

MULTIPLE BLOOD FEEDING BY A LABORATORY COLONY OF
ANOPHELINE MOSQUITOES AND IMPLICATIONS
FOR DISEASE TRANSMISSION

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The intake of a blood meal by a female mosquito is a critical event in its life history. The blood meal provides nutrient and initiates a series of physiological events ending in egg maturation. The blood meal is also an essential step in the life cycle of vector borne pathogens. Aedine and culicine mosquitoes generally use one blood meal for each gonadotrophic cycle, but we have observed that in our laboratory colony of Anopheles tessellatus maintained at $28 \pm 1^{\circ}\text{C}$, 80% R.H. and natural day-night conditions, females take multiple blood meals, to engorgement, before egg development. 3 - 5 day old n. tessellatus are exposed to guinea pigs for either one of multiple blood meals on consecutive days. Following successful engorgement, mosquitoes are held in individual vials, provided with egg-laying surfaces. A blood meal is accompanied by prediuresis and concentration of blood proteins in the midgut of the mosquito. Each blood meal triggers the maturation of only one batch of eggs. Mosquitoes feeding once generally lay all their eggs at one time, while eggs resulting from multiple blood meals are laid in groups over several days. Multiple blood meals lead to greater fecundity and the possibility of a higher rate of disease transmission. Fecundity in An. tessellatus was not markedly different after feeding on either human, rabbit or guinea pig blood, with the mean numbers of mature oocytes resulting from these feeding being 63.3 ± 5.2 , 64.9 ± 9.3 and 73.4 ± 6.1 respectively. The dynamics of disease transmission may be simply described as follows (MacDonald 1961).

$$Z = \frac{ma^2 b p^n}{-t \log_e p}$$

Where Z is the basis reproduction rate of the disease, m the number of vectors (fecundity), a, probability of biting man and b the proportion of infected vector bites. As a consequence of multiple blood meals in each gonadotrophic cycle, as observed in the laboratory colony, the values m, a and b are increased. This leads to an increase in the rate of disease transmission, according to the model.

MacDonald, G. (1961). Public Health Report 76 753-764.