fedyk S., Chętnicki W., 2007. Preferential segregation of metacentric chromosomes in simple Robertsonian heterozygotes of *Sorex araneus*. Heredity 99: 545–552.
Ferguson-Smith M.A., Trifonov V., 2007. Mammalian karyotype evolution. Nat. Rev. Genet. 8: 950–962.
Fontana F., Rubini M., 1990. Chromosomal evolution in cervidae. Biosystems 24: 157–174.
Gallardo M.H., Bickham J.W., Honeycutt R.L., Ojeda R.A., Köhler N., 1999. Discovery of tetraploidy in a mammal. Nature 401: 341.
Garagna S., Zuccotti M., Thornhill A., Fernandez-Donoso R., Berrios S., Capanna E., Redi C., 2001. Alteration of nuclear architecture in male germ cells of chromosomally derived subfertile mice. J. Cell Sci. 114: 4429–4434.
Gazave E., Catalan J., Ramalhinho M.D.G., Mathias M.D.L., Nunes A.C., Dumas D., Britton-Davidian J., Auffray J.-C., 2003. The non-random occurrence of Robertsonian fusion in the house mouse. Genetics Res. 81: 33–42.
Giménez M.D., Förster D.W., Jones E.P., Jóhannesdóttir F., Gabriel S.I., Panithanarak T., Scascitelli M., Merico V., Garagna S., Searle J.B., Hauffe H.C., 2016. A Half-Century of Studies on a Chromosomal Hybrid Zone of the House Mouse. J. Heredity 108(1): 25–35.
Graphodatsky E.S., Trifonov V.A., Stanyon R., 2011. The genome diversity and karyotype evolution of mammals. Mol. Cytogenet. 4: 22.
Hima K., Thiam M., Catalan J., Gauthier P., Duplantier J.M., Piry S., Sembène M., Britton-Davidian J., Granjon L., Dobigny G., 2011. Extensive robertsonian polymorphism in the african rodent *Gerbillus nigeriae*: geographic aspects and meiotic data. J. Zool. 284(4): 276–285.
Hoffmann A.A., Rieseberg L.H., 2008. Revisiting the impact of inversions in evolution: from population genetic markers to drivers of adaptive shifts and speciation?. Ann. Rev. Ecol. Evol. Syst. 39: 21–42.
Honda H., Miharu N., Samura O., He H., Ohama K., 2000. Meiotic segregation analysis of a 14; 21 Robertsonian translocation carrier by fluorescence in situ hybridization. Hum. Genet. 106: 188–193.
Imai H.T., Satta Y., Takahata N., 2001. Integrative study on chromosome evolution of mammals, ants and wasps based on the minimum interaction theory. J. Theor. Biol. 210: 475–497.
Imai H.T., Satta Y., Wada M., Takahata N., 2002. Estimation of the highest chromosome number of eukaryotes based on the minimum interaction theory. J. Theor. Biol. 217: 61–74.
Kaback D.B., 1996. Chromosome–size dependent control of meiotic recombination in humans. Nat. Genet. 13: 20–21.
Kaback D.B., Guacci V., 1992. Chromosome size-dependent control of meiotic recombination. Science 256: 228.
King M., 1982. A case for simultaneous multiple chromosome rearrangements. Genetica 59: 53–60.
Kirkpatrick M., Barton N., 2006. Chromosome inversions, local adaptation and speciation. Genetics 173: 419–434.
Kong A., Gudbjartsson D.F., Sainz J., Jonsdottir G.M., Gudjonsson S., Richardsson B., Sigurdardottir S., Barnard J., Hallbeck B., Masson G., 2002. A high-resolution recombination map of the human genome. Nat. Genet. 31: 241–247.
Malik H.S., Henikoff S., 2002. Conflict begets complexity: the evolution of centromeres. Curr. Opin. Genet. Dev. 12: 711–718.
Martinez P.A., Jacobina U.P., Fernandes R.V., Brito C., Penone C., Amado T.F., Fonseca C.R., Bidau C.J., 2016. A comparative study on karyotypic diversification rate in mammals. Heredity 118(4): 366–373.
McClintock B., 1993. The Significance of Responses of the Genome to Challenge. World Scientific Pub. Co., Singapore.
Menotti-Raymond M., David V.A., Schäffer A.A., Tomlin J.F., Eizirik E., Phillip C., Wells D., Pontius J.U., Hannah S.S., O'Brien S.J., 2009. An autosomal genetic linkage map of the domestic cat, *Felis silvestris catus*. Genomics 93(4): 305–313.
Munch K., Mailund T., Dutheil J.Y., Schierup M.H., 2014. A fine-scale recombination map of the human-chimpanzee ancestor reveals faster change in humans than in chimpanzees and a strong impact of gcbiased gene conversion. Genome Res. 24: 467–474.
