Déterminants physiologiques des variations individuelles de survie et de sénescence chez la grande oie des neiges

Thèse

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Résumé

Les variations de populations animales dépendent de quatre composantes clé, la survie, la reproduction, l'émigration et l'immigration. Sur l'axe de stratégie d'histoire de vie « lent-rapide », les espèces longévives, généralement classées lentes, sont caractérisées par une survie adulte élevée et stable et une reproduction plus faible et variable. La survie adulte est le trait avec la plus grande contribution relative à la dynamique de population (plus grande élasticité) chez ces espèces, et peut varier différemment selon les causes de mortalité, la réponse au stress ou l'état physiologique de l'individu. Nous avons exploré les déterminants de la survie à l'échelle individuelle chez la grande oie des neiges (*Anser caerulescens atlanticus*), une espèce d'oiseau migratrice longévive et chassée, bénéficiant d'un suivi démographique par capture-marquage-recapture (CMR) actif depuis 1990. Grâce aux données morphométriques, physiologiques et démographiques de deux jeux de données complémentaires, nous avons pu étudier les rôles respectifs de la sénescence, de la pression de chasse, du stress et de la qualité individuelle dans les variations de survie. La longueur des télomères peut notamment être utilisée comme un indicateur de la qualité individuelle. Nous avons utilisé les données de 30 ans de CMR et suivi de la colonie à l'Île Bylot (Nunavut), ainsi que celles d'une expérience réalisée à l'Île-aux-Oies suivie de 13 ans de recaptures et report de bagues par les chasseurs pour estimer des probabilités de survie annuelle en fonction des différentes variables individuelles : l'âge, la condition corporelle, la taille et la longueur des télomères.

Nos résultats montrent des variations de survie selon l'âge des individus, malgré la forte pression de chasse et la stabilité de ce paramètre démographique chez les espèces longévives. Nous montrons pour la première fois le patron de sénescence actuarielle chez cette espèce. La sénescence s'amorce aux alentours de 11 ans avec une diminution progressive de la survie, appuyée par une diminution similaire de la condition corporelle post-reproduction avec l'âge. La survie est fortement affectée par l'accumulation de stresseurs et peut varier avec l'intensité de la réponse hormonale au stress, la condition corporelle, ou la taille corporelle. La mortalité à la chasse semble être compensatoire et affecter des individus de moindre qualité, mais devient additive et affecte tous les individus dans les cas extrêmes d'accumulation de stresseurs. La longueur des télomères ne module

pas la survie annuelle, mais modifie la vulnérabilité à la chasse chez les individus en captivité. Nous suggérons que les réponses contrastées de la mortalité à la chasse en fonction de la longueur des télomères témoignent d'une interaction entre qualité individuelle et décisions reproductives. Nous trouvons par ailleurs que la longueur des télomères varie principalement avec l'âge et la taille corporelle, ainsi que marginalement avec la réponse hormonale au stress.

Cette thèse met en évidence l'importance de prendre en compte la physiologie dans l'étude des variations individuelles en démographie, car les interactions entre traits individuels et mortalité extrinsèques sont complexes mais influentes. Trois principaux déterminants de la survie chez la grande oie des neiges émergent ici : les conditions environnementales lors du développement qui déterminent la taille corporelle la modulation de la vulnérabilité à la chasse selon l'état physiologique et l'âge. Ce travail renforce nos connaissances de cette espèce structurante de l'écosystème arctique et d'intérêt cynégétique en donnant une perspective intégrative et détaillée des variations individuelles de survie.

Abstract

Variations in wild animal populations depend on four key components, namely survival, reproduction, emigration and immigration. On the "slow-fast" life history continuum, long-lived species are generally classified as "slow" and are characterized by high and stable adult survival and low and variable reproduction. Adult survival has the highest relative contribution to these species' population dynamics (highest elasticity). It can vary depending on mortality causes, such as hunting, stress response, or the individual's physiological condition. We have explored the determinants of survival in the greater snow goose (*Anser caerulescens atlanticus*) at an individual scale. We have 30 years of active capture-mark recapture (CMR) demographic data on this long-lived migratory species. Using morphometric, physiological and demographic data of two complementary datasets, we were able to study the respective roles of senescence, hunting pressure, stress and individual quality in the variations of survival. Telomere length is used as an indicator of individual quality. We used 30 years of CMR data and colony monitoring at Bylot Island (Nunavut), as well as data from an experiment that took place in 2009 at Île-aux-Oies, followed by 13 years of monitoring. We estimate annual survival probabilities in response to diverse individual variables: age, corticosterone concentration, body condition, body size and telomere length.

Our results establish the actuarial senescence pattern of this long-lived species for the first time, survival varies strongly with age in spite of high hunting pressure and the stability of this demographic parameter. Senescence sets in around 11 years with a progressive decrease in survival thereafter, accompanied by a decrease in post-reproductive body condition as well. Survival is strongly affected by the accumulation of stressors and can vary with the intensity of hormonal stress response, body condition, and body size. Hunting mortality seems to be compensatory and affect lower quality individuals but affects all individuals in an additive manner in situations of extreme stressor accumulation. Telomere length does not modulate annual survival but modifies vulnerability to hunting in individuals that were kept in captivity. We suggest that the contrasted response of hunting mortality along the telomere length gradient point to an interaction between individual quality and reproductive decisions. We also found that telomere length mainly varies with age and body size, but also marginally with the intensity of hormonal stress response.

This thesis highlights the importance of considering physiology in the study of individual variation of demographic parameters, because the interactions between individual traits and extrinsic mortality are complex but influential. Three main determinants of greater snow goose survival emerge here: early life development, the interaction between physiology and vulnerability to hunting, and age. This work reinforces our knowledge on this species of interest because of hunting activity and its a structural role in the arctic ecosystem, by giving an integrative and detailed perspective on individual variations in survival.

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Liste des abréviations, acronymes

95%CI : 95% confidence intervals / intervalles de confiance à 95%
ADN (**DNA** en anglais) : Deoxyribonucleic acid / Acide désoxyribonucléique
AIC : Akaike information criterion / Critère d'information d'Akaike

- ➔ **AICc** : AIC corrigé
- ➔ **QAIC** : Quasi-AIC
- ➔ **WAIC** : Bayesian AIC

ANOVA : analysis of variance / analyse de la variance
CMR : capture-mark-recapture / capture-marquage-recapture
COND : body condition / condition corporelle
CORT : corticosterone / corticostérone
Cq : quantification cycle / cycle de quantification
GC : glucocorticoid / glucocorticoïde
GOF : goodness-of-fit tests / test d'ajustement des données
HHS (**HPA** en anglais) : l'axe hypothalamo-hypophysaire surrénalien
ICC : intraclass correlation coefficient / coefficient de corrélation inter-classes
ID : identification
LM : linear model / modèle linéaire
LMM : mixed linear model / modèle linéaire mixte (avec effets aléatoires)
PC1 : première composante d'une ACP
PCA (**ACP** en français) : principal component analysis / analyse en composantes principales
qPCR : quantitative polymerase chain ceaction / Réaction en chaîne par polymérase quantitative
RAG1 : Gène activant la recombinaison 1
ROS : reactive oxygen species / dérivés réactifs de l'oxygène
RTL : relative telomere length / longueur de télomères relative
SD : standard deviation / écart-type
UV : ultra-violet
VIF : variance inflation factor / facteur d'inflation de la variance
g : gramme
µL : microlitre
mL : millilitre

Liste des sigles

CEBC: Centre d'Études Biologiques de Chizé
CEN : Centre d'Études Nordiques
CFX Maestro : Logiciel de gestión des données de qPCR de BioRad.
CPAUL : Comité de Protection des Animaux de l'Université Laval
E-SURGE : Logiciel d'analyses de capture-marquage-recapture
FRQNT : Fonds de recherche du Québec Nature et Technologie
IUCPQ: Institut Universitaire de Cardiologie et Pneumologie de Québec
NSERC (CRSNG) : Natural Sciences and Engineering Research Council of Canada
U-CARE: logiciel de test "Goodness-of-fit" pour les analyses de capture-marquage-recapture

Glossaire

Allostasie : processus d'ajustements métaboliques par lequel l'organisme atteint un nouvel état d'équilibre interne, possiblement différent de l'équilibre initial homéostable, en réponse à des changements ou à des contraintes.

Charge allostatique : coût physiologique associé à la répétition ou la chronicité de ces ajustements allostatiques, pouvant mener à une usure des systèmes de régulation.

Élasticité : contribution relative d'un paramètre démographique au taux d'accroissement

Fitness ou valeur adaptative: contribution d'un individu à la génération suivante. Se compte généralement en nombre de descendants recrutés à la génération suivante et dépend donc d'une combinaison entre survie et succès reproducteur.

Glucocorticoïdes: hormones stéroïdiennes produites par les glandes surrénales (par ex. le cortisol chez les mammifères, la corticostérone chez les oiseaux) qui régulent le métabolisme de l'énergie et jouent un rôle central dans la réponse au stress.

Hétérogénéité individuelle : différences interindividuelles de valeurs de traits, pas nécessairement reliés au fitness. Ces différences peuvent résulter d'effets génétiques, maternels, des conditions pendant la croissance, de la trajectoire de vie, et peuvent varier avec l'âge.

Homéostasie : capacité de l'organisme à maintenir un état interne relativement stable (par ex. température, glycémie, pH) malgré les variations externes.

Performance : valeur composite qui reflète la capacité ou l'efficacité d'un organisme à accomplir une tâche. Par exemple, le nombre de jeunes à une année est un critère de performance.

Qualité individuelle : cas particulier d'hétérogénéité individuelle lorsque les traits sont fortement positivement corrélés au fitness.

Réponse au stress : Cascade d'ajustements physiologiques et comportementaux amorcée par des sécrétions hormonales élevées comme l'adrénaline ou les glucocorticoïdes.

Sénescence : dégradation progressive de l'organisme avec l'âge. On parle de sénescence actuarielle lorsque l'on observe une diminution de la probabilité de survie avec l'âge, et de sénescence reproductive lorsque l'on observe une diminution du succès reproducteur avec l'âge. Le taux de sénescence est la vitesse à laquelle le trait étudié (survie ou reproduction) diminue avec l'âge.

Stresseur : Stimulus nocif ou imprévisible provoquant une réponse au stress.

Stress : État de débalancement allostatique causé par un stresseur lors duquel une cascade d'ajustements physiologiques et comportemental se met en place comme la sécrétion d'adrénaline, de glucocorticoïdes, un comportement de fuite, combat ou vigilance, une suppression des fonctions non-essentielles dans l'immédiat comme la digestion ou l'immunité. On distingue deux cas de figure :

- **Le stress aigü** : réaction physiologique et comportementale de l'organisme face à un événement imprévisible, intense et de courte durée. Le retour à l'équilibre se fait dans un délai relativement court (quelques minutes ou quelques heures).
- **Le stress chronique** : état résultant d'une exposition répétée ou prolongée à des stresseurs. Lorsque la réponse au stress est activée trop souvent ou trop longtemps, entraîne une surcharge des systèmes physiologiques et peut avoir des effets négatifs à long terme sur la survie et la reproduction.

Je dédie ce travail à mes parents et grands-parents, qui m'ont toujours soutenue dans mes aventures les plus folles.

Ich widme diese Arbeit meinen Eltern und Großeltern, die mich immer bei meinen verrücktesten Abenteuern unterstützt haben.

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« Papi, raconte-moi comment tu es venu en France, raconte-moi pourquoi tu es parti et comment tu es arrivé ici. »

Le vieil homme ne s'y attendait pas. Cela faisait si longtemps… si longtemps qu'il était assis, là, dans son fauteuil… Il doutait d'avoir jamais été cet homme un jour qui avait fait ce si long voyage. Il regarda sa petite fille. Elle attendait, ses yeux dans les siens.

