# ST. KILDA SOAY SHEEP PROJECT: ANNUAL REPORT 2008

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## POPULATION OVERVIEW

The sheep population on Hirta entered 2008 at a moderately high level and, as a result, there was a slightly higher level of mortality than normal in a non-crash year. 60 tagged sheep were found dead within the study area between March and May of 2008. Lambing began on the 23rd of March with 79.2% of lambs born surviving (Fig. 1).

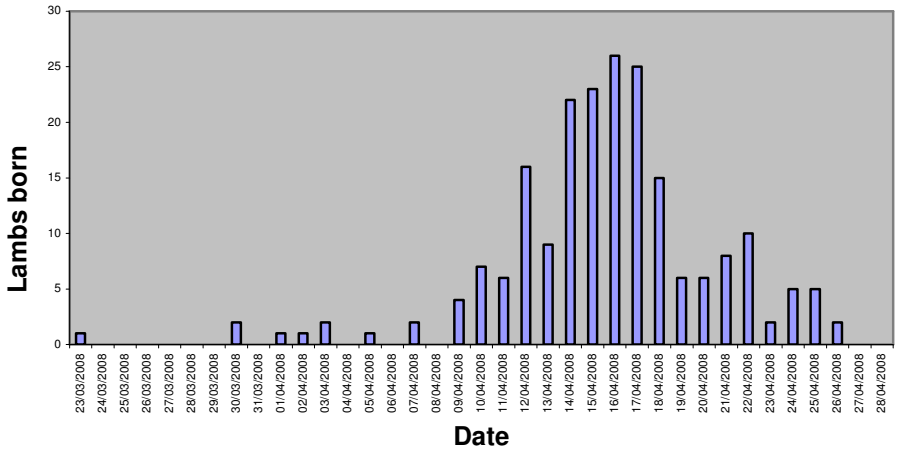


***Figure 1.*** *The temporal distribution of lamb births during 2008.*

In December 2008, 729 tagged sheep were believed to be alive on Hirta, of which 567 regularly used the study area, a total increase of 27% using the study area since the previous year. The age distribution of the population is shown in Fig. 2 and changes in sheep numbers in the study area over time are shown in Fig. 3.

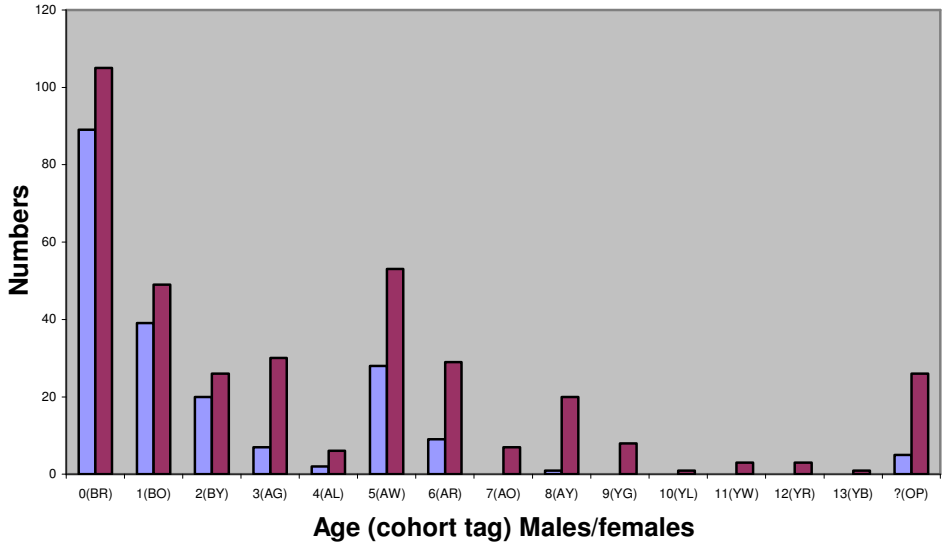


***Figure 2.*** *Age distribution of tagged Soay sheep presumed to be alive at the end of 2008.*

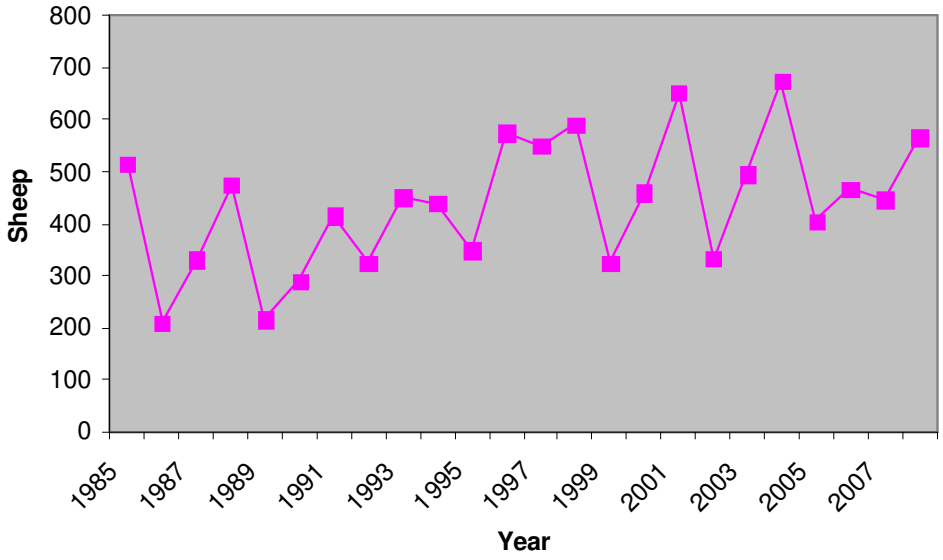


***Figure 3.*** *The number of tagged sheep regularly using the study area since 1985.*

One whole-island count yielded 1909 tagged and untagged sheep, with the details displayed in Table 1. The total population had increased by just over 24% since summer 2007, when it was at 1538. This gives a delta (calculated as ln ($N_{t+1}/N_t$)) of 0.216.

