POACHING IMPACTS

Ivory poaching and the rapid evolution of tusklessness in African elephants

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Understanding the evolutionary consequences of wildlife exploitation is increasingly important as harvesting becomes more efficient. We examined the impacts of ivory poaching during the Mozambican Civil War (1977 to 1992) on the evolution of African savanna elephants (*Loxodonta africana*) in Gorongosa National Park. Poaching resulted in strong selection that favored tusklessness amid a rapid population decline. Survey data revealed tusk-inheritance patterns consistent with an X chromosome–linked dominant, male-lethal trait. Whole-genome scans implicated two candidate genes with known roles in mammalian tooth development (*AMELX* and *MEP1a*), including the formation of enamel, dentin, cementum, and the periodontium. One of these loci (*AMELX*) is associated with an X-linked dominant, male-lethal syndrome in humans that diminishes the growth of maxillary lateral incisors (homologous to elephant tusks). This study provides evidence for rapid, poaching-mediated selection for the loss of a prominent anatomical trait in a keystone species.

The selective killing of species that bear anatomical features such as tusks and horns is the basis of a multibillion-dollar illicit wildlife trade (*1*) that poses an immediate threat to the survival of ecologically important megafauna worldwide (*2*, *3*). Megaherbivores are especially vulnerable to overharvesting because of their large habitat requirements, small population sizes, and long generation times (*4*, *5*). As ecosystem engineers, these species also behaviorally regulate ecological processes (*5*–*8*); anthropogenic selection on phenotypes that influence these behaviors may, therefore, have cascading effects on ecosystem functioning. However, most work that details human-driven selection has focused on smaller species in which evolutionary change is more readily studied (*9*, *10*). It remains unclear to what extent, at what rates, and through what mechanisms harvest-induced phenotypic change occurs in the world's largest land animals.

Warfare is associated with intensified exploitation and population declines of wildlife throughout Africa (*11*), and organized violence has long been intertwined with the ivory trade (*12*–*14*). In Gorongosa National Park, the Mozambican Civil War (1977 to 1992) reduced large-herbivore populations by >90% (*15*), and armies on both sides of the conflict targeted elephants for ivory (*15*, *16*). Intensive poaching in Africa has been associated with an increase in the frequency of tuskless elephants, exclusively (or nearly so) among females (table S3). No record of tuskless male elephants within Gorongosa National Park exists (table S2). Analyses of historical video footage and contemporary sighting data (supplementary materials) show that the precipitous decline of the Gorongosa elephant population was accompanied by a nearly threefold increase in the frequency of tuskless females, from 18.5% (n = 52) to 50.9% (n = 108) (two-sample equality of proportions test with continuity correction, $P < 0.001$) (Fig. 1A).

To test whether the increased frequency of female tusklessness was a chance event associated with the severe population bottleneck (*17*), we simulated the observed population decline in Gorongosa from 1972 (n = 2542 individuals) to 2000 (n = 242) (*15*) under a scenario of equal survival probabilities for tusked and tuskless females (see methods). On the basis of these simulations, the observed increase in tusklessness is extremely unlikely to have occurred in the absence of selection (hypergeometric distribution, $P = 1.8 \times 10^{-15}$) (Fig. 1B). The relative survival of tuskless females across this 28-year period was estimated to be more than five times that of tusked individuals (maximum-likelihood estimate = 5.13, 95% confidence interval 3.98 to 6.60) (Fig. 1C). Thus, we conclude that the population bottleneck in Gorongosa was accompanied by strong selection favoring the tuskless phenotype.

If there were strong selection against tusked elephants, we might also observe divergent genomic signatures of population-size change between the two tusk morphs. We sequenced whole genomes from blood samples of 18 female elephants (n = 7 tusked, 11 tuskless). We mapped sequence reads to the annotated African savanna elephant genome (Loxafr3.0) and generated alignments with ~30× coverage for 13 samples and 14× coverage for 5 samples (supplementary materials). Using the 30× coverage samples (n = 6 tusked, 7 tuskless), we calculated Tajima's D (*18*) genome-wide in nonoverlapping 10-kb windows. Both groups displayed a slight excess of rare variants, indicated with negative D values (tuskless: −0.27, tusked: −0.2). However, tusked samples had significantly fewer rare variants than tuskless samples (Welch's two-sample t test: $P < 0.0001$) (Fig. 1D and supplementary materials), which is consistent with a more severe population contraction of tusked individuals.

