POLYMORPHISM

Adaptive spread of a sexually selected syndrome eliminates an ancient color polymorphism in wall lizards

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Genetically determined color morphs are found in many animals. Polymorphism can be maintained by social selection if competitive interactions allow each morph to increase in frequency when rare. This reliance on negative frequency–dependent selection should make color polymorphism vulnerable to the appearance of novel phenotypes that disrupt competitive interactions among morphs. We show that the origin and adaptive spread of a sexually selected syndrome in common wall lizards (*Podarcis muralis*) selectively eliminates alleles coding for alternative color morphs that have been maintained for millions of years. The results demonstrate how the arrival of a novel phenotype can disrupt balancing selection, providing a link between rapid phenotypic evolution and the loss of color polymorphisms.

Color polymorphisms occur throughout the animal kingdom, including in insects (*1*), fish (*2*), and reptiles (*3*, *4*). Within a population, multiple discrete morphs can be maintained if their relative fitness is influenced by environmental factors that vary or when social and sexual competition favors morphs that are rare (*5*, *6*). However, the phylogenetic and geographic distribution of polymorphism reveals that color morphs are frequently lost (*1*, *7*–*9*). Although the causes of such losses are poorly understood, polymorphism maintained by social selection should be particularly vulnerable to the appearance of novel phenotypes or "strategies" that change the competitive interactions among morphs (*10*). Thus, rapid evolution of traits used in intraspecific communication or competition could disrupt balancing selection, causing selection against established morphs and the loss of polymorphism.

To study this process, we explored the recent origin of a sexually selected syndrome in the color-polymorphic common wall lizard (*Podarcis muralis*) (*11*–*13*). The results show that the geographic expansion and between-lineage introgression of traits used in sexual and social competition caused selective elimination of alleles coding for two of the three color morphs, resulting in a rapid transition from polymorphism to the fixation of a single morph.

Color polymorphism in wall lizards has been maintained for millions of years

Several species of Mediterranean wall lizards (genus *Podarcis*) exhibit a within-population color polymorphism consisting of a white, yellow, or orange throat and ventral region (Fig. 1A) (*3*, *4*, *14*). Both males and females express all three morphs. White, yellow, and orange individuals can differ in morphology, physiology, and behavior, and field and experimental research suggest that social and sexual competition contributes to the maintenance of color polymorphism even in the absence of alternative, discrete, reproductive strategies (*15*–*23*). Occasional loss of polymorphism within the *Podarcis* genus (e.g., *24*, *24*–*29*), including local fixation of the white morph in *P. muralis* on the Italian peninsula (Fig. 1, B and C), suggest that balancing social selection can be disrupted. However, the underlying processes of morph loss are unknown.

To reconstruct the evolutionary history of color polymorphism within *P. muralis*, we collected data on morph frequencies for 237 populations from western Europe, the southern Alps, and the Italian and Balkan peninsulas, and surveyed the literature for additional data (see supplementary text and table S1). These data show that the three ventral color morphs co-occur within all major genetic lineages, suggesting that they are ancestral to the species and have persisted within these lineages ever since their divergence up to 6 million years ago (Mya) (Fig. 1B and supplementary text) (*30*, *31*). Moreover, within-population polymorphism occurs throughout the species' distribution, which covers a range of habitats and climates from hot-summer Mediterranean climates to substantially cooler and highly seasonal environments in central Europe and mountainous areas (>2000 m altitude) (*32*). This persistence of color polymorphism under variable conditions and over millions of years supports previous inferences that balancing selection is not caused by environmental factors alone but also relies on social and sexual competition (*16*, *18*, *26*, *33*).

