

CHROMOSOME STUDIES

III. INEQUALITIES AND DEFICIENCIES IN HOMOLOGOUS CHROMOSOMES: THEIR BEARING UPON SYNAPSIS AND THE LOSS OF UNIT CHARACTERS

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FOURTEEN FIGURES (THREE PLATES)

In November, 1910, while preparing a larger paper upon the problem of synapsis in the germ cells of certain grasshoppers belonging to the subfamily Tettigidae (Robertson '15), I found some individuals in which there occurred a pair of unequal homologous chromosomes. I made a series of drawings of these pairs at the time, intending to incorporate them later into my larger paper. That paper is not yet completed, owing to the duties of teaching during the last year at Kansas University. The importance of these inequalities in members of a chromosome pair, however, seems to warrant publishing the results in brief form. In this account I wish to point especially to a possible relation between these unequal chromosomes and the behavior of certain unit characters in breeding; also to the bearing which the permanency of such chromosomes has upon the problem of parasynapsis.

In the Tettigidae, a subfamily of the short-horn grasshopper family Acrididae, I have found, for all the species of at least four different genera which I have examined, the number of chromosomes to be uniformly 14 in the female (figs. 1, 11) and 13 in the male (figs. 4, 9). I have also found all to have, with limited variation, among the autosome (ordinary chromosome) series, two extremely long pairs of chromosomes, the 7's and 6's (figs. 1, 3, 4), two intermediate pairs, the 4's and 3's (figs. 1-3) or 5's and 4's (figs. 9, 10), and two very small pairs, the 2's and 1's (figs. 1-3, Tettigidea) or the 3's and 2's (figs. 9-10, Acridium).

The sex chromosome—single in the male and paired in the female—may rank in size as No. 5 (*Tettigidea*), No. 3 (*Paratettix*) or No. 1 (*Acridium*), depending upon the genus, but this variation between different genera does not seem to be accompanied by any very considerable difference in the relative sizes of the ordinary chromosomes.

Of the constancy of these size relations, I am very certain, having examined a large number of individuals in each species. Being certain of the size relations, I was very ready to recognize any abnormal variations in this respect when they appeared. I found such variations in two genera: two cases in *Tettigidea parvipennis*, only one of which is given (figs. 4–8), and two in *Acridium granulatus* (figs. 11–13).

1. A DEFICIENT NO. 4 CHROMOSOME IN *TETTIGIDEA* *PARVIPENNIS*

To understand the abnormal chromosomes we must first examine the normal ones. Figures 1 to 3 are of cells from normal individuals. Figure 1 is from the wall of an egg tube. It shows 14 chromosomes, the number characteristic of the female. The chromosomes are numbered and paired according to size. Two of the chromosomes (nos. 1 and 2) have been drawn at one side for convenience. This figure is also typical for male 2x cells, with this exception, that the sex-chromosome, No. 5, is always unpaired in the male. Figures 2 and 3 are lateral views of the first maturation division in the male germ cells. These show the members of pairs about to separate from each other. The members are still in contact at the distal ends in most cases. The No. 7 pair in figure 2 has formed a cross and is still paired through a greater extent than in the other pairs. The sex chromosome ranks fifth in size in *Tettigidea* and may be seen passing undivided to one pole in these cells. Among the autosomes (ordinary chromosomes) of figures 2 and 3 there can be seen a very slight difference in size between the 1's and 2's, a considerable difference between the 3's and 4's, and a much greater difference between the 6's and 7's. We are concerned at present with the relative sizes of the 3's and 4's only.

Since the diameters of the chromosomes are uniform throughout the series in any given cell, the relative sizes can be learned best by measurement of lengths. My measurements have been made from drawings outlined by means of a camera lucida, the image being projected at the level of the base of the microscope. A 2 mm. Leitzoil-immersion objective and an $18\times$ Zeiss compensating ocular were used for outlining. I feel reasonably certain that my drawings show approximately the correct relative sizes of the chromosomes in each cell.

The average length (doubled) of the No. 3 chromosomes in the figures ($\times 2600$) of six cells from two normal individuals of *Tettigidea parvipennis* is 15 mm. The average length of the No. 4 chromosomes under the same conditions in the same six cells is 17.05 mm. The ratio of the No. 3 to the No. 4 chromosomes is therefore as 1 to 1.14. This may be taken as approximately the normal size relation of the No. 3 and No. 4 chromosomes; that is, No. 4 is about one-seventh longer than No. 3.

In figures 4 to 8 are cells from a male individual of the same species, in which one member of the No. 4 pair of chromosomes (4-) is abnormally small. The average length of the No. 3 tetrad is here 13.1 mm. This is shorter than in the preceding case of six cells, but in this instance all chromosomes are affected similarly, the reduction in size being due probably to the cells having been in slightly different stages when killed. This does not affect the relative lengths of the chromosomes, however. The average (doubled) length of the larger (no. 4) diad (figs. 5-8) in the same cells is 15.5 mm. The ratio therefore of the No. 3 tetrad in these cells to the larger diad of the abnormal No. 4 tetrad, or to a No. 4 tetrad made up of two such diads, would be as 1 to 1.18; i.e., the ratio of the No. 3 chromosomes to the normal member of the No. 4 pair here is as 1 to 1.18. This is not far from the normal ratio, 1.14, and the difference is quite within the range of the probable error due to inaccurate measurement, etc. But the average (doubled) length of the smaller diad of the abnormal No. 4 tetrad is 12.7 mm., instead of 15.5 mm. The ratio therefore of the No. 3 tetrad to a tetrad made up of two such No. 4 diads would be as 1 to 0.97; i.e., the ratio of the

No. 3 chromosomes to the smaller No. 4 chromosome is as 1 to 0.97. This shows the smaller No. 4 diad to be even smaller than the No. 3 chromosomes. I believe that the larger diad of this abnormal No. 4 tetrad is the normal No. 4 chromosome, since its ratio, 1.18, is so near to the normal ratio, 1.14, and that the smaller diad is abnormal and has lost a part of its distal end; (my reason for thinking the distal end deficient will appear later). Its ratio, 0.97, instead of 1.14, gives it a shortage of 0.17, or nearly one-sixth of 1.14. It seems therefore to have lost one-sixth of its normal length.

The size of this deficient No. 4 chromosome is constant for this individual. All division figures which occurred in lateral view showed the members of this unequal pair in practically the same relative sizes. Not only the germ cells but also somatic cells (fat body) exhibited the same proportions. The fact that the size ratio is constant in all germ cells and even in body cells seems to point to its germinal origin; i.e., it must have been present in the fertilized egg from which this animal developed.

There is no constant relation between the members of this unequal pair of chromosomes and the sex chromosome, as the cells of the maturation division show (figs. 5, 6, 8), for the sex chromosome passes as often to the pole which receives the small No. 4 chromosome as to the pole which receives the large member.

