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The authors retract the above-referenced manuscript on the basis of the fact that one of the major conclusions, regarding the function of paramyosin in myoblast fusion, is incorrect. The authors stand by all of the other data with the exception of one correction. See below for details.

In this paper, a myoblast fusion defect was attributed to mutation of the *Drosophila melanogaster* paramyosin gene. We now show that the fusion defect instead apparently arises from the *TM3* balancer chromosome. Experiments subsequent to publication using a *TM3*-containing line that lacked a paramyosin mutation suggested that we had misidentified balancer chromosome–containing individuals before the late embryonic stage as paramyosin mutant homozygotes (*prm^1/prm^1*). By using a lacZ-expressing version of *TM3*, staining experiments unambiguously identified young *prm^1* homozygotes and showed them all to lack the fusion defect, implying that the fusion defect is due to the presence of the *TM3* chromosome. We have not tested whether this is a homozygous or heterozygous effect of *TM3*. The apparent rescue of the fusion defect by the wild-type paramyosin transgene is presumably due to the absence of the *TM3* chromosome, instead of the presence of the paramyosin transgene. We conclude that embryos identified as *prm^1* homozygotes in Figs. 3 and 4 of the paper, as well as on the cover of the journal, are not of this genotype. We thus retract the conclusion that paramyosin is important for myoblast fusion. In contrast, *prm^1/prm^1* late-stage embryos and larvae were unambiguously identified by mouth hook color, and so our conclusion regarding myofibril defects in *prm^1* homozygotes remains valid.

As a result of a PCR artifact and an error in notation, the deletion in the *prm^1* allele was stated to be 4 kb in size. Reanalysis of the data as well as additional cloning and sequencing indicate that the deletion is actually 1.05 kb in length and is internal to the *P* element. The deleted region encompasses portions of the upstream region and first exon of the *$white^+$* gene, removing 163 codons. Thus, the paramyosin coding region itself is not disrupted in either *prm^1* or its parent line and both contain a *P* element insert in the transcribed, but not translated, region of *prm* exon 1. Since the normal *prm* transcription start site is upstream of the *P* element, *prm* transcripts likely are produced by transcription from the element into the *prm* coding region or by use of a cryptic transcription start site in the 174 bp between the insertion site and the coding region. Internal deletion of a portion of the *P* element in *prm^1* may reduce transcriptional read through or initiation, accounting for reduced expression compared with its parent line. Some internally deleted *P* elements have stronger mutant phenotypes than their undeleted parental element (Staveley et al., 1995; Hodgetts and O'Keefe, 2001).

The authors stand by the data as to the location of the *P* element disrupting the paramyosin gene (Fig. 1), the effects of *prm^1* and rescue on paramyosin and miniparamyosin expression (Fig. 2), the expression pattern detected by the anti-paramyosin antibody (Figs. 5 and 6), the myofibrillar defects found in *prm^1* homozygotes and the rescue of these defects (Figs. 7, 8, and 9). However, the editors require retraction of the entire paper, and it is consequently marked as such.

The authors sincerely regret these errors and appreciate the opportunity to correct them. We are indebted to Qin Yu, William Kronert, and Jennifer Suggs (San Diego State University, San Diego, CA) for their insights and for carrying out the experiments reported here.

References:

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