when compared to the preliminary data of allele diversity of the same loci in 13 *D. mediopunctata* lineages (Laborda *et al*., 2009a). The mean allele number found by Laborda *et al*. (2009a) in *D. mediopunctata* for the loci amplified in *D. ornatifrons* was approximately 6.4, and for the loci that amplified in *D. maculifrons* was approximately 6.1. This fact, *i.e*., the higher allele diversity of these loci in heterologous amplifications than in the species from they were first described, could be due to the use of laboratory lineages of *D. mediopunctata* against the use of natural populations for the species of the *guarani* group analyzed in this work.

The results obtained suggest that the microsatellite loci that showed positive and good amplification quality are useful markers to be applied in population genetics studies using both species of the *D. guarani* group. The low amplification rate of the analyzed microsatellite loci in population samples from Araucaria Forest fragments suggest an accentuated genomic differentiation of these populations to the *D. maculifrons* and *D. ornatifrons* populations previously analyzed by Laborda *et al*. (2009b).

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Rhodopsin traffic investigation with the heat shock promoter.

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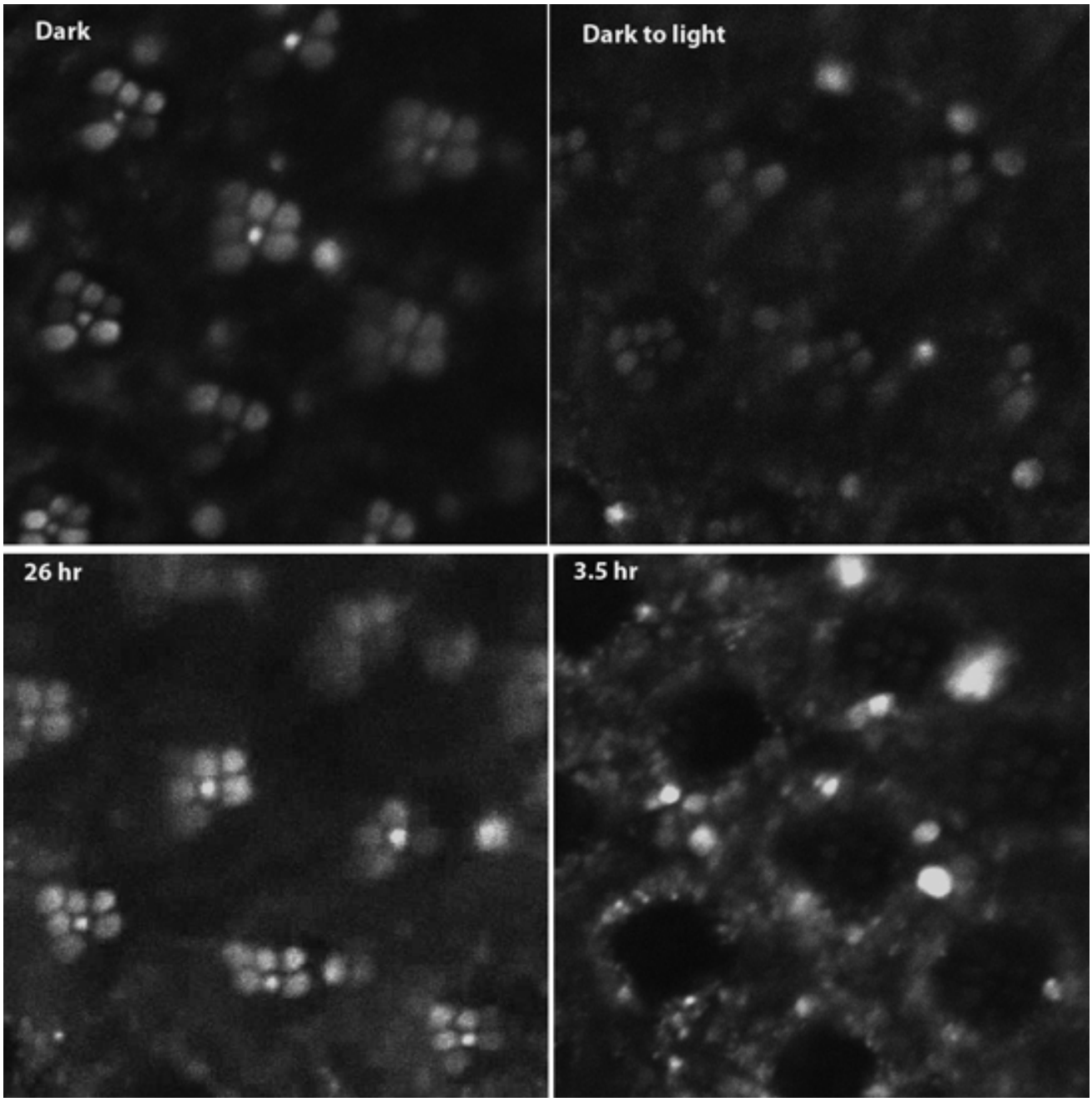
This laboratory has a long-standing interest in rhodopsin turnover in *Drosophila* (Stark, *et al*., 1988); rhodopsin is cleared from the photoreceptive organelle (rhabdomere) via coated pits then multivesicular bodies (MVBs) and lysosomes and imported into rhabdomeres via membranous vesicles. This ultrastructural description has had many molecular elaborations (*e.g*., Chinchore *et al.*, 2009) since our early work. Later, we showed that white-eyed flies maintained in the dark had considerably more rhodopsin than flies kept on a light-dark cycle (Zinkl, *et al.*, 1990; Selimovic, *et al.*, 2010). A white-eyed stock in which R1-6 rhodopsin (Rh1) attached to green fluorescent protein (GFP) was driven by a heat shock (hs) promoter (hs-Rh1-GFP, Belliveau, 2008) allowed us to visualize aspects of rhodopsin traffic using optical neutralization of the cornea in the confocal microscope (Stark and Thomas, 2004).