Another very noticeable evidence of the abnormality and defectiveness of this No. 4 chromosome is shown in its manner of contact with its larger normal mate in the first maturation spindle (figs. 5, 6, 7 c). In these three figures its attachment is not strictly terminal, as it should be. This, it seems to me, is evidence of a deficiency at the distal end of this chromosome. This will be better understood if I describe briefly the method of chromosome pairing in the Tettigidae (Robertson '15). During the period of synapsis, which occurs in the early part of the growth period of the first spermatocyte, homologous chromosomes pair side to side (parasynapsis), as shown in figures A₁, A₂ (pl. 3). After this period of synapsis, during the growth period which follows, a separation of the pairing chromosomes takes

place. This separation begins at the proximal ends of the pair and gradually moves along toward the distal ends (figs. A_3 to A_5). By the proximal end I mean that from which the spindle fiber springs and that which travels in advance toward the pole. The opposite, blunt end is the distal end. The proximal ends of the pair diverge, each through an angle of 90° ; i.e., until they are 180° apart (figs. A_3 to A_5). The proximal ends now point in opposite directions, and the pair, thus attached at their distal ends, form a rod. In this condition the pair (tetrad) enters the first maturation spindle (fig. A_5).

The defective tetrad, like the others, has gone through this process. But a portion of the distal end of one of the chromosomes being gone, the chromosome cannot conjugate properly with its mate and so has a tendency to slip to one side when the tetrad reaches this stage, as figures 5 to 7, and E_2 to E_5 show. The normal conjugant (diad) finds at its distal end no corresponding portion in the defective mate with which to pair. The chromosomes were probably paired normally at the proximal ends and in all parts which are present, leaving the distal part of the larger normal member extending beyond the shortened distal end of the defective mate (fig. E_2). In the process of separation which follows conjugation the defective chromosome (diad) had its shortened distal end rotating on the side of the distal end of the longer, normal mate. When the diads had rotated apart 180° this shortened end was as a result out of line with the end of its longer mate (figs. E_2 to E_5). This is the condition in which the defective chromosome of figures 5, 6 and 7 c is seen.

2. AN UNEQUAL PAIR OF ANOTHER TYPE OCCURRING IN ACRIDIUM GRANULATUS

While working out the chromosomes in dividing ovarian follicle cells of the female individuals of *Acridium granulatus*, I found one animal which showed among its 14 chromosomes five long members (fig. 11) instead of four (the two 6's and the two 7's). This puzzled me in my pairing of the chromosomes. The material was laid aside for the time. A few weeks later I found a male individual which showed in its first maturation divisions

the same five long chromosomes. My problem was solved at once when I saw that this extra long chromosome paired in maturation with one of the ordinary chromosomes of the complex, the No. 1 (figs. 12-13).

This unequal pair appears to be of a type different from the deficient tetrad just described, and so must be treated separately. It is of primary importance in its bearing on questions of synapsis and of maturation division. Only two animals having it were found, a male and a female. These, and most others of this species worked upon, were collected from a spot 75 feet square near Waverly, Massachusetts, and are probably related individuals.

Figures 9 and 10 show cells from normal individuals exhibiting the typical condition of the chromosomes in *Acridium granulatus*. There are two long pairs (6's and 7's), two intermediate pairs (5's and 4's), two short pairs (1's and 3's), and the sex chromosome, which in size ranks No. 2 in this genus. A small, faintly staining, fragmentary body is shown (dotted) in figure 9. It is not present in all cells, and is probably a nucleolar structure of some sort. The normal chromosomes paired for the first maturation division may be seen in figure 10. Their size relations are clearly shown there: two large pairs (6, 7), two intermediate pairs (4, 5) and two small pairs (1, 3), as well as the small 'accessory' chromosome (2).

Figures 11 to 13 are of the abnormal male and female. In figure 11 (female) the five, instead of four, long chromosomes are clearly shown. The fifth long chromosome (1) appears odd because it has no equal chromosome with which to pair. The first maturation division in the male (figs. 12-13), however, shows it pairing with the small (no. 1) chromosome. In the male cells, moreover, it shows a constriction at a region about as far from its distal end as the length of the small (no. 1) chromosome with which it is paired.

The other chromosomes are quite normal in these animals (figs. 11-13). In the female (fig. 11) there are the two long pairs (7's and 6's), the two intermediate pairs (5's and 4's), the short pair (3's), the 2's (which are the sex chromosomes and are

paired in the female), one normal No. 1 chromosome, and the abnormal (long) No. 1. In the male cells (figs. 12-13) there are two large pairs (7 and 6), two intermediate pairs (5 and 4), one small pair (3), and the sex chromosome (2) unpaired; the other small tetrad is represented by one small No. 1 chromosome paired with the abnormal long chromosome. It is evident from these two figures that this long structure is connected with one of the small (No. 1) chromosomes.

The long abnormal diad bears no constant relation to the sex chromosome in its distribution to the second spermatocyte cells, as figures 12 and 13 show. A large number of dividing cells examined showed that in the reduction division it passed as often to the pole which did not receive the sex chromosome as to that which did. I feel certain, therefore, that this chromosome bears no relation to the sex-determining chromosome.

2a. Discussion

What is the origin of the long chromosome?

The partially constricted off distal portion of this long body (figs. 12-13) is probably a No. 1 chromosome and in synapsis it was possibly paired side to side with the opposite normal No. 1, leaving the remainder of the long chromosome, the proximal end, to extend beyond it (figs. B₁ and B₂). In the Tettigidae, as I have said above, side-to-side pairing in synapsis occurs during the bouquet stage. Following this period the chromosomes of a pair separate from each other, beginning at the proximal ends. Remaining in contact at the distal ends, they continue to rotate apart until the pair appears upon the first maturation spindle as a long rod frequently constricted in the middle (figs. 10, 12, 13 and figs. A₁ to A₅). That is exactly what has occurred in the case of this abnormal tetrad, but to the end of one of the No. 1 members is attached this extra $1\frac{1}{2}$ portion, which increases the length of this member to two and one-half times its normal dimension (figs. B₁ to B₄). Unfortunately no satisfactory prophase stages are present to show the behavior of this element in synapsis, but, judging from the constriction

