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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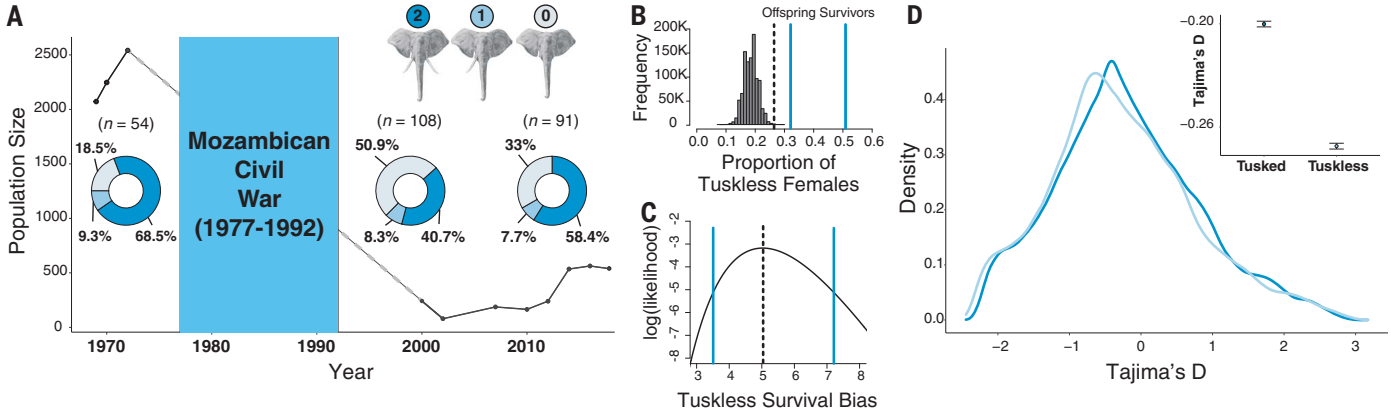

Fig. 1. Demographic shifts during the Mozambican Civil War and evidence of selection for tusklessness in Gorongosa National Park. (**A**) Change in population size and tusk morphology. The line depicts the minimum elephant population size in Gorongosa National Park by year from aerial censuses (*30*); 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

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.

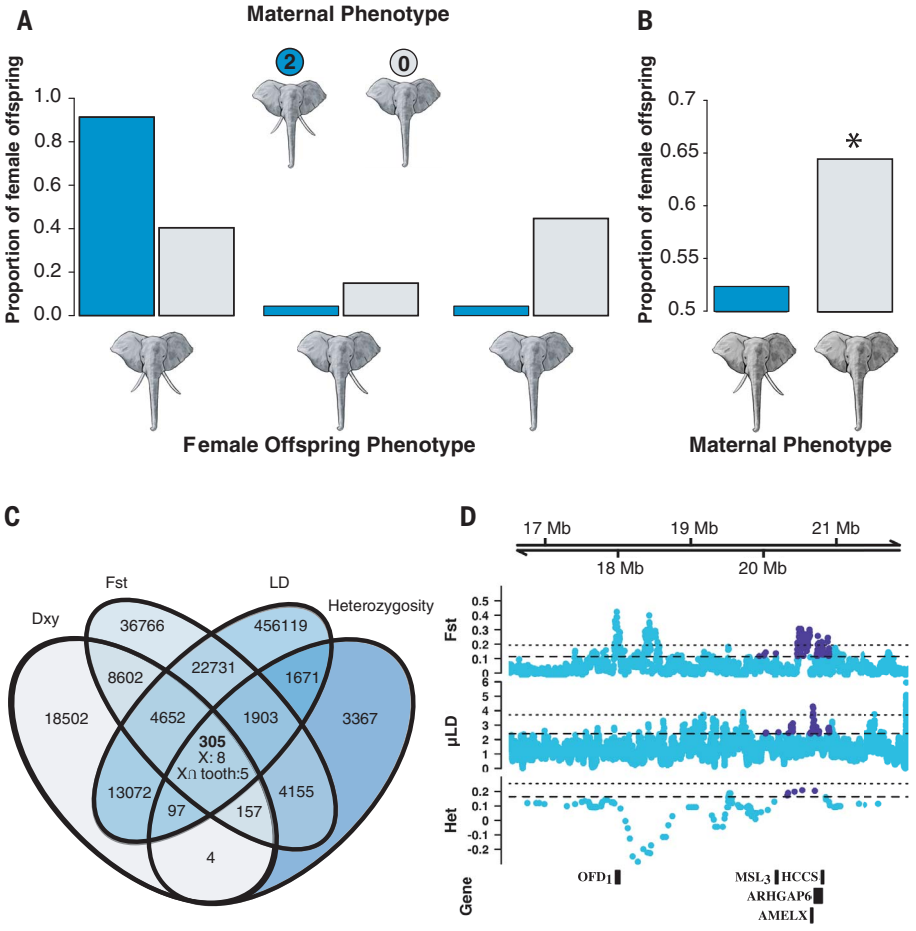

Fig. 2. Evidence for X-linked dominant inheritance with male lethality and an underpinning candidate locus. (**A** and **B**) Inference of inheritance patterns from population offspring survey. (A) Observed phenotypic distribution of female offspring with two-tusked (dark blue) and tuskless (light blue) 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