Murphy W.J., Larkin D.M., Everts-Van Der Wind A., Bourque G., Tesler G., Auvil L., Beever J.E., Chowdhary B.P., Galibert F., Gatzke L., Hitte C., 2005. Dynamics of mammalian chromosome evolution inferred from multispecies comparative maps. Science 309(5734): 613–617.
Nachman M., Myers P., 1989. Exceptional chromosomal mutations in a rodent population are not strongly underdominant. Proc. Natl. Acad. Sci. 86: 6666–6670.
Nam K., Ellegren H., 2012. Recombination drives vertebrate genome contraction. PLoS Genetics 8: e1002680.
Nash W., Wienberg G., Ferguson-Smith J., Menninger J., O'Brien S.J., 1999. Comparative genomics: tracking chromosome evolution in the family ursidae using reciprocal chromosome painting. Cytogenet. Genome Res. 83: 182–192.
Navarro A., Barton N.H., 2003. Chromosomal speciation and molecular divergence-accelerated evolution in rearranged chromosomes. Science 300: 321–324.
Nevo E., 1991. Evolutionary theory and processes of active speciation and adaptive radiation in subterranean mole rats, *Spalax ehrenbergi* superspecies, in Israel. Evol. Biol. 25: 1–125.
Nevo E., 1998. Molecular evolution and ecological stress at global, regional and local scales: The Israeli perspective. J. Exp. Zool. 282: 95–119.
Nevo E., Filippucci M., Redi G., Korol C., Beiles A., 1994. Chromosomal speciation and adaptive radiation of mole rats in Asia Minor correlated with increased ecological stress. Proc. Natl. Acad. Sci. 91: 8160–8164.
Nirchio M., Oliveira C., 2006. Citogenética de Peces. Universidad de Oriente, Cumaná, Venezuela.
O'Brien S.J., Menninger J.C., Nash W.G., 2006. Atlas of Mammalian Chromosomes. John Wiley & Sons.
Odenthal-Hesse L., Berg I.L., Veselis A., Jeffreys A.J., May C.A., 2014. Transmission distortion affecting human non-crossover but not crossover recombination: a hidden source of meiotic drive. PLoS Genetics 10(2): e1004106.
Ogur G., Van Assche E., Vegetti W., Verheyen G., Tournaye H., Bonduelle M., Van Steirteghem A., Liebaers I., 2006. Chromosomal segregation in spermatozoa of 14 robertsonian translocation carriers. Mol. Hum. Reprod. 12: 209–215.
Olmo E., Capriglione T., Odierna G., 2002. Different genomic evolutionary rates in the various reptile lineages. Gene 295: 317–321.
Paigen K., Petkov P.M., 2010. Mammalian recombination hot spots: properties, control and evolution. Nat. Rev. Genet. 11(3): 221–233.
Paigen K., Petkov P.M., 2018. PRDM9 and its role in genetic recombination. Trends in Genetics 34(4): 291–300.
Parsons P., 1987. Evolutionary rates under environmental stress. In: Hecht M.K., Wallace B., Prance G.T. (Eds.) Evolutionary biology. Springer, Boston, MA. 311–347.
Peppers J.A., 1998. A meiotic analysis of the genus *Reithrodontomys* in the United States. Doctoral Thesis. Texas Tech University, Lubbock. TX.
Perry J., Slater H.R., Choo K.A., 2004. Centric fission-simple and complex mechanisms. Chrom. Res. 12: 627–640.
Phillips R., Rab P., 2001. Chromosome evolution in the Salmonidae (Pisces): an update. Biol. Rev. Cambridge Phil. Soc. 76: 1–25.
Prentis P.J., Wilson J.R., Dormontt E.E., Richardson D.M., Lowe A.J., 2008. Adaptive evolution in invasive species. Trends Plant Sci. 13: 288–294.
Proença S., Serrano A., Collares-Pereira M., 2002. An unusual karyotype with low chromosome number in Megacephalini, a basal group of tiger beetles (Coleoptera, Cicindelidae): cytogenetic characterisation by C-banding and location of rDNA genes. Hereditas 137: 202–207.
Qumsiyeh M.B., 1989. Chromosomal fissions and phylogenetic hypotheses: cytogenetic and allozymic variation among species of *Meriones* (Rodentia: Gerbillidae). Occ. Pap. Mus. Texas Tech Univ. 132: 1–16.
Qumsiyeh M.B., 1994. Evolution of number and morphology of mammalian chromosomes. J. Heredity 85: 455–465.
Qumsiyeh M.B., 1995. Impact of rearrangements on function and position of chromosomes in the interphase nucleus and on human genetic disorders. Chromosome Res. 3: 455–465.
Qumsiyeh M.B., 1996. Mammals of the Holy Land. Texas Tech Press, Lubbock, Texas.
Qumsiyeh M.B., Hamilton M., Dempster E.R., Baker R.J., 1991. Cytogenetics and systematics of the rodent genus *Gerbillurus*. J. Mammal. 72: 89–96.