To evaluate the evolutionary response to selection, we quantified the frequency of tusk phenotypes among adult females born after the war (estimated birth years 1995 to 2004). We found that tusklessness among female offspring of survivors (33%, n = 91) remained significantly elevated over the pre-conflict proportion (18.5%, two-sample equality of proportions test with continuity correction, $P = 0.046$) (Fig. 1A) and was greater than expected in the absence of selection (hypergeometric distribution, $P = 4.3 \times 10^{-8}$) (Fig. 1B). These results indicate a heritable genetic basis for tusklessness and an evolutionary response to poaching-induced selection in Gorongosa.

Given the evidence for heritability and female-specificity of tusklessness in Gorongosa, we hypothesized that the phenotype is genetically inherited through a sex-linked locus (*17*, *19*–*21*). We therefore searched for a pattern of inheritance that could explain the observed variation in tusk morphology. Phenotypes displaying extreme female bias are commonly attributed to X chromosome–linked dominant inheritance with male lethality (*22*). Accordingly, we used mother-offspring phenotype surveys in Gorongosa to test the a priori hypothesis that tusklessness is an X-linked dominant, male-lethal trait governed by a single locus. Under this hypothesis, we expect two-tusked females (X_+X_+) and males (X_+Y) to carry only the unaffected allele (X_+). As such, X_+X_+ mothers should exclusively produce X_+X_+ daughters. Furthermore, tuskless females should appear only in the heterozygous state (X_+X_-) owing to male lethality (females would always inherit the unaffected allele from X_+Y fathers); thus, X_+X_- mothers should produce daughters with a 1:1 ratio of X_+X_- and X_+X_+ phenotypes, and only 50% of male offspring (X_+Y) conceived by X_+X_- mothers should be viable. As a result, two-thirds of offspring born to X_+X_- mothers should be female, assuming that all

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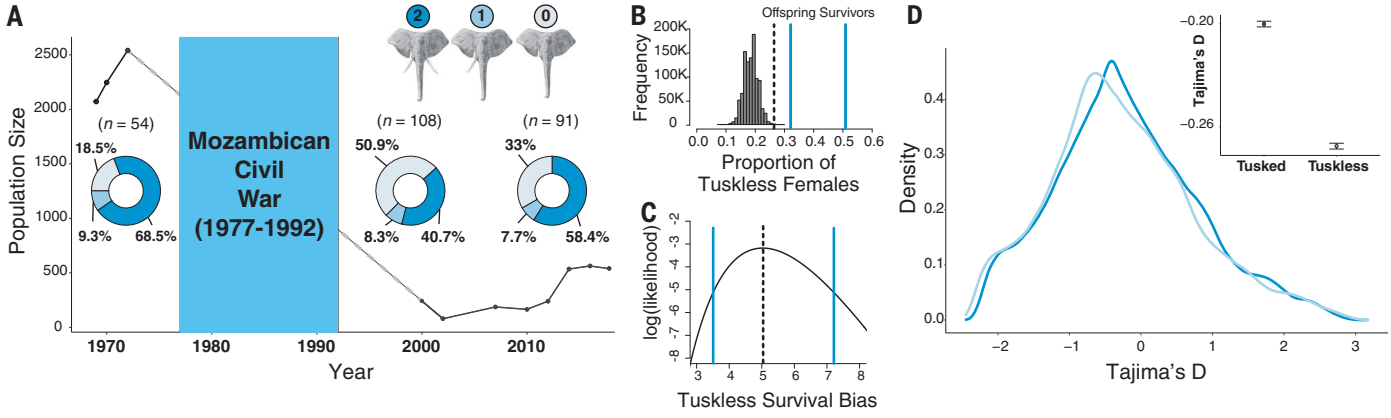

Fig. 1. Demographic shifts during the Mozambican Civil War and evidence of selection for tusklessness in Gorongosa National Park. (**A**) Change in population size and tusk morphology. The line depicts the minimum elephant population size in Gorongosa National Park by year from aerial censuses (*30*); the dashed segment indicates a period for which no robust census data are available. The pie charts show the proportion of two-tusked (dark blue), single-tusked (blue), and tuskless (light blue) females observed prewar (n = 54), in survivors of the war (n = 108), and in the first generation born postwar (n = 91) on the basis of contemporary surveys and historical photos. Individuals for which tusk morphology could not be accurately determined are not represented. (**B**) Simulated samples from a Wallenius distribution of the proportion of female tusklessness expected after the population bottleneck, assuming no selection for tusklessness. Solid vertical lines indicate the observed proportions of tuskless females among survivors of the war and the first postwar generation (born between 1995 and 2004). The dashed line shows the upper 1% quantile of the simulated distribution. (**C**) Log(likelihood) of the increased survivorship of tuskless females observed in Gorongosa, given a range of survival odds of tuskless relative to tusked females. The x axis represents the odds ratio of survival for tuskless individuals compared with that of their tusked counterparts. The dotted line shows the maximum-likelihood estimate; solid blue lines indicate the 95% confidence interval. (**D**) Density plot of genome-wide estimates of Tajima's D for two-tusked (dark blue) and tuskless (light blue) morphs, calculated in 10-kb windows. (Inset) Mean ± 1 SE of genome-wide D for each tusk morph.

