males occasionally stopped answering any pattern or flew away, and those tested undoubtedly differed with respect to age, condition of ovaries, number of successful predations, exposure to flashes of foreign males (kinds and numbers), and genetic makeup.

Apparently the mimicry is not perfect, although comparative figures cannot be given since attraction rates for conspecific interactions are unknown. One female captured the 12th *macdermotti* male she answered. Another answered 20 *congener* males, and then moved to a different perch several meters away and answered more than 20 additional males before she captured one. Another female caught the 21st *congener* male that she was observed to answer. Capture rates were higher for prey belonging to other species: on five occasions I observed the demise of *Photuris A* males; two females captured the first male answered, one caught the second, one the tenth, and one female got the 11th, although she had seized the seventh male and it had escaped. Two other females captured the fifth *tanytoxus* males that they answered.

What is the evolutionary origin of the false signals? Two independent sources are suggested. The flashed responses to *Photuris A, Photinus tanytoxus,* and *Photinus macdermotti* males appear to be similar in delay timing to the predator's own mating responses. False signals could have been derived originally from mating responses and subsequently modified. Responses to the flashes of *Photuris congener* males are similar to the flashes that the predaceous females, and those of many other *Photuris* species, commonly emit when they walk, land, or take flight (*9*). These "locomotion" flashes would need little if any modification to attract some *congener* males. (The flashes of the *congener* female, unlike those of other species, do not bear a specific relation to each flash of the male.) I am able to attract about one male in ten to the 0.08-second flashes of a free-running oscillator with a period like that of the males. I once observed a lycosid spider eating a *congener* male that continued to emit his rhythmic pattern; two additional *congener* males were attracted to the flashes of the captive, and were also seized by the spider. I offer this not as an example of a tool-using spider, for I doubt that it is repeated with regularity, but as an indication of how a physiologically inappropriate but trophically fortuitous activation of the locomotion flash mechanism by the flashes of a passing *congener* male could immediately put the female into the aggressive mimicry role. These observations indicate that the capabilities of the firefly brain are more complex than hitherto suspected.

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2. J. E. Lloyd, *Science* **149**, 653 (1965).
3. E. G. Farnworth, thesis, University of Florida (1973).
4. *Photuris versicolor* is a complex of several morphologically similar species which are widely distributed in the eastern and central United States. Extensive field investigations indicate that probably only one species is present in Gainesville.
5. The mating signals of prey species are discussed in more detail in J. E. Lloyd, *Univ. Mich. Mus. Zool. Misc. Publ. No. 130* (1966), pp. 1–95; *Fla. Entomol.* **52**, 29 (1969).
6. This *Photuris* is apparently a new species. Revisional studies and a Latin binomen will be reported at a later date (J. E. Lloyd, in preparation).
7. These interposed flashes were occasionally observed during actual predation of this species on *macdermotti* (*2*) and could be eliminated from the responses of some females when the stimulus patterns were spaced at intervals of 8 to 10 seconds.
8. Both 0.16 and 0.24 second are within the range of *macdermotti* flash responses. The 0.24-second flash was intermediate only with respect to the responses this female emitted.
9. J. E. Lloyd, *Entomol. News* **79**, 265 (1968).
10. I thank E. G. Farnworth and R. S. Lloyd for assistance in the field; T. J. Walker for helpful discussion, comments on the manuscript, and the loan of photographic equipment; A. Owens for photographic technical assistance; and the National Science Foundation (grant GB 7407) for financial assistance. Florida Agricultural Experiment Station Journal Series No. 5447.

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Erythrocytes in Human Muscular Dystrophy

The observations reported by Matheson and Howland (*1*) appeared to fill the need for a simple and reliable method for detecting heterozygous carriers of Duchenne muscular dystrophy (DMD). Dramatic surface deformation of erythrocytes in scanning electron micrographs was observed by these authors in both DMD patients and heterozygous carriers. This observation was consistent with previous results indicating possible abnormalities in cation transport (*2*), fatty acid patterns (*3*), and sphingomyelin levels (*3*) in erythrocyte membranes in human DMD, and also with abnormal erythrocyte morphology described in murine muscular dystrophy (*4*).

On the basis of these reports we studied the erythrocyte morphology in five individuals with DMD, three obligatory carriers, and two normal controls. The methodology was identical to that utilized by Matheson and Howland. Table 1 summarizes our findings and the data of these authors. Only one of our five DMD patients had a percentage of distorted erythrocytes falling just outside the normal range. All carriers demonstrated normal values. In addition, erythrocytes from DMD patients and obligatory carriers also exhibited normal morphology in routine peripheral blood smears stained with Wright's solution, normal osmotic fragility, and normal resistance to peroxide hemolysis.

Table 1. Comparison of results obtained by Miale *et al.* (this comment) with results reported by Matheson and Howland (*1*). Distorted red blood cells are abbreviated Dist. RBC's.

Miale *et al.*			Matheson and Howland		
Sex	Age	Dist. RBC's (%)	Sex	Age	RBC's Dist. (%)
		Normal controls			
M	32	0	M	43	3.4
F	38	1	M	45	3.3
			M	36	7.4
			F	32	4.0
			F	20	3.2
		Obligatory carriers			
F	42	6	F	32	35.1
F	39	5	F	37	39.9
F	37	0	F	32	34.0
		DMD patients			
M	13	5	M	12	65.4
M	11	0	M	15	98.4
M	10	0	M	14	40.6
M	9	24	M	1	20.6
M	6	7			

In summary, our data do not support the adequacy of scanning electron microscopic analysis of erythrocytes for the detection of heterozygous carriers of DMD.