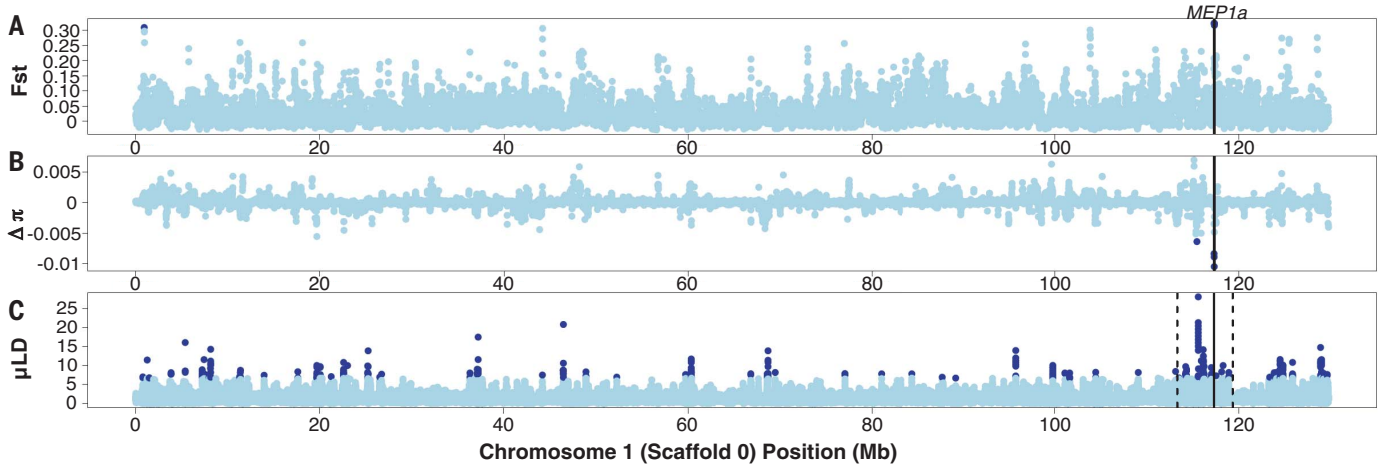

Fig. 3. Autosomal candidate locus for tusklessness in Gorongosa elephants. (**A** to **C**) Scaffold 0 within African elephant chromosome 1, which highlights a candidate locus for tusklessness, *MEP1a*. (A) Genetic divergence (F_{ST}) between two-tusked and tuskless morphs. (B) Relative diversity ($\Delta\pi = \pi_{tuskless} - \pi_{two\text{-}tusked}$) 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).

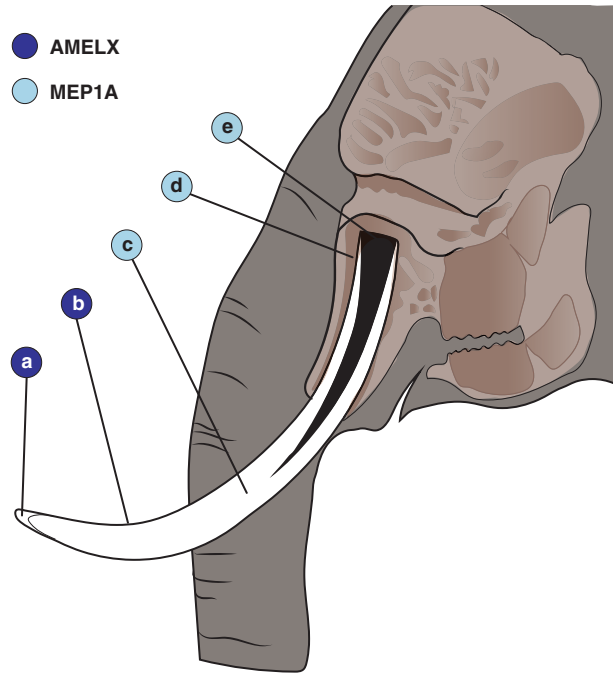

Fig. 4. Putative functional effects of candidate loci on tusk morphology. A cross section of an African elephant tusk shows the anatomical position of (a) enamel, (b) cementum, (c) dentin (ivory), (d) periodontium, and (e) root of the tusk. Dark blue circles indicate regions known or proposed to be affected 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).

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

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.

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SUPPLEMENTARY MATERIALS

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Materials and Methods
Supplementary Text
Figs. S1 and S2
Tables S1 to S8
References (*53*–*72*)
MDAR Reproducibility Checklist

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Supplementary Materials for

Ivory poaching and the rapid evolution of tusklessness in African elephants

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The PDF file includes:

Other Supplementary Material for this manuscript includes the following:

Materials and Methods

Gorongosa National Park Elephant Database

Gorongosa population surveys - ElephantVoices (J.P. and P.G.) completed ten ~3-4 week field surveys in Gorongosa (2011–2019). ElephantVoices built and populated The Gorongosa Elephant Who's Who & Whereabouts Database (http://www.elephantvoices.org/gorongosaelephants/), an online database housing a searchable registry of individually known elephants ('Who's Who') and geospatial records of sightings of observed elephant groups as well as those captured on trail cameras ('Whereabouts'). Users can, for example, use the database to identify an elephant by its distinguishing physical characteristics or to search for the number of tuskless individuals in the population by family, age, or sex. This database is password-protected as a precaution against poachers who might use the geospatial data.

Identifying and registering individual elephants - We used individual identification as the basis for monitoring the elephant population. Elephants were characterized and registered in the Gorongosa database following methods developed by the Amboseli Trust for Elephants (53) and used by ElephantVoices for over four decades. To individually identify elephants, we used sex, age, age accuracy (within 10 years, 5 years, 2 years, 1 year, 6 months, 1 month, 1 week, 1 day), family, family accuracy (4=known, 3=good idea, 2=educated guess, 1=unknown), mother, mother accuracy (4=known, 3=good idea, 2=educated guess, 1=unknown), and physiognomic features including tusk shape and configuration, ear shape, notches, tears and holes, tail, and other body markings.