$X\_$ carriers display the phenotype (i.e., complete penetrance).

We found that 91.3% of daughters born to two-tusked mothers carried two tusks (n = 21 two-tusked, 1 tuskless, 1 one-tusked) (Fig. 2A). By contrast, the daughters of tuskless mothers displayed approximately equal proportions of tusked and tuskless phenotypes [n = 19 two-tusked (40.9%), 21 tuskless (44.7%), two-sample equality of proportions test with continuity correction, P = 0.42] (Fig. 2A). The mothers of both tusk morphs were observed with daughters displaying an intermediate one-tusked phenotype [two-tusked mothers: n = 1 (4.3%), tuskless mothers: n = 7 (14.9%), equality of proportions test P = 0.37]. Tuskless mothers also displayed a biased offspring sex ratio (≤5 years old, n = 67); 65.7% were female, which differs significantly from the null hypothesis of equal sex ratios (exact binomial test, P = 0.027) and is statistically indistinguishable from the 66.7% female bias expected under complete male lethality (P = 0.90) (Fig. 2B). We found no evidence for sex bias among tusked females (54.2% female offspring, n = 48, P = 0.67) (Fig. 2B), and previous research has shown no general sex-biased birth in African elephants (*23*), which suggests that the observed skew in offspring sex ratio is correlated with expression of the tuskless phenotype.

Altogether, 87.1% of mother-offspring phenotypic associations were consistent with a single-locus X-linked dominant model of inheritance, and the sex bias associated with tusklessness was within 1% of that expected under complete male lethality. The unexplained variability in the trait, including the presence of unilateral tusklessness (if genetic), suggests that epistatic interactions between at least two loci may influence the expression of tusk morphology. Genotype-phenotype relationships associated with variation in dental morphogenesis are known to be highly variable, and distinctive mutations have been identified as population or even family specific (*24*). Furthermore, epigenetic patterning and mosaic X-chromosome inactivation can result in the variable phenotypic expression of identical mutations associated with dental agenesis, even between monozygotic twins (*25*). Nevertheless, the Gorongosa data support the hypothesis that the tuskless phenotype is controlled by at least one X-linked dominant, male-lethal locus of large effect, with possible additional modifier loci affecting phenotypic expression of the trait.

We used whole-genome data to identify the putative major-effect locus underpinning the hypothesized X-linked dominant inheritance of the tuskless phenotype and searched for signatures of selection associated with recent intensive poaching. We analyzed genomes to identify the strongest candidate loci for selection on a sex-biased, male-lethal trait. We first conducted a genome-wide scan for loci that exhibit evidence of strong recent selection, specific to the tuskless phenotype, and focused on genomes of tuskless individuals (n = 11). Although tusklessness has not swept to fixation in the Gorongosa population (Fig. 1), we would expect strong signals of recent selection when analyzing genomes from the tuskless subgroup.

Using a sliding window analysis of 10-kb windows with a step size of 2 kb, we scanned for two signatures that we expected under the hypothesis of selection on an X-linked dominant, male-lethal trait. Using 30× coverage samples (6 tusked, 7 tuskless), we first searched for genomic regions that displayed excess heterozygosity within alleles private to the tuskless morph and quantified heterozygosity as deviations from Hardy-Weinberg equilibrium. We next searched for loci displaying highly correlated mutations among tuskless samples [measured as linkage disequilibrium (LD)]. We quantified LD by using single-nucleotide polymorphism (SNP) vectors (μLD), as implemented in the RAiSD (*26*) software.

Subsequently, we compared all tusked (n = 7) and tuskless (n = 11) genomes to search for

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patterns of genetic divergence between tusk morphs. Under the hypothesized model of inheritance, we expected the genomes of tusked individuals to lack the specific mutation(s) causing tusklessness. Tusked individuals should also be less likely to harbor SNPs that are linked to the causal mutation(s). We quantified genetic differentiation by using both normalized differences in allele frequency, F_{ST} (*27*), and the average number of pairwise differences, D_{XY} (*28*). Genomic windows that were significant outliers (above the 95% quantile of the genome-wide distribution) for all four of these summary statistics (heterozygosity, LD, F_{ST}, and D_{XY}) were considered candidate loci for selection on tusklessness. The overlap of outliers across these four statistics revealed 305 candidate windows (Fig. 2C).

