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An invasive non-native mammal population conserves genetic diversity lost from its native range

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Abstract

Invasive, non-native species are one of the major causes of global biodiversity loss. Although they are, by definition, successful in their non-native range, their populations generally show major reductions in their genetic diversity during the demographic bottleneck they experience during colonization. By investigating the mitochondrial genetic diversity of an invasive non-native species, the stoat *Mustela erminea,* in New Zealand and comparing it to diversity in the species' native range in Great Britain, we reveal the opposite effect. We demonstrate that New Zealand stoat population contains four mitochondrial haplotypes that have not been found in the native range. Stoats in Britain rely heavily on introduced rabbits *Oryctolagus cuniculus* as their primary prey, and were introduced to New Zealand in a misguided attempt at biological control of rabbits, which had also been introduced there. While invasive stoats have since decimated the New Zealand avifauna, native stoat populations were themselves decimated by the introduction to Britain of Myxoma virus as a control measure for rabbits. We highlight the irony that while introduced species (rabbits) and subsequent biocontrol (myxomatosis) have caused population crashes of native stoats, invasive stoats in New Zealand, which were also introduced for biological control, now contain more genetic haplotypes than their most likely native source.

Key Words: *Mustela erminea*, stoat, bottleneck, myxomatosis, epizootic, genetic variation

Running title: An invasive ark for genetic diversity

Introduction

The number of individuals within a population substantially influences the amount of genetic variation maintained over time, with rapid reductions in population size significantly lowering genetic diversity (Nei et al. 1975, Lynch & Gabriel 1990, Lande 1998). Examining contemporary patterns of genetic diversity may therefore allow us to infer the historical sizes of populations, and the processes that caused historical demographic bottlenecks (Avise 2004, Frankham et al. 2010, Allendorf et al. 2012). Introduced populations provide an unplanned opportunity to investigate the genetic consequences of population bottlenecks, with a general prediction of reduced genetic diversity compared to their source population, due to their small founding sizes. For most introduced populations this is the observed pattern, though higher numbers of individuals introduced, more introduction events, and more source populations may buffer against such genetic losses (Dlugosch & Parker 2008).

In 1884, stoats (*Mustela erminea*) were introduced to New Zealand from Britain for the purposes of invasive rabbit (*Oryctolagus cuniculus)* control (Thompson 1922). Over the following 20–30 years they arrived in large numbers on a series of shipments, organized both by the New Zealand government and private associations (King & Murphy 2005). In the first two years of introduction, at least 224 stoats were released in New Zealand (King 1984), and in 1885 around 3,000 stoats and weasels (*Mustela nivalis*) were sent from Lincolnshire alone (King & Moors 1979). Determining the exact number of stoats that were successfully liberated in New Zealand is impossible from historical records, as shipments often did not separate weasels from stoats in tallies, and the records of most liberations have been lost; however the total number of stoats liberated is likely to have been several thousand. The bulk of known shipments were to New Zealand's South Island where rabbits were particularly numerous (Thompson 1922, King 1984). Stoats were liberated in sufficient numbers and in so many places that they had spread throughout both main islands of New Zealand by the end of the nineteenth century (King & Murphy 2005).

As part of an investigation of the colonization history of New Zealand, we examined patterns of the distribution of genetic variation of stoats in New Zealand and compared this with Great Britain. This comparison has the potential to elucidate the relative demographic histories of each population subsequent to their separation. Because the size of the British stoat population has fluctuated significantly subsequent to the separation of the two populations in response to fluctuating rabbit numbers – with a known severe demographic bottleneck in the 1950s due to myxomatosis (King & Powell 2007), these specific variations were accounted for in our analysis.

Methods

Sample collection and DNA extraction

Spatially representative stoat samples were collected as part of pest control operations from 16 locations throughout New Zealand (DRYAD: doi:10.5061/dryad.h77tb). Five individual stoats were sampled from each location yielding a total of 80 stoats sampled. Tissue samples were taken from each individual and DNA was isolated using a Bio-Rad AquaPure Genomic Tissue Kit (Cat# 732-6343) following the manufacturer's protocol.