We used eight age categories: 0A (0-4.9 years), 0B (5-9.9 years), 1A (10-14.9 years), 1B (15-19.9 years), 2 (20-24.5 years), 3 (25-34.5), 4 (35-49.9 years), 5 (50+ years) and defined 'adult' as ≥15 years old. We focused on the identification and registration of adult individuals until 2017, when we began to register the immature elephants of well-known families. By the end of 2019 we had identified 350 adult elephants of which 198 were adult females.

Before registering a new elephant, we carefully searched the database for any matching individual to avoid duplication. We assigned each registered individual a unique alphanumeric ID code starting with "gf" for females and "gm" for males. We uploaded up to six photographs to the digital ID card, adding the photographer's name and year taken as metadata.

Locating and recording elephants - We have photographic records of known mothers and their calves going back to 2011. We drove the main tracks to look for elephant groups and used the cellphone application GPS-Trk to keep a record of our route. Gorongosa is extensively wooded and its elephants are highly defensive. Family groups in particular typically react to the approach of vehicles with vigilance, avoidance, confrontation, or mobbing and attacking. Therefore, when we sighted an elephant or group of elephants, we made an immediate attempt to photograph as many individuals as possible. We used a Canon 6D with a 200-400 lens and a 1.4 extender to take ID photographs from a considerable distance so as not to disturb the elephants.

Geospatial records of elephant groups ('sightings') were collected using the Gorongosa EleApp, a custom Android application designed by ElephantVoices that directly records date, time, and GPS location. We manually entered the observer, place name (if known), group type (all-male group, family group, family group with associating males), number of individuals, accuracy of the count (exact, good estimate, guess), family name if known, and individual males and females recognized. Once connected to WiFi, sightings recorded in EleApp were uploaded to the online database. Field notes were typically recorded on a mobile phone and later transcribed into the online record. Field notes included the reaction of the elephants to the vehicle and other behavior, and,

where possible, a census of the group, including adult females present and the age and sex of their offspring. Elephants that we were unable to recognize in the field were later identified using the database, and their IDs were added to the sighting record. New individuals were registered as described above. We used Adobe Bridge to keyword each photograph with the registration number of all elephants pictured.

Analyses of sighting data

Historical tusk phenotype proportions – To estimate the frequency of tusklessness in Gorongosa prior to the civil war, JP categorized the tusk status of individual elephants from fourteen historical videos recorded within the park prior to the conflict (before 1972; Table_S1.xlsx). Across all video footage, 149 individual adult elephants were identified (89 male, 54 female). Individuals for which tusk category could not be reliably determined (due to body position or time on screen) were categorized as "unclassified". To estimate the frequency of tusklessness after the civil war, we used registered individuals in the Gorongosa elephant database (Table_S2.xlsx). This database included 217 registered females and 174 registered males.

Contemporary mother-offspring observations - For purposes of this study we searched our field notes, 5 TB of video footage, and 30,000 photographs taken during observations of 280 elephant groups to identify calves whose mothers were known with a high degree of accuracy. We entered every immature elephant that we could assign a mother-accuracy score of ≥2 into a spreadsheet. The spreadsheet included the following information: mother's name and ID code, mother's tusk configuration (2 tusks, 1 tusk, tuskless), mother accuracy (using the categories described above), field notes, date the calf was first recorded, estimated age of the calf at the first record, calf ID code, estimated calf birthdate, birthdate accuracy score, calf sex, and calf tusk configuration (unknown, 2 tusks, 1 tusk, tuskless). Immature elephants up to 8 years of age are found within 5 meters of their mothers 80% of the time, with the median distance being ~2 m (*54*). As calves age they tend to be found at greater distances from their mothers, particularly for juvenile males. Due to this differentiation between the sexes, we limited our analysis to calves that were ≤ 5 years of age at the first record. Calves that were first recorded as infants (estimated to be ≤ 1 year of age) and were seen in close association with an adult female, along with calves or juveniles that were observed, filmed, or photographed suckling were assigned a mother-accuracy score of 4. Calves and juveniles observed in close association with, orienting toward, and/or persistently following a particular adult female were assigned a mother-accuracy score of 3. For analysis, we included only calves with mother-accuracy scores of 3 or 4. Of the 270 calves entered into the spreadsheet, 130 were of known sex, had a mother accuracy of 3 or 4, and were ≤ 5 years of age at the time of first record. Statistical analyses in the main text exclude individuals with single tusks (numbers and percentages are reported) because single-tusked individuals may occur due to tusk breakage and therefore represent an ambiguous category for purposes of understanding heritability of genetically based tusklessness. These mother-offspring sighting data are included in Table_S4.xlsx mother-offspring, and all R code used to filter and analyze these data is archived on GitHub (and will be made publicly available upon publication).

Selection on tuskless females in Gorongosa - With pre- and post-war population size estimates, and conservatively assuming an equal sex ratio of males and female offspring, we used the Wallenius distribution (BiasedUrn R package) to model the odds of tuskless females surviving the war. Using an odds value of 1 corresponding to no selection, we first simulated the expected proportion of tuskless females following the war (Figure 2a). We then calculated the likelihood, or the probability

of observing our data given an odds value, for odds ranging from 1 to 10 in intervals of 0.001. An odds value of 10 means that tuskless females are 10 times more likely to survive the war than tusked females. This approximate likelihood curve (Figure 2b) was used to infer the maximum likelihood estimate of the odds value, along with the 95% confidence interval of this estimate using a drop in 1.92 log likelihood units from the maximum likelihood estimate. All R code used for these calculations is archived on GitHub (and will be made publicly available upon publication).