If tusklessness is an X-linked dominant trait, then the major-effect locus should reside on the X chromosome. We therefore filtered the 305 candidate windows for those located on the X chromosome and found 8 windows that fell within two contiguous genomic intervals. Five of these windows overlap an ~100-kb region that contains the X-linked isoform of amelogenin (*AMELX*) (Fig. 2D), which encodes an extracellular matrix protein involved in biomineralization of enamel and putatively regulates periodontium formation and cementum-associated genes (*29*, *30*). Several mutations within this locus are associated with enamel hypomineralization and tooth brittleness in humans (*30*).

A genomic deletion in the syntenic region of the human X chromosome (Xp22.2), which encompasses *AMELX* and several adjacent genes, results in amelogenesis imperfecta accompanied by an X-linked dominant, male-lethal syndrome (*31*). In such cases, women display several craniofacial abnormalities, including microdonty and/or agenesis of the maxillary lateral incisors (*31*), which are homologous to elephant tusks. Notably, skewed X-chromosome inactivation in amelogenesis imperfecta contributes to pronounced phenotypic variation in heterozygotes (*32*). Previous studies have shown that male survival can be rescued in mice engineered with a deletion in this region by forced expression of the human holocytochrome c-type synthetase (*HCCS*) gene, which lies directly adjacent to *AMELX* (*33*). The high degree of LD that we observed across this region of the tuskless elephant X chromosome (Fig. 2D and fig. S1) suggests that physical proximity between *AMELX* and neighboring male-lethal loci may underlie the inferred association between tusklessness and male lethality in the Gorongosa population. The remaining three candidate windows of the X chromosome encompass the unprocessed pseudogene *FAM115B* and an adjacent intergenic region. We have found no known link between *FAM115B* and odontogenesis or craniofacial development.

