CAROTENOID REPLACEMENT IN *Drosophila*: FREEZE-FRACTURE. W.S.Stark\*, G.Brown, D.Hombs, J.S.Christianson & R.White±. Div. Biol. Sci., Univ. of Missouri, Columbia, MO 65211 & +Dept. of Biology, Univ. of Massachusetts, Boston, MA 02125.

Carotenoid deprivation in Drosophila reduces visual pigment, opsin, size of the rhabdomere and P-face particle density; replacement by feeding carrot juice rapidly restores visual pigment (Sapp et al., 1991, Exp. Eye Res. 53:71). Our data indicate that this effect is mediated by retinoid-activated opsin gene transcription (Stark et al., 1992, Invest. Ophthal. Vis. Sci. 33: 1398). Here we report that P-face particle density also increases in rhabdomeric microvilli in the early days of replacement therapy to 3000 particles/\mu m<sup>2</sup> by 1 day, reaching the control level of over 4000 by day 2. Our vistas reveal a continuity of the microvilli with the adjacent retinula cell plasmalemma between the adhering junction and rhabdomere. This plasmalemma reflects the rhabdomeric Pface particle density. Freeze-fracture preparations of Drosophila photoreceptors also displayed autophagic coated pits budding from bases of microvilli and from plasmalemma as well as multivesicular bodies and Golgi apparatus. Recovery in Drosophila is considerably faster and more complete than recovery induced by 11-cis retinal in similarly deprived Manduca (Bennett & White, 1991, Vis. Neurosci. 6: 473). Further, there are substantial differences in the endomembrane traffic in deprivation vs. replacement. Support: NSF BNS8811062 & NIH EY07192 (WSS) & NSF BNS91 10672 (RW).