at one end and knowing the behavior of the unequal tetrads of other individuals (figs. 4-8), I believe that here we probably have the result of an unequal division of some preceding generation where a No. 1 tetrad failed to divide at the proper place, in the middle between the two members, but instead so divided as to give a $1\frac{1}{2}$ portion of a No. 1 pair to one pole and a half-portion to the other pole (C_1 and C_2). As a result of this, it might be imagined that there would be some generations of individuals with a sesqui-valent ($1\frac{1}{2}$ -valent) No. 1 chromosome present pairing with the normal No. 1 (figs. D_1 , D_2). The normal No. 1 might pair with the normal part, or with the fragment. In order to get a $2\frac{1}{2}$ -valent No. 1 chromosome the parts of which are largely oriented in one direction it will now be necessary to suppose that, at some reduction division of the germ cells in succeeding generations of animals, a normal No. 1 chromosome became attached during the synapsis period to this sesqui-valent No. 1 chromosome. It might do this by becoming fused with, or by failing to separate from, the fragmentary (cut off) end of the sesqui-valent member after some method as is indicated in figures D_1 to D_6 , giving as a result a $2\frac{1}{2}$ -valent chromosome (fig. D_6), such as we have in figures 10 to 13. All portions of this chromosome, except the sesqui-valent fragment in the middle, would then be oriented in one direction (see arrows in figs. C_1 , C_2 , and D_1 to D_6). My reason for thinking that at least the terminal parts, which make up the greater part of the long chromosome are oriented in the same direction, is based upon the fact that I always see the chromosome arranged on the spindle in perfectly normal position, pointing directly toward the pole to which it is about to go, and upon the fact that the mantle fiber of this chromosome always springs from the longer ($1\frac{1}{2}$) portion of the chromosome (figs. 12, 13, and B_1 to B_5). The chromosome always travels with the larger, sesqui-valent portion in advance (figs. 12, 13, B_4 and B_5).

In this animal evidently the normal No. 1 chromosome paired during synapsis with the distal No. 1 portion of the $2\frac{1}{2}$ -valent chromosome (figs. 12, 13, and B_1 to B_5). It should pair also

with the sesqui-valent portion, but I have seen no evidence that this occurs. Possibly the sesqui-valent portion has become so non-functional that it fails properly to attract the normal No. 1 chromosome in the synapsis period.

This explanation is the best I can propose, in the light I now have, to account for the origin of this long chromosome. The facts are that it is approximately two and one-half times the length of a normal No. 1 chromosome, all or most of its parts are evidently oriented in one direction, and a constriction near the distal end marks off a portion which evidently pairs in synapsis in the normal manner with the No. 1 chromosome and separates from this chromosome in the normal manner in the first maturation division (figs. 12, 13, and B_1 to B_5).

An essential fact is that we have here cases of an abnormally large chromosome, which is constant in size in individuals of both sexes. Its relative size is the same in all the dividing germ cells found in the male, and likewise in the somatic cells (follicle) of the female. I believe that it is a permanent structure, so far as these two individual animals are concerned.

A second important fact is that this abnormally large chromosome alternates with a normal chromosome and it may or may not be present in either sex. Theoretically, we may have, depending on the presence or absence of this chromosome, three sorts of male individuals and three sorts of female individuals; those containing two normal No. 1 chromosomes, those containing one normal and one abnormal No. 1 chromosome and those containing two abnormal No. 1's. I have found the first two cases in both sexes. The latter case has not yet been found in either sex. We have therefore a basis for a Mendelian ratio. The presence of one long and one short chromosome might be considered the cell condition of the heterozygote, or hybrid; the presence of two short chromosomes or of two long ones would give the homozygotes, recessive and dominant, if such relations may be imagined in so far as these chromosomes are concerned.

3. ON SYNAPSIS AND REDUCTION

In their behavior these unequal tetrads throw a good deal of light on the nature of the synapsis of chromosomes and their separation from each other in the reduction division. In regard to the beginning of this process, the individuals furnishing these abnormal chromosomes do not have much to show, as I have said above. This beginning, however, I determined from normal spermatogenesis, and have worked it out in my second paper.

I have found the pairing process in the Tettigidae to start as a parasynapsis and to end in a telosynapsis (end-to-end pairing). In the synizesis period following the last spermatogonial division, the members of each pair of chromosomes, six pairs in all, having assumed the thread condition, are seen to arrange themselves in side-by-side fashion. The number of threads is at first twelve, in addition to the accessory chromosome. The twelve threads are seen to become six double and finally six single threads. During this period the sex chromosome usually lies at one side of the nucleus. This condition of pairing continues into the later growth period, when the members of each pair move apart, the separation beginning first at the proximal ends (figs. A_1 to A_4), the chromosomes of a pair remaining attached to each other at the distal ends (figs. A_4 , A_5). Each rotates through 90° and the two then form a rod (fig. A_5), which may show, by a slight constriction, the point of contact of the two members. In this condition the paired chromosomes (tetrads) enter upon the first maturation division.

In the behavior of these unequal pairs I feel perfectly certain that the same thing must have taken place, for I see no reason to think that these abnormal pairs should behave differently from the normal chromosomes during this process. Their behavior on the maturation spindle and in the anaphase following is in every way similar to that of the normal chromosomes, and we have every reason to believe that a parasynapsis ending in a telosynapsis has taken place. The unequal pairs appear on the maturation spindle in the telosynapsis condition.

Attention, first of all, should be called to the permanency of size of these abnormal pairs, especially of the abnormal members

of each pair. In the first type of inequality which I described (figs. 4-8), all dividing cells examined showed the members of the No. 4 pair unequal and constantly of the two respective sizes. The normal No. 4 chromosome was always of the size normal for No. 4 chromosomes and its deficient (No. 4) mate was likewise of a constant size but uniformly one-sixth less than the normal No. 4 in all cells, both somatic and germinal. The constancy of this relative inequality was very clearly shown, especially in those figures of the first spermatocyte division which appeared in lateral view (figs. 5-8). A similar uniformity of sizes I found in the unequal pair of the second type (figs. 12, 13) in both male and female animals. Abundant evidence of the relative sizes was given, especially in the tetrads of the first spermatocyte divisions. Every cell showed the No. 1 portion of the abnormal tetrad to be of the normal No. 1 size (figs. 10-13) and the longer $2\frac{1}{2}$ -valent portion to be likewise of a constant size. It was not only of uniform size in all the cells of the male individual, both somatic and germ cells, but in another individual, a female, it was also found to be of the same length (fig. 11).

These facts indicate that in the latter case this abnormally long chromosome must have been handed on as such, not only through many generations of cell division, but also through many generations of individual animals. In the former case the deficient No. 4 chromosome—which was of practically the same size, not only in a large number of spermatocyte cells, the germ cells, but also in the body cells—indicates again that such an abnormal chromosome maintains its identity from generation to generation, from fertilized egg to fertilized egg.