Genomic data analyses

Animal capture and genetic sampling **-** During May–August 2018 we chemically immobilized 18 female elephants (n = 7 tusked, n = 11 tuskless) by remote injection (darting) from a helicopter using a combination of thiafentanil oxalate and azaperone. Dosage was based on the approximate size and age of the individual, which we assessed visually at the time of capture. We carefully monitored each elephant during handling and measured cardiac rate (normal: 25–30 bpm), respiratory rate (normal: 4–6 breaths/minute), and rectal temperature (normal: 36–37 °C) at 5-min intervals. We also covered the eyes of each individual to minimize exposure to external stimuli. While immobilized, each elephant was fit with an iridium GPS collar (Savannah Tracking, Nairobi, Kenya) to track movements as part of a related study. We obtained a blood sample (10 mL) from each individual by venipuncture of one of the major veins of the ear. When sample collection was complete, we antagonized the thiafentanil with naltrexone and observed each elephant from a safe distance until it regained its footing and walked away from the area. All animal-handling procedures were approved by the Animal Care and Use Committee at the University of Idaho (protocol #2015-39) and were in accordance with guidelines established by the American Society of Mammalogists (*55*).

DNA extraction, library preparation, and sequencing **-** We extracted DNA from individual blood samples using PureLink™ Genomic DNA Mini Kit (Thermo Fisher). Extracted DNA was submitted to The California Institute for Quantitative Biosciences (University of California, Berkeley) for library preparation and whole-genome sequencing. We prepared these DNA samples for 150 cycles of paired-end sequencing on an Illumina NovaSeq.

Sequence alignment and variant calling **-** Reads were processed through a Snakemake workflow (https://github.com/harvardinformatics/shortRead_mapping_variantCalling) to create a VCF file containing high-quality SNPs for downstream analyses. Specifically, reads were first filtered with fastp (*56*) using the "detect_adapter_for_pe" option to remove low-quality reads and trim off any remaining adapter sequences. Afterwards, reads were mapped to the Loxafr3.0 genome assembly for *Loxodonta africana* using the BWA-MEM algorithm (*57*) (along with the -M option); PCR duplicates were marked for downstream analyses using Picard (http://broadinstitute.github.io/picard). Various summaries of the read filtering and mapping process are provided in Table_S5.xlsx, including the fractions of high-quality reads, high-quality read alignments, and PCR duplicates, along with the mean genome-wide sequencing depths. We sequenced 13 samples to higher depths (mean ~30X), and the remaining 5 samples to intermediate depths (mean ~10-15X) due to low DNA yields.

We then used aligned reads to detect SNPs and small indels using the GATK (v. 4.1.8) HaplotypeCaller workflow. Low-quality SNP sites were filtered out using the expression "QD < 2.0 || FS > 60.0 || SOR > 3.0 || MQ < 40.0 || MQRankSum < -12.5 || ReadPosRankSum < -8.0", and we did not filter out sites with excess heterozygosity to avoid discarding sites potentially relevant to this study. After filtering, we had 11,931,133 SNPs. Lastly, we annotated variants in the final VCF using snpEff (*58*) (Loxafr3.99 database). Although we mapped reads to the Loxafr3.0 reference, we

used information from the LoxAfr4 assembly to group scaffolds into chromosomes for particular analyses.

Quality control - We used vcftools (*59*) to calculate the relatedness (unadjusted Ajk statistic) of individuals to one another to verify that samples did not originate from the same family (Table_S6.xlsx). Two individuals, 0045B and 2986A, appeared to be siblings (Ajk ≈ 0.5), so we only used individual 0045B for downstream analyses to prevent relatedness from biasing results.

In addition to the per-site SNP filter applied above, we applied additional filtering per individual for analyses that relied on high-quality genotypes: Tajima's D, LD (using SNP vectors in RAiSD), heterozygosity, and relative diversity ($\pi_{tuskless}$ - $\pi_{two\text{-}tusked}$), described below. For these analyses, we only used the 13 samples (7 tuskless, 6 tusked) that had high depths (~30X; Table_S5.xlsx) and only considered variable sites in which each of these individuals had at least 8X depth but no more than 80X depth. We did not consider any sites that had missing data. Notably, our sequencing-depth restrictions per individual and intolerance of missing data did not discard a significant number of sites; of the ~13.5 million SNPs that satisfied a lower minimum-depth threshold of 4X per individual, 97% were retained when using our higher minimum depth of 8X per individual (Table_S7.xlsx). The only analyses that used all samples, high- and intermediate-depth, involved ANGSD (*60*) to evaluate differentiation between tuskless and tusked samples (F_{ST} and D_{XY}). This program uses genotype likelihoods to account for uncertainty within regions of lower sequencing depth. For a list of which samples were used for which analyses, please see Table_S8.xlsx.

To define genomic windows for analyses, we sought blocks of SNPs in relatively high LD. We used vcftools (*59*) to quantify genotype LD by site (measured as r^2) for our high-depth samples and found that blocks of 10kb typically had r^2 values > 0.5 (Figure S1). Since genomic windows for many of our analyses were defined based on SNPs, we chose SNP-window sizes such that the mean window length, in base pairs, was approximately 10kb or less (Table_S8.xlsx).