***Table 1.*** *Demographic and geographic distribution of sheep observed during the count of Hirta on August 8th 2008. Coat colours are DW = dark wild, DS = dark self, LW = light wild, and LS = light self.*

| Location | Females | | | | Males | | | | Lambs | Total |
|---|---|---|---|---|---|---|---|---|---|---|
| | DW | DS | LW | LS | DW | DS | LW | LS | | |
| **Conachair/Oiseval** | 185 | 10 | 75 | 7 | 88 | 3 | 21 | 0 | 199 | **588** |
| **Mullach Bi/Cambir** | 219 | 19 | 82 | 9 | 85 | 3 | 12 | 3 | 227 | **659** |
| **Ruaival/Village** | 230 | 12 | 78 | 3 | 58 | 1 | 27 | 0 | 253 | **662** |
| **Total** | **634** | **41** | **235** | **19** | **231** | **7** | **60** | **3** | **679** | **1909** |

## REPORTS ON COMPONENT STUDIES

### Vegetation.

Mick Crawley.

There were 1909 animals in the whole-island count in August 2008. The 2007 count of 1538 predicted a population increase (delta) = 0.216 and hence a predicted whole island count of 1974 (which turned out to be too high by just 3.4%). Using the same simple model with this year's population of 1909 predicts a major crash (delta = -0.74) and hence a whole island count of only 911 in August 2009. We await with interest to see whether or not the predicted crash occurs. The vegetation in August 2008 certainly predicted hard times ahead for the sheep, with average sward height on the inbye of 8.35cm compared with the long-term average sward height of 11.5cm. Only one previous year has had a lower mean sward height (8.21cm) and this was the post-crash August of 1999 (2002 was close at 8.84cm, another post-crash August). Gap cover is another indication of relatively high grazing pressure, and the gap cover measured in August 2008 (38.3%) was the highest ever measured at this time year. Palatable grass biomass (mean = 4.43g per 20x20cm quadrat) was the lowest ever measured by a large margin (the next lowest was 7.43g in 2007 and 7.97g in 2000), because mean gap cover was so high in 2008.

Flowering in the grasses was relatively low (but not record-low in any of the main species) but the three commonest herbs were all at record low flower-stem densities (*Leontodon*, *Ranunculus* and *Potentilla*). Productivity (as measured by mean pluck-down inside the pyramids) was down to record lows at Gun Meadow and St Columba's, presumably as a result of the very low rainfall during the rapid growth phase (May and June). The more productive grasslands (Mid Fields and West Meadow) had lower values than 2008 in all years prior to 2005.

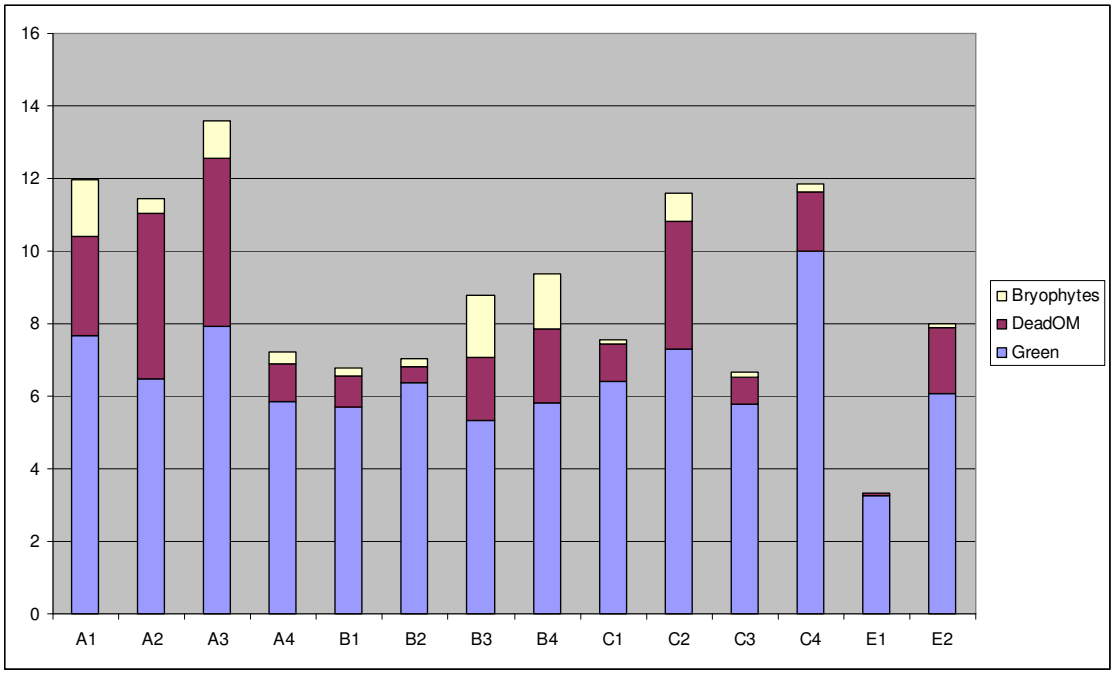


***Figure 4.*** *Spatial heterogeneity in biomass (mean g dry matter per 20x20cm quadrat) and sward composition at the 14 inbye sample locations on transects A, B, C and E. Total green leaf material (grass plus herbs = Green) varied by a factor of more than 2, and dead organic matter (Dead OM) was also highly variable.*

There was substantial spatial heterogeneity in total biomass and sward composition (Fig. 4) along the transects within the Head Dyke (the inbye swards). The seaward end of transect E (E1) was exceptionally closely grazed, with virtually no dead organic matter or bryophyte, while station 3 on transect A (just above The Street) had high amounts of standing dead material within the sward.

The predictions in the first paragraph were made in August 2008, and there may have been sufficient late-season grass growth in autumn 2008 and winter 2009 to rescue the sheep population from a crash. We shall know by March 2009.

**Dynamics of Phenotypic Change and the Shrinking Sheep.**

Arpat Ozgul, Shripad Tuljapurkar, Tim Benton, Josephine Pemberton, Tim Clutton-Brock and Tim Coulson.

Increasing numbers of empirical studies have reported rapid change in trait values in wildlife populations. Such trends could be explained by a range of ecological and evolutionary effects. One interesting question for ecologists and evolutionary biologists is how much of a role evolution plays in the observed change. However, ecological and evolutionary effects are intimately intertwined in changing environments, and separating these effects has proven to be a difficult analytical challenge. As a consequence, we do

not have a good understanding of the dynamics of phenotypic traits in natural environments.