« Là-bas, c'était un petit village. Il y avait beaucoup de prés et de champs, et moi, l'après-midi, je devais garder les oies. C'était mon travail. Un jour que j'étais parti dans un pré assez éloigné du village avec toutes mes oies, je m'allongeai sous un arbre pour me reposer. Bientôt, je sentis le sommeil venir. Alors pour ne pas que mes oies se sauvent pendant que je dormais, je pris une longue ficelle que j'avais toujours dans ma poche et j'attachai toutes mes oies entre elles par les pattes, et puis je nouai la ficelle à mes pieds. Et je m'endormis l'esprit tranquille. Je fis de longs et beaux rêves de voyage… Mais quand je me réveillai, je compris pourquoi j'avais fait ces rêves de voyage. Mes oies aussi avaient envie de voir du pays. Elles s'étaient envolées et m'avaient emporté avec elles. Je me retrouvai dans un pré inconnu. Mais j'apercevais au loin la pointe d'un clocher. Alors je me dis que c'était peut-être le clocher de mon village. Je me mis à marcher dans sa direction. Malheureusement, ce n'était pas mon village. Je pensais que mes oies m'avaient emporté bien loin. Mais j'aperçus à nouveau au loin un autre clocher et j'avançai dans sa direction. Mais ce n'était encore pas mon village. Je continuai ainsi à marcher de clocher en clocher. Quand j'en eus assez de marcher, je demandai à un homme où je me trouvais. Il me répondit que j'étais en France ! Je me dis qu'ici aussi c'était joli et qu'on pouvait bien y garder des oies comme chez moi, en Russie. Alors je décidai de m'arrêter de marcher et de rester en France. »

Adam cessa de parler et la petite fille ne dit rien. Elle rêvait à son grand-père, volant, traversant des étendues immenses, emporté par des dizaines d'oiseaux blancs. Cette histoire, elle en était sûre, elle ne l'oublierait jamais.

Histoire de famille écrite par ma mère

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Avant-propos

Cette thèse de doctorat a été évaluée par Dr. Creagh Breuner, Dr. Nadia Aubin-Horth et Dr. Sandra Hamel. Il s'agit d'une thèse par articles composée de six parties. L'introduction et la conclusion générales sont rédigées en français. Les quatre chapitres sont des articles publiés ou en voie de l'être et sont donc rédigés en anglais. Je suis la première auteure des quatre chapitres présentés dans cette thèse. L'introduction générale est un état de l'art de la recherche sur les différents sujets abordés au cours de la thèse. Les concepts essentiels aux chapitres et les liens entre ces concepts y sont établis.

Le chapitre 1 intitulé « **Evidence of actuarial senescence in a hunted long-lived migratory bird** » est en préparation pour soumission à une revue scientifique. Frédéric LeTourneux et moi avons fait les analyses, avec l'appui de Gilles Gauthier et Pierre Legagneux. J'ai rédigé le manuscrit en incluant les révisions de Frédéric LeTourneux, Gilles Gauthier et Pierre Legagneux, qui sont les trois co-auteurs. Tous les auteurs ont contribué à la rédaction de la version finale de l'article. Ce chapitre est le premier dans la thèse mais le dernier à avoir été réalisé et écrit. Les résultats reportés sont le plus à jours possible, mais les modèles continuent à rouler sur Calcul Canada pour améliorer la convergence. L'inclusion de l'hétérogénéité individuelle dans les modèles est également en cours.

Le chapitre 2 intitulé « **Manipulating individual state during migration: carry-over effects of cumulative stress on survival** » a été publié dans *Ecology and Evolution* en juin 2025. Joël Bêty, Gilles Gauthier et Pierre Legagneux ont conçu l'expérience et collecté les données. Pierre Legagneux a analysé les échantillons en laboratoire. J'ai analysé les données avec les retours de Gilles Gauthier, Frédéric LeTourneux et Pierre Legagneux. J'ai rédigé le manuscrit en incluant les révisions de Gilles Gauthier, Frédéric Angelier, Joël Bêty, Frédéric LeTourneux et Pierre Legagneux, qui sont les cinq co-auteurs. Tous les auteurs ont contribué à la rédaction de la version finale de l'article.

Le chapitre 3 intitulé « **Telomere length decreases with age and reflects early-life environment but not adult condition in a long-lived migratory bird** » a été soumis à *Ecological and Evolutionary Physiology* en août 2025. J'ai conçu l'étude avec Pierre Legagneux et Gilles Gauthier. Gilles Gauthier et Pierre Legagneux ont dirigé la collecte de

données, à laquelle j'ai participé. J'ai analysé les échantillons en laboratoire pour extraire les données de longueur de télomères, avec l'apport de Frédéric Angelier, Cristoforo Silvestri et Glenn Yannic. Charline Parenteau a analysé les échantillons de plasma pour quantifier la corticostérone plasmatique. J'ai analysé les données avec l'appui de Gilles Gauthier et Pierre Legagneux. J'ai rédigé le manuscrit en incluant les révisions de Gilles Gauthier, Frédéric Angelier, Glenn Yannic, Charline Parenteau, Cristoforo Silvestri et Pierre Legagneux, qui sont les six co-auteurs. Tous les auteurs ont contribué à la rédaction de la version finale de l'article.

Le chapitre 4 intitulé « **Variations in survival in relation to markers of different life stages in a long-lived bird** » est en préparation pour une soumission dans *Journal of Avian Biology cet automne*. J'ai conçu l'étude avec Pierre Legagneux et Gilles Gauthier. Joël Bêty, Gilles Gauthier et Pierre Legagneux ont collecté des données, lors de la même expérience que le chapitre 2. J'ai analysé les échantillons en laboratoire pour extraire les données de longueur de télomères, avec l'apport de Frédéric Angelier, Cristoforo Silvestri et Glenn Yannic. J'ai analysé les données avec l'appui de Gilles Gauthier et Pierre Legagneux. J'ai rédigé le manuscrit en incluant les révisions de Gilles Gauthier, Frédéric Angelier, Glenn Yannic, Cristoforo Silvestri et Pierre Legagneux, qui sont les cinq co-auteurs. Tous les auteurs ont contribué à la rédaction de la version finale de l'article.

Dans la conclusion générale, je fais une synthèse des principaux résultats de la thèse. Je reviens également brièvement sur les limites du projet et je propose des perspectives à court et long terme. La documentation supplémentaire pour chaque chapitre est apportée à la fin de la thèse.

Introduction

Les variations de populations animales sont couramment observées par les biologistes et naturalistes, mais leur description et leur compréhension restent complexes. Elles reflètent en effet des changements sous-jacents de survie, reproduction, et de mouvements d'émigration/immigration, des paramètres déterminants de la dynamique des populations et qui sont intrinsèquement reliés à l'écologie et l'évolution de chaque espèce (Dobson & Oli, 2007). La définition des stratégies d'histoire de vie a permis aux écologistes d'appréhender ces dynamiques complexes, en plaçant les espèces sur des continuums définis par la contribution relative de ces différents paramètres aux variations de population. Le plus utilisé aujourd'hui en écologie est l'axe « lent-rapide » ou slow-fast continuum en anglais (Stearns, 1992; Gaillard et al., 2005; Healy et al., 2019). Les espèces situées à l'extrémité « rapide » du continuum sont généralement de petite taille, atteignent la maturité sexuelle précocement, présentent un taux de reproduction élevé et une faible longévité. À l'inverse, les espèces dites « lentes » se reproduisent plus tardivement, produisent moins de jeunes par événement reproducteur et vivent plus longtemps (Dobson & Oli, 2007). Chez les espèces longévives généralement classées « lentes », la survie varie moins que la reproduction d'année en année, mais les éventuelles variations de survie adulte peuvent avoir des conséquences plus marquées sur les populations (Saether & Bakke, 2000; Gaillard & Yoccoz, 2003). On parle alors d'une plus forte élasticité du paramètre de survie, c'est-à-dire d'une plus grande contribution relative de la survie au taux d'accroissement de la population (Lebreton & Clobert, 1991; Gaillard et al., 1998; Saether & Bakke, 2000).

Cette forte sensibilité de la dynamique des populations à la survie chez les espèces longévives en fait un paramètre essentiel à étudier pour comprendre les mécanismes sous-jacents. Parmi les potentielles causes de mortalité, on distingue les causes extrinsèques telles que la prédation, les maladies ou la chasse, et les causes intrinsèques liées à la physiologie des individus (Koopman et al., 2015). Ces causes intrinsèques et extrinsèques de mortalité interagissent et créent des variations individuelles de survie (Ricklefs & Wikelski, 2002; Koopman et al., 2015). En particulier, lorsque cette mortalité augmente avec l'âge, on parle de sénescence, c'est-à-dire d'une dégradation progressive de l'organisme avec l'âge. On observe également une variabilité individuelle du patron de sénescence qui peut résulter de

caractéristiques intrinsèques, ainsi que de l'accumulation des expériences de vie des individus (Nussey et al., 2013; Koopman et al., 2015).

Théories évolutives de la sénescence

La sénescence a longtemps été négligée dans l'étude des populations sauvages, en partie parce que son étude nécessite d'accumuler des données à long terme, surtout pour les espèces longévives. Elle a depuis été démontrée dans ces populations et est maintenant largement étudiée sur de nombreuses espèces de vertébrés (Holmes & Austad, 1995; Nussey et al., 2013; Jones et al., 2014; Bouwhuis & Vedder, 2017). La sénescence a d'abord été observée à l'échelle de la cellule qui arrête de se multiplier après un nombre limite de réplication, appelé limite de Hayflick (Hayflick & Moorhead, 1961). À l'échelle de l'organisme, on observe aussi une diminution de la survie et de la reproduction des individus avec l'âge (Medawar, 1952; Williams, 1957). On distingue la sénescence actuarielle, qui est une augmentation du taux de mortalité intrinsèque, de la sénescence reproductive qui désigne une diminution d'un paramètre de reproduction avec l'âge (Holmes & Austad, 1995).

Des variations de taux de sénescence sont observables entre espèces, populations et individus (Bérubé et al., 1999; Bronikowski & Promislow, 2005; Bleu et al., 2015), et peuvent être expliquées par plusieurs théories évolutives (Jones et al., 2014; Jones & Vaupel, 2017; Roper et al., 2021). Celles-ci reposent sur deux éléments fondamentaux. Premièrement, la force de la sélection naturelle doit diminuer avec l'âge car un individu âgé aura déjà transmis ses gènes à sa descendance (Hamilton 1966). Deuxièmement, elles reposent sur des compromis d'allocation de l'énergie limitée, définis par la théorie des traits d'histoire de vie (Gavrilov & Gavrilova, 2002).

La théorie d'accumulation de mutations (Medawar, 1952) attribue la baisse de performance individuelle, par exemple la baisse du nombre de jeunes d'un individu lors d'un évènement de reproduction (Arnold, 1983), à l'accumulation de mutations délétères actives à un âge avancé. Ces mutations ne seraient pas contre-sélectionnées à cause de la sélection décroissante avec l'âge (Medawar, 1952). La sénescence serait ainsi une conséquence de ces mutations délétères accumulées qui vont avoir un effet négatif sur le fonctionnement de l'organisme uniquement à un âge avancé. La théorie de la pléiotropie antagoniste (Williams,

1957) ajoute à ces mutations, ayant des effets délétères à des âges avancés, des effets positifs en débuts de vie, favorisant le maintien de ces mutations dans la population. Selon cette théorie, l'âge de première reproduction devrait être positivement corrélé à l'âge d'amorce de la sénescence. Ces prédictions ont été testées et validées expérimentalement grâce à la comparaison de deux lignées de *Drosophila melanogaster* ayant subi de la sélection artificielle sur leur taux de sénescence (Rose, 1984).

La théorie du « soma jetable » ou « *disposable soma* » (Kirkwood, 1977) explique la sénescence par des compromis d'allocation d'énergie. Celle-ci propose que, les ressources étant limitées, le maintien de la lignée germinale, dont l'ADN est transmis à la descendance, serait favorisé au détriment de la lignée somatique. Cette économie d'énergie sur la lignée somatique provoquerait l'accumulation de dommages responsables du déclin appelé sénescence. Cette dégradation a été reliée à des marqueurs physiologiques, comme la longueur des télomères, séquences non codantes protégeant les extrémités des chromosomes (Blackburn, 1991). En effet, les télomères restent stables dans les cellules de la lignée germinale mais raccourcissent avec l'âge dans les cellules de la lignée somatique, comme prédit (Blackburn, 1991; Counter et al., 1992; Eastwood et al., 2019). Cette théorie complète très bien celle de la pléiotropie antagoniste en y ajoutant la notion d'un compromis énergétique entre reproduction et maintien de l'organisme (Monaghan et al., 2008; Nussey et al., 2013). Le cadre formé par cette association est soutenu par des nombreuses observations empiriques et forme la base de la littérature récente sur la sénescence (Nussey et al., 2013).