Ventral color polymorphism is lost following the invasion of a sexually selected phenotype

In central Italy, common wall lizards differ from the ancestral phenotype in a suite of derived and coexpressed quantitative traits, including extensive melanization of the body, green dorsal coloration, exaggerated color ornaments, large body and head size, and dominant behavior ("nigriventris syndrome"; Fig. 2A, lower left) (*13*, *34*–*36*). This difference between ancestral and derived phenotypes applies to both sexes but these characters are generally more exaggerated in males. The nigriventris syndrome evolved within the central Italy lineage of *P. muralis* and has since spread via sexual selection throughout most (but not all) of this lineage (*12*, *34*). The entire suite of characters has also introgressed into parts of the distantly related southern Alps lineage (*11*–*13*, *37*). As a result, both the ancestral and nigriventris phenotypes now occur in genetic lineages that diverged >5 Mya, with the characteristic color and morphological traits of the nigriventris syndrome varying consistently together across the landscape (*13*, *34*) (Fig. 2A).

Populations of *P. muralis* from the region around Rome on the Italian peninsula exhibit the most exaggerated phenotype (originally described as *P. muralis nigriventris*; Fig. 2A, lower left). Sampling 180 lizards from three locations throughout this region revealed that these nigriventris populations are fixed for the white morph (Fig. 2A). This is in contrast to 153 individuals from four populations of the same central Italy lineage, east of Rome, that are fixed for the ancestral *P. muralis* phenotype; here, all three ventral color morphs co-occur (Fig. 2A). We therefore hypothesized that the nigriventris syndrome caused loss of the yellow and orange morphs, resulting in the fixation of the white morph.

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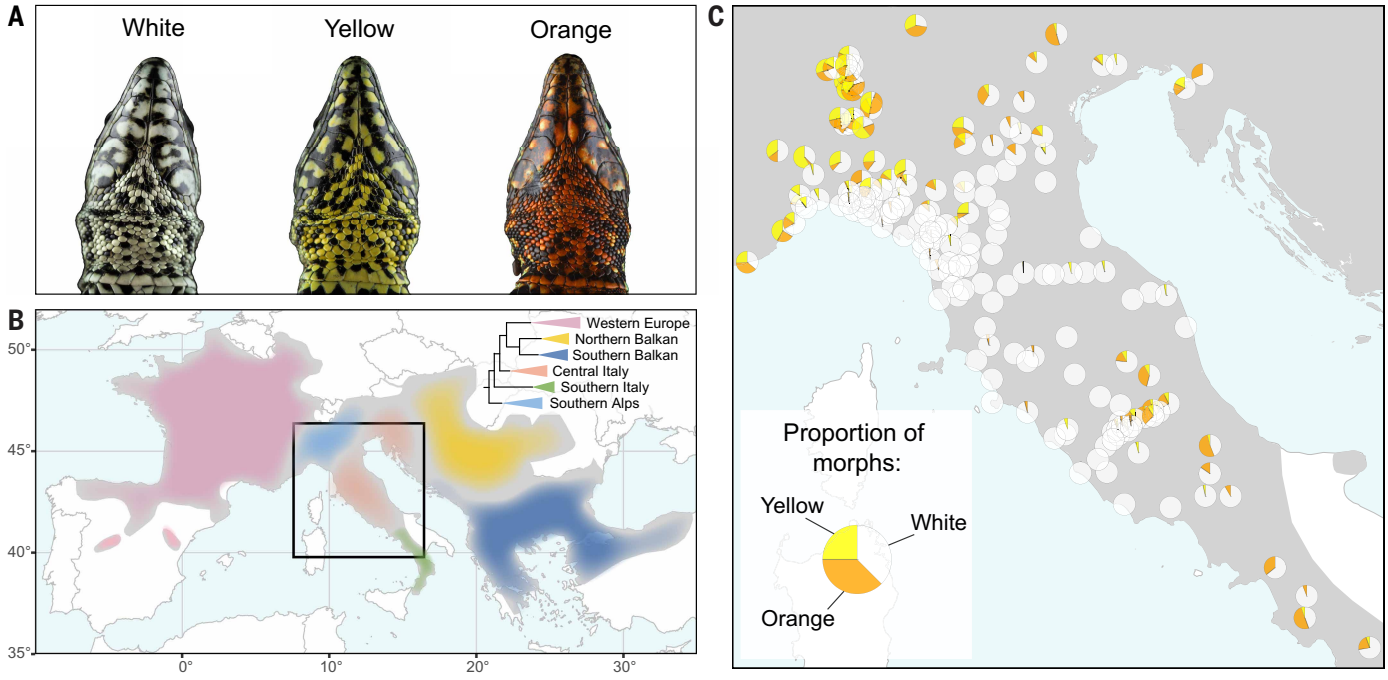

