sults: the absence of a rise in blood pressure following carotid occlusion or sectioning of the buffer nerves in the decerebrate cat. Since we obtained equally active vasomotor reflexes in the intact and in the mid-collicular decerebrate cat, the conclusion is inescapable that the basic control mechanism for baroreceptor reflexes must reside in the brainstem and that the influence exerted by suprapontine structures is not essential.

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Our conclusions were based on four observations: the two well-established facts that (i) decerebration by itself does not result in a fall of blood pressure, and (ii) section of the buffer nerves results in a sustained elevation of blood pressure; and (iii) our positive result that decerebration in animals with three or four severed buffer nerves results in an immediate and sustained fall of blood pressure (which Katz *et al.* appear also to have observed in their vagotomized animals, as their records in the cited references indicate), and (iv) our "negative result" that section of the buffer nerves in decerebrated animals fails to result in a sustained rise of blood pressure, although a transient rise immediately following nerve section has been observed. It is not clear from the correspondents' comments whether the blood pressure rise which they observed after buffer nerve section persists after the minimal 30 minutes interval which we used as our criteria. Without this essential information, a true difference between our results and theirs cannot be established.

The "negative result" used in support of our conclusions, and published elsewhere, was that the pressor response to occlusion of one carotid artery proximal to the only innervated carotid sinus was inhibited. Since the pressor responses which Wang and his associates clearly found unchanged before and after mid-collicular decerebration were elicited by bilateral carotid occlusion, the experiments are not comparable.

Finally, we do not claim that the mechanism of baroreceptor reflexes does not reside in the lower brainstem, that is, in the pons and medulla. Hence, we are not in disagreement with Wang and his colleagues on this point. It is our contention, supported by our facts, that the excitability of these reflexes may be modified by suprapontine structures and that this reflex excitability may be changed without changing the resting mean blood pressure. It is through a modulation of this reflex mechanism that we propose that rostral brain structures exert some tonic control of blood pressure. We have not addressed ourselves to the essentiality of this control. We have merely pointed out its presence.

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19 August 1964

"Cytoplasmic" Sterility

The report on "cytoplasmic" sterility by Ehrman (10 July, p. 159) has some fascinating implications for the field of gene-cytoplasm interactions in general. One possible explanation of his results would be that the Mesitas and Santa Marta cytoplasms have some common structures which interact with genes affecting male fertility; further, that these structures occur in different proportions in the two cytoplasms, the particular ratio in either one being a response to natural selection for effective interaction with the genome. Cytoplasmic structures do not seem to replicate by the same system as the nuclear genes, and it is not necessary to assume that only two kinds of cytoplasmic "alleles" can be present for any one genetic locus, that equal distribution must occur at mitosis or meiosis, or that all of them necessarily multiply at the same rate under all conditions.

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3 August 1964

Meyer's explanation is very close to a working hypothesis which I am planning to test. The Mesitas strain consistently carries a heavy infection of microsporidia, while the Santa Marta strain is free of them. It is, of course, possible that other symbionts or parasites of various kinds (protozoans, bacteria, viruses) may also be discovered in these flies. Suppose, then, that each of the six morphologically indistinguishable races or incipient species of the *Drosophila paulistorum* complex carries a symbiont to which it is adapted, and that this hereditary "infection" is transmitted via the egg cytoplasm. The nonhybrid genome keeps the infection under control so that it does not interfere with male fertility. The genotype of the hybrid disrupts this control, and the male hybrids are sterile. The symbionts are controlled by the genotype of the race in which they occur, but they may get out of control in individuals of hybrid genotypes. This may, then, be a causative factor which brings about the reproductive isolation between these incipient species.

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2 September 1964

Wild and Domestic Animals as Subjects in Behavior Experiments

In a recent report, Kavanau (*1*) sets forth several generalizations which he says "have important bearings on the rationale and design of experiments on learning and reinforcement." Two of these generalizations seem especially likely to mislead those readers who are not actively engaged in behavioral research. They imply that a new era has arrived in which wild animals must wholly replace domestic animals as subjects in learning experiments.

I would agree that there certainly are differences between wild and domestic animals—differences in rearing and living conditions, in structure, in physiology, and in underlying genetic factors—and that, as a consequence, there are behavioral differences as well (*2*). Granting these does not concede Kavanau's position.

Consider first his statement concerning evolutionary processes: