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The *white*$^{-}$ mutation provides a growth advantage for mosaic clones in *D. melanogaster* and *D. simulans*.

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In this short communication we demonstrate that a *white* mutation can provide a positive bias to a mosaic clone homozygous for a recessive marker linked with *white*$^{-}$ compared to its twin clone homozygous for another marker linked with *white*$^{+}$. The effect is not very strong and requires for its observation a large sample or a strong mutagenic treatment generating enough clones. At the same time, the effect remains persistent and statistically significant in two fly species (*D. simulans* and *D. melanogaster*), with different alleles of *white* and mosaic clone markers and with different mutagenic agents applied. The protein White is a part of ATP-dependent nucleotide and amino acid transporters, particularly for guanine and tryptophan, the precursors for eye pigments (Evart *et al*., 1994). The gene *white* is also known to affect courtship behavior and response to anesthesia (Svetec *et al*., 2005; Campbell and Nash, 2001). No function relevant to cell growth has been reported for this gene by now.

Genes. y^2 – *D. simulans yellow*^{2}, *yellow* chaetae, normal wings, the mutation of spontaneous origin in the *vermilion* strain; *sn* – *D. simulans singed*, "singed" chaetae, homozygous females are sterile, radiation-induced mutation in the *vermilion* strain; *v* – *D. simulans vermilion*, vermillion, brilliant red eye color; nH^{+} – an autonomous *D. simulans* transposable element located in X-chromosome, the genetic instability H^{+}-factor, n represents the number of H^{+} copies in the X chromosome; *w* – *D. simulans white*, white eye color; *wy* – unspecified *D. simulans wavy* allele, grey wavy wings, the mutation was originated spontaneously in the *sn v* strain; *y* – *D. melanogaster* unspecified *yellow* allele; *w* – *D. melanogaster* unspecified loss-of-function *white* allele; w^{1118} – *D. melanogaster* loss-of-function *white* allele, homozygotes have white eyes with only 1% red pigment (compared to 100% pigment in wild type); sn^3 – *D. melanogaster singed*^{3} allele, homozygous viable and fertile.

Stocks and Crosses. *Drosophila simulans*: (1) y^2, H^{+}, (2) *sn v*, H^{+} / $\overset{\wedge}{XX}$, (3) y^2 *w*, (4) *sn v wy*, $2H^{+}$/ $\overset{\wedge}{XX}$. *Drosophila melanogaster*: (1a) *y*, (2a) *w* sn^3, (3a) *y* w^{1118}; (4a) sn^{MR2}. The following crosses were performed to obtain *y* + +/+ + *sn*, *y w* +/+ + *sn* and *y* + +/+ *w sn* heterozygotes: (I) ♀ y^2, H^{+} × ♂ *sn v*, H^{+} /*Y* ; (II) ♀ y^2 *w* × ♂ *sn v wy*, $2H^{+}$ /*Y* (*D. simulans*); (III) ♀ *y* × ♂ *w* sn^3 ; (IV) ♀ *y* w^{1118} × ♂ sn^{MR2} (*D. melanogaster*).

Mutagenesis. To generate mosaic clones by somatic recombination, we used the following procedure. The F_1 48-hrs old larvae of the cross (III) were treated with 0.3 ml per vial 0.5% water solution of phosphemide (CAS # 882-58-6). Analogously, the larvae from the cross (IV) were treated

with 2 mg/ml water solution of oxoplatin (CAS # 53261-25-9). F_1 larvae from the cross (III) were treated by γ-rays (600, 900 or 1200r) after 72, 96, or 120 hrs of development. Untreated F_1 cross (III, IV) larvae were used for a control. H^+-dependent mutagenesis in the offspring of the crosses (I) and (II) occurred spontaneously.

Observation

Within the F_1 of all the crosses the y + +/+ + *sn*, *y w* +/+ + *sn* and *y* + +/+ *w sn* heterozygous females were examined for *yellow* and *singed* clones on head, notum, and humeri with MBS-10 stereomicroscope. The clone number and frequencies were calculated. The clones were ranked according their size in terms of the macrochaetae number (clone number / flies population) × 100%. In *D. simulans* individuals heterozygous for the H^+-factor the clone frequency on the humeri was registered separately as a result of the humeri-specific *H*-factor mutagenesis.