In Soay sheep, mean body size has fluctuated substantially over the past 25 years, and has, on average, declined in all age classes, providing a perfect example of a systematic trait change in a wild population (Fig. 5). Using a novel analytical approach (the age-structured Price equation) and the exceptional long-term demographic data, we have decomposed the observed change in this phenotypic trait into different contributing processes, including selection and response to selection.

Our novel approach provides new insight into the dynamics of phenotypic traits. We show that evolutionary change has contributed relatively little to the observed dynamics; selection and response to selection explained less than 10% of the observed fluctuations in mean body weight. The majority of annual variation is caused by processes other than selection; in particular, fluctuations in the population structure and the growth response of younger sheep to changing environmental conditions explained 88% of the observed variation.

To find out why sheep are getting smaller, we examined trends in different components of the observed change in mean body weight. During their first year of life, lambs have been growing more slowly than they used to (on average by 93 g per year; Fig. 6). We also found that lambs grew more slowly in years of high population size and bad weather. Winters on St. Kilda have warmed and shortened over the course of the study, which has resulted in an increase in lamb survival and a subsequent gradual increase in population size on the island. These findings suggest that a change in the interplay between climate and population size has had interesting phenotypic consequences.

Our research provides the most detailed and exact analysis of phenotypic trait dynamics ever undertaken. We show that both the long-term declining trend and annual fluctuations in mean body size are primarily a consequence of environmental variation. These results also indicate that phenotypic trends are not necessarily evidence for evolutionary change and reinforce the need for a dynamic theory of evolution.

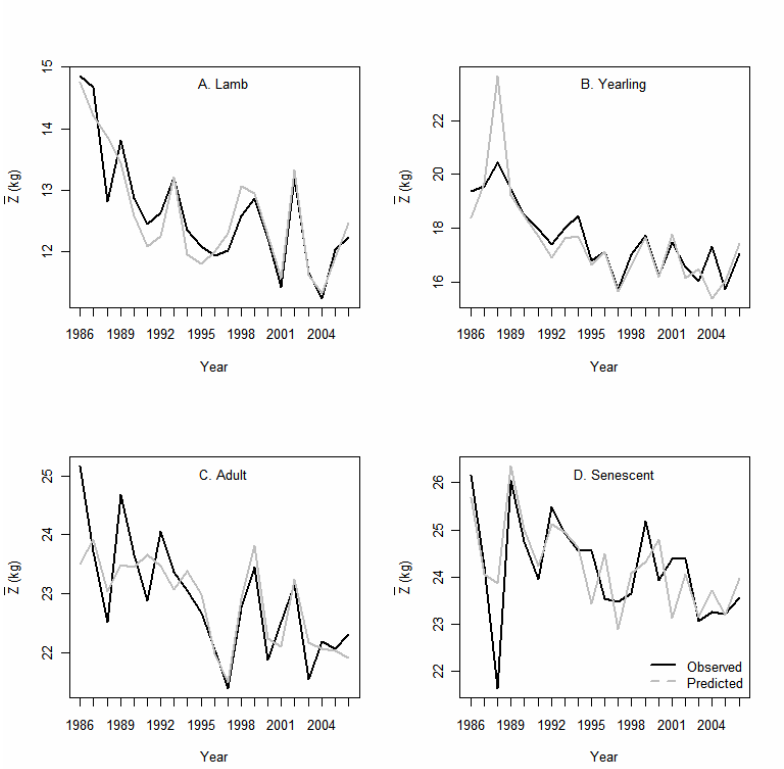


**Figure 5.** *Mean annual August weights for (A) lambs, (B) yearlings, (C) adults and (D) senescent female Soay sheep. The black lines show the observed fluctuations and long-term decline in mean weight, and the grey lines show those obtained from the application of the age-structured Price equation.*

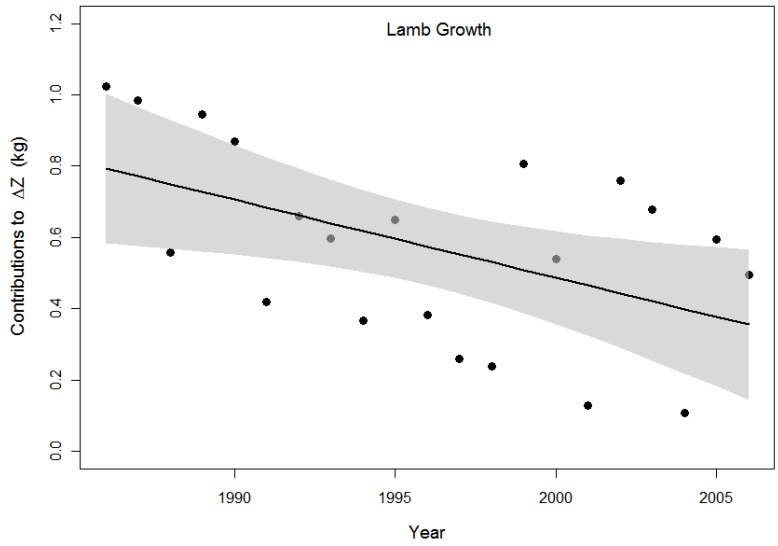


***Figure 6.*** *Temporal trend in the contributions of growth during the first year of life to changes in mean body weight. Shaded area indicates the 95% confidence limits.*

**Environmental stress predicts senescence in parasite resistance more effectively than chronological age in wild Soay sheep.**

Adam Hayward, Alastair Wilson, Jill Pilkington, Josephine Pemberton, and Loeske Kruuk.

Biological senescence, often simply referred to as ageing, is an intrinsic decline in performance and fitness that affects virtually all organisms. However, it is still unclear as to whether the ageing process is entirely intrinsic, and can be measured by age in years ('chronological' age), or whether external factors such as environmental conditions and stresses experienced through life are more important. True 'biological age' remains an unquantifiable concept, and is estimated by approximate measures of ageing, of which 'chronological' age is just one, and of which 'environmental' age, as we term it here, is another.