L'association des précédentes théories entre elles et avec la classification des espèces selon l'axe de stratégies d'histoire de vie « lent-rapide » crée un cadre théorique intégrateur, parfois appelé « théorie du train de vie » ou « *pace of life theory* » (Speakman & Król, 2010; Nussey et al., 2013). Tout d'abord, l'axe « lent-rapide » est directement relié aux compromis énergétiques entre reproduction immédiate et survie pour la reproduction future (Jones et al., 2008; Monaghan et al., 2008; Nussey et al., 2013). Ces compromis sont parallèles au compromis entre maintien de la lignée somatique ou de la lignée germinale, défini dans la théorie du *disposable soma* (Stearns, 1992; Jones et al., 2008; Monaghan et al., 2008). Les stratégies d'histoire de vie dépendent également du métabolisme (Williams et al., 2010;

Speakman & Król, 2010) car les espèces “rapides” ont généralement un métabolisme plus élevé, qui s’accompagne d’une plus forte production de sous-produits délétères comme les dérivés réactifs de l’oxygène (ROS pour *Reactive Oxygen Species*). Ces ROS entrainent des dommages cellulaires (Kristal & Yu, 1992; Speakman, 2005), notamment en accélérant l’attrition des télomères (Boonekamp et al., 2017). On s’attend alors à ce que les espèces lentes aient une sénescence plus lente que les espèces rapides (Jones et al., 2008).

Ainsi, ce cadre théorique relie stratégies d’histoire de vie, métabolisme, dynamique des télomères et sénescence (Monaghan et al., 2009; Hall et al., 2010; Monaghan, 2024). Plus l’âge avance, moins il y a d’individus de cet âge, moins ces individus vont se reproduire à l’avenir, et plus ils auront accumulé des dommages internes. La sélection devient alors moins forte et moins efficace, permettant l’accumulation de mutations avec des effets délétères retardés dans la vie de l’individu. Les mécanismes de maintien de l’organisme deviennent alors moins efficaces, l’organisme se dégrade et la performance baisse avec l’âge (Medawar, 1952; Williams, 1957; Williams et al., 2010).

Questionnement du cadre théorique de la sénescence

Les principes fondamentaux de la sénescence pensée inévitable et liée aux compromis d’histoire de vie sont cependant remis en question (Cohen et al., 2020; Roper et al., 2021). Par exemple, l’hypothèse du train de vie prédit une corrélation négative entre les traits de fitness associés aux compromis d’histoire de vie (Kirkwood & Rose, 1991; Lemaître & Gaillard, 2017), principalement entre la survie et la reproduction (Kirkwood & Austad, 2000). Ces prédictions ont été confirmées par plusieurs études, ce qui appuie l’existence d’un continuum de traits d’histoire de vie chez de nombreuses espèces (Wiersma et al., 2007; Williams et al., 2010; Boonekamp, Salomons, et al., 2014; Healy et al., 2019). Cependant, des variations à l’échelle des espèces, populations ou individus ont ouvert de nouvelles pistes de réflexion et de nouveaux niveaux de complexité.

Dans un premier temps, une grande diversité de patrons de sénescence a été décrite, montrant que certaines espèces échappent à la sénescence et améliorent même leurs performances avec l’âge (Vaupel et al., 2004; Jones & Vaupel, 2017; Roper et al., 2021). Les espèces avec des croissances asymptotiques, comme certains poissons ou reptiles, sont des

exemples très parlant de non-compromis (Reichard, 2017) car leur taille, leur survie et leur fécondité augmentent avec l'âge (Uriarte et al., 2016). Par ailleurs, le coût de la reproduction, attendu par une baisse de survie, s'est montré plus faible qu'attendu chez plusieurs espèces d'oiseaux (Winder et al., 2025). Il a également été montré que des mutations avec des effets délétères faibles en début de vie pouvaient se maintenir dans la population malgré des effets délétères plus forts en fin de vie (pléiotropie positive; Maklakov et al., 2015). Cela remet en question la théorie de la pléiotropie antagoniste qui repose sur les effets positifs de la mutation en début de vie pour maintenir la mutation dans la population (Williams, 1957).

Ces différences intraspécifiques et intra-populationnelles de variations de survie et de reproduction ont montré que la théorie du train de vie peut difficilement être élargie pour expliquer les variations individuelles qui sont pourtant très marquées au sein des population (Cohen et al., 2020; Van De Walle et al., 2023; Winder et al., 2025). En effet, certains individus globalement plus performants parviennent à avoir une meilleure survie et un meilleur succès reproducteur que d'autres individus de la population. Ainsi, la prise en compte de l'hétérogénéité individuelle dans ces traits liés au fitness est essentielle pour compléter les hypothèses liées au continuum de traits d'histoire de vie (Cohen et al., 2020; Van De Walle et al., 2023; Winder et al., 2025). Par exemple, les compromis d'histoire de vie entre longévité et nombre de descendants chez le fulmar (*Fulmarus glacialoides*) n'ont pu être décrit qu'après la prise en compte de l'hétérogénéité individuelle non observée (Jenouvrier et al., 2018).

Composantes de l'hétérogénéité individuelle et qualité

De nombreux traits présentent une forte hétérogénéité individuelle (Vindenes & Langangen, 2015; Cam et al., 2016; Hamel, Gaillard, & Yoccoz, 2018). Si l'on décompose cette hétérogénéité individuelle, on peut distinguer qu'elle résulte de l'assemblage d'une composante fixe et d'une composante dynamique (Figure 0.1). À la naissance, un individu hérite d'un bagage génétique de ses parents qui lui confèrera un phénotype plus ou moins favorable selon les contraintes environnementales (Ellegren & Sheldon, 2008; Bouwhuis et al., 2015). Pendant le développement pré- et post-natal, les parents vont avoir un effet durable sur leurs descendants, par exemple via l'épigénétique (Frésard et al., 2013; Asghar, Bensch, et al., 2015) ou via leur propre niveau de sensibilité au stress (Breuner, 2008), ce qu'on

appelle des effets maternels ou effets parentaux. Pendant leur croissance, la qualité de l'environnement auquel l'individu a accès jouera également un rôle sur son futur phénotype, que l'on peut résumer en parlant d'effets de cohorte (Nussey et al., 2007; Hamel, Gaillard, et al., 2009; Cam & Aubry, 2011). Un environnement défavorable peut par exemple induire une diminution directe de la croissance (Gauthier et al., 2006; Doiron et al., 2015; Vedder & Beccardi, 2025) ou une forte réponse hormonale au stress avec des répercussions sur le développement (Blas et al., 2007; Crino & Breuner, 2015). Chez les espèces à croissance déterminée, les effets parentaux ou de cohorte intervenant avant la fin de la croissance détermineront la composante fixe de la qualité individuelle (Figure 0.1). Ils auront ainsi des effets à long terme sur le fitness des individus (Van De Pol et al., 2006; Hamel, Gaillard, et al., 2009; Douhard et al., 2014; Briga et al., 2017).

Après la fin de la croissance et durant l'ensemble de la vie de l'individu, les conditions environnementales, l'acquisition d'expérience ou l'accumulation d'événements stressants pourront faire varier la performance phénotypique d'un individu (Moyes et al., 2009; Wilson & Nussey, 2010; Hau et al., 2015). La stochasticité individuelle, c'est-à-dire les évènements aléatoires inhérents au cycle de vie, affectera la croissance, la reproduction et la survie d'individus qui partagent autrement les mêmes caractéristiques (Caswell, 2009). Certains auteurs comme Caswell (2009) distinguent stochasticité individuelle et hétérogénéité individuelle. Ils considèrent que l'hétérogénéité individuelle est une caractéristique inhérente à l'individu (aussi appelée « *frailty* »*)*, équivalente à la composante fixe (Figure 0.1). Considérant que l'hétérogénéité peut varier au cours de la vie d'un individu selon son âge et la stochasticité individuelle (Wilson & Nussey, 2010; Vindenes & Langangen, 2015), nous incluons la composante dynamique dans l'hétérogénéité individuelle, comme définit précédemment.

Cette hétérogénéité et ses effets sur le fitness se retrouvent parfois dans la littérature sous le terme de « qualité individuelle » (Wilson & Nussey, 2010). La qualité individuelle se définie comme l'hétérogénéité individuelle des traits relatifs au fitness, et plus généralement comme « une propriété du phénotype qui est positivement, mais pas nécessairement parfaitement, corrélée au fitness » (Wilson & Nussey, 2010). D'autre part, le terme « hétérogénéité individuelle » est plus largement utilisé, et peut aussi inclure des traits sans

lien fort avec le fitness (Hamel, Gaillard, & Yoccoz, 2018). Les traits pouvant être considérés comme des marqueurs importants de l'hétérogénéité de fitness (ou qualité individuelle) sont par exemple l'âge à la première reproduction, le nombre de descendants produits à chaque évènement de reproduction (Hamel, Côté, et al., 2009; Le Vaillant et al., 2015), ou encore la longévité (Lescroel et al., 2009). La qualité individuelle, comme l'hétérogénéité individuelle, résulte donc d'une composante fixe qui comprend les effets génétiques, parentaux et de cohorte, et d'une composante dynamique qui résulte de la stochasticité individuelle et de l'âge (Vindenes & Langangen, 2015; Wilson & Nussey, 2010; Figure 0.1)

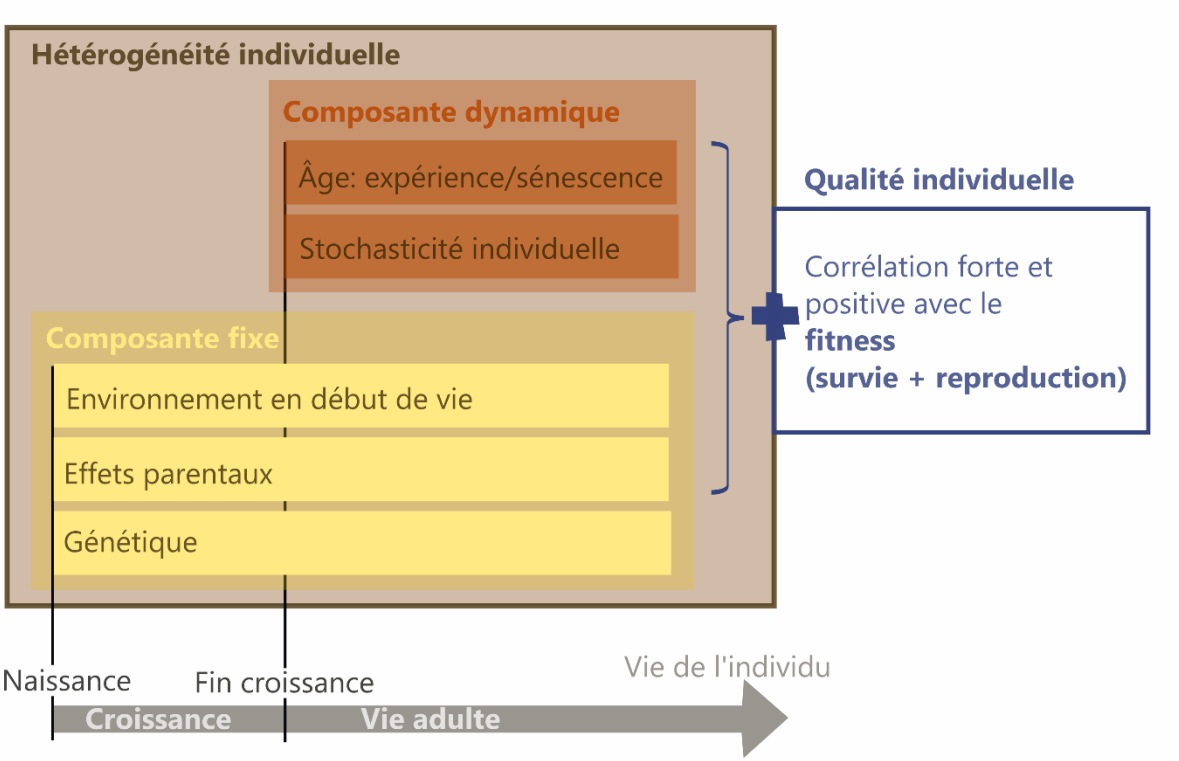

Figure 0.1: Illustration des différentes composantes de la qualité individuelle, que l'on définit ici comme un type particulier d'hétérogénéité individuelle positivement corrélée avec le fitness. Voir une illustration simplifiée en Annexe S0.1.