Fig. 1. Ventral color polymorphism in the common wall lizard *P. muralis*. (**A**) The common wall lizard *P. muralis* exhibits a color polymorphism with three discrete morphs that differ in their throat and ventral coloration: white, yellow or orange. Photographs by R. García-Roa. (**B**) *P. muralis* is distributed across central and southern Europe (gray areas mark the species distribution according to the International Union for Conservation of Nature Red List Data). The taxonomic relationships of the major lineages (*30*) are reproduced in the top right panel (note that the species outgroup is omitted) and color-coded areas on the map mark the approximate distribution of each genetic lineage. The black square indicates the position of the detailed map shown in (C). (**C**) Overview of morph frequencies from 220 sampling locations ("populations") in the focal area, covering most of the distribution of the lineages in the southern Alps, central Italy, and southern Italy. For this graphical presentation, populations within 5 km^2 were merged, resulting in 177 pie charts representing an average of 56.8 individuals per population. Individuals displaying both yellow and orange were counted as 0.5 for each color.

To test this hypothesis, we first investigated whether the geographic expansion of the nigriventris syndrome was associated with an increase in the frequency of the white morph. We followed previous work and used the average dorsal greenness of adult male lizards as an estimate of the population-level expression of the nigriventris syndrome (*11*–*13*). To test whether the spread of the nigriventris syndrome throughout the central Italy lineage and its introgression into the southern Alps lineage has the same consequence for morph frequencies, we included lineage as a fixed effect in the statistical model.

In both lineages, the geographic expansion and intensity of expression of the nigriventris syndrome was closely associated with an increase in the frequency of the white morph (greenness: $\chi^2 = 264.3$, df = 1, $P = 2.0 \times 10^{-16}$; lineage: $\chi^2 = 4.58$, df = 1, $P = 0.03$; greenness×lineage: $\chi^2 = 0.44$, df = 1, $P = 0.51$; Fig. 2, B to D). That yellow and orange morphs become increasingly uncommon in the wake of the introgression of the syndrome into the distantly related southern Alps lineage suggests that the sexually selected spread of the nigriventris syndrome is the cause for this association.

Loss of polymorphism is concordant with changes in allele frequencies at morph-determining loci

To establish why the invasion of the nigriventris phenotype caused loss of polymorphism, we capitalized on knowledge of the genetic basis of the color morphs. In *P. muralis*, ventral color polymorphism is determined by two unlinked loci that regulate the production of pigments deposited in xanthophores (*14*). Yellow individuals are homozygous (yy) for one allele at a locus associated with the gene *BCO2*, which encodes the beta-carotene oxygenase 2 that oxidizes carotenoids to colorless apocarotenoids (*38*, *39*). Individuals with uniform orange coloration or an orange-white mosaic are homozygous (oo) for one allele at a locus associated with the gene *SPR*, which encodes a sepiapterin reductase involved in pterine metabolism (*40*, *41*). Yellow-orange individuals are homozygous at both loci (yy and oo). White individuals are heterozygous or homozygous for an alternative allele (Y and O, respectively) at each of these two loci.