The Soay sheep population of St Kilda provides an ideal system for studying ageing, since it has been monitored intensely for over twenty years, and a range of data are available for many hundreds of individuals, documenting their life histories, parasitism, population dynamics, and environmental conditions. Individuals may be infected with a number of parasite species, including single-celled protozoa, intestinal worms, and external biting parasites, particularly wingless flies known as keds. The most prevalent parasite species in the population are strongyle nematodes, a group of intestinal helminths which contribute to loss of condition and mortality over winter. Strongyles lay eggs in the gut lumen, and adult worm burden can be estimated by faecal egg counts (FEC). FEC is known to be highest in the youngest individuals, before reaching a stable lower level by the age of two. It is, however, unknown how FEC changes during adulthood and in particular during later life. Work in other study systems has shown a decline in immune performance with increasing age, but no study as yet has identified how within-individual parasite burden changes with age in a wild system. In our study, we used two measures of ageing to determine how FEC changes with age in the Soay sheep population, a 'chronological' and an 'environmental' measure.

By using a statistical technique known as mixed-effects modelling, we were able to identify how within-individual FEC changes with age in both male and female Soay sheep. We also identified a number of other factors which influence parasite burden, such as population density, climatic conditions, and seasonality.

Surprisingly, we detected a significant decline in FEC with increasing chronological age in both female and male sheep (Fig. 7). This runs counter to our predictions, since one would expect ageing individuals to become increasingly susceptible to parasitism as immune system function declines with age. It is noticeable that the decline in males is shallower than in females, and that males thus suffer higher parasite burdens throughout life than females.

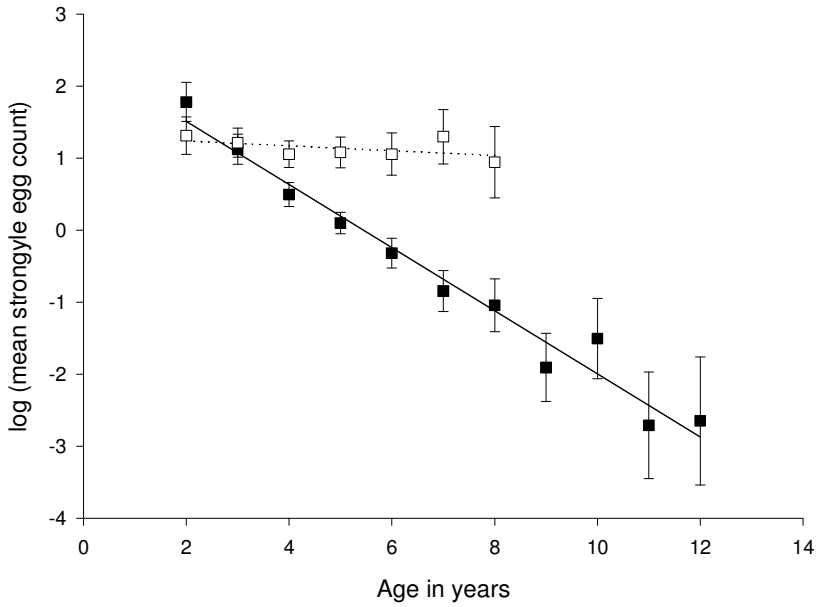


***Figure 7.*** *Both female (filled symbols, solid line) and male (open symbols, dotted line) show a significant decline in FEC with increasing age.*

We used two measures of environmental age. The population has unusually unstable dynamics, with periodic fluctuations in population size caused by rapid periods of growth and then large over winter mortality events, known as 'crashes'. Individuals surviving crashes are often in very poor condition following the winter, and so we used number of crashes experienced as a measure of the amount of environmental stress an individual has experienced, and as our first measure of 'environmental age'. In both males and females, and with age taken into account, individuals that experienced more crashes exhibited higher FEC, though this increase was only significant in females (Fig. 8). This indicates that individuals that have suffered a higher degree of environmental stress harbour higher parasite burdens. The failure to detect any effect of number of crashes experienced in males may be due to low sample sizes in the zero- and two-crash categories.

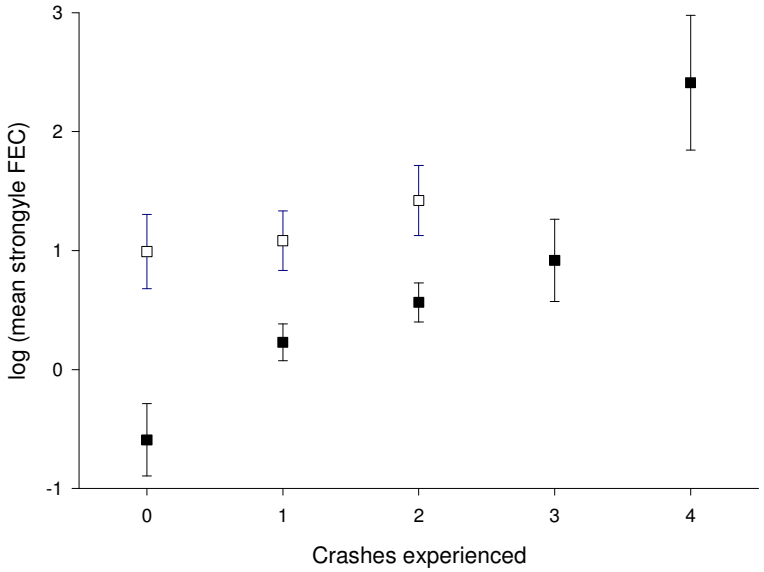


***Figure 8.*** *Female sheep (filled symbols) show a progressive increase in FEC with increasing number of population crashes experienced. In males, however, the increase is non-significant, possibly due to smaller sample sizes and therefore larger standard errors.*

The crudeness of this measure led us to a second measure of environmental age, which we refer to as cumulative environmental stress (CES). This measure was derived by summing the proportion of lambs dying in each year across an individual's life, with the assumption that years of high lamb mortality correspond to a poor and more stressful environment. Thus, individuals of the same age but of different cohorts (i.e. born in different years) will have experienced different levels of CES, with higher CES indicating experience of poorer environmental conditions. Our results showed a significant positive effect of CES on FEC in both female sheep, and a marginally non-significant positive effect on FEC in male sheep (Fig. 9). Thus, although 'chronological' age does not predict an increase in FEC in this population, two measures of 'environmental' age do predict an increase.

