Is the slow worm a Batesian mimic?

SIR,—Smith[1] has suggested that juvenile slow worms (*Anguis fragilis* L.), and also those adult females which retain the dark vertebral stripe, are Batesian mimics of the adder (*Vipera berus* L.). His main evidence for this view is circumstantial; parturition occurs at the same time in the two species, but is usually about one week earlier in the viviparous lizard (*Lacerta vivipara* Jacquin). Smith suggests that parturition time in the slow worm has evolved such that the species gains maximum advantage from the mimicry.

There is, however, a simpler explanation of the time of parturition. The viviparous lizard is a strictly diurnal heliotherm, maintaining a 'preferred body temperature' of about 30° C whenever solar radiation is sufficiently intense to enable it to do so[2]; it can achieve this relatively high temperature rapidly[3]. Adders, in contrast, are both diurnal and crepuscular[4], and the slow worm, as Smith admits, is a burrowing animal which is not often seen in daylight. Body temperatures of the adder are consequently variable; they do not maintain a high temperature for long periods. Slow worms do not usually maintain a preferred body temperature at all; their body temperature is nearly always near that of the ambient air (unpublished observations).

It is not surprising, therefore, that embryological development in viviparous lizards should proceed more rapidly, and it is significant in this connection that the optimum temperature for development of viviparous lizard embryos *in vitro* is 27–28° C, whereas for slow worms it is 18° C (ref. 5).

I conclude that there is no justification for regarding the slow worm as a Batesian mimic of the adder, and adduce as additional evidence the fact that neither my children nor I—two predators with very different levels of experience—ever confuse the two species in the field.

Yours faithfully,

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1 Smith, R. H., *Nature*, **247**, 571 (1974).
2 Avery, R. A., *J. anim. Ecol.*, **40**, 351 (1971).
3 Avery, R. A., and McArdle, B. H., *Br. J Herpet.*, **5**, 363 (1973).
4 Smith, M. A., *The British Amphibians and Reptiles*, (Collins, London, 1951).
5 Maderson, P. F. A., and Bellairs, A. d'A., *Nature*, **195**, 401 (1962).

MR SMITH REPLIES:—Herpetologists have questioned my suggestion[1] that the dorsal zigzag of some female slow worms (*A. fragilis L.*) mimics that of adders (*V. berus L.*), since anyone familiar with the animals can easily distinguish them. Dr Bellairs (personal communication) notes the different behaviour of slow worms and adders when threatened, unlike other mimetic lizards[2], and Avery here suggests that the coincidence of parturition times is explained by body temperature and habits. The usual predators of slow worms are not humans, however, but are more likely to be small, colour-blind mammals which must decide quickly whether or not to attack. Thus a character associated with a previous unpleasant experience (an adder bite) may cause momentary hesitation, giving a slightly increased chance of escape and a small selective advantage.

Whether mimicry evolved in response to concurrent parturition times or *vice versa* is incidental to the increased advantage of a birth period around the peak of the adder's when gravid females are vulnerable to visual predation as they bask in the sun. Nevertheless, it is interesting that, although slow worms mate mainly in May, fertilisation does not occur until June[3] and embryonic development begins around the birth time of viviparous lizards (*L. vivipara* Jacquin), so that 'preferred body temperature' is only part of the explanation.

Thus, Batesian mimicry seems to be the best, current explanation of the dorsal zigzag pattern and its presence in some adult females only.

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1 Smith, R. H., *Nature*, **247**, 571 (1974).
2 Bustard, H. R., *Br. J. Herpet.*, **4**, 22 (1968).
3 Smith, M. A., *The British Amphibians and Reptiles*, (Collins, London, 1951).

Silent alleles and haploid expression in deficiency mapping

SIR,—If a portion of one of the chromosomes is missing, and a primary gene product is reduced in amount, this indicates that either one locus determining this enzyme is missing or that it is ineffective due to a silent allele[1].

The evidence can be evaluated in the conventional currency used in human linkage studies, the lod, or logarithm of the odds ratio[2,3], by the following simple procedure.

We suppose that some transformation of the enzyme level is normally distributed with unit standard deviation, and is d units in the diploid state and h units in the haploid state: this may be derived from data on 'silent' alleles, on the assumption that they are completely silent, or 'non-leaky', or inferred by halving the diploid level. If the parent conveying this hypothetical silent allele has a level a units, after transformation, the likelihood that this is due to haploid expression is the ordinate of the normal curve of unit variance with mean h or $\sqrt{(1/2\pi)}\exp[-\frac{1}{2}(h-a)^2]$ while the likelihood that it is due to diploid expression is $\sqrt{(1/2\pi)}\exp[-\frac{1}{2}(d-a)^2]$; the natural logarithm of the ratio of these ordinates is $\frac{1}{2}[(d-a)^2-(h-a)^2]$; and the logarithm of this ratio, or lod, is $0.217\ [(d-a)^2-(h-a)^2]$. The antilogarithm of this is the likelihood ratio or odds ratio.

On the data of Ferguson-Smith *et al.*[1] the mother had a mean level of 124 units; the mean level for controls with the same phenotype was 119 units and, taking the conservative estimate that a silent allele would reduce activity by 40%, we have $d' = 119$, $d = 15.4$; $h' = 71.4$, $h = 11.9$; $a' = 124$, $a = 15.7$ where the transformation $x = \sqrt{(2x')}$ is used. This gives an approximately unit standard deviation on the data of Spencer *et al.*[4]. The log of the likelihood ratio, or the lod, is then $0.217\ ((a-h)^2-(a-d)^2) = 3.1$ giving an odds ratio of about 1250:1 in favour of a nonsilent allele.

Supplementary information, such as the size of the deficiency, and the frequency of silent alleles, may also be incorporated. This is most simply done by working in logarithms throughout, so that the numerical results are additive.

The failure of the sister of the propositus to show evidence of a silent allele, which she would have a 50% chance of doing if one were there, provides a further lod of log ($\frac{1}{2}$), or 0.30, favouring deficiency.

I thank Professor M. A. Ferguson-Smith for discussions.

Yours faithfully,

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1 Ferguson-Smith, M. A., Newman, B. F., Ellis, P. M., and Thomson, D. M. G., *Nature new Biol.*, **243**, 271 (1973)
2 Barnard, G. A., *J. R. Stat. Soc.*, **B11**, 115 (1949).
3 Morton, N. E., *Am. J. Human Genet.*, **7**, 277 (1955).
4 Spencer, N., Hopkinson, D. A., and Harris, H., *Nature*, **201**, 299 (1964).