The exact causal mutations for the yellow and orange morph have not been established to date. Therefore, we targeted a candidate single-nucleotide polymorphism (SNP) in the "yellow" *BCO2* locus and a candidate 38-bp (base pair) insertion in the "orange" *SPR* locus (*14*). We successfully genotyped 679 individuals at the yellow locus and 728 individuals at the orange locus. Phenotypic morph and allelic variants matched in 97.35% of individuals for the yellow locus and in 98.08% of individuals for the orange locus (Table 1; yellow: 18 mismatches out of 679 genotyped individuals; orange: 14 mismatches out of 728 genotyped individuals). The association of these molecular markers with color morph identity across deeply divergent lineages of *P. muralis* is consistent with a conserved genetic architecture of morph determination in this species (*14*). The frequency of mismatch was not higher for white-morph phenotypes, which constituted 75% of all observed mismatches compared with 87% of all genotyped individuals. Similarly, individuals with a green dorsum accounted for only 28% of the total number of mismatches compared with 61% of all genotyped individuals (see table S2 for more details). This rules out low penetrance as an explanation for morph loss following the invasion of the nigriventris phenotype.

Similar to the loss of yellow and orange morphs (Fig. 2B), the frequencies of the recessive alleles at the yellow and orange loci were strongly negatively associated with the population-level expression of the nigriventris syndrome in both lineages (yellow locus, greenness: $\chi^2 = 166.1$, df = 1, $P < 2.2 \times 10^{-16}$; lineage: $\chi^2 = 10.9$, df = 1, $P < 0.001$; greenness×lineage: $\chi^2 = 0.49$, df = 1, $P = 0.48$; orange locus, greenness: $\chi^2 = 133.2$, df = 1, $P < 2.0 \times 10^{-16}$; lineage: $\chi^2 = 3.0$, df = 1, $P = 0.09$;