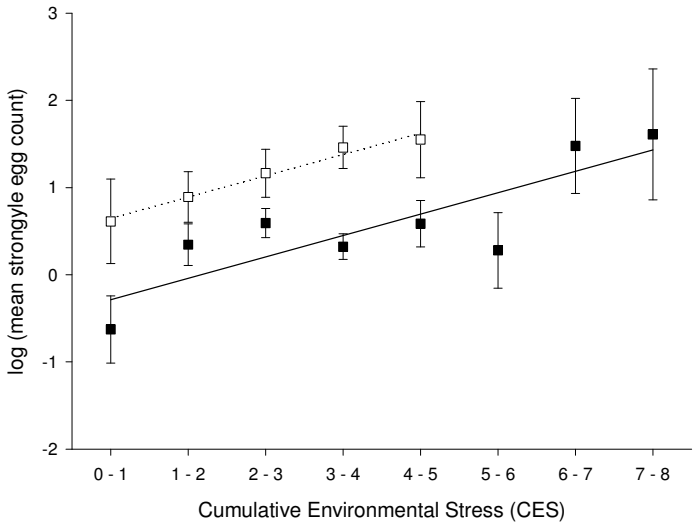


***Figure 9.*** *Female sheep (filled symbols, solid line) show a highly significant increase in FEC with increasing experience of environmental stress. Male sheep (open symbols, dotted line) show a similar increase, although it is marginally non-significant. As noted above, this may stem from a smaller sample size in males.*

These results, the first of their kind in a wild population, show that chronological age may not always correspond to biological age, particularly in variable environments. Whether controlling for chronological age or not, environmental age predicted an increase in FECs contrary to the decrease predicted by chronological age. While this is not evidence for biological senescence *per se*, it does emphasize that changes in characteristics with age are very much dependent on the external factors an individual experience as it ages, and that individuals that have suffered more cumulative external stress show reduced ability to resist parasite infection. In these circumstances, measures of age that capture environmental stresses experienced by an individual over time may be useful for understanding the process of senescence. The challenge now is to unpick precisely which environmental variables are responsible for the effects noted here, and to identify at what stage in the life of an individual these variables have the greatest effect.

**The environmental conditions on St. Kilda influence relationships between characters.**

Matt Robinson, Alastair Wilson, Jill Pilkington, Tim Clutton-Brock, Josephine Pemberton and Loeske Kruuk.

The amount of food available to the Soay sheep of Hirta varies substantially from year-to-year as a result of weather conditions and the number of sheep on the island. This means that there are substantial differences between individuals in the conditions they experience throughout their life which can create differences between the sheep in their growth, in their resistance to parasite infection, in the length of horns that they grow, and in the length of time they survive. The sheep may also differ from one another because they have a different genetic make-up, or in other words, there might be a genetic basis to

the differences we see between individuals within this population, with some individuals inherently better than others at gaining food and dealing with parasites.

Most of the characteristics of the sheep that we measure are related to each other. For example, an individual with fewer parasites will be able to get more nutrition from the food they eat and so may be heavier than an individual with many parasites, and better able to invest energy into growing horns. So parasite abundance, weight and horn length will be related to each other to some degree and we may expect that differences between individuals in one character, whether created by the environment or by genes, should create the same differences in all related characters. However, this may not be true in all of the environmental conditions that the sheep experience. For example if environmental conditions are favourable and food is abundant, then the weight of a sheep may not be influenced to the same degree by how resistant they are to being infected by parasites. Therefore, having genes which reduce the chances of having parasites may not influence weight in the same way across all environments.

We examined this by testing whether individual differences in parasite load, body weight and horn length were constant across all of the different environmental conditions that a sheep may experience throughout its life. Environmental conditions were said to be good if many of the lambs born in a given year survived and poor if many of those born died. We found significant genetic relationships between weight and horn length and between weight and parasite resistance in males and some evidence that these relationships changed depending upon the environmental conditions that the males experienced (Fig. 10). This provides evidence that genes which influence particular characters in one environment may not always have the same effects across all environments. This is important because previous work has found that these characters may influence the chances of a sheep surviving or reproducing across its life and it may be that the same genes may not always have the same influence across all environmental conditions.

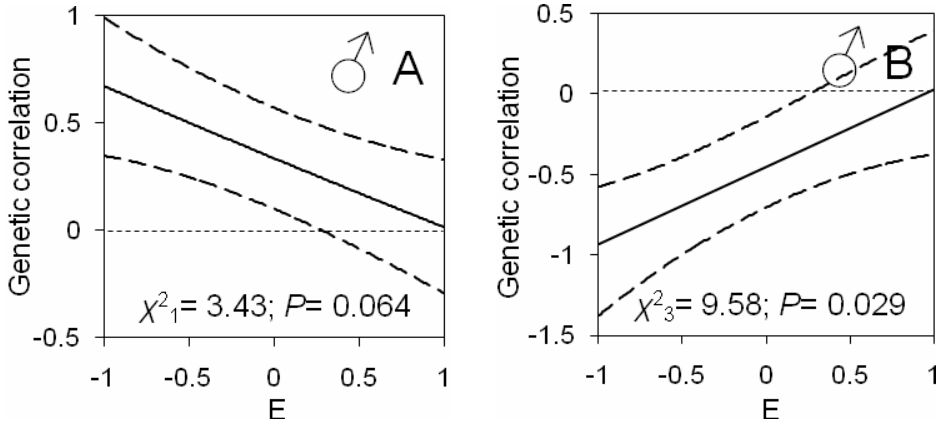


***Figure 10.*** *The genetic relationship between (A) body weight and horn length and (B) between body weight and parasite infection in males across environments (E) with -1 being a poor environment and +1 being a good environment. The graphs show strong genetic relationships in poor environments (genes for larger weight being associated with genes for larger horns and genes for fewer parasites) and weak relationships in good environments.*

**The genetic basis of coat pattern in Soay sheep.**

Jake Gratten, Jill Pilkington, Emily Brown and Jon Slate

Coat pattern is polymorphic in Soay sheep; individuals are either uniformly coloured, a phenotype referred to as 'self', or they are darker on the back than the belly, rump and around the eyes and mouth, a phenotype referred to as 'wild'. Inheritance of coat pattern is consistent with the action of a single gene at which the wild-type allele ($A^+$) is completely dominant to self ($A^a$). In previous Annual Reports (2004, 2006) we have shown that this gene is *Agouti signalling protein* (hereafter *Agouti*). What we have not yet characterised is the causal mutation (or mutations) at *Agouti* underlying the recessive self phenotype. This knowledge would enable us to differentiate wild-type sheep that have two copies of the $A^+$ allele from those with one copy of each allele, something that cannot be done visually. This is important because we could then measure the genetic relationship between coat pattern and key fitness traits more accurately and determine if selection plays a role in the maintenance of the polymorphism.

