3-3-7. Characterization of Cholesterol Transport from Midgut to Fat Body in *Manduca sexta* Larvae

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Using *in vitro* methods, we investigated the transfer of cholesterol from larval *Manduca sexta* midgut to the hemolymph lipoprotein, lipophorin, and the transfer of cholesterol from lipophorin to larval fat body. In the midgut, transfer of free cholesterol shows saturation kinetics, but the apparent Km is higher than the measured Kd for the midgut lipophorin-receptor complex. In addition, the transfer is unaffected by suramin, which binds to the receptor and inhibits lipophorin binding, and by antibodies to the lipid transfer particle, which is required for export of diacylglycerol from the midgut to lipophorin. In the fat body, transfer of free cholesterol also shows saturation kinetics, and the apparent Km is higher than the measured Kd for the fat body lipophorin-receptor complex. Suramin and anti-lipid transfer particle antibodies exert only a small (20%) inhibitory effect. In both tissues it seems that the most likely mode of cholesterol transfer is via aqueous diffusion, which is also an important mechanism in vertebrate cells. Based on these results, we propose that cholesterol homeostasis in larval *M. sexta* is maintained by a mass action mechanism in which cholesterol is freely transferred between lipophorin and tissues depending on the needs of the tissues. This simple mechanism is ideally suited to insects, which can neither make cholesterol nor internalize lipophorin, the two mechanisms that vertebrate cells use to control their cholesterol content.