PCR and sequencing

As mitochondrial DNA (mtDNA) is a particularly sensitive indicator of population bottlenecks for the maternal lineage (Avise 2004), the hypervariable region 1 (HVR1) of the mitochondrial control region was chosen for this study. This was amplified with the primers DS1 and MER-R (Kurose *et al.* 1999). PCR amplifications were performed in 25µl reactions containing 1µl of DNA extract, GeneAmp 1x PCR Gold buffer (15mM Tris-HCl, 50mM KCl, pH 8.0), 2.5mM MgCl2, 200 µM each dNTP, 0.5µl of 10µM each primer and 0.3µl of AmpliTaq Gold DNA Polymerase (Applied Biosystems). Amplification was carried out in a GeneAmp PCR System 9700 thermocycler (Applied Biosystems) under the following conditions: 95 ^{o}C 7 min, followed by 35 cycles of 94 ^{o}C 1min, 55 ^{o}C 1 min, 72 ^{o}C 2 min, and a final extension at 72 ^{o}C 10min. PCR Products to be sequenced were

purified using a High Pure PCR Product Purification Kit (Roche Diagnostics). Purified products were then sequenced on an automated Applied Biosystems Model 310 sequencer (Applied Biosystems, Inc).

Haplotype networks

Sequences were aligned in Geneious 7.1.5 (Biomatters) using geneious global multi-alignment assuming 93% similarity. A maximum parsimony network was then created of these New Zealand control region haplotypes, along with 186 haplotypes from stoats from across Eurasia collected and sequenced by Martínková and colleagues (2007) using the software TCS (Clement et al. 2000). The level of difference in haplotype composition between populations was tested for using Fisher's exact test. The detection threshold for haplotype frequency was determined using the equation $(1-f)^n = \alpha$ where f = true frequency in the population, n = number of samples and α = alpha level.

Genetic drift simulations

Two sets of Wright-Fisher genetic drift simulations were then run in the software R to investigate the effect of the two demographic bottlenecks (1. arrival of stoats in New Zealand, 2. myxomatosis in Britain) on mitochondrial haplotype frequencies. The R code for these simulations can be obtained at (DRYAD doi:10.5061/dryad.h77tb). Simulations were set to run from the year 1883 (before the introduction of stoats to New Zealand in 1884) until 2000. All simulations were repeated with 1,000 iterations, and population numbers are for female stoats only to represent the sampling of the maternally inherited mtDNA haplotypes.

The first simulations were performed to investigate if the population bottleneck caused by the introduction of stoats to New Zealand, and subsequent expansion of the New Zealand population, could have led to a significant change in allele frequency. From a British population of 300,000 (King & Powell 2007) with the current New Zealand haplotype proportions, two simulations were run – one with a founding population of 100 females, and one with 500 females. The known number of stoats released in New Zealand was 224 in the first two years

(King 1984), and the large number of unrecorded releases subsequent to this mean the true total is likely to be far greater (King & Powell 2007); therefore the first simulation is for a very conservative (small) bottleneck scenario, and the second a more realistic one (though still conservative). The simulated population then grew, doubling every year until it reached 100,000, at which point it fluctuated around this value with a normal distribution N(100,000, 10,000). This value of 100,000 stoats in New Zealand was derived from a pre-breeding population estimate of 200,000 (King and Powell 2007).

The second set of simulations was performed to investigate how severe the demographic bottleneck needed to be for the British stoat population to show the observed haplotype proportions (if a genetic bottleneck was responsible for the difference in haplotype frequencies). As the first simulation indicated that the New Zealand stoat population bottleneck is unlikely to have changed the haplotype proportions greatly (see Results), these were used as the initial starting values for the British stoat population. The pre-bottleneck population (1883–1952) was defined as normally distributed N(300,000, 3,000). The minimum population size was then varied (100, 500, 1,000) with durations from 1–3 years. The population size was then modelled to be normally distributed N(20,000, 5,000) for the subsequent 10 years, and this was followed by an increase to a normal distribution N(100,000, 10,000). The values for the population sizes were derived from an assumption that the current British stoat population is roughly equal to the New Zealand population: this is a conservative estimate as current estimates have it as larger (King and Powell 2007). The relative sizes of the population over time were estimated from the trapping records (Tapper unpublished in King and Powell 2007), assuming that these can be extrapolated to the population as a whole.

