SHORT NOTE

Genetics of polymorphism in the Little Shag

John Dowding and Michael Taylor's paper on "Genetics of Polymorphism in the Little Shag" (*Notornis* 34 (1):51-57) is a brave effort at going beyond the mere collection of data to attempt to analyse and explain. They propose a genetic model which predicts the ratios of black and pied offspring to be expected from the various pairings of white-throated, smudgy, or pied adult birds. As the numbers they actually observed were consistent with those expected under their model they infer that their model is valid: "Our evidence supports the conclusion that plumage polymorphism in the Little Shag is controlled by two alleles at a single genetic locus, the allele specifying 'dark' being incompletely dominant."

However, it seems to me that there are defects in their analysis which seriously undermine the validity of this conclusion. One could question their assumption that the population is at genetic equilibrium when there is a gradient in morph frequencies from north to south of New Zealand, suggesting, as they point out, "that one or more selective forces are constantly in operation." But the basic problem is one of logic. Dowding and Taylor's hypothesised genetic model is a plausible one and their observed data are indeed consistent with it – or, in statistical language, not significantly different from those expected under their hypothesis. But such a finding does not necessarily mean that the hypothesis is true. Statistical hypothesis testing proceeds by a process of rejection: a hypothesis is accepted only if all possible alternatives can be rejected. I suggest that in the case of the Little Shag there are other possible alternatives which Dowding and Taylor did not test – other genetic models which are equally plausible, and which cannot be rejected on the basis of their observed data.

In particular, they could also have considered the reverse model to theirs: that 'pied' is partially dominant over 'white-throated', with all the smudgy and some of the pied birds being heterozygous (see model B in Table 1). By my calculations the expected ratios of black and pied offspring are not greatly different under either model. The expected results are given in Table 2, along with those under Dowding and Taylor's model (model A). I have

TABLE 1. Genetic models for polymorphism in the Little Shag: frequencies of hypothesised genotypes

Genotypes XX, xx = homozygous; Xx = heterozygous. Model A is basically that of Dowding and Taylor (1987); Model B its inverse.

	Model A (*Wh* partially dominant)	Model B (*Pd* partially dominant)
Adult morphs		
White-throated (*Wh*):	.188 XX + .324 Xx	.512 xx
Smudgy (*Sm*):	.167 Xx	.167 Xx
Pied (*Pd*):	.321 xx	.081 XX + .240 Xx

TABLE 2. — Comparison of observed frequencies of black and pied offspring of Little Shags with those expected under genetic models A and B.

Parental cross	No. of Black offspring			No. of Pied offspring		
	Obs	Exp A	Exp B	Obs	Exp A	Exp B
1. *Wh x Wh*	17	15.3	17	0	1.7	0
2. *Wh x Sm*	11	9.3	7.8	0	1.7	3.2
3. *Wh x Pd*	19	17.1	15.8	6	7.9	9.2
4. *Sm x Sm*	1	1.5	0.9	1	0.5	1.1
5. *Sm x Pd*	1	3.0	2.4	5	3.0	3.6
6. *Pd x Pd*	0	0	0.33	1	1	0.67
Totals	49	46.2	44.23	13	15.8	17.77

recalculated the latter because of the inconsistency in their method of calculating the proportion of heterozygotes among the white-throated birds. Their method implicitly assumes that there is also the same proportion of heterozygotes among the smudgy birds, but elsewhere they assume that *all* smudgy birds are heterozygotes. I have followed the latter assumption, although again the differences in the expected numbers are small. When the two models are tested against the observed results using a similar chi-squared goodness-of-fit test to that used by Dowding and Taylor (grouping results to avoid small expected numbers), in both cases the differences are not significant ($x^2 = 7.3$ and 6.6 respectively; with 6 d.f., $P > 0.50$ in both cases).

Therefore neither Dowding and Taylor's model nor the alternative model can be rejected on the basis of the observed numbers of black and pied offspring. There may well be other, more complex models which would also explain the observed pattern. More observations are required before any single genetic model for the polymorphism of the Little Shag can be unequivocally accepted.

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Galbreath raises a number of matters in his letter. First, he questions our assumption that the population is at genetic equilibrium. There are two main points to be made here; the first is that although there is little evidence either way, it does appear that the cline we describe (more pied birds in the north, more white-throated in the south) has been in place for some time. This suggests some stability in the population, if only at a gross level; hence our deliberate statement that "we have no evidence that it is not" (at equilibrium). He is probably aware by now that we are attempting to accumulate data on this question via the OSNZ Little Shag survey. This is being carried out at present and again, it is hoped, in five or ten years time. Second, morph frequencies such as those discussed here very rarely change rapidly – for the practical purposes of genetic analysis most populations are at genetic equilibrium, unless a dramatic selective force is operating. Obviously we have had to make this assumption (which we believe to be a reasonable one) or we would not have published our model.

The second matter is a somewhat philosophical one, concerning statistical hypothesis-testing. Galbreath declares that "the basic problem is one of logic". If he is suggesting that we were illogical in putting forward the conclusion we did, then I must disagree with him. He states that "a hypothesis is accepted only if all possible alternatives can be rejected". This suggests, incorrectly in my view, that our model cannot stand as a *hypothesis* until all others have been considered. This may be the case for a *theory* but it is clearly not accepted practice for testing hypotheses in the biological sciences. We have put forward for discussion a hypothesis (which is consistent with the known facts) and it remains valid until disproved.

Third, he points out an error in our calculation of the proportion of heterozygotes among white-throated birds, for which I am grateful. The equations shown (our paper, p.54) should read:

DD/DD + Dd = 0.19 / 0.19 + 0.32 = 0.37

Dd / DD + Dd = 0.32 / 0.19 + 0.32 = 0.63

The expected values of black and pied offspring in our Table 3 therefore become 17.1 and 7.9 respectively (as shown in Galbreath's Table 2, Exp A). This makes our expected and observed results for Wh x Pd crosses slightly closer and causes no change to our conclusions.

Fourth (and most significant), Galbreath proposes an alternative model in which 'pied' is partially dominant over 'dark'. He calculates expected numbers of black and pied offspring and shows (his Table 2) that his model is as valid *statistically* as ours. In doing this, however, he makes an additional assumption which I believe renders his model less likely than ours. In our paper, we showed that it is probable that black juveniles develop into both white-throated and smudgy adults, with pied juveniles developing into pied adults. Galbreath has clearly accepted this in calculating his expected values (Exp B) in Table 2; he assigns his heterozygous offspring to the black and pied categories in the proportion 0.167 / 0.407 black: 0.240 / 0.407 pied (heterozygote frequencies from his Table 1). However, this results in a situation where some heterozygotes must develop into black chicks (and become smudgy adults) and others must develop into pied chicks (and pied adults). Thus individuals of the same genetic constitution would be required to display two distinctly different juvenile phenotypes. Some explanation as to how this might occur is necessary (none is provided) and I suspect it would result in a much more complex model. The situation proposed in our model, in which all heterozygotes develop into black chicks, seems the more likely.

Finally, Galbreath suggests that there may be other, more complex models which would explain our observations. I agree, and I am sure the situation is indeed more complicated than either of us has suggested – for this very reason we stated in the Discussion of our paper that "there may be additional genetic effects at other loci...". What we were attempting to analyse in our paper, however, were the primary loci responsible for the observed polymorphism. Our model may well be proved incorrect (or incomplete) in the future; I believe however that it is more likely than Galbreath's alternative, and that it stands at present as a valid working hypothesis.

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