Il semblerait que la confusion sur les termes souvent rapportée dans la littérature (Cam et al., 2016; Hamel, Gaillard, & Yoccoz, 2018) vient de leur utilisation dans différentes disciplines au sein de la biologie. L'étude des compromis entre traits d'histoire de vie utilise plutôt le terme de « qualité » car l'impact sur le fitness, en particulier la reproduction, est au cœur des réflexions. En parallèle, en dynamique des populations, les modèles prennent en compte l'hétérogénéité individuelle qui masque parfois les processus populationnels mais ne

font pas nécessairement un lien direct vers le fitness des individus. Ici, nous parlerons de qualité individuelle comme d'une hétérogénéité individuelle particulière, en lien avec le fitness, tel que défini par Wilson et Nussey (2010). Par ailleurs, nous parlerons d'hétérogénéité individuelle lorsque ce lien avec le fitness ne peut être établi, ou pour parler de l'hétérogénéité observée dans un trait en particulier (Hamel, Gaillard, & Yoccoz, 2018). La qualité individuelle correspond ainsi à la part de l'hétérogénéité individuelle liée au fitness, et résulte à la fois de composantes fixes et dynamiques, comme défini précédemment (Vindenes & Langangen, 2015).

L'allocation d'énergie sous-jacente à l'hétérogénéité

Lorsque l'on essaye de rassembler ces réflexions, l'importance de l'énergie et de son allocation se démarque. En effet, un organisme va dépendre de l'équilibre entre l'acquisition et la dépense d'énergie pour son fonctionnement. L'homéostasie est l'état d'équilibre dynamique dépendant de la disponibilité des ressources et des demandes énergétiques de l'organisme (Romero et al., 2009). On appelle charge allostatique l'énergie nécessaire pour le fonctionnement prévisible de l'organisme au fil des saisons (McEwen & Wingfield, 2003; Romero et al., 2009). Selon les saisons et leurs variations de disponibilités ou exigences en ressources, cette charge allostatique peut augmenter ou diminuer, entrainant des ajustements de physiologie et de comportement pour maintenir l'organisme en homéostasie (Jacobs & Wingfield, 2000; Wingfield, 2008). Une gestion efficace de ces ajustements implique un équilibre dynamique entre acquisition des ressources, capacité de stockage et absorption des coûts associés (Wingfield & Kitaysky, 2002). En effet, certains modèles théoriques d'optimisation d'énergie permettent de prédire l'investissement reproducteur selon la capacité des individus à stocker leurs réserves énergétiques (Rowe et al., 1994). Ces modèles ont ensuite été validés par des études empiriques chez les eiders à duvet (*Somateria mollissima*) et les grandes oies des neiges (*Anser caerulescence atlanticus*; Rowe et al., 1994; Bêty et al., 2003; Descamps et al., 2011; Hennin et al., 2016).

Lorsque les ressources ne sont pas limitantes, par exemple pour des individus en captivité nourris *ad libitum*, certains compromis attendus ne sont plus observés. Par exemple, l'investissement reproducteur ne se fait plus au détriment de la survie (Ricklefs & Cadena, 2007). Winder et al. (2025) montrent chez plusieurs espèces d'oiseaux que les coûts de la

reproduction sur la survie n'apparaissent que lorsque l'augmentation expérimentale de la taille de ponte dépasse ce qui serait observable à l'état naturel. Similairement, Grandmont et al. (2023) montrent que les grandes oies des neiges peuvent sauter un évènement de reproduction en réponse à un évènement imprévisible, particulièrement lorsque les conditions environnementales sont défavorables et ne permettent pas de récupérer de cet évènement stressant. Par conséquent, certains individus pourraient être plus efficaces que d'autres à acquérir des ressources et stocker des réserves, telles que les réserves endogènes ou sous forme de cache de nourriture. Ceux-ci pourraient alors être moins affectés par ces compromis (Bourg et al., 2019) et pourraient en théorie allouer leur énergie en maximisant à la fois leur reproduction et leur survie (Maklakov et al., 2015; Winder et al., 2025).

Étudier les variations d'acquisition, de stockage et d'allocation d'énergie permet ainsi de mieux comprendre les variabilités inter- et intra-individuelles. L'hétérogénéité individuelle d'acquisition d'énergie peut expliquer les différences de qualité individuelle (Vedder & Bouwhuis, 2018). Une dégradation multifactorielle de l'homéostasie au fil de la vie serait ainsi sous-jacente à la sénescence (Cohen, 2016). Additionnellement, la réponse face à des évènements de stress imprévisibles peut varier selon les stades de vie ou les individus, selon l'état physiologique de l'individu et ses capacités d'acquisition et stockage de l'énergie (McEwen & Wingfield, 2003; Landys et al., 2006).

Les effets des évènements de stress

Un évènement imprévisible de stress, aussi appelé stresseur, met l'organisme dans un état d'urgence et déclenche une cascade d'ajustements physiologiques (Wingfield et al., 1998; Landys et al., 2006). Une sécrétion massive d'adrénaline immédiatement après la perception de l'événement stressant entraîne la réponse « combat-fuite » ou « *fight or flight* » (Jacobs & Wingfield, 2000; Koolhaas et al., 1999). Cela engendre une hausse du rythme cardiaque, l'arrêt de la digestion, la mobilisation des ressources énergétiques et leur redirection vers les muscles (Koolhaas et al., 1999; Romero et al., 2009) afin d'affronter un danger immédiat pouvant avoir des conséquences sur la survie. Le même stimulus stressant va également activer l'axe hypothalamo-hypophysaire surrénalien (HHS ou HPA en anglais), qui viendra augmenter la sécrétion de glucocorticoïdes dans le sang (Romero & Wingfield, 2001). Ainsi, quand l'adrénaline diminue, environ trois minutes après le stresseur initial, les

glucocorticoïdes permettent la poursuite des ajustements nécessaires pour augmenter la survie immédiate : vigilance accrue, augmentation du rythme cardiaque, mobilisation des ressources (Buchanan, 2000; Romero et al., 2009). Par une boucle de rétroaction négative, les concentrations de glucocorticoïdes reviennent éventuellement à leurs niveaux basaux, accompagnés du retour à l'homéostasie des autres paramètres mentionnés (Wingfield et al., 1998; Romero, 2004).

Étant très énergivore, cette cascade d'ajustements repose sur la mobilisation de réserves endogènes par l'augmentation du taux métabolique, générant une augmentation du stress oxydatif (Monaghan et al., 2009). Or, le stress oxydatif peut engendrer des dommages à l'organisme, par exemple à l'ADN et au système immunitaire (Monaghan et al., 2009; Speakman & Selman, 2011). Ainsi, l'équilibre entre une forte réponse au stress pour maximiser la survie immédiate et une limitation du stress oxydatif pour favoriser la survie future est au cœur des questionnements sur les compromis d'histoire de vie (Monaghan et al., 2009; Buttemer et al., 2010; Hall et al., 2010). L'accumulation ou la prolongation de cette réponse au stress énergivore peut entrainer une surcharge allostatique (McEwen & Wingfield, 2003) et une altération de la boucle de rétroaction négative (Romero, 2004). La sécrétion accrue et prolongée de stress oxydatif qui en découle explique l'influence de l'accumulation des évènements de stress au cours de la vie sur la qualité des individus (Hau et al., 2015; Lin & Epel, 2022).

Il existe aussi une grande variabilité interindividuelle de l'intensité de la réponse au stress, liée à la qualité individuelle (Breuner et al., 2008; Ouyang et al., 2016). La sécrétion de glucocorticoïdes elle-même dépend à la fois des conditions de développement (Loiseau et al., 2008; Crino & Breuner, 2015), des effets maternels (Breuner, 2008), du stade d'histoire de vie (Schoenle et al., 2018), des interactions sociales, de l'environnement actuel, de l'âge et de la condition physiologique (Breuner et al., 2008). De plus, à la fois l'intensité de la réponse hormonale et l'augmentation du métabolisme qu'elle provoque dépendent des ressources disponibles dans l'environnement et dans les réserves de l'individu (Breuner et al., 2008; Schoenle et al., 2018). Ainsi, l'intensité de la réponse hormonale au stress peut être un indicateur des compromis individuels entre survie immédiate et survie future, dépendant de la condition physiologique des individus (Crespi et al., 2013; Angelier et al., 2018). Des

variations interindividuelles seront observées selon la condition physiologique des individus qui affectera l'ampleur de la surcharge allostatique, particulièrement si celle-ci se prolonge (Edes et al., 2018; Schoenle et al., 2018, 2021). Selon l'ampleur des dommages, le fitness pourrait être affecté à court, moyen ou long terme.

Les télomères, marqueurs de l'état physiologique

Pour étudier les dommages quantifiables provoqués par le stress oxydatif, le suivi de l'attrition des télomères s'est imposé dans les dernières décennies. Les télomères sont des séquences répétitives (TTAGGG chez les vertébrés) non-codantes situées aux extrémités des chromosomes pour protéger les séquences codantes de l'ADN (Blackburn, 1991; Blasco, 2007). En effet, lors de la réplication de l'ADN, le dernier fragment du brin d'ADN qui sert de guide à la ribonucléase, enzyme réplicative, ne sera lui-même pas répliqué, donc l'ADN se raccourcit. Parallèlement, il a été montré que le stress oxydatif généré par le métabolisme accélère l'attrition des télomères *in vitro* (Von Zglinicki, 2002; Richter & Von Zglinicki, 2007), mais ce mécanisme est encore sous investigation *in vivo* (Boonekamp et al., 2017). Les télomères servent ainsi de tampon pour absorber ces raccourcissements sans affecter le fonctionnement des cellules. Leur séquence répétitive permet également leur réparation par la télomérase (Blackburn, 2005), dont l'activité dépend notamment de l'espèce (Haussmann et al., 2007), de l'âge, du niveau d'expression de l'enzyme et de l'allocation d'énergie aux mécanismes de réparation (Smith et al., 2022).

La longueur des télomères découle donc de l'équilibre entre raccourcissement à la réplication, dommages dus au stress oxydatif et activité de la télomérase (Blackburn, 2005; Monaghan & Haussmann, 2006). Quand les télomères atteignent une longueur critique, la cellule entre en apoptose, dont la conséquence est un arrêt de la multiplication. Ce mécanisme est lié à la limite de Hayflick présentée précédemment (Hayflick & Moorhead, 1961; Allsopp et al., 1992). On peut ainsi prédire la capacité réplicative des cellules et expliquer la variabilité individuelle observée dans les cultures cellulaires (Allsopp et al., 1992). Dans le contexte de la recherche biomédicale sur les humains, ce mécanisme a été élargi à l'échelle de l'organisme et les télomères sont largement considérés comme des marqueurs de vieillissement (López-Otín et al., 2023).

Étudié dans le cadre de l'écologie évolutive en comparant diverses espèces de vertébrés, ce lien entre longueur des télomères et sénescence a aussi suscité l'intérêt des biologistes (Bekaert et al., 2005). La corrélation avec la longévité a encouragé son utilisation comme indicateur de l'état de sénescence d'un organisme (Bize et al., 2009; Tricola et al., 2018). En particulier, la longueur des télomères en début de vie semble prédire la durée de vie d'un individu (Heidinger et al., 2012; Eastwood et al., 2019) et la stratégie d'histoire de vie (Dantzer & Fletcher, 2015). Le raccourcissement des télomères d'un même individu au cours de sa vie a bel et bien été observé (Vedder et al., 2022). Cependant, la longueur des télomères mesurée à un temps *t* ne peut pas nécessairement prédire l'âge, due à l'importante variabilité interindividuelle (Remot et al., 2022). L'âge seul n'explique qu'une faible part de cette variabilité, ce qui a progressivement amené l'interprétation de la longueur des télomères comme indice de la qualité individuelle plutôt qu'un proxy de l'âge (Bauch et al., 2013; Le Vaillant et al., 2015; Angelier et al., 2019). Ceci est appuyé non seulement par les limites de leur utilisation comme indicateur de la sénescence (Simons, 2015), mais également par leur lien avec le succès reproducteur (Bauch et al., 2013; Heidinger et al., 2021; Pepke, Kvalnes, Ranke, et al., 2022).