Genomic analysis of demographic differences between tusk phenotypes – We used vcftools (*59*) to calculate Tajima's *D* in 10kb nonoverlapping windows for tusked and tuskless groups. Using all 10kb windows for which estimates were available, we compared the mean difference in *D* between groups using a Welch's two sample *t*-test. To ensure that observed differences in mean *D* were not significantly affected by differences in sample size between groups (7 tuskless, 6 tusked) or the choice of specific individuals, we reran the analysis seven additional times, iteratively down-sampling the tuskless group to six individuals by removing a different individual. The results of down-sampled analyses remained statistically significant in each case. Therefore, we reported the original comparison between tusk morph groups using all high-depth samples.

Selection scans and linkage disequilibrium - To detect signatures of selection within tuskless elephants, we used RAiSD (*72*), which computes several metrics that are sensitive to sweeps. We computed these metrics in 50-SNP sliding windows along the genome (with default values for other parameters) and only used the metric that quantifies LD using SNP vectors (μ_{ld}). The individual scores per window are constructed such that highly positive values are consistent with recent selection (i.e., high LD).

Identification of differentiated loci - We used a sliding window analysis along the genome to quantify genetic differentiation between tuskless and tusked samples. This analysis used two population genetic statistics: F_{ST} and D_{XY}. F_{ST} uses normalized differences in allele frequencies between groups of samples. While F_{ST} is a popular way to quantify differentiation, its values strongly depend on levels of diversity, such that genomic regions with low diversity within groups are expected to have higher values (*60, 61*). Thus, to ensure we detected genomic regions that were truly

differentiated between tuskless and tusked samples, and not just regions of low diversity, we also used D_{XY} (*62*), which is independent of sequence diversity within groups. This statistic is computed by the average number of pairwise differences between sequences from two groups, ignoring all comparisons between sequences within groups.

To compute these statistics, we used ANGSD (60), which utilizes genotype likelihoods to account for uncertainty within regions of lower sequencing depth. Thus, we used all 18 samples for these analyses, since ANGSD takes genotype uncertainty into account for the intermediate-depth samples (Table_S5.xlsx). We had ANGSD calculate F_{ST} in 10kb windows along the genome, and we also used it to compute minor allele frequencies within tuskless and tusked samples, excluding sites that had a minor allele frequency (across all 18 samples) < 10%, and also excluding low-quality bases (BQ < 20) and low-quality mapped reads (MQ < 20). Using 50-SNP sliding windows (with a step size of 10 SNPs), we computed D_{XY} as $\sum(p1\times(1-p2) + p2\times(1-p1))$ where $p1$ and $p2$ represent allele frequencies in tuskless and tusked groups, respectively. Values of D_{XY} were normalized by the number of SNPs in each window (63,64). These 50-SNP windows used to compute D_{XY} had similar length, in base pairs, to other analyses (Table_S8.xlsx).

Heterozygosity analyses - To study levels of heterozygosity along the genome, we used cyvcf2 (26) to parse our VCF file and calculate local deviations of observed heterozygosity from proportions expected under Hardy-Weinberg equilibrium ($2p \times (1\text{-}p)$, where p is the frequency of the allele in the sample; Figure 4d).

For these heterozygosity analyses, we only considered SNPs that were common in tuskless samples (15-85% frequency) but absent in tusked samples. While we expect an X-linked, male-lethal mutation to be heterozygous in females, SNPs linked to this mutation may not, especially if these linked SNPs had intermediate frequencies when the causal mutation first arose and were present on both mutant and wild-type genetic backgrounds. However, linked SNPs that had very low frequencies, such as those unique to the genetic background in which the causal mutation arose, may be enriched for heterozygous genotypes after the X-linked, male-lethal mutant allele experienced positive selection and increased the frequency of these linked SNPs. Thus, to target this subset of linked SNPs that may also be enriched for heterozygous genotypes, we only considered SNPs that were common in tuskless samples (15-85% frequency) but absent in tusked samples. Since this sub-setting procedure increases the inter-SNP distance of the SNPs that are ultimately analyzed, we used 10-SNP windows (with a step size of 2 SNPs), which corresponded to genomic windows that were roughly 10kb in size. This smaller SNP window size was chosen to make the size of these windows (in base pairs) more comparable to the other analyses (Table_S8.xlsx).

Genetic diversity analysis - Genetic diversity was calculated for tusked and tuskless individuals separately in 10kb nonoverlapping windows using vcftools. Relative diversity was calculated as the difference in diversity observed between the tuskless group and the tusked group ($\pi_{tuskless}$ - π_{tusked}). If the candidate locus underpins the tuskless phenotype, we would expect significantly lower genetic diversity, compared to the genome-wide average, in the candidate region due to recent, strong selection within tuskless individuals. Alternatively, diversity within tusked individuals should not be significantly lower than the genome-wide average. Mean π genome-wide was nearly identical for both groups, with a mean of 0.001666 for tusked samples (stan. dev. = 0.00295) and 0.001668 for tuskless samples (stan. dev. = 0.00287). Within the 3 candidate windows associated with MEP1a (scaffold_0: position 117320001 – 117330000, position 117330001 – 117340000 and position 117340001 – 117350000), π estimates were 0.009, 0.009, and 0.011, respectively, for the tusked group, and 0.0002, 0.0004 and 0.0004, respectively, for tuskless group. Thus, while we observed decreased diversity within tuskless samples, tusked samples also had elevated diversity within the

candidate region. This is driven by a set of 246 non-reference variants that are all fixed within tuskless samples but present at intermediate frequencies in tusked samples (see "*Candidate region on chromosome 1*" below).