Statistics. Hypotheses regarding the yellow and singed clone proportion were estimated in a standard χ2 – test. All the biased *y*:*sn* clone proportions given in the figures differ from 1:1 at least at $P < 0.05$.

Results and Conclusions

1. In flies heterozygous for *white* and other X-linked markers, the marker linked to the *white* mutation in *cis*- possesses higher mosaic clone frequency than the marker in *trans*-. For example, in *y* + + / + *w sn* heterozygotes the *singed* clone frequency is higher than the frequency of the twin *yellow* clones, and *vice versa*, in *y w* + / + + *sn* heterozygotes *yellow* clones are more frequent than *singed* ones. In absence of the *white* mutation (in *y* +/+ *sn* flies), frequencies of *yellow* and *singed* clones are equal. Thus, the mosaic clones homozygous for a *white* mutation have a growth advantage on a *white*/+ background compared to twin clones homozygous for $white^+$.

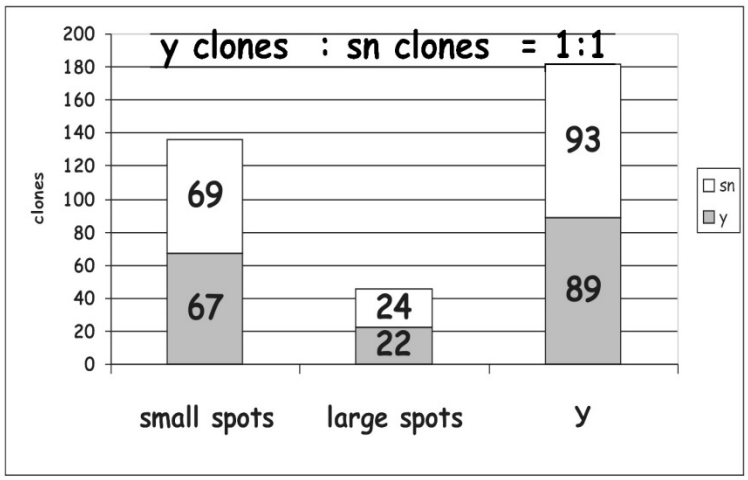


Figure 1. In the absence of the *white* mutation, (*y*) to (*sn*) clone ratio is 1:1.

Flies: *y* + / + *sn*, 8819 *D. simulans* females, spontaneous mosaicism.

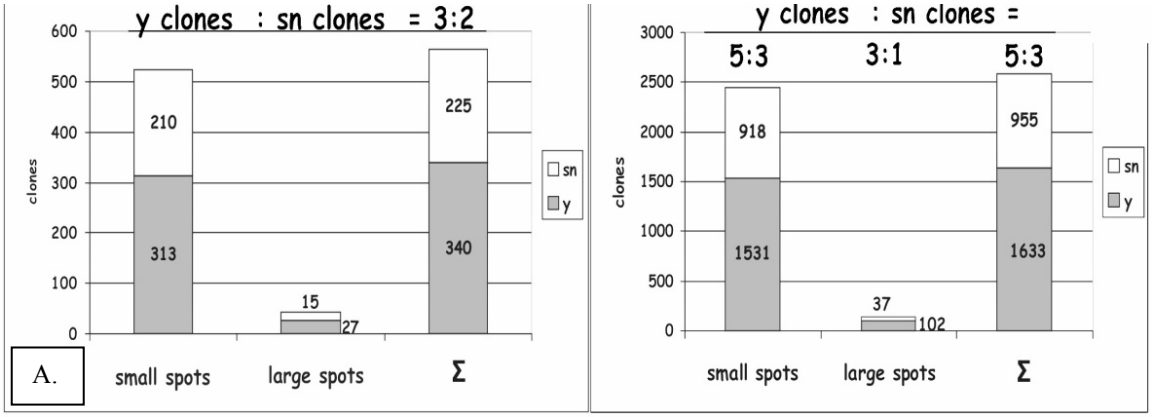
2. This effect was observed in two *Drosophila* species, *D. melanogaster* and *D. simulans*, and within a species it is true for different alleles of *white*, *yellow*, and *singed*.