To determine whether the recessive *Agouti* mutation for self phenotype is a structural change affecting protein function, rather than a regulatory mutation that stops gene expression, we sequenced the entire coding region of the *Agouti* gene (comprising 402 base pairs spanning exons 2, 3 and 4) in six wild and six self-type sheep. This revealed a 5 base pair deletion at position 100-104 in exon 2 and two single base changes in exon 4; a G→C substitution at position 5051 and a T→A substitution at position 5172. These mutations have also recently been described in Australian domestic sheep. The exon 2 deletion (hereafter referred to as the 'deletion') is a frame-shift mutation that results in a premature stop codon in exon 3 and the production of an *Agouti* protein less than half as long (64 amino acids) as the wild-type protein (134 aa). This is likely to cause a loss of function, and as expected all six self-type sheep carried two copies of this mutation (Table 2). The G→C substitution in exon 4 has no effect on the amino acid sequence and can be ignored. On the other hand, the T→A substitution at position 5172 (hereafter referred to as the 'exon 4 mutation') is predicted to cause a cysteine to serine substitution in the functionally important signaling domain of *Agouti* and may also lead to loss of function.

***Table 2.*** *Coding sequence variation in the Soay sheep Agouti gene. Dots indicate identical sequence to that in the top row.*

| Sheep ID | Coat pattern | Nucleotide position | | |
|---|---|---|---|---|
| | | Ex 2: 100-104 | Ex 4: | Ex 4: 5172 |
| 6107 | Wild | AGGAA/AGGAA | G/G | T/T |
| 6109 | Wild | AGGAA/Deletion | G/C | T/A |
| 6112 | Wild | . | . | . |
| 6118 | Wild | AGGAA/Deletion | G/C | T/A |
| 6145 | Wild | . | . | . |
| 6182 | Wild | . | . | . |
| 6028 | Self | Deletion/Deletion | C/C | A/A |
| 6095 | Self | Deletion/Deletion | C/C | A/A |
| 6138 | Self | Deletion/Deletion | C/C | A/A |
| 6140 | Self | Deletion/Deletion | C/C | A/A |
| 6165 | Self | Deletion/Deletion | C/C | A/A |
| 6188 | Self | Deletion/Deletion | C/C | A/A |

All six self sheep carried two copies of the exon 4 mutation, in addition to two copies of the deletion (Table 2). This means that the two mutations are inherited together (in what is known as a haplotype), at least in this small sample of sheep, and consequently it is unclear whether the exon 4 mutation is also causal.

To examine this question in greater depth, we genotyped both mutations in 621 sheep belonging to the largest families in the Soay sheep pedigree. We then worked out paternal and maternal haplotypes for each individual and compared the observed distribution of wild and self-type sheep in each of the 10 possible haplotype classes to that expected under two inheritance models: (i) the deletion is the only loss of function mutation, (ii) both mutations are causal (Table 3). The two models differ with respect to the predicted phenotype of sheep in haplotype classes 5, 6 and 7; under the deletion-only model these sheep would be wild-type, since there is at most only one copy of the deletion, whereas under the two mutation model they would be self-type because there is either two copies of the exon 4 mutation, or this mutation occurs in combination with the deletion. Only six sheep were observed in these three haplotype classes, but all were self. This provides support for the two mutation model because a self phenotype is generated when there are two copies of the exon 4 mutation, even in the presence of one functional copy of exon 2.

***Table 3.*** *Association between Agouti coding sequence haplotypes and coat pattern in Soay sheep.*

| *Agouti* haplotype combination | Predicted phenotype | | Observed phenotype | | Total |
|---|---|---|---|---|---|
| | Deletion only | Two mutations | Wild | Self | |
| 1. AGGAA-T/AGGAA-T | Wild | Wild | 372 | 2 | 374 |
| 2. AGGAA-T/AGGAA-A | Wild | Wild | 16 | 0 | 16 |
| 3. AGGAA-T/Deletion-T | Wild | Wild | 1 | 0 | 1 |
| 4. AGGAA-T/Deletion-A | Wild | Wild | 193 | 2 | 195 |
| 5. AGGAA-A/AGGAA-A | Wild | Self | 0 | 0 | 0 |
| 6. AGGAA-A/Deletion-T | Wild | Self | 0 | 0 | 0 |
| 7. AGGAA-A/Deletion-A | Wild | Self | 0 | 6 | 6 |
| 8. Deletion-T/Deletion-T | Self | Self | 0 | 0 | 0 |
| 9. Deletion-T/Deletion-A | Self | Self | 0 | 0 | 0 |
| 10. Deletion-A/Deletion-A | Self | Self | 1 | 28 | 29 |
| Total | | | 583 | 38 | 621 |

It is important to note that the two mutation model fails to explain coat pattern for all sheep; one wild-type and four self-type sheep carry haplotypes that would be predicted to produce the alternative phenotype. The wild-type mismatch is probably a sample mix-up because it is impossible to generate a wild phenotype with two copies of the deletion. Alternatively, it is conceivable that other recessive mutations for the self pattern are segregating in the population. We sequenced the entire coding region of each of the mismatching sheep and found no novel mutations. Consequently, if a third mutation exists it must occur in a regulatory region of *Agouti*, or at another gene. A recessive regulatory mutation for self pattern has been described at *Agouti* in domestic sheep. Unfortunately, we are unable to test for differential *Agouti* expression in the mismatching Soays because they are long deceased and we do not have RNA for gene expression studies. Our data suggest that at least two and possibly three recessive mutations for self pattern are segregating in Soay sheep on Hirta. It is hoped that this knowledge will help to quantify the relationship between coat pattern and fitness in Soay sheep.