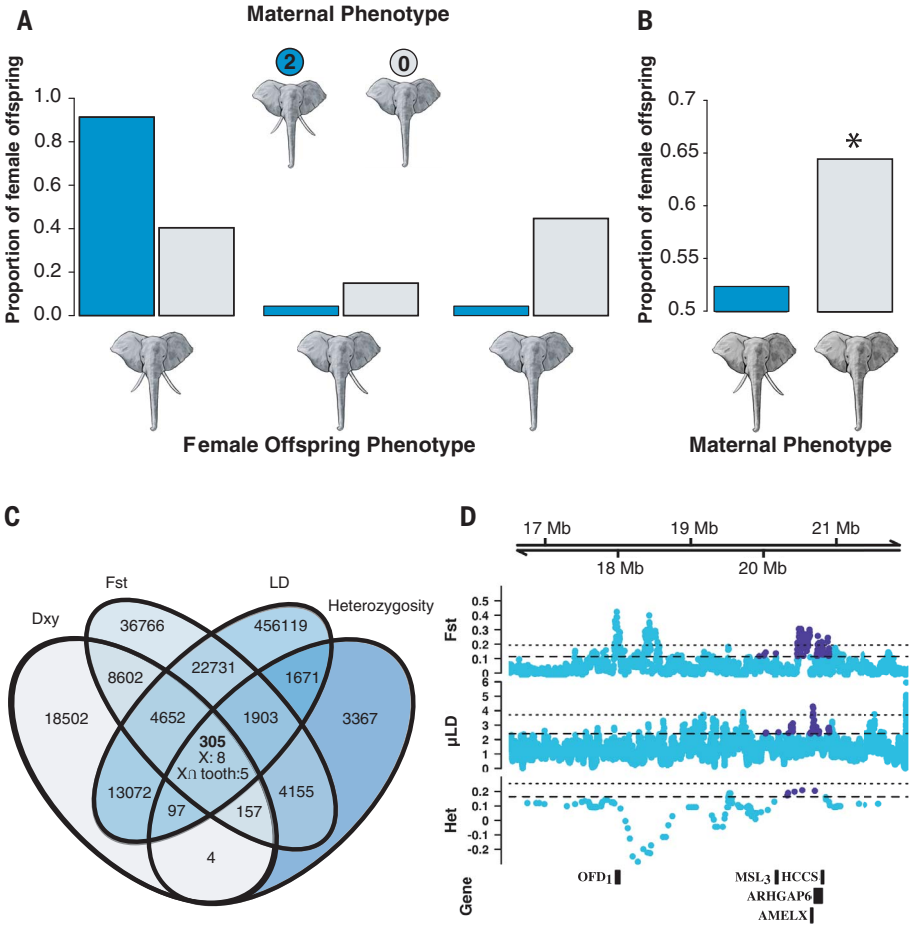

Fig. 2. Evidence for X-linked dominant inheritance with male lethality and an underpinning candidate locus. (**A** and **B**) Inference of inheritance patterns from population offspring survey. (A) Observed phenotypic distribution of female offspring with two-tusked (dark blue) and tuskless (light blue) mothers. (B) Observed sex ratio of offspring for tusked and tuskless females. Asterisk indicates significant deviation from a 1:1 sex ratio among tuskless mothers (binomial test, $P = 0.027$) but not tusked mothers ($P = 0.67$). (**C** and **D**) Evidence for selection on sex-linked candidate locus. (C) Venn diagram of four summary statistics computed in sliding windows across the genome, showing the numbers of overlapping windows in the 5% tails of each statistical distribution. Summary statistics include genetic differentiation between tusked and tuskless samples (F_{ST} and D_{XY}), along with LD and deviation in heterozygosity from Hardy-Weinberg equilibrium ("Heterozygosity") within tuskless samples. Included in the four-way intersection are the number of windows on the X chromosome and the subset of these that overlap known tooth genes (X ∩ tooth). (D) Magnified Manhattan plots of F_{ST}, LD, and heterozygosity (Het) show the genomic location of five X ∩ tooth windows that are contiguous and overlap *AMELX* and flanking regions. Dashed and dotted lines indicate upper 5% and 1% quantiles, respectively. Dark blue dots represent outlier windows ($P < 0.05$) within the candidate region. Light blue dots represent nonoutlier windows. The candidate region contains 23 genes, but for clarity, only genes known to be involved in tooth development and/or male lethality are labeled.

Given the potential influence of epistasis on expression of tusk morphology in Gorongosa, we conducted additional genome-wide scans for regions displaying extreme genetic divergence between tusk morphs (F_{ST} or D_{XY}, $P < 0.001$) (Fig. 3A) and low genetic diversity specific to the tuskless morph (relative diversity = $\pi_{tuskless} - \pi_{tusked}$, $P < 0.001$. Three contiguous genomic windows met these two criteria (F_{ST} $P < 0.001$, relative diversity $P < 0.001$) (Fig. 3B). This region overlaps with a single autosomal gene on chromosome 1: *MEP1a*. This gene encodes

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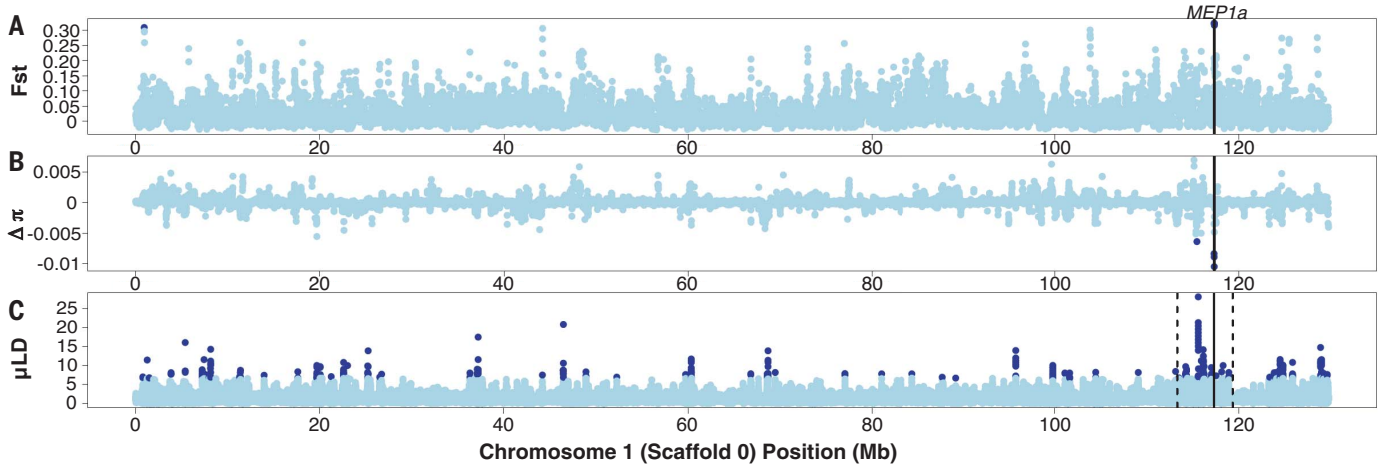

Fig. 3. Autosomal candidate locus for tusklessness in Gorongosa elephants. (**A** to **C**) Scaffold 0 within African elephant chromosome 1, which highlights a candidate locus for tusklessness, *MEP1a*. (A) Genetic divergence (F_{ST}) between two-tusked and tuskless morphs. (B) Relative diversity ($\Delta\pi = \pi_{tuskless} - \pi_{two\text{-}tusked}$) among tuskless individuals. Low values represent regions with low relative genetic diversity in tuskless morphs. (C) Linkage disequilibrium quantified by using SNP vectors (μLD) among tuskless samples. Extreme outliers in each distribution ($P < 0.001$) are indicated with dark blue dots. Light blue dots indicate nonextreme values. Solid vertical lines represent the position of *MEP1a* along chromosome 1 of the elephant genome. Dashed lines represent the boundaries of the surrounding region that displays a high density of elevated LD.

