consequence of an error, either of direction of attraction or of insertion of the chromosomal spindle fibres on the half-spindle spherules. This form of misdivision could be correctly called 'attraction-misdivision' or shortly '*a*-misdivision'.

In other cases, the stretched and converged proximal ends of the telocentrics (Fig. 2) suggest a pushing away of the two arms of the daughter-univalents, produced either by the so-called stem-body or by the forces assumed by Östergren to act within the spindle tactoid[4]. This pushing away of the chromosome arms produces at first the separation of their distal ends, afterwards the stretching and finally the misdivision of the centromere. This mechanism may act too late or not strongly enough, and then the misdivision is completed by the formation of the cell wall[5]. This second type of misdivision can be called 'push-misdivision' or '*p*-misdivision'.

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A New Species of Root Eelworm Attacking Carrots

IN the summer of 1944, cysts of a root eelworm were found on carrots growing on a smallholding at Chatteris in the Isle of Ely. Afterwards, infestations were noted on carrots in several other fields in the same area, some of which were alleged to have grown carrots for twenty years in succession. Where the infestation was heavy, the crop was poor and patchy. Plants lifted from the poor patches were stunted and carried an abundance of lateral roots; but this development of lateral roots was less marked than in sugar beet heavily attacked by the beet eelworm (*Heterodera schachtii*).

So far as I am aware, the only previous record in Great Britain of carrots being attacked by a cyst-forming species of *Heterodera* is that of Triffit[1]. The species appears to be new and undescribed, and a summary of its more important features is given below.

Carrot root eelworm (*Heterodera carotæ*, n.sp.)

Cysts	
Shape	Lemon
Size, length	Max. 0·68 mm.; min. 0·21 mm.
breadth	Max. 0·53 mm.; min. 0·15 mm.
Length/breadth	2·3 to 1·35
Regression coefficients	
Length/breadth	0·6584 ± 0·057
Breadth/length	0·7084 ± 0·059
Colour	White changing to brown on death of female. No intermediate yellow stage, as in potato root eelworm
Gelatinous egg sac	Present and large, in full-grown specimens equal in size to the cyst and containing many eggs
Larvæ	
Length	454 μ
Male cysts	
Size on rupturing cortex	0·33 mm. × 0·09 mm. approximately
Males	
Size, length	1·19 mm.
breadth	31·4 μ
Stylet	28·8 μ
Copulatory spicule	Bi-dentate as in *H. schachtii*
Host range	Only cultivated and wild carrot attacked out of 106 plants tested, including the hosts of other common species of *Heterodera*

H. carotæ falls into the group of *Heterodera* species in which the male has copulatory spicules with bi-dentate tips, as in *H. schachtii* and *H. cruciferæ*. On this character alone it can be distinguished from the other species. From *H. schachtii* it differs considerably in size and shape, and in the possession of a large egg sac into which many eggs are extruded. From *H. cruciferæ* it can be distinguished by its smaller size, by differences in its length–breadth relationships and in host-range. In addition, the larval length of *H. cruciferæ* is given as 418 μ by Franklin[2], whereas that of *H. carotæ* is 454 μ.

An account of *H. carotæ* and of observations on other species of *Heterodera* is being prepared for publication elsewhere.

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Blinking and Sleep

As normal blinking is involuntary, it might be expected that blinking would proceed at its normal or at a somewhat reduced rate during sleep, as with breathing and the heart-beat. This is not so, however, though little seems to have been published on the subject, especially on the quantitative side. Whereas many of the muscles are relaxed during sleep, "the muscular tone of the muscles which keep the eyes closed is undoubtedly increased". Duke-Elder[1] states that the occurrence of bilateral blinking movements "may be used as a test for the simulation of sleep".

My interest in the subject has prompted me to utilize the relatively few opportunities open to a physicist to examine the problem on a number of children and on several adults. In all cases so far examined, it has been found that in sleep the eyelids are quiescent, and show no signs whatsoever of blinking movements.

During a recent long train journey, opportunity was afforded of examining the problem in its quantitative aspects, the subjects being a young man and his wife, both of whom fell asleep several times during the course of the journey. Their interblink periods were determined when awake (eyes open), when resting with closed eyes, and observations were continued for up to 15 min. after they had fallen asleep. In each case the conditions of the individual tests were clearly as nearly identical as was possible, and check determinations could be made; the subjects were quite unaware that they were under observation. The results are incorporated in the accompanying table. It did not prove practicable to obtain an accurate determination of the interblink period of the male resting with closed eyes, owing to the rapid onset of sleep in his case; but accuracy was possible with the female, who rested with closed eyes occasionally for a period greater than ten minutes.