Results

Five haplotypes were found from 80 New Zealand stoats (GenBank Accession # KP307771-KP307775), alignment: (DRYAD doi:10.5061/dryad.h77tb).. The

most common haplotype (NZ_hap_1) is identical to the single haplotype observed in all British stoats, and the other four haplotypes were most closely related to the British haplotype (differing from it by only one base pair), and more distantly related to the mainland European and Irish haplotypes (Figure 1). The range of haplotypes was significantly higher in the South Island of New Zealand, with haplotypes 2–4 only recorded here (Figure 1).

With only one haplotype detected in the British stoat population, the total proportion of all haplotypes other than this haplotype must be <5.12% with 95% confidence, and <7.76% with 99% confidence, given the level of sampling. Given this detection threshold, both haplotypes 2 and 3 should have each been detected in Britain – if the haplotype frequencies were the same in the New Zealand and British populations, and it is extremely likely that at least one of the combinations of haplotypes 2–5 would to be detected. New Zealand as a whole is highly significantly more diverse than Britain ($p < 0.001$), with stoats on the South Island being significantly more genetically diverse than those on the North Island ($p < 0.001$).

The simulation of the New Zealand stoat population haplotype fluctuations illustrates that large increases in the proportions of rare alleles are unlikely (figure 2). In some simulations rare alleles (4 and 5) did however go extinct in New Zealand due to genetic drift (figure 2). From these simulations, over 95% of genetic drift was observed in the first six years when population sizes were small (up to 3,200). Subsequent to this, the haplotype proportions remained reasonably constant (figure 2).

For the simulation of the British stoat population, the size and variability of the populations outside of the acute bottleneck phase had minimal effect on levels of genetic drift; the only important variables that determined the level of drift were the minimum population size (N_{min}) and the duration of this bottleneck – as expected from random genetic drift. The sudden increase in genetic drift caused by the bottleneck associated with myxomatosis can be seen in figure 3. In order to obtain results which reflected the decrease in frequency of haplotypes 2–5

below the detection threshold, an extremely severe bottleneck well below 1% of the original population (with an $N_{e(f)}$ of less than 100) for several years was required (Figure 4). This detection threshold is the probability of detecting at lease one of these four alleles which were not detected in Britain given the sampling effort (57 samples), when randomly sampling a population with the final allele frequencies derived from each bottleneck simulation.

Discussion

Our results show that the introduced invasive stoat population of New Zealand is significantly more diverse than native stoats in Britain, based on mitochondrial DNA control region sequence data. There are five possible mechanisms that could produce this observed pattern: 1) multiple source populations for New Zealand stoats, 2) insufficient sampling of British stoat genetic diversity, 3) genetic drift following foundation causing an increase in previously rare alleles in the New Zealand population, 4) in-situ mutation in the New Zealand stoat population, and 5) a significant genetic bottleneck for British stoat populations in the 20th Century. Our results support the 5th scenario, for reasons outlined below

The available data do not support the establishment of genotypes in New Zealand from outside of Britain, as the New Zealand haplotypes are most similar to the single British haplotype, which differs from all haplotypes detected in mainland Europe by at least two substitutions. This supports the belief that the New Zealand stoat population was founded entirely from one or more British sources - as historical records imply (King & Powell 2007). Sampling in Britain was widespread, therefore it seems unlikely that unsampled spatial structure is responsible for the lack of diversity observed within the British stoat population.

Using the simulation of genetic drift for the introduction to New Zealand we demonstrate that the significant increase in rare alleles required for the observed pattern is unlikely to occur. While rare alleles can surf the expansion

fronts of introduced populations, rapidly expanding in frequency (Excoffier and Ray 2008) it remains improbable that four rare haplotypes were all independently significantly amplified in frequency. Similarly, the probability of four independent mutations arising and independently surfing to high frequency is unlikely, though we cannot rule either of these scenarios out completely due to the many unknown parameters of the population expansion and mutation rate.