meprin subunit alpha, a matrix metalloprotein that plays an important role in dentin mineralization by processing a precursor, dentin sialophosphoprotein (DSPP). Abnormalities in DSPP are associated with several odontogenic disorders, including dentin dysplasia, which results in malformation of the tooth root and premature tooth loss (*34*). *MEP1a*$^{-/-}$ mice display significant alterations in dentin bone mineral density (*35*). These three windows are nested within an extended genomic interval (~6 Mb) that displays elevated LD ($P < 0.001$) (Fig. 3C and fig. S2), which suggests recent positive selection across this region. Together, *AMELX* and *MEP1a* have functional associations with the development of several distinct regions of the mammalian tooth, including enamel, dentin, cementum, and periodontium (Fig. 4). However, analyses of divergent polymorphisms and structural variants (including deletions, duplications, and copy number variants) between tusk morphs did not reveal obvious causal genetic variants for either locus (supplementary materials).

In summary, human-mediated selection for tusklessness during the Mozambican Civil War appears to be driven by recent selection on at least one X-linked locus (*AMELX*) and one autosomal locus (*MEP1a*). Physical linkage between *AMELX* and proximate male-lethal loci on the X chromosome, such as *HCCS* (*31*, *33*), may underpin the proposed X-linked dominant, male-lethal inheritance of tusklessness in the Gorongosa population. If our interpretation is correct, this study represents a rare example of human-mediated selection favoring a female-specific trait despite its previously unknown deleterious effect in males (sexually antagonistic selection). Given the timeframe of selection, speed of evolutionary response, and known presence of the selected phenotype before the selective event, the selection of standing genetic variation at these loci is the most plausible explanation for the rapid rise of tusklessness during this 15-year period of conflict. However, the exact genetic and developmental mechanisms leading to tusklessness and/or male nonviability remain unresolved. Although tuskless males do not occur in Gorongosa or in surveys of large sample sizes from Africa's most intensively studied elephant populations (*17*, *21*, *36*, *37*), there are anecdotal reports of tuskless males in several locations (*20*, *38*, *39*). We are unaware

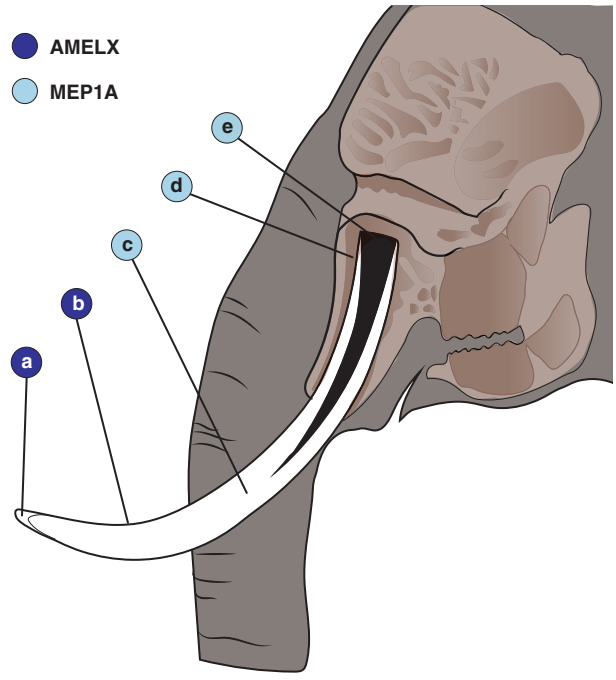

Fig. 4. Putative functional effects of candidate loci on tusk morphology. A cross section of an African elephant tusk shows the anatomical position of (a) enamel, (b) cementum, (c) dentin (ivory), (d) periodontium, and (e) root of the tusk. Dark blue circles indicate regions known or proposed to be affected by candidate gene *AMELX*. Light blue circles are proposed to be affected by candidate gene *MEP1a*. Neither gene is known to affect the formation of the dental pulp (black interior of cross section).