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4. P. F. Morse and J. L. Howland, *Nature (Lond.)* **245**, 156 (1973).

12 August 1974

We thank Miale *et al.* for their interesting comments regarding the use of erythrocyte morphology as a criterion of the carrier state in Duchenne muscular dystrophy. We agree with them that the approach has yet to be shown to have practical utility but we would also hold that a sample series consisting of three proposed carriers and two controls provides little basis for a decision.

The variability of their data as well as the generally lower percentages of distorted cells that they encounter suggest that there are differences of technique underlying the two sets of observations. Indeed, recent unpublished experiments in our laboratories have emphasized the importance of such factors as complete removal of plasma by thorough washing of the cells and the use of fresh glutaraldehyde in a fixing solution of proper final tonicity. In any event, we note that two of their three carriers and the majority of their Duchenne patients exhibited elevated percentages of distorted erythrocytes when compared with their controls. In other words, as far as their data go, they appear to be on the side of supporting our earlier observations.

Finally, with regard to erythrocytes from dystrophic patients, it should be mentioned that Appel *et al.* (*1*) have recently reported erythrocyte distortion from patients with Duchenne and myotonic muscular dystrophy. These investigators employ a preparation technique different from ours, omitting, for example, the saline washing, and the character of the distortion which they observe is likewise different, with dome-shaped cells in the dystrophic case.

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Phosphorus Dynamics in Lake Water: Contribution by Death and Decay

In his report Lean (*1*) appears to extrapolate from results of labeling studies of 1 to 24 hours in duration to conclusions concerning the entire dissolved organic phosphorus (DOP) pool in the natural water system. Although his work is interesting and seems to further elucidate the easily labeled and, thus, apparently the highly labile fraction of the DOP pool, several comments are in order.

First, his words "I identified the forms of ^{32}P in the filtrate . . ." are misleading since he has in fact not identified anything. He has *characterized* three fractions of labeled phosphorus, namely, the original orthophosphate, a high-molecular-weight fraction, and a low-molecular-weight fraction. Each of the latter two could quite reasonably include a host of compounds since Sephadex separations are based primarily upon molecular size differentiation.

Second, Lean proposes that the high-molecular-weight fraction is the result of a combination of the low-molecular-weight organic phosphorus with colloidal material in the lake water. In his model, then, he precludes the direct formation of high-molecular-weight phosphorus in the soluble or colloidal form. For the specific case where only 3 minutes of contact occurred between the organisms and the added $[^{32}P]PO_4$, it is reasonable to argue that the decay of organisms is not a likely source of the soluble organic phosphorus. However, if labeling of organic molecules within the organism can occur within 3 minutes, what evidence is there that release to the surrounding water has occurred by excretion instead of death and cellular lysis? Lean acknowledges that the experimentation took place at maximum biomass, a point where growth and death would be in balance.

On the other hand, if, in fact, not all the cellular organic phosphorus components received ^{32}P labeling in this short period of 3 minutes (that is, if some specific chemical compounds received no ^{32}P incorporation), then the model dynamics may indeed ignore a significant segment of the organic phosphorus pool. This nonlabeled segment could be comprised of an entirely different set of compounds from those represented by the "XP" and "Colloidal P" of Lean's model. However, these compounds could be released into solution predominantly by death and decay. Thus, Lean's repudiation of other authors' claims of release by death and decay (his references 12 and 13) based upon his model and sequence of experiments is not valid.

In fact, there is conclusive evidence (*2*), published prior to the final submission of Lean's report, that a significant fraction of the DOP in both laboratory algal cultures and natural waters is DNA or its fragments (7 to 10 percent of the DOP) capable of exclusion from Sephadex G-75 and G-100 gels. The DNA material represented roughly 50 percent of the total high-molecular-weight fraction. Three distinct responses were used to validate the identity of this isolated, high-molecular-weight material: (i) a deoxyribose-specific fluorescence analysis for DNA, (ii) enzymatic breakdown by deoxyribonuclease of the isolated high-molecular-weight peak, and (iii) conclusive isolation and identification of the bases adenine and guanine by two-dimensional, thin-layer chromatography (including cochromatographing standards and specific color reactions) after perchloric acid digestion of the isolated high-molecular-weight material.

That this material originated from the soluble compartment, independent of cellular damage during processing, was amply demonstrated (*2*) and in one case demonstration relied solely upon diffusive transport across a 0.22-μm membrane into sterile culture media. Although direct evidence to differentiate between direct excretion of DNA fragments by living organisms and release into solution by death and by subsequent decay was not sought, the presence of such fragments would certainly evoke temperance in denying the contribution of death and decay to the DOP pool.

Certainly, if Lean's experiments deal solely with excreted compounds originating from viable organisms, then most likely his results do not pertain to the entire DOP pool. In fact, he states, ". . . I concluded that no high-molecular-weight material was excreted, only XP."

Since the existence of DNA fragments in both algal cultures and several natural water systems has been clearly documented (*2*), these fragments must have been either excreted (contrary to Lean's hypothesis quoted above) or released by death and decay. Lean is