Differentiated SNPs analysis - In addition to our window-based analysis to detect differentiated regions between tuskless and tusked samples (using F_{ST} and D_{XY}, above), we used cyvcf2 to scan our VCF for highly-differentiated, individual SNPs and small indels with allele frequency differences between tuskless and tusked samples that were ≥ 50%. We used only those individuals that had high sequencing depths. We quantified SNP differentiation using the total allele-frequency difference across all non-reference alleles. For instance, if tuskless samples had multiple non-reference alleles not found in tusked samples, each would contribute to the overall allele frequency difference. While most SNPs are biallelic across our samples (95.6%), we used this procedure to accommodate the less-frequent multiallelic sites that may be biologically relevant. Interestingly, despite high sequencing depths and relatively few samples per group, we could not identify a single SNP or small indel (detectable by GATK4) that was fixed in tuskless samples and absent in tusked samples.

Analysis of large structural variants - To identify larger structural variants not detectable by GATK4, we used *sv-callers* (*66*), a Snakemake workflow that runs four structural variant callers: Manta (v1.1.0), DELLY (v0.7.7), LUMPY (v0.2.13), and GRIDSS (v1.3.4). The sv-callers workflow uses the structural variant callers on each individual sample, so we combined calls across samples using SURVIVOR (*67*) and merged calls that were of the same type (e.g. deletion, insertion) and had breakpoints within 1kb of one another. Within these merged VCF files, we used the "SUPP_VEC" field, created by SURVIVOR, in the INFO column. This field indicates the presence or absence of a structural variant within each sample but does not provide genotype information. To focus on variants that might explain the difference in tusk status between tuskless and tusked elephants, we only considered variants that had appreciably higher frequencies within tuskless samples, i.e. frequencies that were at least 50% higher in tuskless samples compared to tusked samples.

Candidate cis-regulatory elements (cCREs) – To assess whether non-exonic SNPs occurred within potential regulatory elements, we searched the ENCODE database for cCREs within the human genome (build hg19). For each gene within the two candidate windows mentioned above (one on the X chromosome, one on chromosome 1), we gathered all cCREs that were (1) within the gene body, (2) between the first and last transcription start site, or (3) up to 50kb upstream of the transcription start site. We then took the coordinates of these cCREs and used the UCSC genome browser's liftover tool to translate the coordinates from the hg19 genome to the elephant genome (loxAfr3.0). While this analysis gives additional insight into the potential impact of non-exonic variants, we do not know if these cCREs are biologically meaningful in African elephants without additional experimental work.

Supplementary Text

Candidate region on the X chromosome - Within the candidate region scaffold_39: 19913744 - 20932476 (Figure 2d), there is a single site with an allele frequency of 50% in tuskless samples but completely absent in tusked samples. This site contains an intergenic, multi-allelic indel in which the reference allele is T, and the alternate alleles are TG, TGG, and TGGG. Four tuskless samples were heterozygous for the TGG allele, one sample was heterozygous for the TG allele, one sample was heterozygous for the TG and TGG alleles (i.e., no reference allele), and the last tuskless sample was homozygous for the reference allele. By comparison, samples from two individuals with two tusks were heterozygous for the TGGG allele, which was not found in any of the tuskless samples. Further away are three SNPs upstream of the HCCS gene, and these involve biallelic non-reference

alleles unique to tuskless samples. One of these SNPs lies within a candidate cis-regulatory element (cCRE, described above). At these three sites, five tuskless samples were heterozygous for the alternate allele, one was homozygous, and one had the reference allele at all three sites. In terms of large structural variants, DELLY detected a 2.6kb deletion within the interval, but it was present in 4 of 7 tuskless samples and 1 of 6 tusked samples. Outside of the candidate interval, both DELLY and LUMPY detected a 1kb deletion 133kb upstream of the interval, present in 3 of 7 tuskless samples and absent in tusked samples.

Candidate region on chromosome 1 - Within the candidate region scaffold_0:117320001-117350000 (Figure 3), there is a set of 246 variants (SNPs and small indels) that were all fixed in tuskless samples but present at 50% frequency in tusked samples. These variants are mostly intergenic between MEP1a and PLA2G7 (n = 126) or within MEP1a introns (n = 86). While none of these variants lie within the promoter of MEP1a, n = 89 are within cCREs (described below), including 7 small indels. In addition to these non-coding variants, there are 3 nonsynonymous mutations in the exons of MEP1a. Within this entire window, four out of six tusked samples were heterozygous at all of these variant positions, while one was homozygous for all alternate alleles and another was homozygous for all reference alleles (totaling an allele frequency of 50% in tusked samples across all these variants). Tuskless samples were homozygous for the alternate alleles across all variants in this region. In terms of large structural variants, nothing existed within the candidate region. However, Manta detected a 104bp insertion 411kb upstream of the region that was present in 5 of 7 tuskless samples but absent in tusked samples. Also, DELLY detected a 28bp deletion 364kb downstream of the region that was present in 4 of 7 tuskless samples but absent in tusked samples.