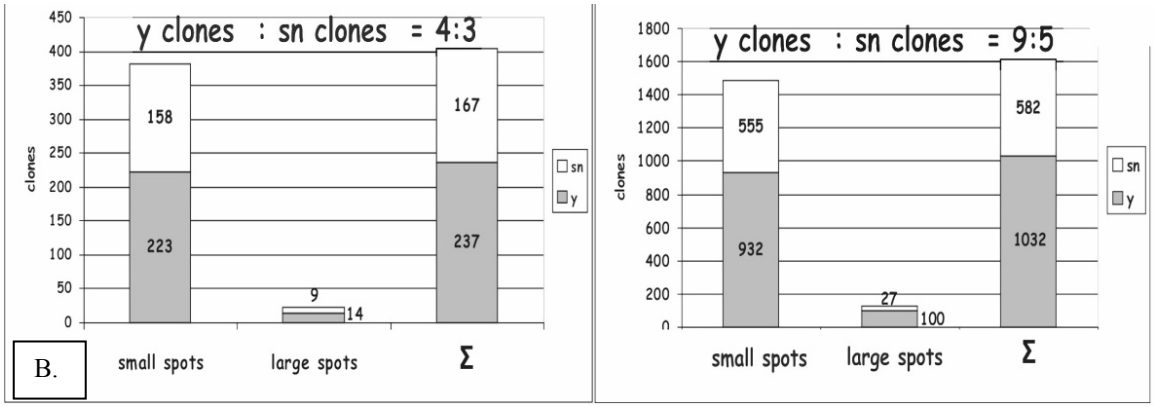
3. The effect is true for different types of mutagenesis – spontaneous, chemical, γ-ray, and genetic instability factor-induced.

4. The manifestation of the effect depends on the developmental stage at which the clones were induced. The effect is clear for later induced (smaller) clones, in this case the proportion of

white-homozygous to *white*$^{+}$-homozygous clones (both marked with either *yellow* or *singed*) is far from 1:1 (up to 4:1). However, for early (large) clones, the proportion of *y* and *sn* clones is near 1:1. As the large clones typically originate early in the development, and most of the small clones originate later, the same regularity is applicable for large and small clones as well.

5. The effect extent depends on the applied dose of mutagen. The stronger was the mutagen, the higher preponderance of white homozygous clones over *white*$^{+}$ homozygous twins we observed.





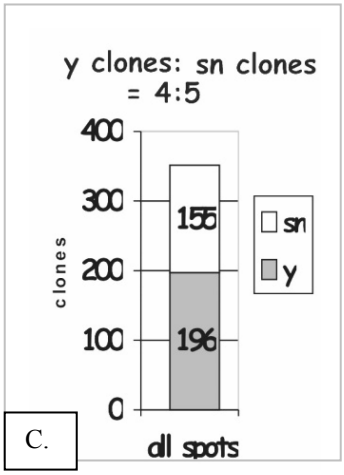


Figure 2. (*y w*) clones dominate over their (+ *sn*) twins. 2A. Spontaneous mosaicism (left panel) and *H*-factor-induced mosaicism (right panel) in the same *D. simulans y w* + / + + *sn*, *1H*$^{+}$ flies. Sample size is 60796 females. 2B. Spontaneous mosaicism (left panel) and *H*-factor-induced mosaicism (right panel) in the same *D. simulans y w* + / + + *sn*, *2H*$^{+}$ flies. Sample size is 20045 females. 2C. Oxoplatin-induced mosaicism in *D. melanogaster y w* + / + + *sn* flies. Sample size is 1250 females.