La longueur des télomères peut en effet refléter les différents aspects de la qualité individuelle présentés précédemment. Celle-ci est héritable (Bauch et al., 2022; Vedder et al., 2022), sujette aux effets maternels ou parentaux (Asghar, Bensch, et al., 2015), aux conditions de croissance (Watson et al., 2015) et aux stades de vie exigeants comme la migration ou la reproduction (Angelier et al., 2013; Bauch et al., 2013). L'exposition répétée au stress peut également accélérer l'attrition des télomères (Hau et al., 2015; Angelier et al., 2018; Chatelain et al., 2020). L'endommagement des télomères lié aux facteurs cités précédemment semble se faire via la production de ROS par le métabolisme accéléré (Chatelain et al., 2020), même si les résultats semblent dépendre des marqueurs de stress oxydatifs utilisés (Beaulieu et al., 2011; Boonekamp et al., 2017). Ainsi, le lien entre la longueur des télomères et la qualité individuelle passerait, au moins en partie, par l'hétérogénéité individuelle de taux métaboliques et défense antioxydantes. Cette hypothèse télomères-qualité (Angelier et al., 2019) est donc prometteuse mais des études empiriques sont encore nécessaires afin d'y apporter l'appui suffisant.

Un individu naît avec quelques attributs innés comme un bagage génétique, une longueur de télomères, une sensibilité au stress, une stratégie d'allocation d'énergie à la survie immédiate ou future, hérités de ses parents. Certaines variations de la performance dans le temps seront prévisibles et communes aux individus d'une même espèce. Par exemple elle peut augmenter lors des premières phases de vie jusqu'à la maturité, puis décroître chez les espèces présentant de la sénescence. Cependant, les parcours de vie de chaque individu influenceront cette performance de manières différenciées, même pour des individus ayant le même baguage génétique. Les télomères pourraient ainsi être de bons marqueurs intégratifs des variations d'une mortalité intrinsèque, liée à la physiologie de l'individu, interagissant avec les expériences de vie pendant et après le développement.

La chasse et ses conséquences sur les populations sauvages

Dans un contexte évolutif, il est important de prendre en compte l'interaction entre la mortalité intrinsèque et la mortalité extrinsèque. Selon les définitions classiques, la mortalité intrinsèque est associée à la physiologie des individus, et le plus souvent à leur qualité individuelle. Au contraire, la mortalité extrinsèque est une cause externe et aléatoire de mortalité (Koopman et al., 2015). Cependant, il a été démontré que la mortalité extrinsèque peut varier avec l'âge (Caswell, 2007), en interaction avec des facteurs habituellement classés dans des causes de mortalité intrinsèque (Koopman et al., 2015). Par exemple, une défaillance du système immunitaire peut augmenter la vulnérabilité à une infection aléatoire. Cette idée de vulnérabilité d'un individu à une cause de mortalité extrinsèque causée par un trait intrinsèque peut se généraliser à de multiples sources de mortalité, comme les accidents ou la prédation.

Comme la prédation, la chasse est une source de mortalité extrinsèque pouvant affecter les populations via une mortalité directe (les individus tués à la chasse) ou via une mortalité indirecte (le dérangement par exemple). Quand la chasse a un effet négatif direct sur la probabilité de survie, cette mortalité est dite additive (Lebreton, 2005), en opposition à la mortalité compensatoire qui affecte des individus qui seraient morts d'une autre cause (Boyce et al., 1999; Lebreton, 2005). Chez les espèces longévives où la probabilité de survie adulte est maintenue à des niveaux élevés, la chasse aura souvent un effet additif direct (Gauthier et al., 2001; Hamel et al., 2006). Ce sont cependant les effets indirects de la chasse

qui interagissent le plus souvent avec d'autres sources de mortalité ou des stress environnementaux (LeTourneux et al., 2022; LeTourneux, 2024). Ils affectent alors des traits liés au fitness comme la condition corporelle (LeTourneux et al., 2021). À l'échelle de la population, la capacité de compenser la mortalité indirecte de la chasse peut donc en partie dépendre de l'hétérogénéité individuelle de différents traits physiologiques et démographiques (Grzegorczyk et al., 2024). Chez la petite oie des neiges (*Anser caerulescens caerulescens;* Fowler et al., 2020) ou le canard colvert (*Anas platyrhynchos*; Szymanski et al., 2013), les caractéristiques de qualité individuelle comme l'efficacité d'acquisition et de stockage d'énergie, représentées par la condition corporelle, affectent la vulnérabilité à la chasse.

Par son influence sur la mortalité et sur divers traits physiologiques, la chasse peut biaiser la détection de la sénescence, les estimations de paramètres démographiques tels que la survie, ou influencer les processus eux-mêmes (Loison et al., 1999; Jones et al., 2008). La chasse peut notamment affecter la sénescence par la sélection d'individus selon leur condition physique ou leur qualité individuelle (Mysterud et al., 2005). Cette sélection est particulièrement évidente dans un contexte de chasse au trophée (Loison et al., 1999; Coltman et al., 2003). Il a malgré tout été possible d'observer des patrons de sénescence chez des espèces de mammifères chassées, comme l'orignal (*Alces alces)* et le chevreuil (*Capreolus capreolus*; Gaillard et al., 2004). Les oiseaux migrateurs chassés comme les anatidés sont fortement étudiés pour l'écologie et la gestion, mais la senescence est souvent un paramètre ignoré (Bouwhuis & Vedder, 2017; Grzegorczyk et al., 2024). Toutefois, l'intérêt en termes de gestion pour ces espèces a mené à des suivis démographiques à long terme, surtout pour les espèces longévives. Lorsque de tels suivis existent, ils constituent de bonnes opportunités pour l'étude de la sénescence.

Modèle d'étude : la grande oie des neiges

La grande oie des neiges (*Anser caerulescens atlanticus*) est une sous-espèce d'oie des neiges qui hiverne sur la côte est des États-Unis, se reproduit dans l'est du haut Arctique canadien et fait une halte migratoire de plusieurs semaines au Québec le long du fleuve Saint-Laurent au printemps et à l'automne (Gauthier et al., 1992; Figure 0.2). Elle est étudiée grâce au programme de suivi systématique de la reproduction et de marquage établi en 1990 à l'Île

Bylot, Nunavut (73° N, 80° W), colonie représentant environ 15% de la population reproductrice de cette sous-espèce (Reed et al., 1998). En complément, des campagnes de marquage ont été menées sur leur halte migratoire au Québec (Île-aux-Oies, 47°00′N 70°33′W) en 2006-2009 et 2019-2023.

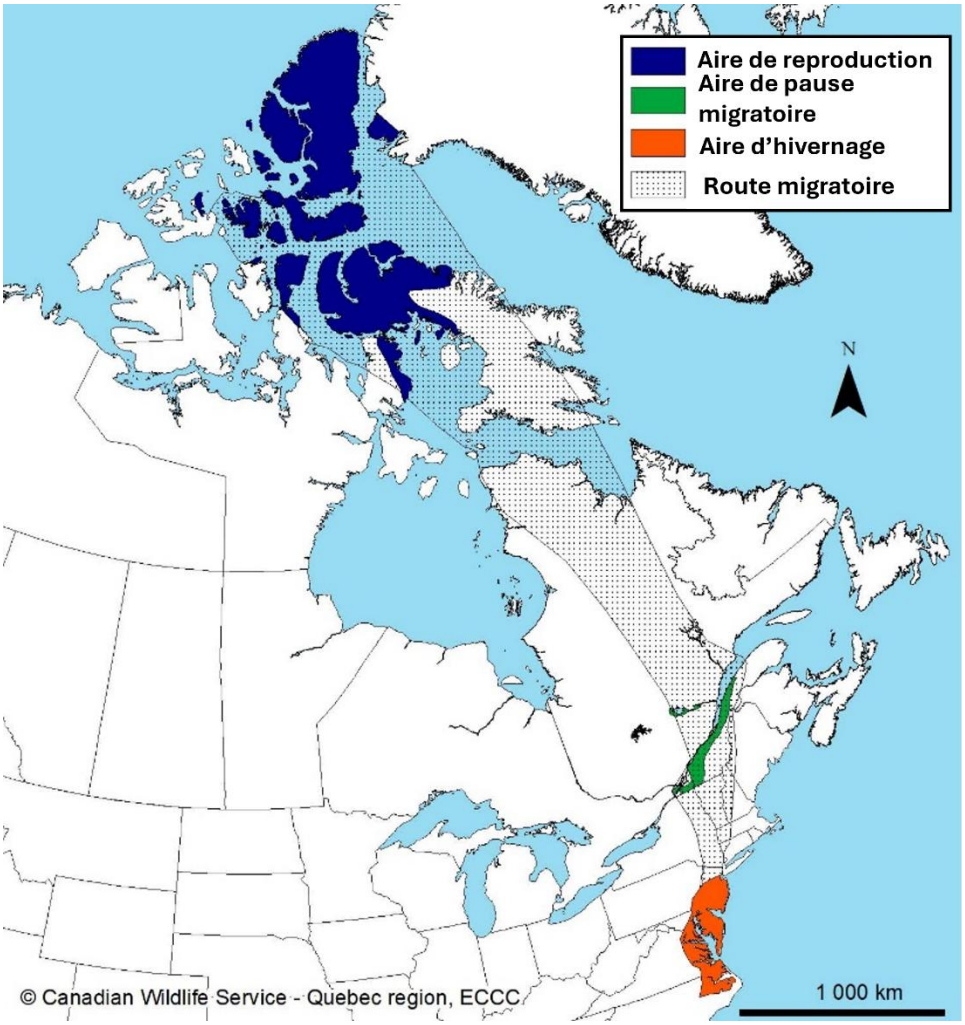

Figure 0.2: Carte de la migration de la grande oie des neiges, tirée de Lefebvre et al. (2017).

Ces haltes migratoires, particulièrement au printemps, sont critiques pour leur cycle annuel, car les oies s'engraissent et accumulent des réserves endogènes qui serviront à la migration et à la reproduction (Gauthier et al., 1992, 2003). La condition corporelle des femelles à la fin de cette période d'engraissement détermine l'investissement reproducteur. Si celle-ci est trop faible, elles sauteront un évènement de reproduction, ce qui maximise leur survie et augmente leur probabilité d'une future reproduction (Bêty et al., 2003; Souchay et al., 2014). Les décisions reproductives des femelles sont essentielles car les oies forment des

couples monogames. Les femelles sont philopatriques, alors que les mâles dispersent et suivent leur femelle vers son site de reproduction (Prevett & MacInnes, 1980). Si la femelle est en trop faible condition pour se reproduire, le couple effectuera une plus courte migration vers un site de mue moins nordique (Reed et al., 2003). Une fois dans le haut Arctique, les conditions environnementales sont déterminantes pour le succès reproducteur (Reed, 2004) et la croissance des jeunes (Lepage et al., 1998; Doiron et al., 2015). Entre autres, un fort décalage entre le pic de ressources végétales et l'éclosion peut affecter la taille des jeunes à l'envol (Doiron et al., 2015) et leur survie dans la première année (LeTourneux, 2024).

La grande oie des neiges est une espèce longévive : son temps de génération est d'environ 6,5 ans (Gauthier & Lebreton, 2004), sa durée de vie maximale observée en conditions naturelles est de 24 ans (Chapitre 2) et le taux de survie adulte est élevé (~0.80) et peu variable entre les années (Gauthier et al., 2001; LeTourneux et al., 2024). Ce taux de survie adulte élevé et stable est caractéristique des espèces longévives avec des stratégies d'histoires de vie lentes (Stearns, 1992; Gaillard & Yoccoz, 2003), chez lesquelles l'investissement reproducteur est modulable et la survie maximisée. Cela signifie que la survie est certes moins variable entre les années, mais que de faibles variations de survie adulte pourraient avoir de gros impacts sur la dynamique de population (Gauthier & Brault, 1998).

Dans les années 1990, la population de grande oie des neiges a augmenté exponentiellement dû à leur utilisation de champs agricoles lors de leur engraissement printanier, ce qui a à la fois augmenté leur survie et leur succès reproducteur (Gauthier et al., 2005). Après deux mesures de gestion en 1999 et 2009, elle est chassée de leur retour des aires de reproduction en septembre jusqu'à leur départ vers l'Arctique en mai pour maintenir la population entre 500 000 et 750 000 individus (Calvert & Gauthier, 2005; Lefebvre et al., 2017; LeTourneux et al., 2022, 2024). Ces mesures de gestion sont efficaces car elles diminuent la survie des juvéniles et des adultes par de la mortalité directe, mais également indirectement par le stress généré par la chasse (Béchet et al., 2004; LeTourneux et al., 2021, 2022).

