whether the effect occurs if abnormal 10 is homozygous.

The preferential segregation of the abnormal chromosome 10 does not influence the segregation of allelic genes if the chromosomes bearing them are knobless, nor does it influence segregation at microsporogenesis.

As a consequence of the preferential segregation described above, genes in different chromosomes, each heterozygous for knobs, segregating simultaneously in the same plant, show a similar, if not identical, phenomenon to the 'affinity' in segregation shown in the mouse. Data are given in Table 3 for coupling backcross progenies involving the pigmy (*py*) and coloured endosperm (*C*) loci.

In maize, the affinity depends upon heterozygosity for heterochromatic parts of the chromosome, the preferential, or polarized, segregation of which is activated, or accentuated, by the presence of a special chromosome, in this case the abnormal chromosome 10. The centromeres are not the active agents in maize, nor is there evidence for their being the agents in the mouse. Two possible mechanisms, not necessarily mutually exclusive, may underlie the promotion of affinity in maize by the abnormal chromosome 10. One may be an increase in the frequency of the non-homologous pairing which is shown by knobs at the pachytene stage of meiosis. The other is the formation of supernumerary centromeres, apparently in the neighbourhood of knobs, which occurs in the presence of the abnormal chromosome 10 at both divisions of meiosis[4].

D. G. Catcheside

Department of Genetics,
Waite Agricultural Research Institute,
University of Adelaide.

[1] Michie, D., and Wallace, M. E., *Nature*, **171**, 26 (1953).
[2] Longley, A. E., *Genetics*, **30**, 100 (1945).
[3] Rhoades, M. M., *Genetics*, **27**, 395 (1942).
[4] Rhoades, M. M., and Vilkomerson, H., *Proc. Nat. Acad. Sci.*, **28**, 433 (1942).

I am grateful to Prof. Catcheside for directing attention to the observations in maize, and for affording me this opportunity of emphasizing an important distinction which I made in my previous communication[1] to which he refers. Of the two concepts which were differentiated under the names 'polar affinity' and 'mutual affinity', the former does little more than add an interpretative gloss to the phenomenon of 'polarized segregation' which has been described under that name by, for example, Catcheside[2]. It is the latter concept which is new, and it is the latter concept only—as can, I believe, be shown from our data[1]—which has application to the anomalous segregations observed in mice.

Now in the data of Longley[3] cited by Prof. Catcheside, polarized segregation involving 'knobbed' chromosomes, particularly chromosome 10, clearly occurs. It also appears that there may be an attraction (which it may seem reasonable to compare with 'mutual affinity') between the neo-centromeres of this chromosome and the neo-centromeres of other chromosomes—at least this seems to be one possible explanation of the enhancement of the polarized segregation of 'knobs' on other chromosomes (and of the attendant quasi-linkages) when abnormal 10 segregates to the preferred pole. Whether or not the term 'affinity' should in such a case be stretched to refer to neo-centromeres is discussed by Mrs. Wallace in the following communication.

I hope to publish a full treatment of these matters elsewhere.

Donald Michie

Department of Zoology,
University College, London.

[1] *Nature*, **171**, 26 (1953).
[2] *Ann. Bot.*, **8**, 119 (1944).
[3] *Genetics*, **30**, 100 (1945).

The observations of Longley and Rhoades on the knobbed chromosomes of maize, to which Prof. Catcheside's letter has directed attention, were considered during the formation of my own views on the observations in mice for which Dr. Michie and I have used the term 'affinity'. There is a superficial resemblance between them in that they are both examples of non-random segregation, and both produce similar (although not identical) apparent linkages. That the same principle may, as Prof. Catcheside has implied, be found eventually to underlie them is possible, but certainly not yet proven. Indeed, a recent paper by Rhoades[1] gives a stronger indication of this probability than is mentioned by Prof. Catcheside, for Rhoades maintains that the centromeres *may*, in fact, be the active agents in maize (as theory proposes them to be in the mouse); he has shown that "the true centric region is involved in the formation of neo-centromeres", the latter occurring in the region of 'knobs'.

However, the resemblance is, at least at present, no more than superficial, for there are several important differences, both observational and theoretical, which make the use of identical terms undesirable.

Two observational differences are at once apparent from the published work. First, the anomaly in maize is confined to the sex in which the two poles of the dividing cell have a different biological destiny —it has been observed only in megasporogenesis, whereas the anomalous segregations have been observed in both males and females in the mouse. Secondly, the false linkages in maize depend on the presence of a particular chromosome (abnormal 10), which shows polar preference even in the absence of other abnormal chromosomes, whereas in the mouse no particular allele marking a point of attraction is known to be essential to the process or to have a polar preference. An important theoretical difference has already been mentioned: the maize phenomenon is thought to be controlled directly by the neo-centromeres and perhaps indirectly by the centromeres, whereas, in the absence of evidence for neo-centromeres in the mouse, the phenomenon here is thought to be controlled directly by the centromeres.

It would seem judicious, therefore, in order to avoid confusion, that separate terms be used for each phenomenon as a whole, namely, 'polarized segregation'—or, as Longley terms it, "preferential segregation"—and, on the other hand, 'affinity', until such time as their true relationship is fully understood. The term 'attraction' appears to be a sufficient description of the relation between neo-centromeres proposed as an explanation of Longley's data[2], as it is also for the relation between centromeres proposed in the mouse.

M. E. Wallace

Department of Genetics,
Cambridge.

[1] *Heterosis*, 66 (1952).
[2] *Genetics*, **30**, 100 (1945).