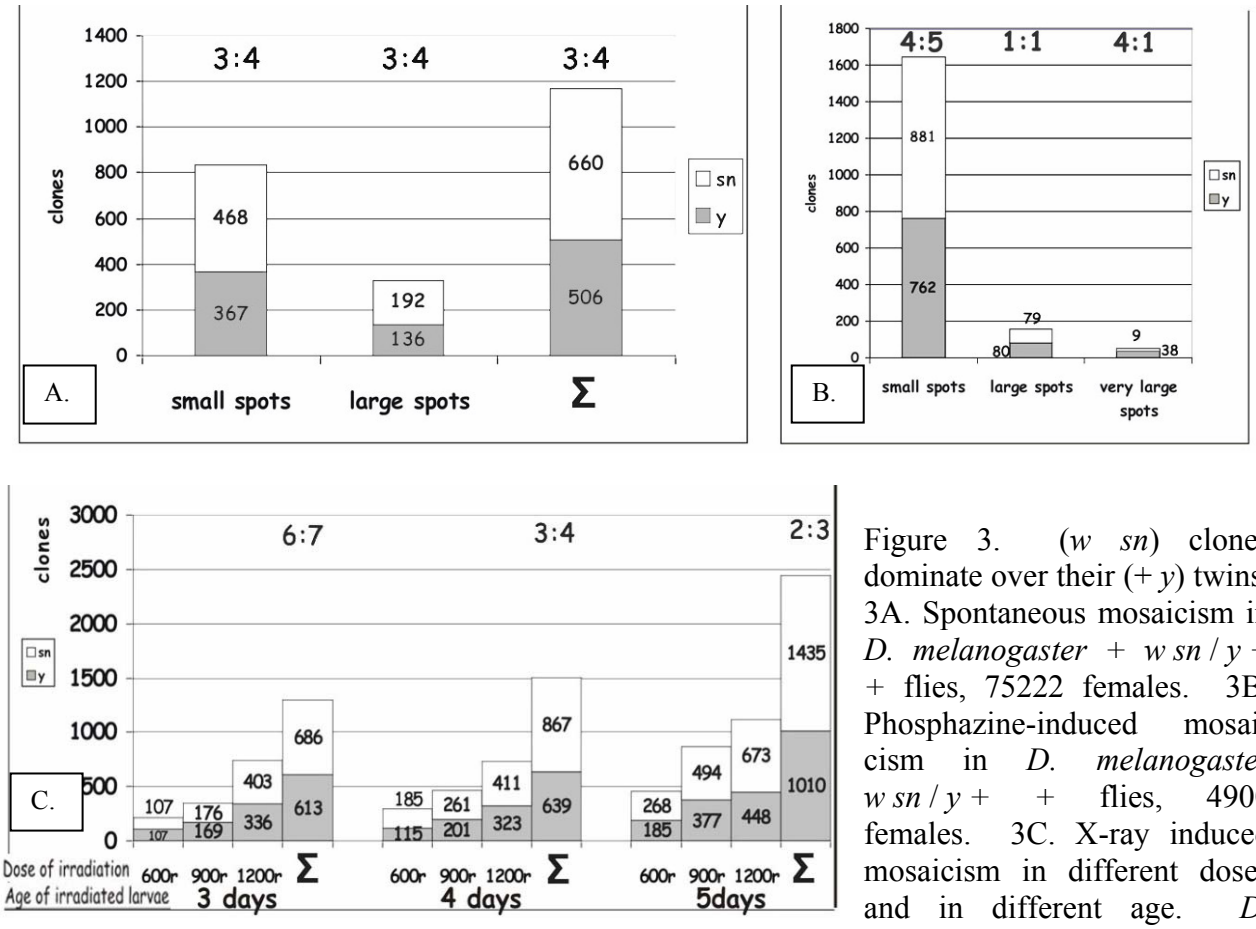


Figure 3. (*w sn*) clones dominate over their (+ *y*) twins. 3A. Spontaneous mosaicism in *D. melanogaster* + *w sn* / *y* + + flies, 75222 females. 3B. Phosphazine-induced mosaicism in *D. melanogaster* *w sn* / *y* + + flies, 4900 females. 3C. X-ray induced mosaicism in different doses and in different age. *D. melanogaster*, 15600 females

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