The permanency of these abnormal chromosomes has an important bearing upon one problem of synapsis; upon the question as to whether or not a complete side-to-side fusion of homologous chromosomes takes place during this period. I have attempted to illustrate my ideas by diagrams (figs. F_1 to F_5 , G_1 to G_6 , and H_1 to H_6). If we suppose that a fusion of the chromosomes takes place, we should expect to find in the case of the unequal No. 4 pair something like figures F_1 to F_5 occurring. The shortened No. 4, pairing with that portion of the normal

No. 4 to which it corresponds, or with which it is homologous, and leaving the distal end of the normal No. 4 projecting beyond (fig. F_1), would, on fusion with it, form a single cylindrical body having one end, the distal, smaller in diameter than the remainder of the cylinder. If a splitting occur at the end of synapsis in a plane formed regardless of the old plane of fusion, having merely for its object the splitting of this single fused body into symmetrical halves, there would result two daughter chromosomes of equal size but with smaller diameter at the distal end than throughout the remainder of the chromosome (figs. F_3 to F_5). There would then be no such uniform inequality of the No. 4 chromosomes preserved in the first spermatocyte divisions of this animal as we have found. Instead of having a normal sized No. 4 and a (say) five-sixth-sized No. 4 in every cell after the first maturation division, there would be two No. 4 chromosomes of equal length but shorter than the normal (fig. F_5). On the contrary every first spermatocyte metaphase and anaphase (figs. 5-7) shows one normal sized No. 4 chromosome of the same fixed relative size and likewise one deficient No. 4 chromosome of a definitely fixed relative size.

If we turn to the abnormally long tetrad (figs. 12, 13), we have even more definite proof that fusion longitudinally and splitting along a new plane in all probability does not occur. The normal No. 1 chromosome evidently has paired with the distal portion of the long No. 1 chromosome in a manner similar to the diagram shown in figures B_1 and B_2 . If in such pairing it fuse completely with its longer mate, its would form a cylinder again, with one end of larger diameter, the opposite of smaller diameter (figs. G_1 to G_4). If, on the splitting of the fused thread, the new split be formed, not along the old plane of fusion, but upon any plane giving two equal daughter threads, we would get two long chromosomes of the same length with diameters large at the distal end instead of a long chromosome and short chromosome, of the same relative lengths and equal diameter, with which we started. Again, if the new split appeared in the fused chromosome in a plane at right angles to that of fusion (figs. H_1 to H_6), we should

get a similar result, which is also contrary to the facts in the case. The chromosomes, when they come out of the pairing process, are evidently of the same size they were on entering the process. The abnormal sized $2\frac{1}{2}$ -valent No. 1 chromosome is of the same size in all the germ cells after the process as in the germ cells of the same individual before the process, and even in the body cells of another individual (fig. 11). The same may be said of its normal mate. No tags or projections were seen in any case to indicate that a new longitudinal plane of division had occurred.

In both types of unequal tetrads we have very strong evidence that homologous chromosomes, on entering the side-to-side pairing process of synapsis, remain as distinct individuals, retain their identity throughout the period and come out of it with at least the same size they had on entering it. Each pairing chromosome maintains its distinct individuality during this period.

This is opposed to the ideas of Jannsens ('09) and Morgan ('11) as expressed in the theory of the 'chiasma type.' In their theory they assume that homologous chromosomes in parasynapsis twist about each other and fuse. On splitting, a plane passes down the fused body, regardless of the previous spiral fusion-plane, resulting in two daughter chromosomes which may not be identical with the two chromosomes which entered the process. Each new one may contain parts of both original chromosomes. If such had been the case, the separation or formation of a short and a long chromosome out of the fused chromosome (B_2 to B_5), with such regularity of size, etc., as we have shown, could not have occurred. Or, supposing that spiral twisting and fusion had occurred and that splitting again was limited to the larger end of the fused chromosome (figs. G_2 to G_4), we should expect that the shorter member resulting (figs. B_2 to B_5) would at least show fragments of the side of the long chromosome attached to its proximal point in those cases where the splitting plane did not coincide with the old plane of fusion between the short and long chromosomes. No such attached fragments were found. All short or normal No. 1 chromosomes in the metaphase and anaphase of the first spermatocyte divisions were of uniform length

and showed that they retained the same size and were as free from terminally attached fragments as they were on entering the pairing process.

In regard to the question of pre- and post-reduction, I have evidence here that the first maturation division is the reduction division so far as regards three of the seven pairs of chromosomes of the Tettigidae subfamily of grasshoppers. These pairs are the 1's, the 4's and the sex chromosomes. The abnormally long No. 1 chromosome is seen in Tettigidea separating from its normal mate (figs. 12, 13), the deficient No. 4 is seen separating from its normal mate (figs. 5-8), and the unpaired sex chromosome (paired in the female, figs. 1, 11) is seen passing over whole to one of the poles in the male cells in similar manner, just as if it had had a mate from which to part at this division, where such parting is being carried out by the members of the ordinary chromosome pairs (figs. 2, 3, 5, 6, 8, 10, 12, 13). The other chromosome pairs (the 2's, 3's, 6's, and 7's) behave similarly to the 1's and 4's in every respect during the synapsis, prophase, metaphase, and anaphase periods of the first spermatocyte divisions. This leads one to think that probably the first spermatocyte division is the reduction division for all pairs of chromosomes, in the Tettigidae subfamily of grasshoppers at least.

4. THE DEFICIENT NO. 4 CHROMOSOME AND THE LOSS OF UNIT CHARACTERS

The most important of these unequal tetrads are those in which one member of the pair is of less than the normal size (figs. 4-8). The importance lies in this, that such a deficiency may be the result of the dropping of a part of a chromosome and in this way may furnish the basis in the germ cell for the loss of unit factors in heredity.

In animal and plant breeding there are many unit characters which might be considered to have resulted from the loss of something from the germ plasm, whether this something be an enzyme of some sort or a substance upon which an enzyme might act.

It has been shown by Castle ('11) and others that in mice, rabbits, and guinea-pigs the ordinary gray color of the wild rodent is not a simple character, but on the contrary, is very complex. It depends—for example, in mice—upon the simultaneous presence of at least seven or eight color factors. The complete dropping of any one of these from the germ plasm modifies the color of the animal.

The factor most commonly dropped is that for the production of color, the animal—whether potentially gray, black, or brown, etc.—being an albino, actually without color. The next factor very commonly dropped is that for barring of the fur. This does not show unless black or brown pigment is present in the hair. The barring of the hair is due to the fact that some factor prevents the development of black and brown pigment granules in a portion of the individual hair immediately below the tip, leaving a black tip and a dark base with a light yellow band between them. When this barring factor is dropped, we get a black or a brown animal instead of a gray. The next most commonly dropped factor is that for the production of black, giving cinnamon or brown animals. By dropping the factor for self color, spotting results, spotting of two sorts, white upon a colored coat, or yellow upon a black, brown, or gray coat,—giving in the extreme cases white with black or brown eyes and yellows with black or brown eyes. By dropping the dark-eyed character we obtain pink-eyed animals with a scarcity of pigment in the fur. By dropping the intensity-of-pigmentation factor we get diluteness of pigment, giving dilute-pigmented gray, black ('blue'), brown ('cream'), or yellow animals. When all of these factors are present we have the wild gray type. By the dropping of any one of them entirely the color may be modified accordingly.

