growth of the innermost layer of the secondary wall, generally known as the S_3 layer, as was suggested by Wardrop[6]. The thickest layer of the secondary wall is normally the S_2 layer, consisting of 30–150 lamellae[7], whereas the S_3 layer is normally much less than 30 lamellae. In callitroid thickenings, however, the S_3 layer is about twice the thickness of the S_2 layer.

A highly magnified section through the wart structure is illustrated in Fig. 5. The wart structure has been described by Wardrop and Davies[8] as a localized thickening of the cell wall in the form of a papilla. While this description is in general verified by Fig. 5, it can be seen that the wart itself has a different structure from the S_3 layer. It can be concluded, therefore, that the wart itself is not simply an extension of the S_3 layer. Examinations of other *Callitris* spp. are being carried out.

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Genetics of Cuckoo Egg Polymorphism

Recent work on avian chromosomes has opened new vistas in the investigation of egg polymorphism in cuckoos. The question of how some of the parasitic cuckoos can consistently parasitize several unrelated host species in one area, usually with eggs well-matched to those of each host[1–3], has long puzzled biologists. Speculations on the genetics involved have been undertaken by Miller[4] and Punnett (in Chance[2]) among others; these need reappraisal in the light of newer ideas and discoveries.

Punnett (*loc. cit.* and ref. 5) discussed among other possibilities that of *Y*-inheritance, and remarked that it would fail to account for the marked deviations from perfect mimicry known to occur in the case of the common cuckoo (*Cuculus canorus*)[3]. With due regard for Occam's Razor and modern cytogenetic knowledge, it is here proposed that the simple *W*-chromosome (to use the modern *Z–W* terminology for avian sex chromosomes) inheritance hypothesis is probably correct. The first positive identification of a *W*-chromosome in birds was reported in 1963 for the budgerigar (*Melopsittacus undulatus*)[6]. Recent work at the City of Hope Medical Center, Duarte, California (unpublished), has disclosed the presence of a large *W* in a Passerine species. Ohno *et al.* have shown that macrochromosomes are remarkably uniform in the avian sub-class Carinatae[7]. Hence it is likely that cuckoos will be found to possess a *W*-chromosome in the heterogametic (female) sex, a highly conjectural possibility prior to 1960 (when all the speculations on cuckoo inheritance occurred).

It is suggested therefore that *W*-inheritance can explain all observed polymorphic phenomena in *C. canorus* specifically, and in other cuckoo species with similar breeding biologies also. Briefly, some of the arguments involved are as follows:

A female cuckoo of a certain host-specific *gens* (egg-monomorphic tribe within a species)[3] would transmit to all her daughters the same egg-colour gene complex that she herself has. According to the *gens* theory, these daughters would be 'imprinted' to their foster-parent species[2,3]. Each daughter would then continue to lay host-matching eggs in nests of that species regardless of the *gens* of her father. Occasional deviations from this pattern can be explained by (1) crossing over (or translocation) between *Z* and *W*; (2) breakdown of imprinting; (3) lack of proper host nests, and other accidental situations. Any of these could explain the occasional cases of mis-matching cited by Southern[3]. It is possible that cases of more frequent misplacement which apparently occur in some areas[1,3] could be ascribed to breakdown in absolute *W*-linkage. For example, a translocation (or cross-over) between *Z* and *W* involving egg-colour genes could become established and spread through a local population because of imperfect discrimination by the host(s) in that area.

The foregoing seems to apply well to the didric cuckoo (*Chrysococcyx caprius*) in South Africa[8–10], in which species, however, *gentes* have not yet been demonstrated. This common bird should prove very suitable for the interesting task which remains: testing the *W* hypothesis cytologically and genetically.

I thank Drs. U. K. Abbott and W. J. Hamilton III, University of California, Davis, and M. B. Markus of Pretoria University, South Africa, for their advice.

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Localization of Ribonucleic Acid in the Cuticle of Nematodes

The demonstration of esterase enzymes in the cuticle of *Ascaris lumbricoides*[1] indicated that this complex structure was metabolically active. The increase in size after the final moult in many nematodes with consequent growth of the cuticle would imply that some biosynthesis takes place during this period. It has been assumed, however, that such biosynthetic activities occurred in the hypodermis[2]. Here I report the demonstration of RNA in the cortical layer of three parasitic nematodes, *Aspiculuris tetraptera*, *Syphacia obvelata* and *Ascaris lumbricoides*.

Two methods for the localization of RNA have been used, namely the acridine orange method of Bertalanffy and Bickis and Kurnick's methyl green-pyronin *Y* method, both methods as given by Pearse[3]. With the former method, fresh frozen 8μ sections were used, and for *A. tetraptera* and *S. obvelata* it was necessary to embed in 2·5 per cent gelatin before sectioning. Sections were cut on a Pearse–Slee cryostat at −20° C. With Kurnick's method, 6μ paraffin sections of worms fixed in Carnoy's fixative (6 : 3 : 1) for 2 h were used. With both methods, controls were pre-incubated in a solution of ribonuclease type IIA (Sigma Chemical Co.) containing 0·5 mg of enzyme per ml.[3]. The substitution of Fenwick's saline[4], at the same *p*H, for Krebs's Ringer solution had no detectable effect on the intensity of staining with acridine orange. In all the three species examined, only the inner cortex of the cuticle was positive; the reaction was absent in all controls including controls of ascarid material pre-incubated in a solution of RNase previously heated at 90° C for 10 min.

The demonstration of RNA in the inner cortex of the cuticle of these nematodes by two different methods precludes the possibility of artefacts and points to the synthesis of proteins in this layer. Chemical analyses of