For heat shock, flies were lightly etherized and placed in a vial in a 37^{o}C water bath for 1 hr. Then, based on what we already knew and how our pilot observations guided us, we put them in a food vial in the dark. R1-6 and R7 rhabdomeres show fluorescence (Figure, top left). Since it was the heat shock promoter, not Rh1's promoter (ninaE), R1-6's rhodopsin (Rh1) should be expected to



be driven ectopically into R7 (and ocellar receptors) as well as into R1-6 (Belliveau, 2008). Some of the R1-6 rhabdomeres were dark, and this striking result was repeatable and unexplained. The fly (Figure, top left) had been maintained in the dark for 4 days, showing that, without light, the rhodopsin remains in the rhabdomere, and, in other work, we extended this obsevation out to 12 days. If, however, flies were kept in the dark for 3 days then in the light for 1 day (Figure, top right), the fluorescence was greatly diminished. This is in keeping with our earlier finding (Zinkl, *et al*., 1990) and Chinchore *et al*.'s (2009) work and reinforces the notion that light is necessary to trigger the clearance of rhodopsin from the rhabdomere.

We also sought to investigate import of rhodopsin into the rhabdomere. In pilot work, we showed that there was no fluorescence 2 hr after heat shock while the fluorescence was nearly fully established at 5 hr. Dissecting this time span, we saw dim rhabdomere fluorescence at 3.5 hr (Figure, bottom right). For a control, we show a fly 26 hr after heat shock (Figure, bottom left); as stated above, both were kept in the dark after heat shock. The striking aspect of the 3.5 hr vista is the haze of fluorescent bodies seen in the cytoplasm of the retinula cells. We presume that we are visualizing membranous vehicles (and, perhaps Golgi apparatus) involved in the import of rhodopsin into the rhabdomere.

All four of our figures, and hundreds of other images we have obtained, show large fluorescent bodies that appear to be in pigment cells between ommatidia. We have always assumed, though we have not proven, that these are the giant unpigmented pigment granules of white eyes (Stark and Sapp, 1988). We thought we should not gloss over this point because, again, with techniques more sophisticated than our 1980's ultrastructural work, there has been a vastly renewed interest in eye color pigment granules.

We hope that our observations are of use to the many research groups using modern techniques and the accessibility of rhodopsin and the compound eye in *Drosophila* to study the broader issue of protein traffic.

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Polytene chromosome analysis in eye color mutants of *Drosophila willistoni* and their hybrids. The H inversion.

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Introduction

During the course of the linkage analysis of (new) spontaneous mutations in *Drosophila willistoni* from populations of Uruguay, Brazil, and Argentina (Soler and Goñi, 2012, Dros. Inf. Serv., this issue), data on the genetic interaction of some eye color mutants were evaluated, among other genetic data to construct the linkage groups. Within the referent eye color mutations, *brown (bw)*, reported to be linked to chromosome 2 by Spassky and Dobzhansky (1950), produces white eye color in young flies to uniformly blotch brownish eye color in old flies with *cardinal (cd*) (Figure 1 in Soler and Goñi, 2012). The presence of this new eye color in F_2 progeny was interpreted as the occurrence of the eye color genetic interaction between the tested mutations and concluded that the