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of any study that has firmly established a frequency of tuskless males beyond what could plausibly be explained by rare injuries or observer error (supplementary text and table S3), but we cannot rule out the possibility of alternative genetic mechanisms and/or genotype–environment interactions. Furthermore, intermediate single-tusked phenotypes commonly co-occur in family groups that also include bilaterally tuskless females (*17*, *20*, *37*). Although the evidence from Gorongosa is consistent with an X-linked dominant, male-lethal trait, continent-wide patterns of tusk expression and heritability may be the result of geographic variation in LD between *AMELX* and adjacent male-lethal loci, additional loci elsewhere in the species' genome, individual variation in patterns of X-chromosome inactivation, or some entirely different genetic mechanism. Further study is needed to establish the exact number and identity of causal variants that encode tusklessness, and comparative studies across multiple populations will be necessary to reveal the geographic structure of genetic variation and inheritance underlying the trait.

Social conflict and commercial harvest can intertwine to devastate animal populations (*11*, *40*). However, most known instances of harvest-induced evolutionary change occur gradually over longer time periods, and the selective effects of harvest can be difficult to disentangle from other factors (*9*, *41*–*44*). Our study shows how a sudden pulse of civil unrest can cause abrupt and persistent evolutionary shifts in long-lived animals even amid extreme population decline. In Gorongosa, recovery of both elephant abundance and ancestral tusk morphology may be crucial for ecosystem restoration. Elephant tusks are multipurpose tools that are used for excavating subterranean food and minerals (*45*, *46*) and gouging and peeling bark, which can kill trees (*47*, *48*). These behaviors can catalyze forest-to-grassland transitions at large scales (*45*) and create habitat for other species at local scales (*49*, *50*). Accordingly, a population-wide increase in tusklessness may have downstream impacts such as reduced bioturbation, shifts in plant species composition, reduced spatial heterogeneity, and increased tree cover—any of which could affect myriad other ecosystem properties. Elsewhere, evolution in species that perform key ecological functions has exerted potent effects on food-web structure, community composition, and nutrient transport (*51*, *52*). Restoration of these functions may require disproportionately longer time scales than the initial selection event (*44*) and may thus constrain the pace of rewilding efforts. Understanding the dynamics of rapid evolution in the Anthropocene is therefore essential, not only for revealing the biological impacts of contemporary human activities but also for designing strategies to mitigate them.