It is evident that these animals lack something. The dropping of the black pigment, for example, gives animals which are cinnamon or brown. No black can be produced in the race until this factor is again added to the mixture. It is entirely absent. In this case black cannot be considered a latent character which has been restricted in some way from attaining its development, as is the case in albinos of gray or black animals;

these on being crossed with brown, or any animal that contains the color factor, will give gray or black, etc., showing the gray or black characters to have been present in substance though not developed. If the black be absent, nothing can restore it except the addition of black again. There is evidently a complete absence of the black pigment material from the fur of the animal.

In the varieties of domestic plants many similar cases may be given. But before mentioning the color varieties I wish to call attention to the cross between the 'cupid' and the 'bush' variety of sweet pea as one of the best examples. The cupid is a dwarf plant whose internodes are very short, making the stem correspondingly short, about 9 to 10 inches. It has the prostrate habit, that of lying on the ground, due to the diverging habit of the branches. The 'bush' variety produces branches which do not diverge but grow upright, making a tall bush growing 42 to 48 inches in height. These varieties, on being crossed, produce in the F_1 generation plants which show a reversion to the habit and size of the wild sweet pea of Sicily (Punnett '11). They have the long internodes and the long stem of the bush variety combined with the prone, prostrate habit of the cupid variety. By inbreeding the F_1 individuals the F_2 show the tall 'bush' variety, the tall procumbent, the short procumbent or original 'cupid,' and the short 'cupid' bush-like variety. The factors concerned are the long internodes versus the short, and the procumbent versus the erect habit. The two varieties evidently owe their origin to the dropping of one or the other of these factors from the germ plasm of the wild type. The bringing of them together in the hybrid supplies the lack in both races (the 'cupid' and the 'bush' varieties), the result being the wild type.

In flower color of sweet peas we have a similar case. Most white sweet peas breed true to white, but there are two varieties of white which on being crossed produce a purple colored variety, like the wild Sicilian species. On being inbred, the F_2 generation shows nine of the colored variety and seven of the white. Of the whites some breed true, some give a 3.1 ratio, i.e., three whites

to one colored, while some give all colored again. This shows that there are two factors concerned. The presence of both is needed to produce the colored flowers, the absence of either one giving white. It is the F_2 result of an ordinary dihybrid Mendelian ratio, in which there are nine cases where both factors are present and colored results, three cases where only one factor is present and white results, three cases where the other factor is present and white results, and one case where neither factor is present and a white which breeds true results.

The colored individuals in the F_2 generation are found to belong to six classes depending upon the presence or absence of a purple factor, a light-wing factor giving a bi-colored flower, and a factor for intensity of pigmentation, in the absence of which a dilutely colored flower results. The wild type is intensely colored, and bi-colored, i.e., purple with blue wings. By dropping the purple a red bi-colored or uniformly colored flower is obtained in both the dilute and intense series, by dropping the bi-color factor uniformly purple or red are obtained, by dropping the intensity factor both varieties of the red are obtained in dilute form, and, finally, by dropping either the color-producing base or the color developer we get any of these color varieties in the albino form. The wild purple Sicilian species contains all these factors. By the dropping of these factors one by one and by inbreeding, all the color varieties of our domestic sweet peas have been obtained.

Now, it seems to me that it would be quite possible to account for phenomena of this kind in plants and animals as the result of an unequal division of the chromosomes in the reduction division, similar to what I have found evidence of in *Tettigidea parvipennis*. If but one member of the pair of chromosomes showed the deficiency it might not give a result in the organism. When both chromosomes of the pair show such a deficiency, a condition which would result only when inbreeding occurs, then such a deficiency might be shown in the somaplasm by some such defect as albinism, blackness, dwarfishness, etc. It is true that such a chromosome would evidently be an abnormality of a deficient kind, but are not all such traits as albinism in plants

and animals, melanism in rodents, retarded mental development, feeble-mindedness in man, dwarfisms, etc., abnormal deviations from the type of the species which may be classed as deficient characters?

The amount which may be dropped from one chromosome, such as I have shown in *Tettigidea*, may be considered too much to allow of the existence of the organism in the homozygous condition, i.e., where both members of the pair lack it. It is conceivable that a limit in the amount that may be absent might occur and that, going beyond that limit, the homozygous condition might be lethal, as in yellow mice. Here the homozygote always dies, or may never be formed, as has been shown by the results obtained from breeding yellows together. The litters are three-quarters the normal size and the young are found to be two-thirds yellow and one-third gray or other colors, never breeding true to yellow. This indicates that the yellow parents were heterozygotes and that the one-fourth pure yellows, which we would expect in F_2 , have never been formed. Baur ('07) found, on crossing two varieties of snapdragon—the green leaved with the golden leaved—that the offspring were 50 per cent green and 50 per cent golden leaved. The greens bred true but the golden variety produced 25 per cent greens, 50 per cent golden and 25 per cent that were almost white. The latter died at the end of germination, when the food in the seed was used up, since they possessed no chlorophyll. The golden variety was variegated with green patches (chlorophyll-containing cells) and so could manufacture its own starchy food.

The yellow condition in mice and in the snapdragon may be due to a greatly deficient chromosome, so greatly deficient that a zygote having two such deficient chromosomes might have too great a deficiency to be able to develop, or, after having developed, may lack some substance, such as the chlorophyll in the snapdragon, necessary for carrying on one of the vital life processes. In addition, this lack might be so great that it would over-ride the normal chromosome in the heterozygous zygote, giving a yellow mouse instead of a gray, black, or brown, as the case may be; or in the snapdragon a golden variety instead of a green

variety. That something of this nature has occurred, we are inclined to believe from the fact that the golden variety is simply a plant which lacks chlorophyll in small variegated patches over its surface, whose color is therefore due to this condition. In the cases here given possibly there is a law at work correlating the amount of material that may be lacking from a chromosome with the recessiveness or dominance of the trait resulting in the organism. A defective chromosome may continue to give a trait which is recessive to the normal condition until the defectiveness of the chromosome reaches that point where its deficiency becomes so great that the homozygous zygote cannot develop. At that point the defect becomes dominant to the normal condition and individuals can exist only in the heterozygous or normal condition.

It is strange that yellow should be dominant in mice while in most other species of domestic animals it is recessive. This may be due to the position of the yellow determinant along the chromosome. In most species it may be thought of as lying near the end of the chromosome and accordingly could be dropped very easily, causing little disturbance and giving a recessive trait. In the mouse it may be conceived of as lying farther from the end of the chromosome. On dropping enough of the chromosome to cause yellow, a greater disturbance would be created and the defective trait resulting would accordingly be dominant to the normal trait. I make this as a suggestion merely.

The chances for such abnormal divisions are limited by the number of pairs of chromosomes in the species and by the varying amounts which may be dropped from each chromosome in each case. In guinea-pigs the number of pairs is twenty-eight (Stevens '11). The chances for abnormal divisions in guinea-pigs are therefore large. Where the number of chromosomes is small the chances are smaller. The amount that may be cut off from each individual chromosome might vary enough to give several varieties due to the variation in this respect in one pair of chromosomes. The same might be said of each of the twenty-eight pairs in guinea-pigs. Any of these varying conditions in a single pair of chromosomes might combine with

all possible combinations of similar variations in the twenty-seven other pairs. Such might be the basis of all of our variations in guinea-pig inheritance.

