wings. The effect of full phenotype expression seems also to modify significantly the axillary and jugal areas of the wing base.

The initial impression was that the new *curly* (*Cy*) phenotype of *D. willistoni* extended the life cycle of the strain to approximately 60 days. However, when the experiments to establish the generation time were performed, by generation F11 of the mutant strain, the developmental time was about 21 days, very similar to the GdH4-1 strain.

There was no significant difference between crosses in both directions (*i. e.*, female *Cy* × male *cy* or female *cy* × male *Cy*), which leads to the conclusion that the *curly* mutation of *D. willistoni* is not linked to the X chromosome. The F1 showed a frequency of 96.02% of *Cy* mutants (241:10), very near to 100% expected for a Mendelian dominant allele inheritance. Since the phenotype is highly variable, the individuals marked as wild types could be in fact mutants with the more subtle phenotype, which could made the F1 mutant frequency raise to 100%. This seems to be a situation very similar to that described by Ward (1923), who described that some phenotypically wildtype flies were in fact mutants in a genetic background that promoted the suppression of the *curly* phenotype. The F2 generation has shown the frequency of 2.34:1, which can be fairly accepted as similar to the expected 3:1 Mendelian proportion. The deviation can be due to partial lethality, since in some crosses a very few individuals were recovered in F2. This lethality is presumably dependent on the individual genetic background. The availability of mutants, as the *curly* described here and the series of eye mutants described by Soler and Goñi (2012) and the chromosome gene arrangement of these mutants (Goñi and Valente, 2012), are very important to make *D. willistoni* a candidate to become an experimental model. Its genomic characteristics distinctive from *D. melanogaster* (Clark *et al.*, 2007; Schaeffer *et al.*, 2008) can increase even more such interest.

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Erupt-like mutants from a natural population of *Drosophila melanogaster*.

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Mutations affecting head structures (eyes and antenna) were isolated from a natural population of *D. melanogaster* from Nalchik (North Caucasus, Russian Federation). The screen for

visible mutations was performed for several years by establishing isofemale lines. The mutations affecting eye and antenna formation were found in screens of several years in average in one of 100 females tested. All are recessive and demonstrate incomplete penetrance and variable expression. Phenotypically the mutations resemble *er* (*erupt*, 3-70.7) mutant (Lindsley and Grell, 1968; Aubele, 1968). Chromosomal localization of our mutants was not identified, so we denoted them as vm (visible mutation).

Here we report the mutant phenotypes.

Three mutants - $vm^{23\text{-}N2013}$, $vm^{28\text{-}N2012}$, and $vm^{8\text{-}N2011}$ have similar manifestation: eruption of underlying hypodermis in center of one or both eyes. Eruption may be segmented and have hairs. In a few flies legs are deformed.

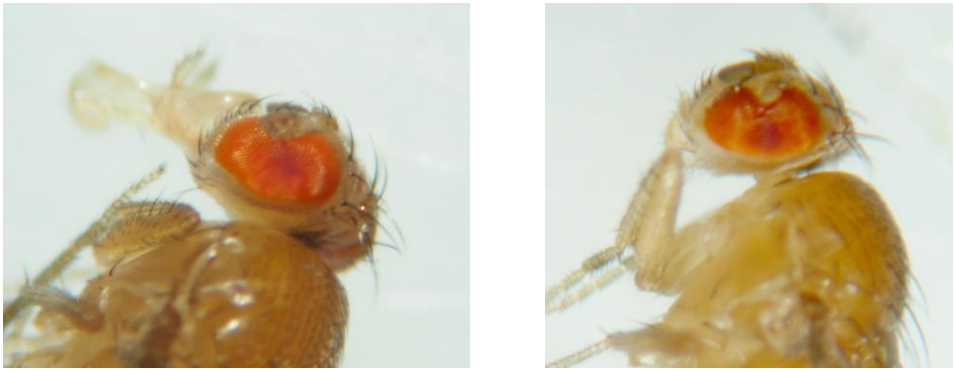

Figure 1. The $vm^{23\text{-}N2013}$ phenotype. A "palp-like" growth protruding from the eye.

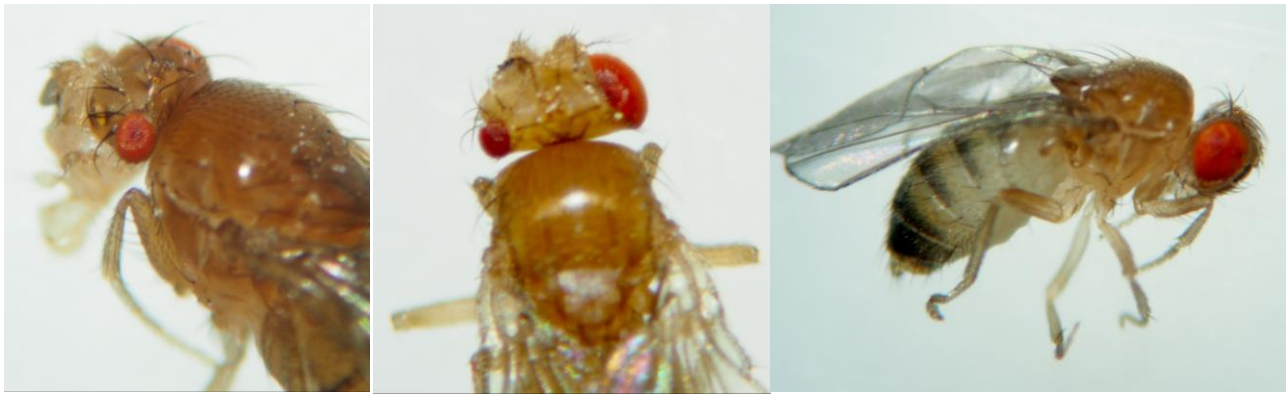

Figure 2. The $vm^{28\text{-}N2012}$ phenotype. The variable expression in eyes and the deformed legs are shown.

The third mutant, $vm^{11\text{-}N2012}$, is different from others. It affects both eyes and antenna. In extreme cases antenna are duplicated and the head expanded. Eyes are malformed. Flies with strong manifestation are sterile, but others have fertility sufficient to maintain a stock. The mutation was mapped to chromosome 2.

Figure 3, see facing page.

References: Lindsley, D.L., and E.H. Grell 1968, Carnegie Inst. Wash. Publ. 627; Aubele, A.M., 1968, Dros. Inf. Serv. 43: 139.

Figure 3. The $vm^{11\text{-}N2012}$ phenotype. In extreme cases the antenna are duplicated on one or both sides.