## An update on the genetic affinities of Soay sheep

Josephine Pemberton & Dario Beraldi

Over the years we have often submitted DNA samples from St Kilda Soay sheep to wide-ranging studies of sheep genetic variation conducted by other research groups, but we have not before reported back on the results.

Mitochondrial DNA is inherited from mother to offspring. Because there is only one chromosome in the mitochondrion (the powerhouse of the cell), mutations are generally inherited in a clonal fashion, providing a powerful tool for tracing ancestry. In a study using 836 sheep from 45 breeds across Europe, the Middle East and Africa and several wild sheep species, Bruford and Townsend (2006; in Documenting Domestication: New Genetic and Archaeological Paradigms ed: Zeder et al, California University Press)

identified three maternal lineages of domestic sheep. Soays are located firmly within the 'A' lineage along with the vast majority of European breeds. However, within this lineage they are distinct: the 23 Soays screened each had one of three closely-related mitochondrial variants which were not found in any other breed.

Two studies of the conventional chromosomes, using different kind of genetic markers and analyses, establish two clear points: Soays have slightly depressed genetic variation compared with most sheep breeds and they are genetically very distinct from other domestic breeds. For example, a study using markers called microsatellites (Lawson-Handley *et al* (2007) Heredity) found that Soays had an average heterozygosity of 0.497 compared with a mean for other European and Middle Eastern breeds of 0.606. In a principle component analysis of the data, Soays were well differentiated from all other breeds in the analysis (Figure 11). In a more recent analysis of 1,400 single nucleotide polymorphisms Kijas *et al* (in Press; PLoS One), 67% of loci were polymorphic in Soays compared with a domestic sheep average of 81%. These loci generally have lower heterozygosity than microsatellites, but again Soays were a little impoverished (heterozygosity 0.223) compared with the domestic breed average (0.295). In this second study, which included 23 breeds from around the world, Soays were by far the most genetically distinctive single breed.

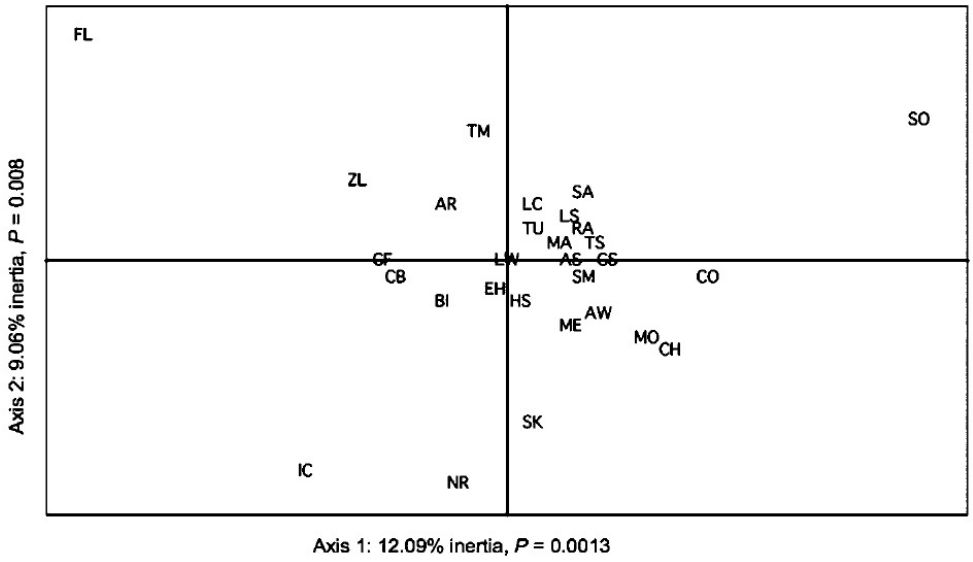


***Figure 11.*** *Principle Component Analysis of 29 sheep breeds using 23 microsatellite DNA markers. The technique places each breed on the two axes according to the genetic similarities and differences between the breeds. Here, as in several such analyses, Soays (SO, top right) are highly distinctive from other breeds. From Lawson-Handley et al (2007) Heredity 99:620-631.*

Finally, researchers at the University of Glasgow Vet School, led by Massimo Palmarini, have been studying an unusual kind of genetic marker in sheep – retroviruses which integrate themselves permanently into the chromosomes. These markers are particularly powerful for tracing evolutionary history and in a currently submitted paper (Chessa *et al*), four such markers are shown to have distributions across breeds which are consistent with the idea of two waves of domestic sheep out of the Middle East. The first wave is

characterised by primitive traits such as dark, hairy, shedding fleeces, and here the markers unite Asian wild sheep, mouflons, Soays and other primitive breeds on Europe's Western seaboard. The second wave is characterised by human selected traits such as woolly, white, non-shedding fleeces and here the markers unite most of the conventional European breeds.

In conclusion, genetic studies of Soays consistently identify that the population includes some highly distinctive, probably 'primitive', genetics. Where large numbers of markers have been studied, Soays are also revealed to have slightly depressed levels of genetic marker variation compared with other domestic breeds, as might be expected for an isolated population on an island. In a previous report, Jon Slate suggested that modern Soays could be an admixture of original Soays and one or more improved breeds. The results reported here do not rule out this theory – indeed some Soays do have retroviruses believed to be characteristic of the second wave of domestic sheep into Europe.

## PUBLICATIONS


Coulson, T., Ezard, T. H. G., Pelletier, F., Tavecchia, G., Stenseth, N. C., Childs, D. Z., Pilkington, J. G., Pemberton, J. M., Kruuk, L. E. B., Clutton-Brock, T. H. and Crawley, M. J. (2008). Estimating the functional form for the density dependence from life history data. *Ecology* **89**: 1661-1674.

Craig, B. H., Tempest, L. J., Pilkington, J. G. & Pemberton, J. M. 2008 Metazoan-protozoan parasite co-infections and host body weight in St Kilda Soay sheep. *Parasitology* **135**: 433-441.

Ezard, T. H. G., Gaillard, J. M., Crawley, M. J. and Coulson, T. (2008). Habitat dependence and correlations between elasticities of long-term growth rates. *American Naturalist* **172**: 424-430.