Nous disposons sur cette espèce de données de captures, de retours de bagues d'oies récoltées à la chasse, de mesures morphométriques et d'échantillons de sang collectées lors

du suivi systématique de cette espèce. La qualité, quantité et la continuité de ces données offrent une opportunité unique d'estimer la survie pour mettre en évidence l'hétérogénéité individuelle de nombreux traits et appuient la pertinence de ce modèle d'étude.

Objectifs de la thèse

L'objectif principal de cette thèse est d'améliorer notre compréhension des variations individuelles de survie chez une espèce longévive soumise à différents stress durant son parcours migratoire dans un cadre théorique éco-évolutif associant sénescence et qualité individuelle. Pour atteindre cet objectif, j'utilise deux jeux de données complémentaires : un provenant de captures pendant la migration à l'Île-aux-Oies en 2009, suivies de ré-observations de 2010 à 2021, et un second provenant du suivi de la population à l'Île Bylot de 1990 à 2022. Ce doctorat combine des données de suivi à long terme de capture-marquage-recapture, ainsi que données de mortalité à la chasse et des mesures morphologiques et physiologiques présentant de fortes variations individuelles. Ces traits sont reliés à la survie, paramètre démographique présentant la plus haute élasticité chez la population de la grande oie des neiges.

Dans le **Chapitre 1**, nous avons examiné le patron de sénescence actuarielle en utilisant les données sur des oies marquées étant juvéniles puis recapturées ou rapportées par les chasseurs les années suivantes. Les patrons de sénescence actuarielle sont désormais bien documentés dans les populations sauvages, chez les mammifères et les oiseaux (Bouwhuis & Vedder, 2017; Gaillard & Lemaître, 2020), mais l'interaction avec la chasse reste encore peu discutée. Les études sur la sénescence des oiseaux sauvages longévifs reposent principalement sur les populations d'oiseaux marins non chassés (Aubry et al., 2011; Froy et al., 2013; Pardo et al., 2013; Bouwhuis & Vedder, 2017). Nous proposons ici d'estimer le patron général de survie en fonction de l'âge en interaction avec la pression de chasse. Nous attendons une augmentation de la survie dans les deux premières années de vie, un plateau de survie maximale de plusieurs années puis un déclin rapide (Ricklefs, 2008), similaire au patron trouvé chez les fous de Nazca (*Sula granti*; Anderson & Apanius, 2003).

Dans les chapitres suivants, nous avons souhaité examiner les mécanismes qui expliquent les variations de survie en se concentrant sur des traits physiologiques comme la

corticostérone, la longueur des télomères ainsi que sur des traits morphologiques comme la condition et la taille corporelle.

Le **Chapitre 2** s'intéresse à l'effet de la réponse au stress sur la survie, particulièrement lors d'une accumulation de stresseurs. Une étude corrélative récente sur la grande oie des neiges a montré que l'accumulation de l'augmentation de la chasse et le port d'un collier affectaient négativement la survie par un effet synergétique (LeTourneux et al., 2022). Ce chapitre permet de tester expérimentalement si l'accumulation de stresseurs peut en effet diminuer la survie malgré les stratégies de maintien de ce paramètre chez cette espèce. Nous avons pour cela examiné les effets d'une expérience effectuée au printemps 2009 à moyen et long termes. Cette expérience visait à réduire la condition des oies au printemps en les maintenant en captivité avec un accès ou non à de la nourriture (Legagneux et al., 2012; Grandmont et al., 2023). Elle permet donc d'évaluer l'effet de stresseurs accumulés sur la survie et comment cette réponse était modulée par l'intensité de la réponse au stress et la condition corporelle. La diminution de survie attendue chez les individus avec la plus grande accumulation de stresseurs passerait par une surcharge allostatique importante. Il serait ainsi cohérent d'attendre un effet positif de la condition corporelle et un effet négatif de la réponse hormonale au stress sur la probabilité de survie annuelle.

Dans le **Chapitre 3**, nous avons examiné les liens entre différents traits associés à la qualité individuelle, leur hétérogénéité interindividuelle et leurs variations avec l'âge. En effet, l'interprétation de la longueur des télomères comme indice de la qualité individuelle s'est développée récemment mais demande encore d'être validée par plusieurs études empiriques. Nous avons spécifiquement examiné les relations entre la longueur des télomères, la réponse au stress, la taille corporelle et la condition corporelle post-reproduction et l'âge des individus. La taille corporelle est un indicateur de la composante fixée de la qualité individuelle (Doiron et al., 2015), alors que la condition corporelle représente un indice des réserves endogènes (Bêty et al., 2003). La réponse au stress, mesurée par les concentrations de corticostérone est un indice de la sensibilité au stress (Wingfield et al., 1998). La longueur des télomères représente un marqueur cumulatif des conditions en début de vie et des expériences vécues tout au long de la vie de l'individu (Bateson, 2016). Nous attentions des corrélations négatives entre longueur des télomères et concentration de

corticostérone, mais des corrélations positives entre longueur des télomères, condition corporelle et taille corporelle (Hau et al., 2015; Bateson, 2016; Angelier et al., 2019). Nous prédisions également une diminution de la longueur des télomères et de la condition corporelle avec l'âge à cause de la sénescence, selon l'hypothèse télomères-qualité (Angelier et al., 2019).

Le **Chapitre 4** s'inscrit dans la continuité du Chapitre 3 et évalue le lien entre la longueur des télomères, la condition corporelle, la taille corporelle et la survie adulte. Pour approfondir la réflexion, nous avons également évalué le lien entre la longueur des télomères et la mortalité à la chasse. Nous prédisions une relation négative entre longueur des télomères et survie, comme chez de nombreuses espèces d'oiseaux, accompagnée d'une plus forte mortalité à la chasse des individus à plus courts télomères (Wilbourn et al., 2018).

Chapitre 1. Evidence of actuarial senescence in a hunted long-lived migratory bird

Île Bylot, Août 2024

En préparation:

Grentzmann I.P., LeTourneux F., Gauthier G., Legagneux P. In prep. Evidence of actuarial senescence in a hunted long-lived migratory bird

1.1 Résumé

La sénescence actuarielle est la baisse de la probabilité de survie avec l'âge, pouvant provenir de processus physiologiques et être influencée par des causes externes de mortalité. Les mortalités intrinsèque et extrinsèque étaient traditionnellement étudiées séparément, mais leur interaction est désormais claire, et mène à des réponses complexes de la mortalité totale. La chasse, source de mortalité extrinsèque, est un outil de conservation depuis plusieurs décennies et une cause primaire de mortalité dans certaines populations sauvages comme la grande oie des neiges (*Anser caerulescens atlanticus*). Malgré son importance, l'interaction entre la chasse et la sénescence n'est que rarement étudiée chez les oiseaux sauvages. Chez la grande oie des neiges notamment, les modèles de population utilisés pour la gestion ne considèrent pas le potentiel effet de la sénescence sur les variations de survie. Notre étude se démarque ainsi en se concentrant sur l'interaction entre la chasse et la sénescence dans une population d'oiseaux sauvage. Nous bénéficions pour cela de données à long terme depuis plus de 30 ans avec de nombreux individus d'âge connu (N=54 693), marqués à l'Île Bylot puis recapturés et repris à la chasse. Nous pouvons ainsi utiliser des analyses de capture-marquage-recapture afin d'établir le patron de sénescence actuarielle et d'explorer son lien avec la pression de chasse. Nous trouvons que la sénescence débute aux alentours de 11 ans et qu'elle n'est pas affectée par la pression de chasse. Ces résultats montrent que des patrons de sénescence sont observables chez des espèces aviaires chassées, et que des mécanismes de compensation de la mortalité ont lieu dans cette population. Des études futures comparant des populations sujettes à des pressions de chasses contrastées seront nécessaires pour approfondir notre compréhension de l'impact de la chasse sur la sénescence d'un point de vue évolutif.

1.2 Abstract

Actuarial senescence is the decrease in survival probability with age, resulting from intrinsic physiological processes and influenced by external causes of mortality. Intrinsic and extrinsic mortality were traditionally studied separately, but their interaction is now clear and leads to complex total mortality responses. Hunting, an extrinsic source of mortality, has been utilized as an important conservation tool for several decades, is a primary cause of mortality in some wild populations, like the greater snow goose (*Anser caerulescens atlanticus*). In spite of its importance, hunting's interaction with senescence is rarely studied in wild birds. In the greater snow goose, population models used for management do not consider the potential effect of senescence in the variations of survival. Our study stands out by its focus on the interaction between hunting mortality and senescence in a wild bird population. For this, we benefit from long-term data covering more than 30 years with numerous known age individuals (N=54 693) marked at Bylot Island and then recaptured and recovered by hunters. We were thus able to use capture-mark-recapture analyses to establish the actuarial senescence pattern and to explore its link with its relationship with hunting pressure. We find that senescence starts at ca. 11 years but is not affected by hunting pressure. These results show not only that senescence patterns are observable in harvested wild bird populations, but also that mortality compensation mechanisms occur in this population. Further studies comparing populations subject to contrasting hunting pressure are necessary to deepen our understanding of the evolutionary implications of harvest on senescence.

1.3 Introduction

Senescence was first characterized at the cellular level (Hayflick & Moorhead, 1961) but is now more largely considered at the organismal level. It can be expressed through two major fitness components: a decline in reproductive output with age (Bouwhuis et al., 2012) or an increase in intrinsic mortality rate, called actuarial senescence (Kirkwood & Rose, 1991). The incidence of both components of senescence in wild populations has been extensively studied over the last 30 years (Holmes & Austad, 1995; Nussey et al., 2013; Jones et al., 2014; Bouwhuis & Vedder, 2017), and a diversity of patterns has been observed across taxa (Jones et al., 2014; Roper et al., 2021). Senescence patterns have been documented through long-term studies mainly in ungulates (Gaillard et al., 2004) and birds (Holmes & Austad, 1995; Holmes et al., 2001; Bouwhuis & Vedder, 2017). For similar life-history strategies, birds tend to be more long-lived and show a more abrupt decline in survival at an advanced age compared to mammals (Ricklefs, 2008). However, research on senescence is still taxonomically limited, with a bias in favour of long-lived species, especially seabirds (Aubry et al., 2011; Foote et al., 2011; Breton et al., 2014) and ungulates (Gaillard et al., 2004; Gaillard & Lemaître, 2020).

The role played by intrinsic and extrinsic mortality sources in actuarial senescence is still unclear, in humans as well as in wild animal populations (Koopman et al., 2015; Grzegorczyk et al., 2024). Intrinsic mortality is usually defined as resulting from internal physiological mechanisms, whereas extrinsic mortality is caused by random external causes such as diseases, predation or extreme environmental conditions (Koopman et al., 2015). However, extrinsic mortality can also be affected by age (Caswell, 2007) or physiological characteristics commonly associated with intrinsic mortality (Koopman et al., 2015). In addition, extrinsic mortality sources can induce indirect and delayed mortality (e.g., through disturbance), which could depend on intrinsic factors such as energy intake or behavior (Pettorelli et al., 2011), causing an apparent increase in intrinsic mortality. Looking at the response in total mortality to a specific mortality source can thus be interesting when investigating variations in life history traits (Péron, 2013). The boundary between the two types of mortalities is thus still unclear and it is accepted that cause-specific mortality will

have a broader impact on total mortality through a combination of compensation mechanisms (Péron, 2013; Koopman et al., 2015).

Population responses to an extrinsic mortality source, such as predation or harvest, vary along a gradient from additive to compensatory mortality (Sandercock et al., 2011; Péron, 2013). Compensation occurs when individuals affected would have died of other mortality causes (Lebreton, 2005; Grzegorczyk et al., 2024), and manifests through the apparent lack of response of the annual survival probability to a cause-specific mortality (Boyce et al., 1999; Lebreton, 2005). It can happen when harvest interacts with intrinsic mortality sources, affecting lower quality individuals, historically called the "doomed surplus" (Errington, 1945). Conversely, additive mortality occurs when individuals would have survived if not for the mortality source of interest.

