

<https://doi.org/10.1126/science.aba7178>

Erratum for the Research Article: “Loci associated with skin pigmentation identified in African populations” by Nicholas G. Crawford, D. E. Kelly, M. E. B. Hansen, M. H. Beltrame, S. Fan, S. L. Bowman, E. Jewett, A. Ranciaro, S. Thompson, Y. Lo, S. P. Pfeifer, J. D. Jensen, M. C. Campbell, W. Beggs, F. Hormozdiari, S. W. Mpoloka, G. G. Mokone, T. Nyambo, D. Wolde Meskel, G. Belay, J. Haut, NISC Comparative Sequencing Program, H. Rothschild, L. Zon, Y. Zhou, M. A. Kovacs, M. Xu, T. Zhang, K. Bishop, J. Sinclair, C. Rivas, E. Elliot, J. Choi, S. A. Li, B. Hicks, S. Burgess, C. Abnet, D. E. Watkins-Chow, E. Oceana, Y. S. Song, E. Eskin, K. M. Brown, M. S. Marks, S. K. Loftus, W. J. Pavan, M. Yeager, S. Chanock, S. A. Tishkoff

Abstract

In the Research Article “Loci associated with skin pigmentation identified in African populations,” the authors made the inaccurate conclusion that *mfsd12a* targeting in zebrafish disrupts xanthophore function. This happened because of a misinterpretation of experimental observations, not from errors in data collection or reporting. In the original experiments, a loss of normal methylene blue staining occurred in 100% of *mfsd12a*^{-/-} embryos at 5 days postfertilization. These *mfsd12a*^{-/-} embryos were derived from matings between mosaic F0 founder fish (*mfsd12a* targeted mosaic F0 x *mfsd12a* targeted mosaic F0). The single guide RNAs for *mfsd12a* were extremely efficient, and no wild-type F1 offspring were obtained from the F0 inbreeding. Therefore, the clutches of 100% *mfsd12a*^{-/-} embryos were compared with embryos derived from matings of wild-type fish (*mfsd12a*^{+/+} x *mfsd12a*^{+/+}) in which loss of methylene blue staining was not observed. In each case, multiple clutches were analyzed from independent matings. Additionally, the wild-type breeders originated from the same colony of TAB5 fish that were used to generate fertilized eggs for Cas9 injections to generate the *mfsd12a* F0 founders. These reproducible results led the authors to conclude that *mfsd12a* was required for normal methylene blue staining of xanthophores. However, in subsequent work, the authors have observed a lack of methylene blue staining in clutches originating from other matings within their facility. They have confirmed that this lack of methylene blue staining segregates with a population variant linked to chromosome 12 within their TAB5 colony. This variant was likely present at a high-enough frequency in their *mfsd12a*-targeted F0 founder fish to affect the outcome. These recent data thus do not support their previous conclusion that *mfsd12a* functions in zebrafish pigmentation (presented in Fig. 7, A and B, of the original manuscript). Importantly, however, it does not alter the main conclusions of the manuscript that MFSD12 is associated with mammalian pigment variation in both human and mouse. The sections of the results referring to the zebrafish knockout have been removed entirely, along with the accompanying figure and cited references. Both the HTML and PDF versions of the text have been corrected.