The symptoms observed within twenty minutes of taking the food were tremors, giddiness, perspiration, inability to speak or swallow. There were no fatalities, and the symptoms disappeared after twenty-four hours, though the persons affected had to be taken to hospital for treatment.

It was difficult to feed monkeys, as they refused to take the food. Two ounces of the millet produced the same symptoms on dogs; tremors and paralysis were characteristic effects, and the dogs died in twenty-four hours. The Stas Otto test was negative, showing the absence of alkaloids. The fat was extracted with petroleum ether (60–95° C.), or chloroform, or ether, and it was found that 1·5 gm. of fat corresponding to 50 gm. of the millet were fatal to dogs. The fat had the following figures (average):

Melting point	42° C.
Refractive index (60° C.)	1·4650
Iodine value	93·6
Saponification value	170·7

The defatted residue was found to be non-toxic. The poison in the fat seems to be decomposed by dilute acid and alkali, as after acid and alkali treatment the treated fat is no longer poisonous. The liquid, decanted after shaking the millet with petroleum ether, develops a characteristic red colour when shaken with concentrated sulphuric acid. But as the fat in the millet after treatment with dilute acid—when it is no longer poisonous—still gives the colour test, it is inferred that the colour observed is not due to the poison in the fat but to a decomposition product of it. The poison seems to be neither an alkaloid nor a glucoside, as it is not extracted with acid, water or 90 per cent alcohol. It seems to be adsorbed chromatographically in silica column. Further work is in progress.

The fat obtained from non-poisonous varieties of *varagu* is quite harmless, and does not give the sulphuric acid test described above.

The most surprising observation is that the fat derived from the poisonous variety develops symptoms of poisoning in dogs and monkeys when injected intramuscularly, 1 gm. of the fat being fatal. Crows seem to be extremely susceptible to the poison in the fat, either when ingested orally or injected intramuscularly. Within ten minutes of oral ingestion, the crow puts down its beak and vomits; even after all the contents have been vomited the crow puts down its beak in an effort to vomit. It loses the mobility of its eyes and soon its power of using its legs, while the wings are spread out and can no longer be brought together. The crow dies in twenty-four hours. The effects are slow when the fat is given by injection; but the collapse is more pronounced and the symptom of vomiting, even when there is nothing to vomit, is a characteristic of the poisoning in the crow.

These observations do not appear to have been recorded so far in the literature. They give a means of distinguishing the poisonous from the non-poisonous variety, and it should lead to the reclamation of an important article of food which, of late, has come into disrepute owing to the ill-effects attending its consumption.

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Rh Phenotypes and Fisher's *CDE* Notation

Since the introduction of Fisher's theory and notation[1] of the *Rh* blood-group system, the tendency has been to express phenotypes in terms of the probable genotype. This method is most convenient for European populations, where every common phenotype consists very largely of a single genotype; but its extension to non-European populations usually involves serious ambiguities. In this and other connexions, there is an undoubted need for an unambiguous and generally understood phenotype notation expressed in Fisher's symbols, which would be available to supplement current practices. Several attempts have been made to express phenotypes with the use of these symbols, probably the earliest being that of Bushby[2]. The present proposals closely resemble those of Bushby; but allowance is here made for the fact that the six typing sera (anti-*C*, *D*, *E*, *c*, *d*, *e*) are not usually all available as Bushby tacitly assumes. No originality is claimed for these proposals; it is probable that other workers have thought of and used them independently; and, in fact, when recently I was discussing an *Rh* problem with Dr. M. Bessis of Paris, we found that we had both for some time been using this system in working out *Rh* problems.

The phenotype formula is derived as follows: considering first the *C* locus of Fisher, if a specimen of blood has been tested with anti-*C* alone and the result is positive, a single *C* is written. If the result is negative, *cc* is written, whether or not the blood has been tested with anti-*c*, since a negative result with anti-*C* indicates the presence of two *c* genes. If the blood has been tested with anti-*C* and anti-*c* and both give positive results, *Cc* is written. If the result with anti-*c* is negative, *CC* is written, and this can be done even in the absence of a test with anti-*C*. The *D* and *E* systems are treated similarly. But if both the antisera required for one particular system (for example, *Ee*) are unavailable, the statement of the results (for example, *Ccdd*) makes this quite clear. To take a concrete example, let it be supposed that blood of the genotype *CDe/cde* is being tested with the commonest four antisera, namely, anti-*C*, anti-*c*, anti-*D* and anti-*E*, the results will, in that order, be + + + − and the phenotype will be written *CcDee*. The only available information which is missing from this expression of the results is as to whether the blood has been tested with anti-*e*; but the negative result with anti-*E*, in fact, makes such knowledge irrelevant. The absence of a second *D* symbol (*D* or *d*) shows that no test has been done with anti-*d*.

The above suggestions only relate to the common antigens *C*, *D*, *E*, *c*, *d*, and *e*, in respect of which all the suggested conventions appear self-evident. In order to deal with the rarer alleles such as *Cw*, it would be necessary deliberately to adopt certain further conventions by agreement between the workers concerned.

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[1] Fisher, R. A., personal communication cited by Race, R. R., *Nature*, **153**, 771 (1944).
[2] Bushby, S. R. M., *Lab. J.*, **8**, 78 (1946).