It is noticeable in any animal or plant which becomes domesticated that very soon there appear whites, blacks, browns, spotted, yellows and others of the color varieties common to domesticated species. The same may be said of other characters of the species, size for instance. On domestication inbreeding occurs. This gives rise to homozygous strains, which may be isolated. In the wild state inbreeding is not so prevalent. Promiscuous mixing occurs. A summing up of the characters results and all normals, which are usually dominant, are present and show. The weaker recessive characters, if present, are covered up. They exist in the single or heterozygous condition and so do not show. If they exist in the homozygous condition they may show, as in albinos, but the organisms may be killed off by natural selection. Under domestication man preserves these recessives.

The loss of parts of chromosomes may explain very easily the appearance of such phenomena in the domestication of species. The fact that there appear always the same or nearly the same color varieties in each species may be due to this, that their chromosomes are more or less similarly organized, that there are approximately the same number and in many respects the same individual chromosomes to be dealt with in each species, and finally, that these chromosome pairs are subject to the same vicissitudes of fortune in division at the maturation period in each species. True, there are minor variations in chromosomes as we pass from species to species. There are also minor variations in color inheritance as we pass from species to species. But these variations are small when we think of the similarities. All species of rodents show grays, all show albinos, all show blacks, all show some form of brown, and yellows. The method of inheritance of the yellows, for instance, might vary from species to species, but they are yellow, nevertheless.

The same spontaneous variations, such as albinism, probably occur not once but over and over again in the same species in various parts of the world entirely independently of each other.

If the basis of albinism lie in an abnormal reduction division of a certain pair of chromosomes, we should expect it to do just that sort of thing. So long as the same number of pairs of chromosomes occurs in a species, so long will the same variations continue to occur. In this way we shall continue to have white animals produced anew by 'mutation'—blacks, yellows, spotted, and all the varieties not only of color but in respect to other properties of the body as well. In the same way we might always expect to have produced a certain percentage of defective human beings, such as the classes of feeble-minded, imbeciles, epileptics, etc., each of which seems to be due, in many cases at least, to something lacking in the germ plasm (Davenport '11).

Germinal variations of this kind, it seems to me, might be at the basis of De Vries' 'eversporting varieties,' which gave such abnormalities as striped flowers, five-leaved clovers and monstrosities of various sorts, such as pistollody, twisted and flattened stems, etc. Again, it seems possible that germinal variations of this sort might lie at the basis of those of De Vries' mutations which he distinguished as retrogressive in character; i.e., which were characterized by the dropping out of some character from the parent species. They might lie at the basis of some mutants which he considered progressive, but which showed some retrogressive traits, such as the brittleness of the stem in *Oenothera rubrinervis*. My reasons for supposing this are as follows: De Vries found his 'eversporting varieties' producing their abnormal individuals continually. He found his parent species, *Oenothera lamarckiana*, continually throwing off the same mutants in certain proportions. "No single parent plant proved ever to be wholly destitute of mutability." In his parent species, *lamarckiana*, he has probably a constant fundamental number of chromosomes to deal with. He has a reduction division taking place every time a germ cell is formed. He has the same possibility of abnormal, unequal, divisions of tetrads at the time of this division, giving a deficient homologous chromosome. He has self fertilization (inbreeding), which would tend to bring such defective chromosomes together. He has frequent cases of sterility in the inbred offspring of his mutants,

as one would expect in instances where a vital factor had been dropped. He has mutations which, with one or possibly two exceptions, are of a retrogressive nature; i.e., lacking something necessary which was present in the parent species. The *gigas* variety was due evidently to a doubling of the number of chromosomes (Gates '09). It seemed to lack nothing, but the other mutants seemed all to lack traits, some more useful, some less useful, which were present in the parent species. These phenomena, it seems to me, point to something for their basis like the abnormal variations in reduction divisions, such as I have described in *Tettigidea parvipennis*.

It is interesting to compare the number of mutations De Vries obtained from *lamarckiana* with the number of chromosomes. His number of chromosomes was 14, seven pairs. He obtained seven mutations from his plants. One of these, *gigas*, was evidently due to a doubling of the number of chromosomes. It, however, was not a defective mutant, and so may be left out of account here. The other mutants seemed to have something lacking, and there were six of them. Of these *scintillans* seems to have been heterozygous, producing on inbreeding, *lamarckiana* and *scintillans*. It also produced, in a small percentage, some of the mutants most frequently produced by *lamarckiana*. Possibly *scintillans* had one over-deficient chromosome, such as I have postulated for yellow mice or the golden snapdragon. Many sterile pollen grains were found. Possibly the cause of sterility lies here. On this hypothesis, the fact that *scintillans* can produce *oblonga*, *lata*, and *nannella* is not surprising. The other chromosome pairs are just as liable to accidents in germ cells of *scintillans* as of *lamarckiana*. *Lamarckianas* may be produced by *scintillans*; why may not the mutants of *lamarckiana* be produced also? Each of the other five mutations might be based respectively upon deficiencies in one of the remaining five pairs of chromosomes. Since these remaining mutants breed true in each case, it would be supposable that, in order to show, they must be in the homozygous condition. Thus we may possibly account for the five remaining mutants. This hypothesis, it seems to me, is worthy of consideration here. I

see many reasons to suppose that De Vries was dealing, in part of his mutants at least, with something similar to what I have described in this paper as deficient homologous chromosomes.

Deficient chromosomes, such as I have found paired with their normal sized mates in *Tettigidea parvipennis*, it seems to me, furnish a sufficient explanation for the loss of unit factors from the germ plasm. Looked at in the light we now have of the behavior of unit characters which belong to this 'loss' group, the hypothesis appears very probable. It seems to me that Professor Morgan and his students, who are working upon *Drosophila*, should also take into consideration the possibility of such deficient chromosomes. It is a good working hypothesis, and I am going ahead with breeding experiments upon this species, *Tettigidea parvipennis*, in the hope of getting some results. As to what connections there may be between these deficient chromosomes and the greater problem—the origin of new characters—it is difficult to imagine. This matter had better be left until we have more knowledge of the behavior of these chromosomes.

The observational work upon which this paper is based was done at Harvard University under the direction of Prof. E. L. Mark. The writing was completed at Kansas University. I wish to express here my gratitude to Dr. Mark for the help and criticism he has so kindly given to me from time to time.

ADDENDUM

After this paper was worked out, a treatise by Miss Carothers ('13) appeared, describing unequal tetrads in three species of the 23-chromosome grasshoppers, *Brachystola magna*, *Arphia simplex*, and *Dissosteira carolina*. I wish to consider her paper briefly here.