Qumsiyeh M.B., Coate J.L., Peppers J.A., Kennedy P.K., Kennedy M.L., 1997. Robertsonian chromosomal rearrangements in the short-tailed shrew, *Blarina carolinensis*, in western Tennessee. Cytogenet. Cell Genet. 76: 153–158.
Qumsiyeh M., Barker S., Dover S., Kennedy P., Kennedy M., 1999. A potential model for early stages of chromosomal evolution via concentric Robertsonian fans: a large area of polymorphism in southern short-tailed shrews (*Blarina carolinensis*). Cytogenet Genome Research 87: 27–31.
Qumsiyeh M.B., 1999. Structure and function of the nucleus: anatomy and physiology of chromatin. Cell Mol. Life Sci. 55: 1129–1140.
Qumsiyeh M.B., Amr Z.S., Srour K.T.A., Al-Fawaghra N., 2014. Karyotype for *Nebo hierichonticus* (Simon 1872) from the Palestinian Territories (Scorpiones: Scorpionidae). Cytologia 79: 277–280.
Rogers J., Gibbs R.A., 2014. Comparative primate genomics: emerging patterns of genome content and dynamics. Nat. Rev. Genet. 15(5): 347–359.
Romanenko S.A., Perelman P.L., Trifonov V.A., Graphodatsky A.S., 2012. Chromosomal evolution in Rodentia. Heredity 108(1): 4–16.
Ropiquet A., Gerbault-Seureau M., Deuve J.L., Gilbert C., Pagacova E., Chai N., Rubes J., Hassanin A., 2008. Chromosome evolution in the subtribe Bovina (Mammalia, Bovidae): the karyotype of the Cambodian banteng (*Bos javanicus birmanicus*) suggests that Robertsonian translocations are related to interspecific hybridization. Chromosome Res. 16: 1107–1118.
Ruiz-Herrera A., Farré M., Robinson T., 2012. Molecular cytogenetic and genomic insights into chromosomal evolution. Heredity 108: 28–36.
Sans-Fuentes M.A., García-Valero J., Ventura J., López-Fuster M.J., 2010. Spermatogenesis in house mouse in a Robertsonian polymorphism zone. Reproduction 140: 569–581.
Shao R., Kirkness E.F., Barker S.C., 2009. The single mitochondrial chromosome typical of animals has evolved into 18 minichromosomes in the human body louse, *Pediculus humanus*. Genome Res. 19: 904–912.
Slate J., Van Stijn T.C., Anderson R.M., Mcewan K.M., Maqbool N.J., Mathias H.C., Bixley M.J., Stevens D.R., Molenaar A.J., Beever J.E. A deer (subfamily Cervinae) genetic linkage map and the evolution of ruminant genomes. Genetics 160: 1587–1597.
Stapley J., Feulner P.G.D., Johnston S.E., Santure A.W., Smadja C.M., 2017. Variation in recombination frequency and distribution across 30 eukaryotes: patterns and processes. Philos Trans R Soc Lond B Biol Sci. 372: 20160455.
von Sternberg R., 1996. The role of constrained self-organization in genome structural evolution. Acta Biotheoretica 44: 95–118.
Stevison L., Woerner A., Kidd J., Kelley J., Veeramah K., Mcmanus K., Bustamante C., Hammer M., Wall J., 2016. The Time Scale of Recombination Rate Evolution in Great Apes. Mol. Biol. Evol. 33: 928–945.
Symonova 2013 is missing!
Szamalek J., Goidts V., Searle J., Cooper D., Hameister H., Kehrer-Sawatzki H., 2006. The chimpanzee-specific pericentric inversions that distinguish humans and chimpanzees have identical breakpoints in *Pan troglodytes* and *Pan paniscus*. Genomics 87: 39–45.
Todd N.B., 1975. Chromosomal mechanisms in the evolution of artiodactyls. Paleobiology 1(2): 175–188.
Trickett A.J., Butlin R.K., 1994. Recombination suppressors and the evolution of new species. Heredity 73: 339–345.
Úbeda F., Russell T.W., Jansen V.A., 2019. PRDM9 and the evolution of recombination hotspots. Theoretical population biology 126: 19–32.
Ventura M., Catacchio C.R., Sajjadian S., Vives L., Sudmant P.H., Marques-Bonet T., Graves T.A., Wilson R.K., Eichler E.E., 2012. The evolution of african great ape subtelomeric heterochromatin and the fusion of human chromosome 2. Genome Res 22: 1036–1049.
White M.J.D., 1978. Chain processes in chromosomal speciation. Systematic Biol. 27: 285–298.
Wilson A.C., Bush G.L., Case S.M., King M.C., 1975. Social structuring of mammalian populations and rate of chromosomal evolution. Proc. Natl. Acad. Sci. USA. 72: 5061–5065.
Wurster D.H., Benirschke K., 1970. Indian muntjac, *Muntiacus muntjak*: a deer with a low diploid chromosome number. Science 168:1364-1366.

Associate Editor: P. Colangelo