A candidate mechanism for genetically-based tusklessness in African elephants - In mammals, females carry twice as many X chromosomes as males but achieve the same gene dosage via X-chromosome inactivation, in which one X-chromosome copy is silenced early in development. Somatic cells may vary in which copy they silence, resulting in phenotypic mosaicism when different alleles on alternative X-chromosomes give rise to different phenotypes. In X-linked dominant male-lethal conditions, including those associated with AMELX, X-inactivation has been identified or hypothesized as a factor determining phenotypic outcomes of genetic variation (*68*–*71*). Phenotypic variability associated with X-chromosome inactivation (XCI) is a strong candidate as one mechanism driving variability in tusk morphology inheritance and presentation in Gorongosa. The sex-linked candidate region is syntenic with the Xp22.2 region of the human X-chromosome, which is associated with MIDAS/XAI disease syndrome (*71*). The X-linked dominant male-lethal inheritance of this disease is commonly associated with segmental aneuploidy across the region. However, variation in trait expression is highly variable within and between families. In certain cases, asymptomatic mothers can give rise to daughters displaying the trait (*70*). In such cases, mother and daughter share the causal genotype, but the asymptomatic individual displays preferential inactivation of the affected chromosome (*70*). Given the homology between the humanXp.22.2 region with the candidate X-linked region of the elephant genome, we propose that skewed X-inactivation may explain aberrant instances in Gorongosa in which a two-tusked mother was associated a tuskless daughter, as observed in a single case in this study.

Literature survey – To assess the frequency of tusklessness in African elephant populations, we systematically reviewed the literature (Table_S3.xlsx). On 20 April 2020, we searched Google Scholar using the phrase ["African elephant" tuskless] and ISI Web of Science using the phrase [(tuskless* OR "missing tusk*" OR "lack* tusk*" OR "tusk* loss") AND eleph*]. These searches yielded 308 and 18 results, respectively, all of which we scanned; those that seemed likely to present

quantitative information on tusklessness in African elephants were retrieved and reviewed. We further scanned the literature cited in these references to identify additional potential sources, and we retrieved data from an online database managed by J.P. and P.G. A small number of potential sources were unobtainable. In total, we reviewed 87 books, papers, and dissertations; the Table_S3.xlsx includes information from 24 sources, which yielded 58 total records. Studies differed in what they reported and how. Whenever possible, we extracted data on sex-specific and overall tusklessness frequencies; the sample sizes from which these frequencies were derived; whether single-tusked individuals were reported; the hunting pressure in the location (qualitatively assessed from information in the sources); whether the authors reported indications that tusklessness is heritable; the authors' hypothesis about the mechanism of tusklessness; the format in which the data were presented; and any pertinent notes about and/or quotes from the primary source. Blank cells indicate that the information was not presented and could not be inferred from the source. Question marks indicate uncertainty (either because of explicit qualifications present in the original source or because we were uncertain of our interpretation of information in the source; in these cases, we explain further in the notes). Only two sources reported appreciable frequencies (>2%) of tusklessness in male African elephants (*72*, *73*), and we regard both of these estimates as uncertain based on statements in the original sources.

Intermediate single-tusked phenotype - One-tusked individuals represent an ambiguous category for our analyses because injuries can result in loss of one tusk, whereas acquired and symmetrical loss of both tusks is vanishingly rare; thus, bilateral tusklessness can safely be assumed to be congenital (*74*, *75*), but instances of unilateral tusklessness are less certain. Various studies include anecdotal reports of such injuries, which can lead to infection and loss of the tooth (*76–79*). To our knowledge, the most extensive quantitative data are from Steenkamp et al. 2007 (*75*), who evaluated photographs of ~1500 animals from 15 populations in 7 countries for evidence of both tusk fracture (n = 2695 tusks examined) and congenital tusklessness (see Table_S3.xlsx). The proportion of fractured tusks was highly variable among populations (range 0–44%), averaging 9.4% among females and 7.4% overall (bilateral breakage was very rare, suggesting that up to ~19% of females and ~15% of all individuals had one broken tusk). However, the proportion of fractured tusks decreased exponentially with rainfall; considering just the populations with rainfall ≥ 800 mm yr^{-1} (comparable to Gorongosa, 840 mm yr^{-1}), only 6 of 301 tusks were fractured (2%, suggesting ~4% of individuals). Thus, the frequency of single-tusked females in Gorongosa (~7.5%) falls within the range of tusk-fracture frequencies across Africa but is higher than expected given rainfall in Gorongosa. Indeed, records in the Gorongosa elephant database indicate that frequency of unilateral tusk breakage across sexes is 3.1% (n = 260), predominantly in older animals, suggesting that injury alone cannot explain the single-tusked phenotype in our study.

Evidence from several studies likewise indicates that single-tuskedness tends to run in families, often those with high proportions of tuskless females, suggesting that there is a heritable intermediate phenotype(*72*, *73*, *80*); but see (*29*). Nonetheless, owing to the uncertainty about the congenital vs. acquired origin of single-tuskedness for any given observation and the comparatively few observations of one-tusked females in the mother-offspring survey data (n = 0 mothers, 8 calves), we refrain from attempting inferences about the basis and inheritance pattern of unilateral tusklessness; we consider this an important avenue for future research.