Gratten, J., Wilson, A. J., MacRae, A. F., Beraldi, D., Visscher, P. M., Pemberton, J. M., and Slate, J. (2008) A localized negative genetic correlation constrains microevolution of coat color in wild sheep. *Science* **319**: 318-320.

Grillo, V., Craig B. H., Wimmer, B. and Gilleard, J. S. (2008) Microsatellite genotyping supports the hypothesis that *Teladorsagia davtiani* and *Teladorsagia trifurcata* are morphotypes of *Teladorsagia circumcincta*. *Molecular and Biochemical Parasitology* **159**: 59-63.

Jones, O. R., Clutton-Brock, T. H., Coulson, T. and Godfray, H. C. J. (2008) A web resource for the UK's long-term individual-based time-series (LITS) data. *Journal of Animal Ecology* **77**: 612-615.

Jones, O.R., Gaillard, J. M., Tuljapurkar, S., Alho, J. S., Armitage, K. B., Becker, P. H., Bize, P., Brommer, J., Charmantier, A., Charpentier, M., Clutton-Brock, T. H., Dobson, F., S., Festa-Bianchet, M., Gustafsson, L., Jensen, H., Jones, C. G., Lillandt, B. G., McCleery, R., Merila, J., Neuhaus, P., Nicoll, M. A. C., Norris K.,

Oli, M. K., Pemberton, J., Pietiainen, H., Ringsby, T. H., Roulin, A., Saether, B. E., Setchell, J. M., Sheldon, B. C., Thompson, P. M., Weimerskirch, H., Wickings, E. J. and Coulson, T. (2008) Senescence rates are determined by ranking on the fast-slow life-history continuum. *Ecology Letters* **11**: 664-673.

King, R., Brooks, S. P. and Coulson, T. (2008) Analyzing complex capture-recapture data in the presence of individual and temporal covariates and model uncertainty. *Biometrics* **64**: 1187-1195.

Morris, W. F., Pfister, C.A., Tuljapurkar, S., Haridas, C. V., Boggs, C. L., Boyce, M. S., Bruna, E. M., Church, D. R., Coulson, T., Doak, D. F. Forsyth, S., Gaillard, J. M., Horvitz, C. C., Kalisz, S., Kendall, B. E., Knight, T. M., Lee, C. T. and Menges, E. S. (2008) Longevity can buffer plant and animal populations against changing climatic variability. *Ecology* **89**: 19-25.

Robinson, M. R., Pilkington, J. G., Clutton-Brock, T. H., Pemberton, J. M., and Kruuk, L. E. B. (2008) Environmental heterogeneity generates fluctuating selection on a secondary sexual trait. *Current Biology* **18**: 1-7.

Wilson, A. J., Pemberton, J. M., Pilkington, J. G., Clutton-Brock, T. H. & Kruuk, L. E. B. (2009) Trading offspring size for number in a changing environment: selection on reproductive investment in female Soay sheep. *Journal of Animal Ecology* 78:354-364.

<u>In press:</u>

Craig, B. H., Jones, O. R., Pilkington, J. G. and Pemberton, J. M. Re-establishment of nematode infra-community and host survivorship in wild Soay sheep following anthelmintic treatment. *Veterinary Parasitology.*

Ezard, T. H. G., Côté, S.D. & Pelletier, F. Eco-evolutionary dynamics: disentangling phenotypic, environmental and population fluctuations. *Philosopical Transactions of the Royal Society London, B.*

Nussey, D.H., Pemberton, J.M., Pilkington, J. G., and Blount, J.D. Life history correlations of oxidative damage in a free-living mammal population. *Functional Ecology.*

Robinson, M. R., Wilson, A. J., Pilkington, J. G., Clutton-Brock, T. H., Pemberton, J. M. and Kruuk, L. E. B. The impact of early environmental heterogeneity on genetic architecture in a wild population of Soay sheep. *Genetics.*

Slate, J., Gratten, J., Beraldi, D., Stapley, J., Hale, M. and Pemberton, J. M. Gene mapping in the wild with SNPs: guidelines and future directions. *Genetica*.

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## APPENDIX A: PERSONNEL CHANGES & SCHEDULE OF WORK

### *Personnel Change*

Emily Brown started a PhD on the evolutionary genetics of Soay sheep at Sheffield, and Philine Feulner started a postdoc on the evolutionary genetics of Soay sheep also at Sheffield.

Chiara Mazzetta joined the project to analyse the long term data series working first at the University of Kent (Canterbury) and now the University of Warwick.

Matt Robinson (Edinburgh University) was awarded his PhD.

### *Schedule of work on St Kilda*

### *Winter - Spring*

Jill Pilkington monitored mortality during the early part of February and with volunteers, throughout lambing. During this period, detailed data were collected on individual sheep found dead, and samples were taken for genetic and parasitological study.

From March 7th until May 6th, Jill Pilkington, Adam Hayward and three volunteers carried out ten population censuses and tagged and sampled lambs for ongoing genetic studies. 207 lambs were born to 187 ewes; these figures include 20 sets of twins (19 ewes held both lambs, 1 lost both lambs). 158 lambs (78 male and 80 female) were caught and tagged; 6 lambs escaped capture; a further 43 lambs died before any tagging attempt.

### *Summer*

Jill Pilkington and two volunteers returned to Hirta on July 11th to carry out ten population censuses, conduct mortality searches (yielding 4 tagged dead animals), and prepare for the main catch-up of study area sheep. The catch-up took place from August 1st – 15th, was led by Josephine Pemberton, and conducted by a team of 11 additional project members and volunteers. 317 sheep were caught and processed, of which 126 were lambs (63 males and 63 females), 51 were yearlings (21 males and 30 females), 21 were adult males, and 119 were adult females. All animals were weighed and measured to monitor growth, and sampled for parasite and genetic analyses. 18 Sheep were retagged because of damaged or missing tags. 32 previously untagged lambs, 4 yearlings and 15 adults were caught and processed. Jill Pilkington and two volunteers remained on Hirta until 29th August to complete parasite counts and vegetation monitoring.

### *Autumn*

From October 17th to December 5th Jill Pilkington and two volunteers carried out ten population censuses, monitored the mating period, capturing and processing 11 incoming tups and 19 resident tups. 45 previously darted, non-resident tups were seen in the study area during this rut. 3 dead sheep were found.

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