Our data is most consistent with a scenario where British stoats went through a very significant genetic bottleneck after the introduction of stoats to New Zealand, resulting in the loss of mitochondrial diversity in the population. Given that trapping records show a very significant demographic bottleneck in British stoats due to myxomatosis in the 1950s (Jefferies & Pendlebury 1968; King & Powell 2007), this event is the most likely cause for this hypothesized genetic bottleneck for British stoats.

European rabbits are not native to Britain, having been introduced around 2,000 years ago – though they did not become naturalized in the wild until around the mid 12th century (Lever 2009). When the myxoma virus arrived in Britain, the UK government supported its spread as a biological control agent for this agricultural pest (Bartrip 2008). The rapid spread and severity of the myxomatosis epizootic detrimentally affected a number of native predator species in Britain, particularly the stoat (Sumpton & Flowerdew 1985). Native British stoats had become particularly specialized and reliant upon the introduced rabbits (McDonald et al. 2000, McDonald 2002), with rabbits forming over 80% of their diet before the myxomatosis epizootic (Southern 1956). From 1953 to 1955 myxomatosis arrived in Britain, causing over 99% of the rabbit population to die (Sumpton & Flowerdew 1985). This loss of prey had a drastic effect on the British stoat population. Poor breeding success for stoats was found immediately after myxomatosis (Lockley 1956). Young stoats died of starvation (Haslam 1955), and fewer pregnant or nursing females were observed than in previous years (Hervey 1955). Significant changes in diet and behaviour were noted, with an increased reliance on invertebrates, birds and rodents, often including prey items not usually eaten by stoats (Cobnut 1955, Brown 1955, Day

1968). These changes in diet did not prevent a dramatic population crash: the average number of stoats caught annually decreased by 84% for the ten years following the myxomatosis outbreak across records kept by game estates (Jefferies & Pendlebury 1968; King & Powell 2007). On some of these game estates, there were no stoats caught during a full calendar year despite continued effort and high and increasing tallies of weasels (*Mustela nivalis*) (King & Moors 1979, King & Powell 2007). Over the whole of Britain, stoats remained scarce with the demographic bottleneck lasting 10–15 years.

The New Zealand and British stoat populations had different demographic histories around the time of their respective population bottlenecks, which are likely to have affected the severity of the genetic bottlenecks experienced. The New Zealand population rapidly expanded after the initial releases (King & Murphy 2005), which would minimize the loss of genetic diversity; while in Britain the severe demographic bottleneck lasted for several generations and recovery was slow: a scenario particularly likely to result in the loss of genetic diversity (Allendorf et al. 2012). If this demographic bottleneck is responsible for the observed difference in haplotype diversity between New Zealand and British stoat populations, our simulations indicate that the British stoat population bottleneck must have collapsed to an effective female population size across Britain of less than a few hundred for several years to account for the present lack of diversity. Due to the simplifying assumptions of the simulations we do not try to interpret this in terms of actual stoat numbers; we simply highlight that the demographic bottleneck must have been extremely severe to cause the implied loss of diversity.

Historical DNA has been obtained from museum specimens of stoats (Martínková & Searle 2006), and further investigation of historical samples may better elucidate the former mitochondrial diversity of British stoats. Using modern methods for capturing ancient DNA (e.g. Bahcall 2013) it should also be possible to obtain nuclear DNA from these historical samples, to examine if nuclear diversity has also been lost from Britain, but potentially retained in New Zealand stoats.