The harvest of wild populations is a source of external mortality that can eventually mask (Loison et al., 1999; Jones et al., 2008) or modify senescence patterns (Loison et al., 1999; Mysterud et al., 2005). Indeed, individual traits like body condition can affect vulnerability to hunting (Szymanski et al., 2013; Fowler et al., 2020). More generally, hunting mortality has been shown to affect evolutionary processes and life history traits (Loison et al., 1999; Gamelon, 2020), in particular in the context of trophy hunting (Loison et al., 1999; Coltman et al., 2003). Senescence was observed in hunted mammals, but harvest is rarely discussed as a central issue (Loison et al., 1999; Gaillard et al., 2004; Gaillard & Lemaître, 2020). In birds, most long-lived species studied for their senescence patterns, such as seabirds, are not harvested (Bouwhuis & Vedder, 2017). Studies on harvested birds such as willow ptarmigans (*Lagopus lagopus*; Israelsen et al., 2020) or wild turkeys (*Meleagris gallopavo*; Norman et al., 2022; Wightman et al., 2024) have detected differences in hunting vulnerability between juveniles and adults, but did not examine senescence patterns in these short-lived species. Harvest on birds is generally not considered a direct selection pressure for morphological traits (Grzegorczyk et al., 2022), but heterogeneity in vulnerability to hunting within populations can affect which individuals are more susceptible to die (Christensen, 2001; Guillemain et al., 2007; Szymanski et al., 2013; Fowler et al., 2020).

In this study, we aim to establish the actuarial senescence pattern of the greater snow goose (*Anser caerulescens atlanticus*) and its relationship with hunting pressure. The greater

snow goose has a known maximum life expectancy of 24 years, making it a relatively long-lived species. It is highly harvested during much of its life cycle, including in autumn, winter and, more recently, spring due to its overabundance (Gauthier et al., 2005; LeTourneux et al., 2022). Hunting is thus an important aspect of the species population dynamics (Gauthier et al., 2001), and is not a random source of mortality. Adult vulnerability to hunting can for instance be increased by stress response or low body condition (Fowler et al., 2020; Chapter 2). The hunting mortality also informs our survival model, giving us additional data about the fate of banded individuals (Gauthier & Lebreton, 2008). This makes the greater snow goose a good model to study the influence of hunting pressure on the senescence pattern. If intrinsic mechanisms of senescence increase vulnerability to hunting, compensation mechanisms could affect older individuals differently than younger ones. Moreover, hunting modified the relationship between mortality and age in the lesser snow goose (*Anser caerulescens caerulescens*; Koons et al., 2014) We thus expected hunting pressure to modify the relationship between survival and age in response to compensation mechanisms varying with age (Péron, 2013). Indeed, if intrinsic physiological mechanisms underlying senescence increases vulnerability to hunting, the effect of hunting pressure should increase with age.

1.4 Materials and methods

1.4.1 Study species, study site and data collection

The greater snow goose is a long-distance migratory species that winters on the East coast of the United States, breeds in the Canadian high Arctic (June-August), and stop-overs along the St. Lawrence River (Québec, Canada) during fall and spring migrations (Lefebvre et al., 2017). Its reproduction has been monitored annually since 1990 at its largest known colony situated on the south plain of Bylot Island, Nunavut (73N 80W). In July, between 300 and 3 000 goslings are marked in the nest with uniquely numbered web-tags within 24 hours of hatching, and between 1 000 and 6 000 geese are captured and marked (Reséndiz-Infante et al., 2020).

Mass banding operations take place near the end of the brood-rearing period, while adults are moulting and flightless (Menu et al., 2001). All captured birds (young,

i.e. individuals born during the summer, and adults, i.e. ≥ 1 year old) are sexed by cloacal examination and banded. All hatching year birds are checked for the presence of web-tags (Reséndiz-Infante et al., 2020). Greater snow geese have long been hunted in eastern Canada in the fall and in the US in winter. They are also hunted in spring in Québec since 1999, and the winter season was prolonged in 2009 in the US.

For this study, we used capture histories from known-age individuals that were captured and banded as young (N=54 693). The only way to know the age of an individual is to mark it during its hatching year. Encounters following initial marking were made up of recaptures of live individuals at Bylot Island during the subsequent summers and dead recoveries reported by hunters both in Canada and the US (Gauthier & Lebreton, 2008).

We used the harvest rate as a quantifiable indicator of hunting pressure. We computed the harvest rate as the total number of harvested geese between two consecutive summers, divided by the fall population size. Annual harvest data during spring and fall is estimated through standard hunter surveys conducted in the Unites States and Canada, adjusted in 1999 and 2009 to each new hunting regulation (Lefebvre et al., 2017). The fall population size is estimated by multiplying the spring population estimate by 0.96, the estimated survival rate during summer (Gauthier et al., 2001). The spring population size estimate used in the calculation of the harvest rate comes from the annual aerial spring survey of the greater snow goose population. This aerial survey is conducted by the Canada Wildlife Service when the population is concentrated along the St. Lawrence River staging area in Québec (Béchet et al., 2004; Lefebvre et al., 2017; LeTourneux et al., 2022). Harvest rate values were calculated separately for juveniles (age 0, the first year of life) and adults (age 1 to 18+) for years 1989 to 2017 (Figure S1.1 in Appendix S1.1).

1.4.2 Capture-mark-recapture model

We estimated age-dependent annual survival using a joint live-and-dead-encounter multievent capture-mark-recapture model (Pradel, 2005), in a Bayesian framework (Servanty et al., 2010; Kéry & Schaub, 2012). We used a parametrization analogous to the one of LeTourneux (2024) to estimate annual survival *(φ)*, live-encounter (*p*), recovery (*r*) and permanent emigration (*E*) probabilities. Age was defined as categories going from 0 to 17+.

The cut-off at 17 years was chosen to enable a clear senescence curve while limiting the computational power needed.

We defined the transition matrix as follows:

	Alive Young	Alive Adult Bylot	Alive Adult Emigrated	Newly Dead	Dead
Alive Young	0	$\varphi_t^Y \times (1 - E_{sex}^Y)$	$\varphi_t^1 \times E_{sex}^Y$	$1 - \varphi_t^Y$	0
Alive Adult Bylot	0	$\varphi_{age,t}^A \times (1 - E_{sex}^A)$	$\varphi_{age,t}^A \times E_{sex}^A$	$1 - \varphi_{age,t}^A$	0
Alive Adult Emigrated	0	0	$\varphi_{age,t}^A$	$1 - \varphi_{age,t}^A$	0
Newly Dead	0	0	0	0	1
Dead	0	0	0	0	1

Where φ_t^Y is the probability that a young survives and enters the adult stage; E_{sex}^Y the sex-specific emigration probability for young; E_{sex}^A the sex-specific emigration probability for adults; $\varphi_{age,t}^A$ adult survival (details below). The "Newly Dead" state lasts for one occasion, results in an inevitable transition to "Dead", and allows for the observation of dead individuals (recoveries).

We compared two models for survival estimation. Our most general model included an interaction between harvest rate (HR) and age and was compared to a reduced model where there was only an additive effect of HR and age. In the most general model, young survival was defined as:

$$logit(\varphi_t^Y) = \mu^Y + \beta_{HR}^Y \times HR_t^Y + \varepsilon_t$$

Where φ_t^Y includes an intercept μ, an effect of harvest on young individuals β_{HR}^Y, and a random effect of time ε_t. HR_t^Y is the harvest rate of young at year *t*.

In this model, adult survival was defined as:

$$logit(\varphi_{age,t}^A) = \mu^A + \beta_{age} + \beta_{HR}^A \times HR_t^A + \beta_{age:HR} \times HR_t^A + \varepsilon_t$$

Where $\varphi^{A}_{age,t}$ depends on age and time; μ^{A} is the average adult survival; β_{age} is the effect of each age category; β^{A}_{HR} is the adult-specific additive coefficient for the effect of harvest rate at year t (HR^{A}_{t}); $\beta_{age:HR}$ is the effect of harvest rate for each age and year t and ε_{t} is the random effect of year t.

Emigration was estimated for juveniles (E^{Y}_{sex}) and adults (E^{A}_{sex}), and males and females separately. Most individuals that emigrate are juvenile males, but adults can also emigrate if, for instance, their mate dies and they follow their new mate to a new breeding location (Lecomte et al., 2009). Detection of these emigrated birds was set at 0 (Appendix S1.2) and emigration was considered irreversible (see transition matrix).

Live-encounter probability (p) was set to 1 for young because marked individuals can only be observed young at their first capture, after which they are in the adult state, conditional on survival. For adults, we included a fixed effect of sex and a random effect of year on p (Appendix S1.2). Recovery probabilities (r) differed for young and adults, and we included a different random effect of year for young and adults on this probability (Appendix S1.2).

Prior to conducting the survival analysis, we tested the goodness-of-fit of the general model with the program U-CARE V2.3.5 (Choquet, Lebreton, et al., 2009). Following guidelines by Gimenez et al. (2018), we separated our data by sex to account for unequal emigration affecting detection probabilities. We also suppressed the first encounter because we estimated a different survival probability for the first year. All tests were non-significant (all p-values > 0.1; overall χ^2 value of 505.2 for 498 degrees of freedom), and overdispersion was negligible ($\hat{c}$ = 1.01), indicating our model fit the data well.

Posterior distributions of model parameters were obtained using Markov Chain Monte Carlo (MCMC) techniques implemented within the *nimble* package and the specialized distributions for capture-recapture modelling from the *nimbleEcology* package in R (Goldstein et al., 2024; de Valpine et al., 2024). We modified the *dDHMMo* function used to compute the marginalized likelihood of each capture history for it to accommodate an age structure of the transition matrices (available on the online repository). This reduced the memory used by the model since it allowed calculating survival probabilities indexed by age

class rather than by individual. We used vague priors for all parameters, except permanent emigration. To facilitate convergence, we used an informative prior for the emigration probability, using estimates from LeTourneux (2024). All used priors are available in Supplementary Materials (Appendix S1.3). For years when no harvest rate value was available (2018-2021), we drew values from a normal distribution for which the mean and standard deviation priors were informative and corresponded to those of our harvest data (Appendix S1.3). We ran 3 chains for 20 000 iterations with a burn-in of 3 000 iterations, and thinned 1/3 of samples to reduce memory use. We assessed convergence of the simulations using trace graphs and the Gelman–Rubin diagnostic ($\hat{R}$ or R-hat; Gelman & Rubin, 1992). We considered R-hat values ≤ 1.1 as evidence of good convergence (Gelman & Hill, 2007; Kéry & Schaub, 2012). We compared our two models using the Watanabe–Akaike Information Criterion (WAIC), a Bayesian equivalent of the Akaike Information Criterion (Gelman et al., 2014). We report all parameter estimates along with their 95% credible intervals. A complete list of model estimates, their 95% credible intervals and a summary of R-hat values are provided in Table S1.1 (Appendix S1.4).

1.5 Results

The preferred model only had the additive effect of harvest rate (WAIC difference between the model with interaction and the addictive model: $\Delta WAIC_{int\text{-}add} = 19.04$). Survival varied strongly with age and suggested the presence of senescence (Figure 1.1). Survival probabilities of juveniles (φ^{Y} = 0.31 [0.25, 0.39]) were much lower than any adult survival probability. Survival increased drastically in the second year (φ^{A}_{age1} = 0.80 [0.75, 0.84]) but did not quite reach prime age survival (e.g., φ^{A}_{age5} = 0.87 [0.84, 0.91]). A first drop appears after age five (e.g., φ^{A}_{age7} = 0.84 [0.80, 0.88]), and a second, steeper, drop in survival occurs around 11 years (e.g., φ^{A}_{age15}= 0.68 [0.57, 0.78]).

Harvest rate did not significantly interact with age as all credible intervals for the interaction included zero (Figure 1.2). As expected, harvest rate had a significant negative effect on survival (β^{A}_{HR}= -0.25 [-0.41, -0.08]; Figure 1.1). A similar marginally significant trend was detected for first-year survival although the effect was weaker (β^{Y}_{HR}= -0.11 [-0.26, 0.03]).