In the twenty specimens of the three species examined she finds the members of one of the three pairs of small chromosomes always unequal in size. The unequal pair occurs in spermatogonial, in first spermatocyte and (separated) in second spermatocyte cells. The members of the unequal pair agree with those I have found, in that they become separated from each other during the first and not during the second maturation division. This is evidence again that the first maturation division is the reducing division. In their passage to the second spermatocytes these unequal members (diads) agree with those of my material in that they are distributed to these cells irrespective of the presence or absence of the sex chromosome. In this respect our unequal pairs differ from those of *Gryllotalpa*, described by Payne ('12), where the longer chromosome was always accompanied by the sex chromosome in the anaphase of the reduction division. Payne may have been dealing with a group of sex chromosomes similar to what he has already worked out in another order of insects ('09).

Miss Carothers' material differs from mine, however, in that she finds the unequal pair present in every animal. I cannot agree with her in this respect, in *Acridium* or *Tettigidea*, as my drawings have shown. Possibly further search will show that the unequal pair is not always present in the species upon which she has worked. If this should be the case, the hypothesis of selective fertilization which she advances would be unnecessary. So far as *Tettigidea* and *Acridium* are concerned, it is not necessary to postulate selective fertilization.

In thinking that the great number of combinations of chromosomes possible is sufficient to account for all variations, I fear Miss Carothers may be mistaken. Gates ('09) has shown that in

Pisum the number of pairs of chromosomes (seven) is not large enough to account for the number of pairs of allelomorphic characters which behave independently of each other in breeding experiments, if we assume that the basis of each member of one allelomorphic pair must be permanently located in one member of a single pair of chromosomes:

In *Pisum* eleven or more pairs of allelomorphs have been observed and the reduced number of chromosomes is only seven; which shows that in this case at least, several characters must reside in one chromosome. The characters must then be confined to separate particles or corpuscles of the chromosomes, and an interchange of homologous particles according to chance during maturation would give the Mendelian combinations.

I am not quite willing to believe that the basis of an allelomorph may slip from one chromosome to another. Yet it is very evident, so far as I can see, that the number of chromosome pairs behaving independently of each other is too small to allow them to be the basis for the number of allelomorphic pairs of characters behaving likewise independently of each other. Possibly some of these extra pairs of allelomorphs may be accounted for by the deficient chromosome hypothesis which I have advanced, or possibly by the 'chiasma' theory of Morgan, though I have evidence against the latter in these unequal chromosomes and in the V-shaped chromosomes of *Chorthippus* and *Jamaicana* (Robertson '15).

Additional instances of unequal chromosomes, so far as I have been able to find in the literature, have been reported by Baumgartner (Science '11), Hartman ('13), and, I have been informed, by Voinov ('12). The last mentioned paper is published in a European journal to which I have been unable to get access. Baumgartner reported an unequal pair in *Gryllotalpa*, but he gave no drawings and no description. Since that time, Payne ('12) has shown that the unequal pair in *Gryllotalpa* is related to the sex chromosome, the larger member of the pair going with it in the reduction division.

A short time ago Mr. F. A. Hartman called my attention to the fact that he had already described unequal divisions of some

of the small chromosome tetrads in the first spermatocyte cells of *Schistocerca* in his paper (March '13) on "Variations in the size of chromosomes." His paper deals chiefly with variations in size of chromosomes due to, what he believes to be, their unequal growth in the cell. Thinking that the whole paper was devoted to 'variation due to unequal growth,' I overlooked the latter part in which he illustrates and describes briefly a few cases of what he considers unequal division in the first spermatocyte. One of these cases (his fig. 86) may possibly be due to faulty conditions of sectioning. The other cases (his figs. 83, 85 and 87 to 91) are probably variations similar to those Carothers has since described in *Brachystola*, *Arphia*, etc. That "size variations may be due to unequal growth" I am inclined to doubt, but Hartman is to be given credit for recognizing the importance of variation in chromosome size in its relation to 'variation' in 'animals,' since his work was done while teaching in a high school away from any university contact and especially since he was entirely ignorant of the Mendelian laws and their relation to variation. Had he known of these and the related work, he would likely have come to the same conclusions that I have.

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EXPLANATION OF PLATES

The drawings of plates 1 and 2 were outlined with an Abbe camera lucida at a magnification of 3900 diameters obtained with a Leitz 2 mm. oil-immersion objective and a Zeiss $\times 18$ compensating ocular, with draw-tube set at 150 mm. and drawing made at the level of the base of the microscope. In the process of reproduction, they have been reduced one-third, and therefore appear at a magnification of 2600 diameters. The numerals affixed to the chromosomes indicate their relative sizes, the smallest being numbered '1.'

PLATE 1

EXPLANATION OF FIGURES

1 to 8 *Tettigidea parvipennis*

- 1 Chromosomes of an oogonial cell; female.
- 2 First spermatocyte; tetrad No. 4 is normal; No. 5 is the sex-chromosome; male.
- 3 Chromosomes of a first spermatocyte of a third animal (male) showing a normal No. 4 tetrad; No. 5 and No. 2 seen somewhat foreshortened.
- 4 to 8 From a fourth animal, male.
- 4 Chromosomes of a spermatogonium; all chromosomes are split and in metaphase; one of the chromosomes of pair No. 4 is deficient.
- 5 First spermatocyte; the deficient No. 4 (4-) separating from its mate (4).
- 6 First spermatocyte; deficient No. 4 (4-) in abnormal, oblique contact with its mate (4).
- 7 Deficient No. 4 tetrads taken from three other first spermatocyte dividing cells in the same animal, showing uniform size but variation in manner of contact of the conjugating chromosomes.