Introduced populations generally lose genetic variation relative to their source populations (Dlugosch & Parker 2008). Of the genetic studies of introduced populations reviewed by Dlugosch & Parker (2008), only 6/76 had increased (>10%) allelic diversity in the introduced population, and in each of these studies there was either a significant sampling bias towards the introduced population, or there were multiple introductions from multiple source populations, which has allowed an accumulation of this widely sampled variation (e.g. Ellstrand & Shierenbeck 2000, Genton et al. 2005, Kolbe et al. 2008, Kennington et al. 2012). Our results suggest that introduced populations can act as an 'ark for genetic diversity' lost from their native populations. This result has similarities to those of Martínková et al. 2014, who found that introduced voles (*Microtus arvalis*) in the Orkney Islands retained ancestral mitochondrial diversity that had been lost from mainland Europe in the 5000 years since their introduction to these islands. Red squirrels (*Sciurus vulgaris*) in Ireland, which are admixed between native and introduced squirrels from Britain, have also retained genetic diversity lost from Britain (Finnegan et al. 2008). Genetic analyses have also shown that supposedly extinct sub-species have survived in introduced populations, with invasive tammar wallabies (*Macropus eugenii*) in New Zealand shown to be a supposedly extinct subspecies from South Australia (Taylor & Cooper 1999). Reintroducing species to parts of their native range where they have been extirpated is now common conservation measure (Ewen et al. 2012). Throughout Europe and in the UK in particular, once common birds such as the grey partridge (*Perdix perdix*), and the cirl bunting (*Emberiza cirlus*), which have introduced populations elsewhere, have been declining dramatically due to changed farming practices (Gregory et al. 2007). As we move toward conserving genetic biodiversity, the genetic diversity conserved in introduced populations may also be considered valuable and therefore worthy of reintroduction.

An important aspect of these results is that we interpreted them in light of a known demographic history for both populations, and the interpretation would potentially be different if we did not have this prior information. Many studies

infer demographic and colonization history based on contemporary patterns in genetic diversity, such as studies of glacial refugia in Europe (e.g. Hewitt 1999, 2000). If our results were obtained from populations where the history of each population was unknown, the interpretation could be that the more diverse population (New Zealand) was a source population or refugium, and the British population was an expansion/introduction from New Zealand. Because of this, care is needed in interfering historical processes from contemporary genetic variation, as it may be difficult to distinguish between a source population losing variation, and an expansion/introduction scenario.

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Data Accessibility

All data and simulations have been uploaded to dryad (doi:10.5061/dryad.h77tb) and genbank (GenBank Accession # KP307771-KP307775) for publication.

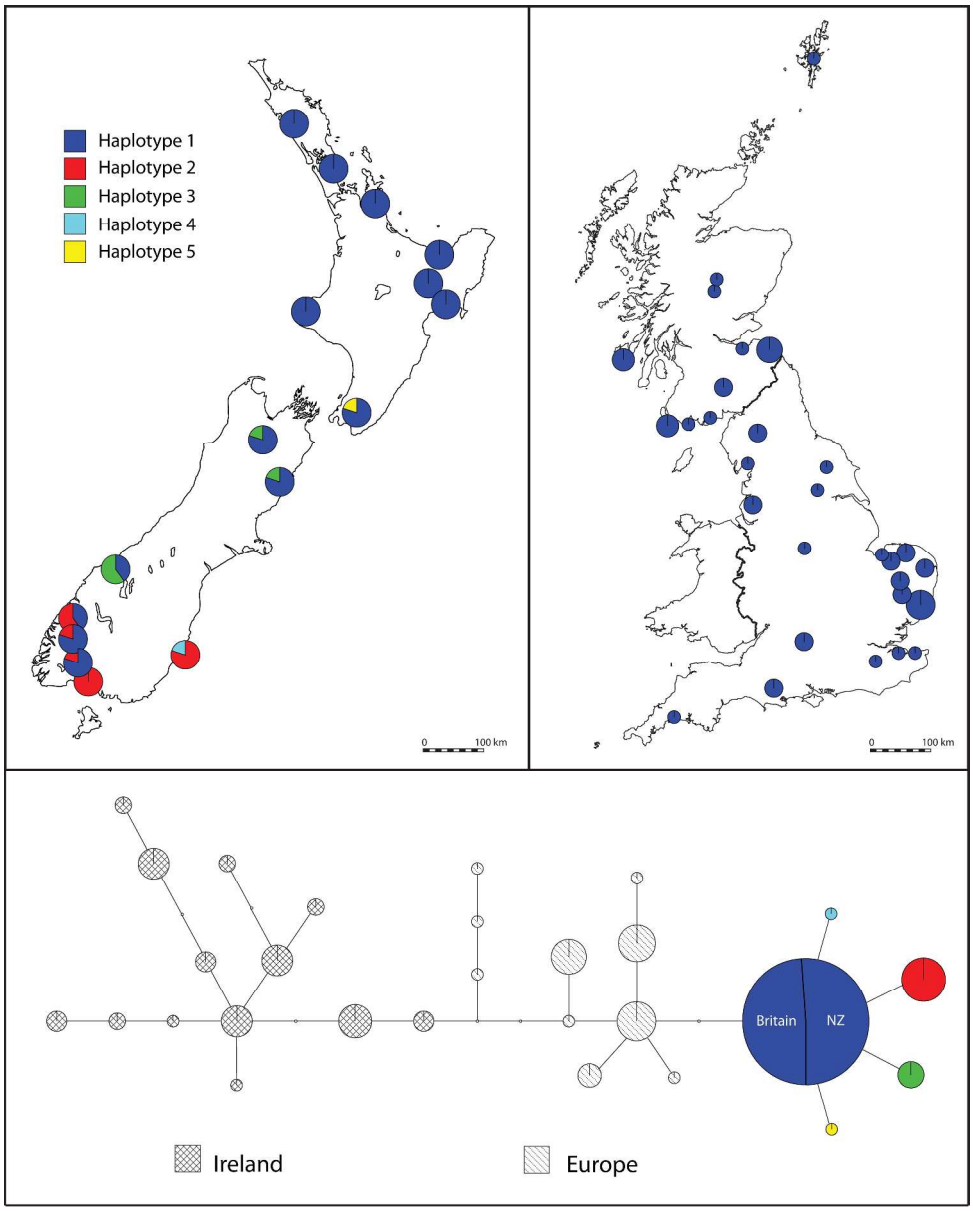
Figure legends:

Figure 1 Distribution and haplotype network of mitochondrial control region haplotypes sampled from across New Zealand and Britain, including samples from mainland Europe and Ireland. The size of each circle represents the sample size from each location.

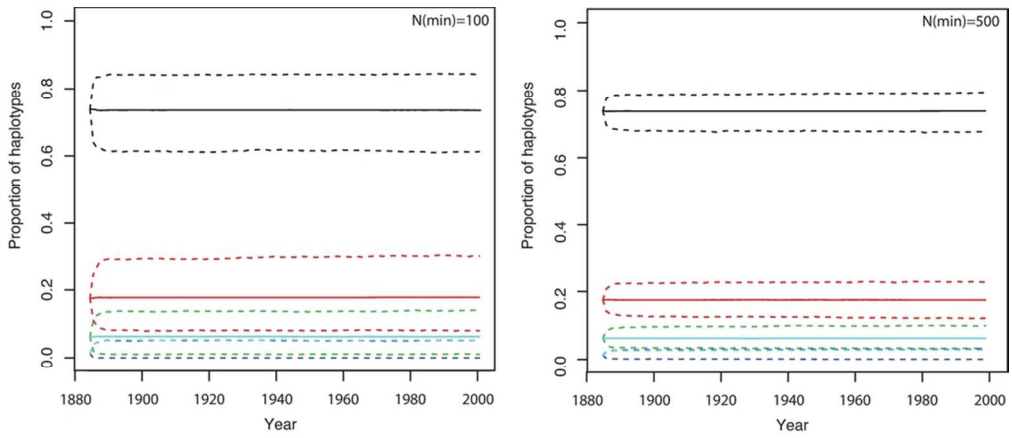
Figure 2 Average proportions and 95% confidence limits of alleles 1 – 5 in two simulated bottleneck scenarios for the New Zealand population assuming the present day sampled proportions of haplotypes were present in 1883. From top to bottom: haplotype 1 (black), haplotype 2 – (red), haplotype 3 (green), haplotype 4 & 5 (blue). Confidence limits show that 95% of all simulations had haplotype frequences within this range.