Condition of subject	Interblink period	
	Male	Female
Awake (eyes open)	2·0 sec.	1·6 sec.
Resting (eyes closed)	*c.* 1·8 sec.	1·6 sec.
Asleep	∞	∞

Interesting conclusions follow from these observations. The interblink periods of each subject when awake and when in the resting condition were sensibly identical, whereas in sleep bilateral lid movements ceased entirely. There was no indication in either case of any slowing down of the blink-rate with the approach of sleep; indeed, on one occasion the female subject ceased blinking immediately after three lid movements each separated in time by less than one second. It appears that the time taken for the transition from consciousness to unconsciousness under natural conditions is not more than one or two seconds. There was no clear indication that the degree of closure of the eyes during sleep was greater than when the eyes were closed voluntarily during resting periods.

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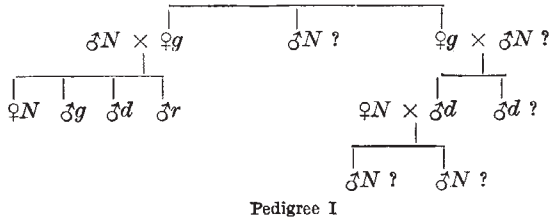
Three Pedigrees for Colour Blindness

THREE pedigrees have been found which are of interest in relation to the two-locus theory of the inheritance of red-green blindness formulated by Waaler[1] and Franceschetti[2], and summarized by Gates[3]. The present hypothesis suggests two series of alleles with near loci on the *X*-chromosome, *N*, *r*, *r'*, *p* and *N'*, *g*, *d'*, *d*, in which *N* and *N'* stand for the normal genes, *r* for red anomaly, *r'* for red anomaly with darkened red[4], *p* for protanopia, *g* for green anomaly, *d'* for deviant or anomalous deuteranopia[5], and *d* for ordinary deuteranopia. The orders of dominance are believed to be as shown above, *N* and *N'* being the most dominant. The two-locus theory is supported by the observation that certain women carrying genes from both series are of normal phenotype[1,2,6,7], and the discovery all over the world of fewer women defectives[3] than would be expected on the single-locus theory by the calculation given by Ford[8].



Pedigree I

In Pedigree I there are two green anomalous sisters whose brother (deceased) was believed to be normal. These sisters married men who were believed to have normal colour vision because they were employed on the railway. One, who was tested by me, was normal; the other (deceased) had not been suspected of any defect. The first sister had four children: a normal daughter, one deuteranope son, one green and one red anomalous, all tested by me. The other sister had two sons, one a deuteranope, exactly like his cousin in every detail of colour vision, married to a normal woman and having two sons believed normal but too young to test. The other (deceased) was believed to have been a deuteranope.

In order to explain this pedigree, we may assume that the two sisters were both double heterozygotes of genotype *rg*/*Nd*, and their brother *NN'*/—, using the system of notation suggested by Ford[8], in which the oblique line separates genes on different chromosomes. Their deuteranope sons would be *Nd*/—, while the green and red anomalous sons would be produced by crossing-over, the green anomalous being *Ng*/— and the red anomalous *rd*/—. The genotype of the red anomalous son is a conjecture based on the assumption that the gene which is more dominant in its own series would be more effective in this combination than the gene of lower dominance in the parallel series. Another conjecture would be that the genes *r* and *d* together would produce only that quality in the phenotype which they could share. The red anomalous colour match in the Rayleigh equation is a possible match for a deuteranope, whereas all other possible deuteranope matches would be impossible for red anomalous vision. The latter conjecture would apply only when the red end of the spectrum is not darkened in red anomalous vision, as in the present case.

The normal daughter might be of genotype *rg*/*NN'*, a double heterozygote in which the two defective genes in different loci have equal and opposite effects, and probably cancel each other out. De Vries has suggested that this combination is of normal phenotype[6].

Pedigree II[9] Pedigree III

Pedigrees II and III illustrate the possibility that a man of normal phenotype may have a colour-blind or anomalous daughter and a normal son. In Pedigree II we may assume that the father was of genotype *pd*/—, and of normal phenotype, which has been suggested for this combination when it occurs in a woman in the form *pd*/*NN'*. If the mother was a normal heterozygote of genotype *pN'*/*NN'*, the daughter would be *pN'*/*pd*, a protanope homozygote and a deuteranope heterozygote together. The son, if normal as believed, would be *NN'*/—, or, if not normal, then a protanope, *pN'*/—.

In Pedigree III the father, believed to be normal, might be of genotype *rg*/—, and of normal phenotype because these genes produce equal and opposite effects, while the mother could be the normal red anomalous heterozygote, *rN'*/*NN'*. The daughter would be *rN'*/*rg*, a red anomalous homozygote and green anomalous heterozygote at the same time, and be of red anomalous phenotype, as found.

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