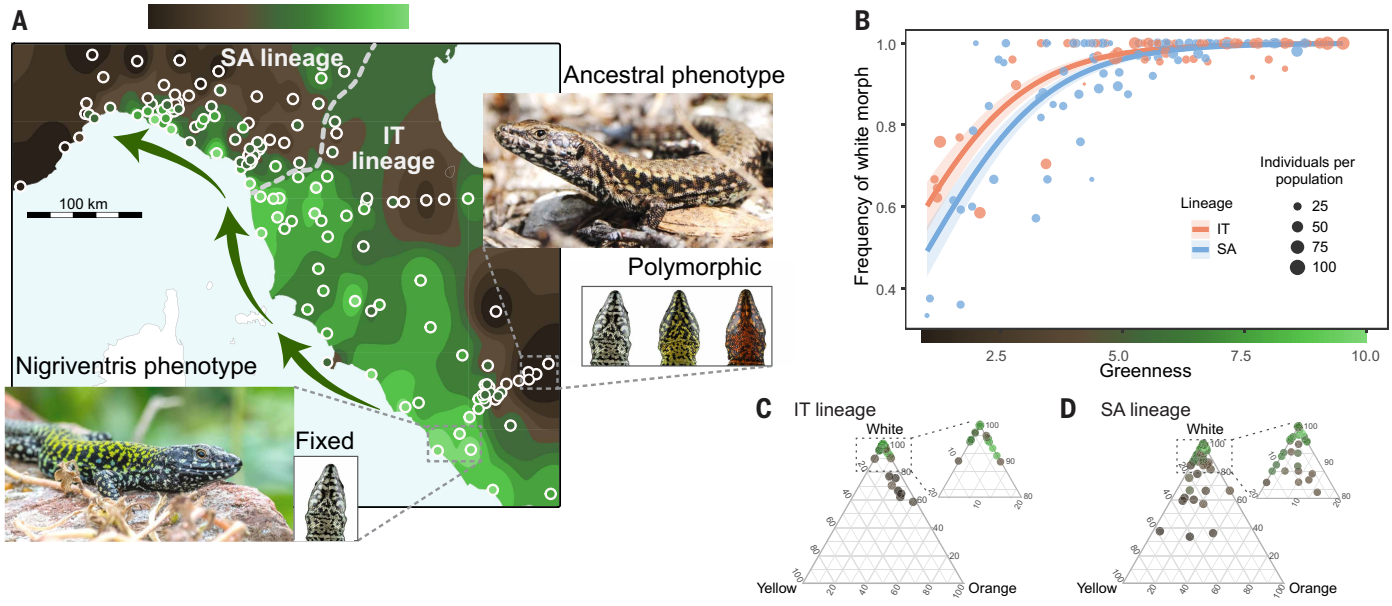

Fig 2. Relationship between the nigriventris phenotype and color morph frequencies. (**A**) Geographic distribution of average male dorsal coloration ("greenness," ranging from 1 to 10), a proxy for the quantitative expression of the nigriventris syndrome in *P. muralis* across 148 populations (N_{males} = 2506). White circles mark the locations of sampled populations. The boundary between the southern Alps and central Italy lineages is indicated with a light gray dashed line (note that the southern Alps lineage here includes populations that belong to the hybrid zone proper, i.e., with an ancestry proportion $0.05 \leq Q \geq 0.95$ (*13*,*34*); see materials and methods for details). Arrows emerge from the putative origin of the nigriventris phenotype within the central Italy lineage and indicate its spread and introgression into the southern Alps lineage (*12*,*13*). At the population level, this spread is manifested as quantitative variation in the suite of coexpressed characters that constitute the nigriventris syndrome (*13*). (Lower left) Male with a highly expressed nigriventris phenotype, typical for the three populations indicated by the box with gray dashed lines. (Lower middle) Close-up shows the throat of a white morph that is fixed in these nigriventris populations. (Top right) Male with the ancestral phenotype, typical for the four populations marked by the box with gray dashed lines. Close-ups below show examples of white, yellow, and orange morphs. Photographs by A. Badiane (nigriventris phenotype), N.F. (ancestral phenotype), and R. García-Roa (throats). IT, central Italy; SA, southern Alps. (**B**) Relationship between frequency of the white morph and average male dorsal greenness in 141 populations (65 belonging to the central Italy lineage and 76 to the southern Alps lineage). size indicates the number of individuals per population that were scored for ventral color morph (minimum 10, mean = 29.8, sd = 13.66). Lines represent fitted values. (**C** and **D**) Ternary plots showing the proportion of white, yellow, and orange morphs in 65 populations belonging to the central Italy lineage and 76 populations belonging to the southern Alps lineage. (Top right) Close-ups show the populations with a frequency of the white morph of at least 0.8. Each population is color coded by average greenness.

Table 1. Relationship between allelic variation and phenotypic morph. Bold indicates mismatches between genotype and expected morph; their percentages are summarized in the text.

Locus	Genotype	Morph				
		White	Yellow	Orange	Yellow-Orange	Total
Yellow (*BCO2*)	Y/Y	500	0	21	0	521
	Y/y	75	**1**	15	0	91
	y/y	**15**	41	**2**	9	67
Orange (*SPR*)	O/O	506	25	0	**1**	532
	O/o	114	20	**1**	**1**	136
	o/o	**9**	**2**	40	9	60

greenness×lineage: $\chi^2 = 0.38$, df = 1, $P = 0.54$; Fig. 3). There was no evidence of gene flow at the two morph loci from the central Italy lineage into the southern Alps lineage, suggesting that this pattern is due to local "white alleles" at the *BCO2* and *SPR* loci going to fixation rather than co-introgression of these alleles and major-effect genes of the nigriventris syndrome (fig. S1). Thus, the invasion of the nigriventris phenotype causes loss of yellow and orange color morphs by eliminating the corresponding alleles from populations, and it does so consistently in deeply divergent lineages.

Loss of yellow and orange alleles is not explained by developmental or genetic linkage

To establish why the yellow and orange alleles disappear from populations following the invasion of the nigriventris syndrome, we investigated the overlap between the color morph loci and the genetic basis of the nigriventris phenotype. If the dominant white alleles contribute to the development of the nigriventris phenotype, or are in close physical proximity (i.e., a linkage block) to genes that do, the fixation of the white alleles requires only selection for nigriventris characters rather than selection against polymorphism. In the absence of developmental and genetic linkage, the loss of polymorphism could be explained by disruption of established forms of social communication or competition that cause balancing selection.