Figure 3 Average proportions and 95% confidence limits of alleles 1 – 5 in the simulated mysomatosis bottleneck scenario for the British stoat population, assuming the present day sampled New Zealand proportions of haplotypes were present in Britain in 1883. Minimum population size = 100 Females for three years. From top to bottom: haplotype 1 (black), haplotype 2 – (red), haplotype 3 (green), haplotype 4 & 5 (blue). Confidence limits show that 95% of all simulations had haplotype frequences within this range.

Figure 4 Final haplotype percentages with 95% condidence intervals for the combined haplotypes 2 – 5 under varying bottleneck scenarios for the British stoat population: (N(min) = 100, 500, 1000), bottleneck duration = (1,2,3 years). Detection thresholds given the sampling in Britain are shown as dotted and dashed lines.



Distribution and haplotype network of mitochondrial control region haplotypes sampled from across New Zealand and Britain, including samples from mainland Europe and Ireland. The size of each circle represents the sample size from each location.
397x495mm (300 x 300 DPI)



Average proportions and 95% confidence limits of alleles 1 – 5 in two simulated bottleneck scenarios for the New Zealand population assuming the present day sampled proportions of alleles were present in 1883. From top to bottom: haplotype 1 (black), haplotype 2 – (red), haplotype 3 (green), haplotype 4 & 5 (blue). Confidence limits show that 95% of all simulations had haplotype frequences within this range.
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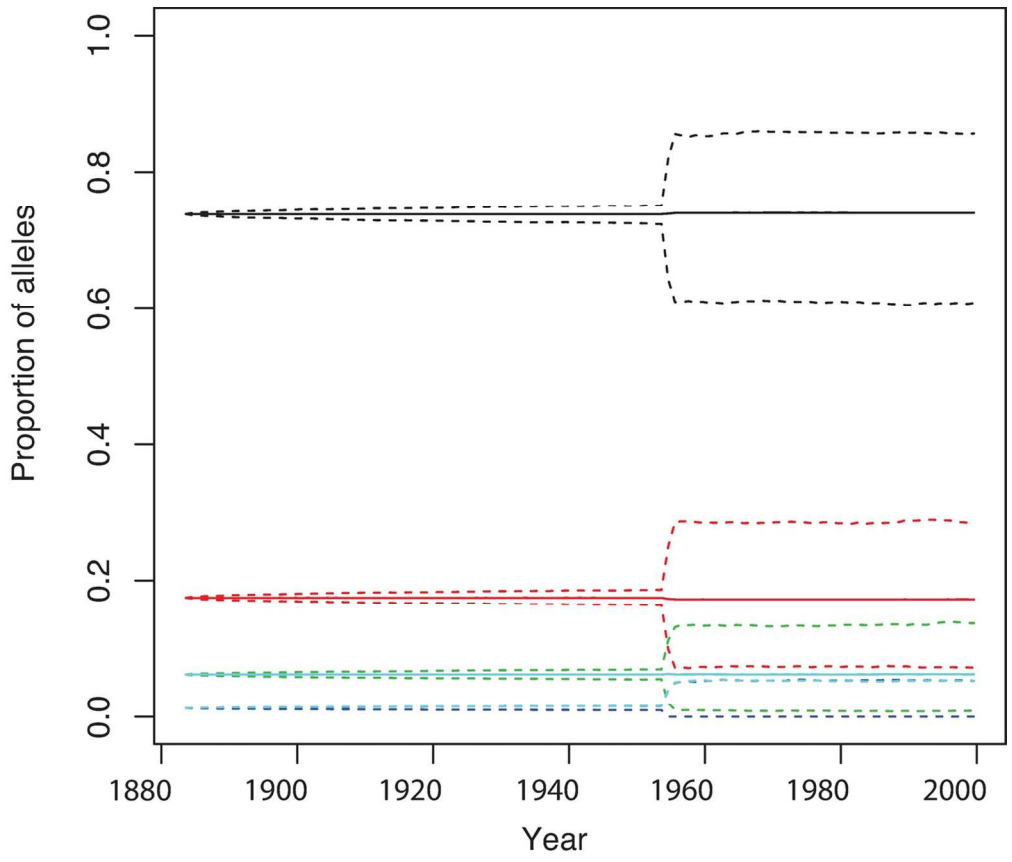

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120x108mm (300 x 300 DPI)