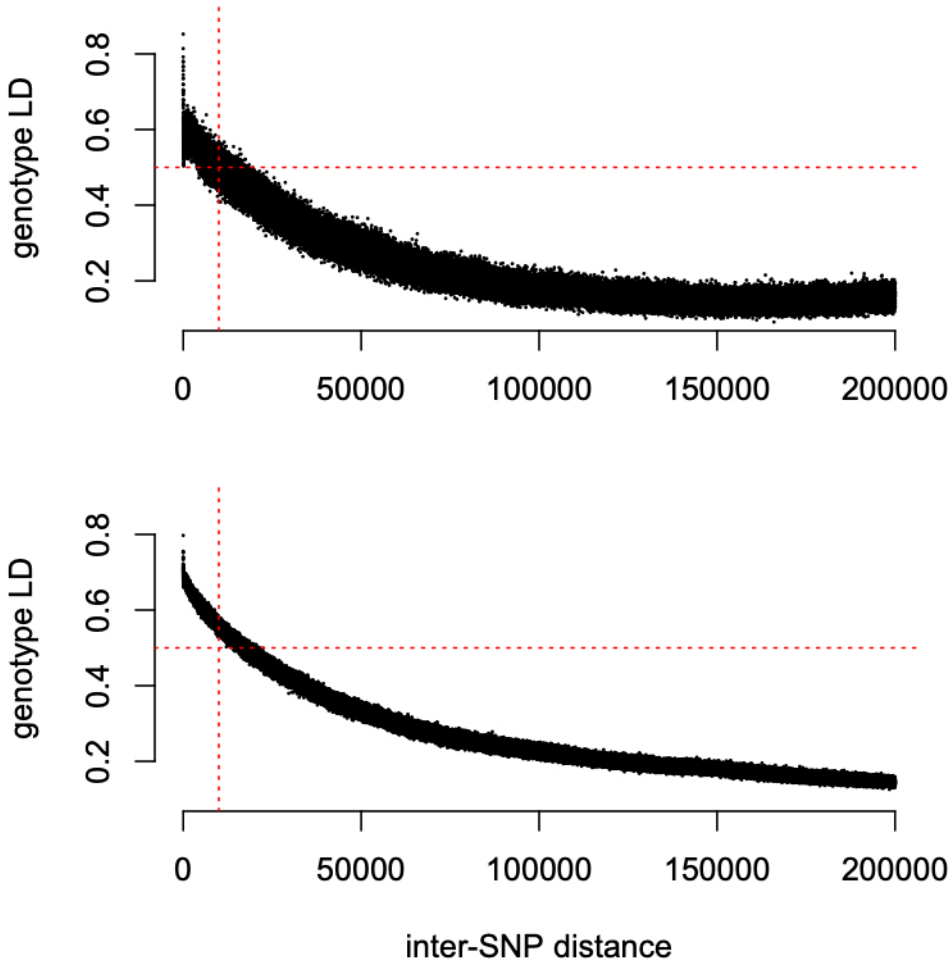

Figure S1. Genotype LD vs distance between SNPs. Linkage disequilibrium (LD) between genotypes, measured as r^2, decayed to approximately 0.5 (horizontal dotted line) for SNPs that were 10kb apart (vertical dotted line). The upper panel shows the decay of r^2 vs inter-SNP distance for the X chromosome, while the bottom panel shows this decay for chromosome 1.

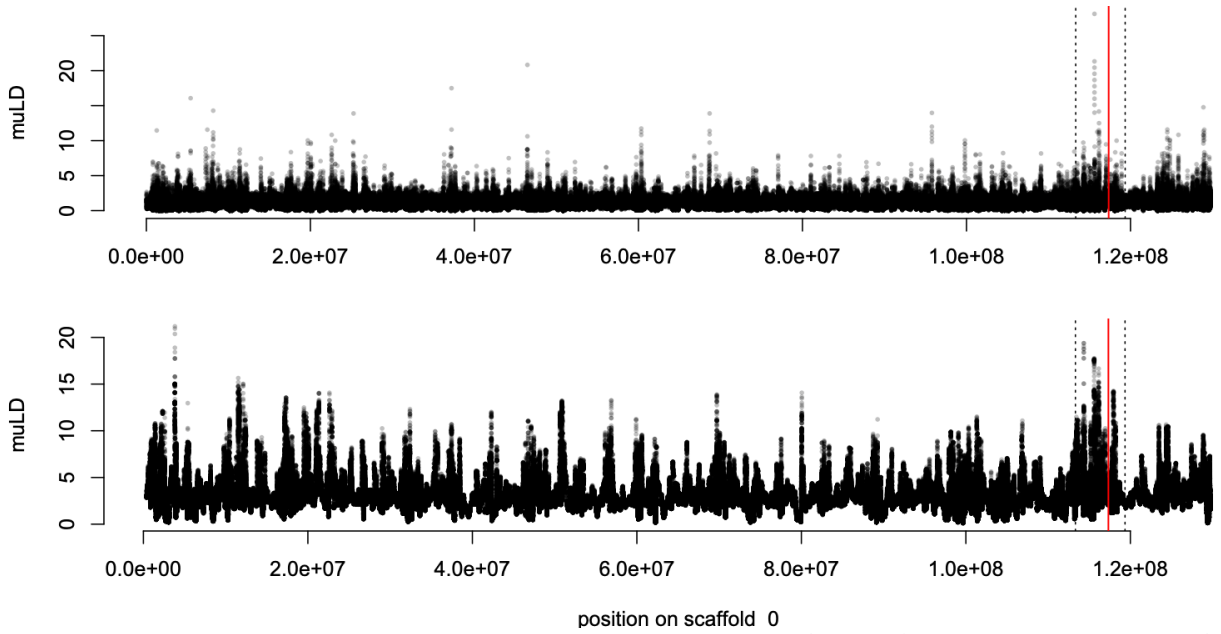

Figure S2. LD along scaffold_0. The top panel shows linkage disequilibrium (LD), as measured with SNP vectors, using 50-SNP windows that have a mean size of 7.8kb. The bottom panel shows larger-scale patterns of LD using 1000-SNP windows that have a mean size of 343kb. The candidate region on scaffold_0 (red line), which is part of chromosome 1, is nested within a larger region (~6Mb; dotted lines) that displays elevated LD.

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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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