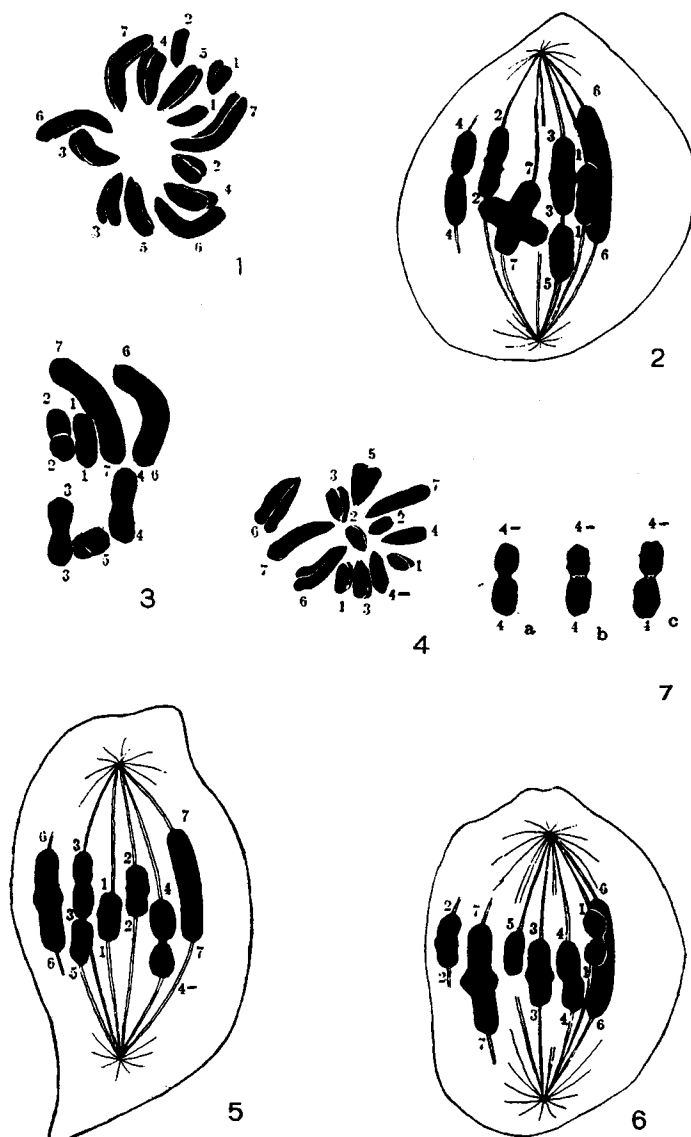
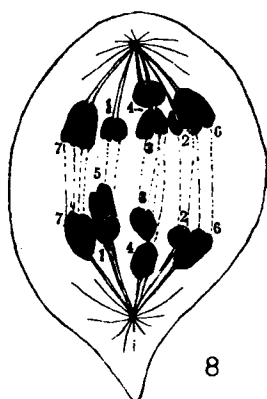


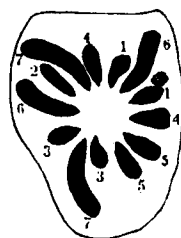
PLATE 2

EXPLANATION OF FIGURES

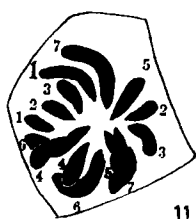
- 8 Deficient chromosome (4—) in the anaphase of the first spermatocyte.
- 9 to 13 *Acridium granulatum*.
- 9, 10 From two normal males.
- 9 Spermatogonium; No. 1 chromosomes normal.
- 10 First spermatocyte, No. 1 tetrad normal.
- 11 Follicle cell of female. One abnormally long ($2\frac{1}{2}$ -valent) No. 1 chromosome.
- 12, 13 From abnormal male.
- 12 First spermatocyte, the $2\frac{1}{2}$ -valent, abnormal, No. 1 (1) separating from its normal mate (1); going with the sex chromosome.
- 13 First spermatocyte, the $2\frac{1}{2}$ -valent No. 1 chromosome not going with the sex chromosome.



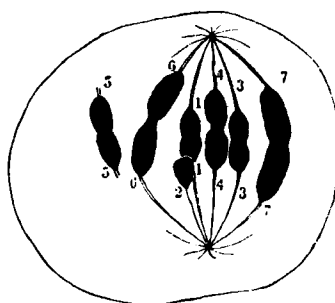
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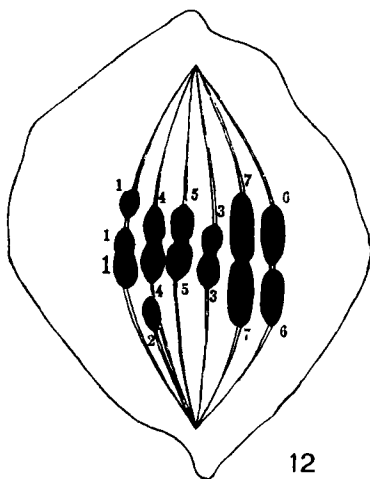
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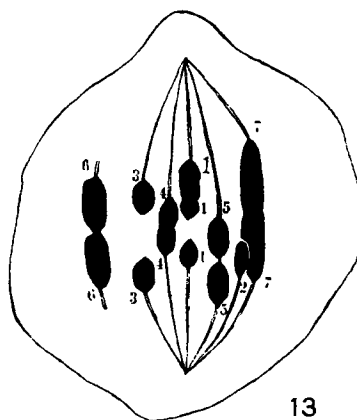
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PLATE 3

EXPLANATION OF FIGURES

A₁ to H₆, schematic

A₁ to A₆ Illustrating pairing in synapsis and separation at first maturation division of homologous chromosomes in Tettigidae. Arrows indicate direction of orientation of the chromosomes; lines and dots indicate respectively maternal and paternal origin of the pairing chromosomes.

B₁ to B₅ Illustrating manner in which the $2\frac{1}{2}$ -valent abnormal No. 1 chromosome pairs with and separates from its normal mate; arrows indicate orientation of parts.

C₁ to D₆ A supposed method of origin for the $2\frac{1}{2}$ -valent No. 1 chromosome.

C₁ C₂ Accidental unequal division in the reduction mitosis, giving a sesqui-valent No. 1 chromosome; arrows indicate orientation of parts.

D₁ Pairing in synapsis of the sesqui-valent No. 1 chromosome of C₂ with a normal No. 1.

D₂ D₃ Separation in late synapsis.

D₃ to D₆ Imagined revolution of the normal No. 1 about the fragmentary end of the sesqui-valent No. 1, giving a $2\frac{1}{2}$ -valent No. 1 chromosome having the two end portions oriented in the same direction.

D₆ Carried over whole in a reduction division giving a $2\frac{1}{2}$ -valent No. 1 chromosome. The distal No. 1 portion corresponds to that part of the long chromosome in B₁ to B₅, which pairs with the normal No. 1.

E₁ to E₆ Evident method of pairing of the deficient No. 4 of figures 4 to 8. Normal No. 4 projects beyond its deficient mate (fig. E₂) at its distal end. In separation the deficient mate evidently has rotated on the side of this projecting end, and has come into position on the metaphase spindle out of line with its normal mate; compare figures 5, 6, 7 b, and 7 c.

F₁ to F₅ Showing result expected if in parasynapsis on the pairing of the deficient No. 4 with its normal No. 4 mate there occurred a complete fusion followed by a splitting of the fused body into two symmetrical parts. The chromosomes would be alike in size in the anaphase of reduction (contrary to fact).

G₁ to G₆ Showing expected result if the same as above took place in the pairing of the $2\frac{1}{2}$ -valent No. 1 with its normal No. 1 mate. There would be two long chromosomes of equal length having clubshaped distal and slender proximal ends, the short No. 1 getting a portion of the long No. 1 (contrary to fact).

H₁ to H₆ Showing the result if the $2\frac{1}{2}$ -valent and 1-valent chromosomes paired and separated in a plane at right angles to the above split (figs. G₁ to G₆), the plane of splitting not agreeing with the plane of fusion of the short and long chromosome (fig. G₂). Two equal chromosomes would result each composed similarly of like parts of both (contrary to fact).