We leveraged data from a previous study (*13*), which established a polygenic architecture of the nigriventris phenotype based on patterns of differentiation and genetic diversity of 90 whole-genome sequences and a genome-wide association study (GWAS) of 685 genomes at reduced representation (*13*). Investigating patterns of linkage disequilibrium surrounding the yellow and orange loci shows correlation decays over short distances, with small linkage blocks of <5 kb (fig. S2). Intersecting these yellow and orange linkage blocks with all genomic regions identified as potentially associated with the nigriventris syndrome (*13*), we find no overlap with the top candidate genomic regions (table S3). One outlier identified by the GWAS analysis on the reduced representation data overlapped with the yellow linkage block (fig. S2, table S3); however, this is not different from what is expected by

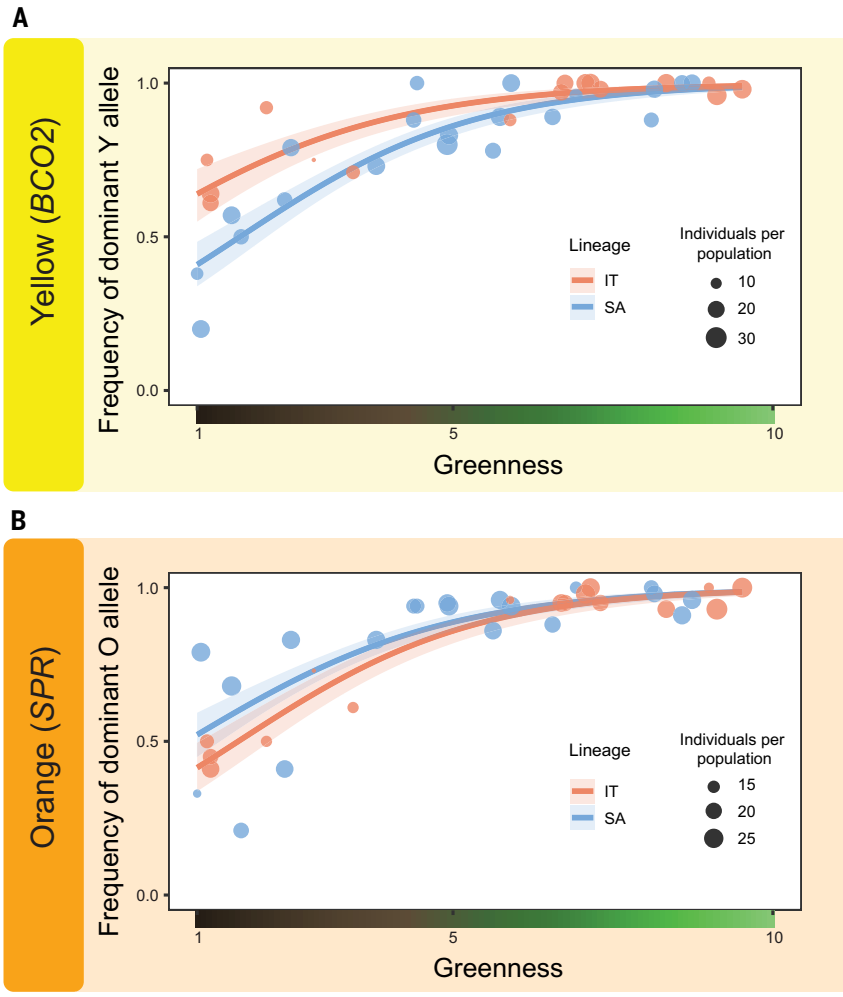

Fig. 3. Relationship between allele frequencies at morph-determining loci and the expression of the nigriventris phenotype. (**A** and **B**) Frequencies of the dominant "white" allele ("Y") at the yellow locus [*BCO2*; (A)] and the dominant "white" allele ("O") at the orange locus [*SPR*; (B)] in 36 populations (16 belonging to the central Italy lineage and 20 to the southern Alps lineage) increase with increasing average greenness of males, a proxy for the population-level expression of the nigriventris syndrome. Dot size indicates the number of individuals per population that were genotyped (yellow locus: minimum 6, mean = 18.9, sd = 4.87; orange locus: minimum 11, mean = 20.2, sd = 4.34). Lines represent fitted values. IT, central Italy; SA, southern Alps.