REFERENCES AND NOTES

1. S. K. Wasser *et al*., *Sci. Adv.* **4**, eaat0625 (2018).
2. S. Wasser *et al*., *Science* **327**, 1331–1332 (2010).
3. D. Biggs, F. Courchamp, R. Martin, H. P. Possingham, *Science* **339**, 1038–1039 (2013).
4. M. Cardillo *et al*., *Science* **309**, 1239–1241 (2005).
5. W. J. Ripple *et al*., *Sci. Adv.* **1**, e1400103 (2015).
6. J. J. Midgley, D. Balfour, G. I. Kerley, *S. Afr. J. Sci.* **101**, 213–215 (2005).
7. G. P. Asner *et al*., *Proc. Natl. Acad. Sci. U.S.A.* **106**, 4947–4952 (2009).
8. M. S. Waldram, W. J. Bond, W. D. Stock, *Ecosystems* **11**, 101–112 (2008).
9. C. T. Darimont *et al*., *Proc. Natl. Acad. Sci. U.S.A.* **106**, 952–954 (2009).
10. V. Fugère, A. P. Hendry, *Proc. Natl. Acad. Sci. U.S.A.* **115**, 10070–10075 (2018).
11. J. H. Daskin, R. M. Pringle, *Nature* **553**, 328–332 (2018).
12. I. Douglas-Hamilton, *Oryx* **21**, 11–24 (1987).
13. R. L. Beyers *et al*., *PLOS ONE* **6**, e27129 (2011).
14. V. Varun, T. Ewing, "Ivory's curse: The militarization and professionalization of poaching in Africa" (Born Free USA, 2014); www.bornfreeusa.org/campaigns/wildlife-trade/ivorys-curse-the-militarization-and-professionalization-of-poaching-in-africa-2014/.
15. M. E. Stalmans, T. J. Massad, M. J. S. Peel, C. E. Tarnita, R. M. Pringle, *PLOS ONE* **14**, e0212864 (2019).
16. J. O. J. Hatton, M. Couto, "Biodiversity and war: A case study of Mozambique" (Biodiversity Support Program, 2001).
17. A. M. Whitehouse, *S. Afr. J. Zool.* **257**, 249–254 (2002).
18. F. Tajima, *Genetics* **123**, 585–595 (1989).
19. J. H. Poole, in *The Ivory Trade and the Future of the African Elephant. Volume 2: Technical Reports* (Ivory Trade Review Group, 1989).
20. H. Jachmann, P. S. M. Berry, H. Imae, *Afr. J. Ecol.* **33**, 230–235 (1995).
21. G. Steenkamp, S. M. Ferreira, M. N. Bester, *J. S. Afr. Vet. Assoc.* **78**, 75–80 (2007).
22. D. Germain, A. Mehta, M. Beck, G. Sunder-Plassmann, in *Fabry Disease: Perspectives from 5 Years of FOS*, M. Atul, M. Beck, G. Sunder-Plassmann, Eds. (Oxford PharmaGenesis, 2006).
23. P. C. Lee, V. Fishlock, C. E. Webber, C. J. Moss, *Behav. Ecol. Sociobiol.* **70**, 337–345 (2016).
24. N. Chhabra, M. Goswami, A. Chhabra, *Med. Oral Patol. Oral Cir. Bucal* **19**, e112–e119 (2014).
25. M. Varela, M. J. Trujillo-Tiebas, P. García-Camba, *Eur. Arch. Paediatr. Dent.* **12**, 318–322 (2011).
26. N. Alachiotis, P. Pavlidis, *Commun. Biol.* **1**, 79 (2018).
27. K. E. Holsinger, B. S. Weir, *Nat. Rev. Genet.* **10**, 639–650 (2009).
28. M. Nei, J. C. Miller, *Genetics* **125**, 873–879 (1990).
29. H. L. Viswanathan *et al*., *J. Periodontol.* **74**, 1423–1431 (2003).
30. C. W. Gibson *et al*., *J. Biol. Chem.* **276**, 31871–31875 (2001).
31. G. M. Hobson *et al*., *Am. J. Med. Genet. A.* **149A**, 1698–1705 (2009).
32. X. Duan *et al*., *J. Dent. Res.* **98**, 870–878 (2019).
33. S. K. Prakash *et al*., *Hum. Mol. Genet.* **11**, 3237–3248 (2002).
34. S. Malik, S. Gupta, V. Wadhwan, G. P. Suhasini, *J. Oral Maxillofac. Pathol.* **19**, 110 (2015).
35. P. Arnold *et al*., *Cell Tissue Res.* **367**, 351–358 (2017).
36. S. R., J. Sarukhan, A. Whyte, R. Hassan, "Millennium Ecosystem Assessment: Ecosystems and human well-being" (Island Press, 2006).
37. C. J. Moss, *Elephant Memories: Thirteen Years in the Life of an Elephant Family* (William Morrow, 1988).
38. I. J. Whyte, A. Hall-Martin, *Pachyderm* **55**, 31–40 (2018).
39. E. Abe, *Pachyderm* **22**, 46–47 (1996).
40. J. S. Brashares *et al*., *Science* **345**, 376–378 (2014).
41. J. B. S. Haldane, *J. Genet.* **44**, 296–304 (1942).
42. K. H. Andersen, K. Brander, *Proc. Natl. Acad. Sci. U.S.A.* **106**, 11657–11660 (2009).
43. M. Heino, B. Díaz Pauli, U. Dieckmann, *Annu. Rev. Ecol. Evol. Syst.* **46**, 461–480 (2015).
44. F. W. Allendorf, J. J. Hard, *Proc. Natl. Acad. Sci. U.S.A.* **106** (suppl. 1), 9987–9994 (2009).
45. J. S. Weir, *J. Zool.* **158**, 293–310 (1969).
46. W. H. Elder, *Zool. Africana* **158**, 293–310 (1969).
47. A. H. K. Beuchner, H. C. Dawkins, *Ecology* **42**, 752–766 (1961).
48. L. D. Wing, I. O. Buss, *Wildl. Monogr.* **19**, 3–92 (1970).
49. R. M. Pringle, *Ecology* **89**, 26–33 (2008).
50. T. C. Coverdale *et al*., *Ecology* **97**, 3219–3230 (2016).
51. E. P. Palkovacs, M. T. Kinnison, C. Correa, C. M. Dalton, A. P. Hendry, *Evol. Appl.* **5**, 183–191 (2012).
52. K. B. Oke *et al*., *Nat. Commun.* **11**, 4155 (2020).

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SUPPLEMENTARY MATERIALS

science.org/doi/10.1126/science.abe7389
Materials and Methods
Supplementary Text
Figs. S1 and S2
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MDAR Reproducibility Checklist

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Ivory poaching and the rapid evolution of tusklessness in African elephants

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Lose the tusks

Harvest and poaching of wildlife have increased as the human population and our technology have grown. These pressures now occur on such a scale that they can be considered selective drivers. Campbell-Staton *et al*. show that this phenomenon has occurred in African elephants, which are poached for their ivory, during the 20-year Mozambican civil war (see the Perspective by Darimont and Pelletier). In response to heavy poaching by armed forces, African elephant populations in Gorongosa National Park declined by 90%. As the population recovered after the war, a relatively large proportion of females were born tuskless. Further exploration revealed this trait to be sex linked and related to specific genes that generated a tuskless phenotype more likely to survive in the face of poaching. —SNV

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