chance (permutation test; $P = 0.17$). These results suggest that the genetic basis of the ventral color polymorphism is independent of the genetic basis of the nigriventris syndrome. This conclusion is corroborated by phenotypic data from the introgression front of the nigriventris syndrome: Across 14 populations in which all three morphs co-exist with nigriventris characters, there was no difference in the intensity of the characteristic nigriventris coloration between white and non-white individuals (greenness: Kruskal-Wallis test $\chi^2 = 0.57$, df = 1, $P = 0.45$; blackness: $F_{1,218} = 1.08$, $P = 0.30$). Taken together, these results show that developmental and genetic linkage play at best a minor role for the loss of color polymorphism following the geographic expansion of the nigriventris syndrome.

Discussion

The ventral color polymorphism of common wall lizards has persisted for millions of years, during range contractions and expansions, and across a wide range of environmental and climatic regimes. However, our results show that two of the three alternative colors—yellow and orange—and their corresponding alleles are rapidly and consistently eliminated following the invasion of a novel, sexually selected phenotype. Neither developmental nor genetic linkage explain the fixation of the white alleles at the two morph-determining loci. Instead, these results suggest that the evolution and geographic spread of sexually selected characters disrupted the balancing selection that maintained alternative color morphs, resulting in the elimination of the orange and yellow morphs.

How does the invasion of the nigriventris syndrome disrupt balancing selection? Males with nigriventris characters are dominant over males with the ancestral phenotype, gaining higher access to high-quality territories and females (*11*, *35*, *36*). The appearance and selection for the nigriventris characters in a population could therefore modify established forms of social competition that favor rare color morphs (*42*, *43*), prevent opportunities and benefits of morph-disassortative mating (*6*, *44*–*46*), or eliminate the function of ventral coloration in social communication (*47*–*49*). The consistent fixation of the white morph suggests that such changes in social and sexual interactions not only weaken the strength of balancing selection for polymorphism but consistently result in selection against yellow and orange morphs.

Prevailing theory suggests that polymorphisms can constrain evolutionary change by maintaining genetic correlations and enforcing negative frequency–dependent selection (*6*, *8*, *42*). Thus, major evolutionary phenotypic change is expected only after morphs are lost and these constraints are removed (*8*, *50*, *51*). Our results contrast with this expectation. This presents an apparent paradox—how can rapid divergence in coloration, morphology, and behavior occur if balancing selection limits evolutionary change? We propose that this paradox is resolved if the nigriventris syndrome initially evolved in isolation on an island before it became established on the Italian peninsula. For example, paleogeographic reconstructions of the central Tyrrhenian Sea support the existence of paleo-islands in the Rome Basin during the Pliocene and Pleistocene (*52*–*54*). Extant populations of wall lizards on small islands can become fixed for a single ventral color morph (*24*, *25*, *29*, *55*) and often show substantially different coloration, morphology, and behavior compared with populations on the mainland (*32*, *48*), differences that can be driven by drift or changes in selection due to low predation pressure and high population density (*24*, *56*, *57*). Under this scenario, island populations "escape" the constraints on phenotypic evolution imposed by polymorphism, enabling the subsequent introduction to the mainland of phenotypes that disrupt balancing social selection. We suggest that this process has contributed to the historical and geographic turnover of color polymorphism in Mediterranean wall lizards.

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SUPPLEMENTARY MATERIALS

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Materials and Methods; Supplementary Text; Figs. S1 and S2; Tables S1 to S3; References (*58*–*64*); Data S1 to S11

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