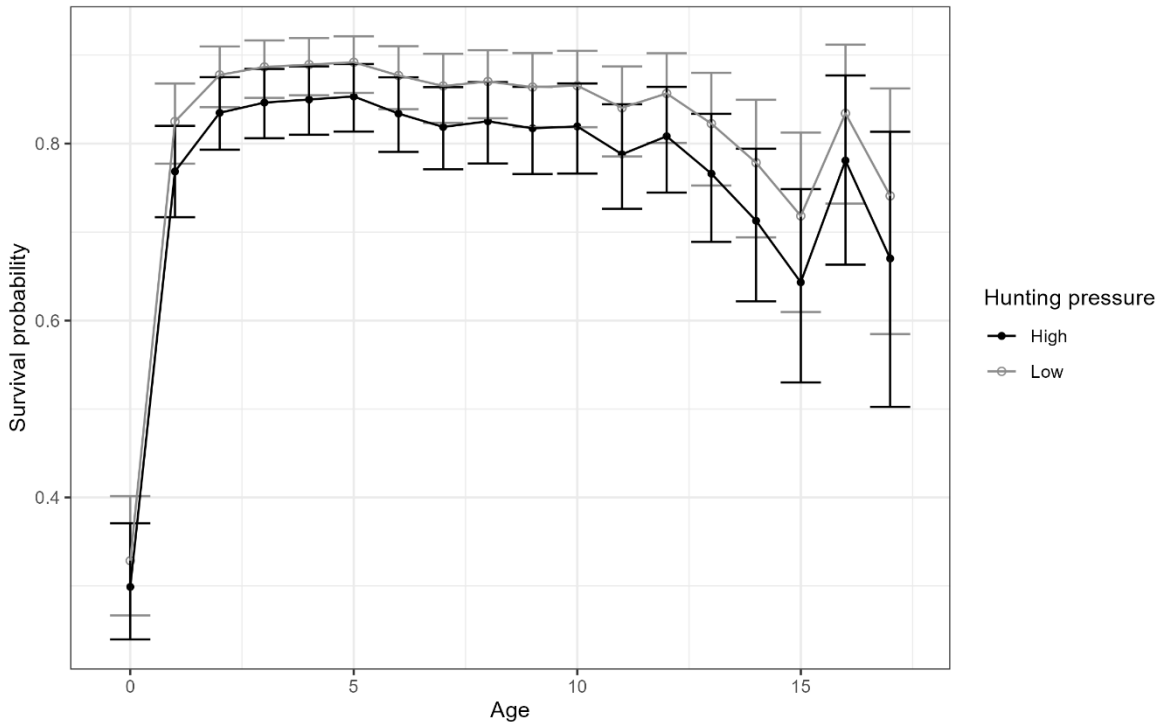

Figure 1.1: Model predictions for age-dependent survival probabilities of greater snow geese (points) along with their 95% credible intervals (error bars) for high (3rd quartile, Q3, in black) and low harvest rates (1st quartile, Q1, in grey).

Emigration probabilities differed for each of our four groups (Figure S1.2 in Appendix S1.5). Maximal emigration was highest in young males (E_{YM} = 0.96 [0.95, 0.97]), followed by young females (E_{YF} = 0.56 [0.50, 0.61]). Emigration of adult males was rare (E_{AM} = 0.01 [4.80e-04, 0.04]) and virtually null for adult females (E_{AM} = 4.71e-04 [1.21e-05, 1.72e-03]). Live encounter probability was slightly higher for males than for females ($\beta_{males\ vs.females}$= 0.34 [0.09, 0.59]) and varied between years (Figure S1.3 in Appendix S1.5). Recovery probabilities were significantly higher for adults than for juveniles ($\beta_{adults\ vs.juveniles}$= 1.04 [0.64, 1.43]), with more temporal variations (Figure S1.4 in Appendix S1.5).

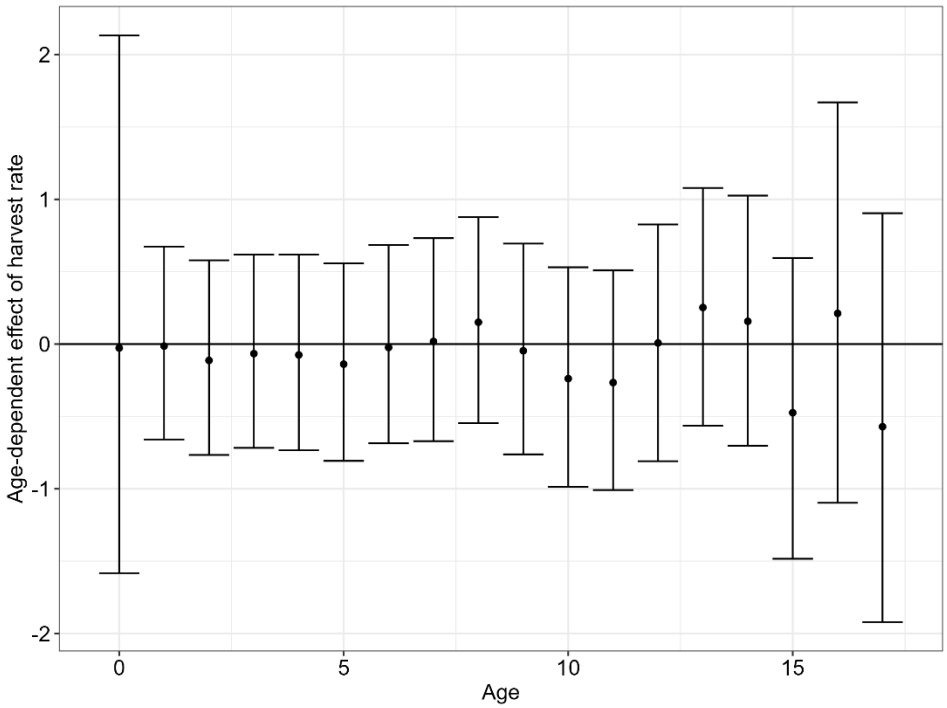

Figure 1.2 : Estimates of the interaction between harvest rate and age from model with interaction, with 95% credible intervals.

1.6 Discussion

We quantified age-dependent survival probability in response to variable hunting pressure across 54 693 known age individuals in a long-lived bird. Our data support actuarial senescence starting at ca. 11 years, and while hunting pressure significantly affected mortality, it did not interact with age. Actuarial senescence has been described in both short- (eg. Fox et al., 2006; Keller et al., 2008; Marzolin et al., 2011) and long-lived bird species (Anderson & Apanius, 2003), and in a multitude of long-lived mammal species (Gaillard et al., 2004; Bergeron et al., 2023). Senescent survival is characterized by increased variability, as has been shown in eastern grey kangaroos (*Macropus giganteus*; Bergeron et al., 2023) and Nazca boobies (*Sula granti*; Anderson & Apanius, 2003). This variability, however, is better characterized in mammals than in birds (Gaillard et al., 2000; Bouwhuis & Vedder, 2017; Bergeron et al., 2023). Senescence and the associated variability can be difficult to assess given the decline in individuals at each age; increased variability could thus be due to the shrinking sample size. Nevertheless, the hypothesis that individuals show important

heterogeneity in senescence rate remains an interesting and biologically meaningful hypothesis to be explored.

Although hunting can be a major source of mortality and has been discussed as a potential confounding factor in the study of senescence (Loison et al., 1999; Mysterud et al., 2005; Jones et al., 2008), it is usually treated as a nuisance and rarely directly investigated or discussed (Loison et al., 1999; Lemaître & Gaillard, 2017). Most research on birds avoids the issue altogether because species studied for senescence are not hunted (Bouwhuis & Vedder, 2017), whereas hunted bird species are rarely the subject of studies on senescence (Grzegorczyk et al., 2024 but see Koons et al., 2014). However, the impact of extrinsic mortality on senescence patterns has been theorized (Williams, 1957; Caswell, 2007), and hunting has been linked to variation in life history traits such as longevity and reproductive success in some species (e.g., wild boars *Sus scrofa;* Gamelon, 2020).

We expected an interaction between hunting pressure and age, as Péron (2013) noted that compensation or additivity dynamics of anthropogenic mortality could vary with age in some bird species. Our hypothesis rested on intrinsic mechanisms of senescence increasing vulnerability to hunting, resulting in greater effects of hunting at older ages. As such, older individuals could either be more, or less, sensitive to an increase in hunting pressure compared to younger individuals, depending on the compensation mechanisms involved. For example, older individuals could be expected to skip reproduction more frequently (Souchay et al., 2014), reducing hunting mortality risk due to the absence of young accompanying the parents (Giroux et al., 1986; Calvert et al., 2005). Alternatively, geese have shown their capacity to adapt their behavior to hunting activity (LeTourneux et al., 2023). Older individuals, who are more experienced, may better avoid hunters and thus be less susceptible to hunting mortality regardless of the presence of young. Under these scenarios, the interaction coefficients should be negative for young individuals and closer to zero for older ones. We cannot rule out that the absence of such interactions in our results could be due to a lack of power with the decreasing number of individuals with age. However, it could also signify that hunting mortality is compensatory to other sources of mortality. Intra-year seasonal compensatory mechanisms have been shown in this population, as a decrease in survival during one hunting season was followed by an increase in survival during the

following season (LeTourneux et al., 2024). Hunting thus seems to target more vulnerable individuals, and those surviving hunting mortality had higher probabilities of surviving during the following season. Our results show that the effect of hunting pressure is not age-dependent. They could thus be in agreement with these previous results, targeting weaker individuals across ages. Moreover, if hunting targets individuals that would die of other causes if not hunted, it could explain why the hunting pressure does not interact with age in the overall age-dependent survival pattern.

It is important to note that harvest rate increased substantially after 1999 due to changes in hunting regulations (Lefebvre et al., 2017; Figure S1.1). Since monitoring began in 1990, no known-age individual reached senescence under the earlier hunting period of lower harvest rates. This may limit our ability to detect a potential interaction between hunting pressure and age. However, our range of harvest rates still varied twofold (from 0.08 to 0.16; Figure S1.1), even when considering only data between 2000 and 2017. If the interaction between hunting pressure and age played a major role in the senescence observed, we would expect to observe a corresponding response within this range.

Emigration and recovery probabilities were in agreement with previous studies on the same species (LeTourneux et al., 2024), though adult recoveries were more variable than previous results. This can be explained by the small proportion of recaptured and recovered adults in our dataset. Since most of the individuals in our dataset are banded young, many die in the first year. Additionally, recapture probabilities are not very high. LeTourneux et al. (2024) used birds marked as adults in the database, which results in more birds surviving for longer and thus more recaptures, explaining why their estimates are less variable. Similarly, our live encounter probability is slightly higher for males than for females, which is inverse to what was previously found. This could also be explained by the very high proportion of young in our dataset, with almost certain emigration of young males. As a result, very few adult males remain and thus the live encounter probability of the very few males remaining could simply be higher by chance.

1.7 Conclusion

The originality of this study lies in the focus on the interaction between hunting mortality and senescence using rich long-term data. Our results suggest that hunting activity is not a factor involved in the senescence pattern observed in snow geese, possibly due to compensatory mechanisms. We found no interaction between hunting mortality and age, but other intrinsic characteristics could well interplay with hunting at any given age. For example, body condition was previously shown to influence vulnerability to hunting (Fowler et al., 2020). Further investigation of this question on an evolutionary standpoint would require contrasting populations of the same species subjected to different hunting pressures across the entire life of the individuals.

1.8 Acknowledgements and data availability

We are grateful to Gérald Picard, Marie-Christine Cadieux and Christian Marcotte for assistance in the field and Marie-Christine Cadieux for managing the database and the Bylot Island field research station. We thank Parks Canada for the access to Sirmilik National Park and support in the field. We are grateful to the Mittimatalik community for their welcome. We further thank all the Inuit, students and field assistants who contributed to goose banding and colony monitoring. We acknowledge funding from the Sentinelle North Research Chair from the Canada First Research Excellence Fund; the Fonds de Recherche du Québec—Nature et Technologies (FRQNT) [2020-NC- 271544]; the Natural Sciences and Engineering Research Council of Canada (NSERC); The Arctic Goose Joint Venture (Environment Canada); ArcticNet from the Networks of Centres of Excellence of Canada; Polar Continental Shelf Program. This research was conducted according to the relevant national and institutional regulations on animal welfare and was approved by the Comité de Protection des Animaux de l'Université Laval (CPAUL), the Canadian Bird Banding Office, the Mittimatalik Hunter and Trapper Organization and Parks Canada.

Data Availability Statement

All data and codes will be available upon publication.

1.9 Supplementary Materials

- Annexe S1.1: Harvest rate data
- Annexe S1.2: Observation matrix
- Annexe S1.3: List of priors used in the Bayesian capture-mark-recapture model.
- Annexe S1.4: Complementary information on parameter estimation
- Annexe S1.5: Emigration and detection probabilities

Chapitre 2. Manipulating individual state during migration: carry-over effects of cumulative stress on survival

Île-aux-Oies, Mai 2021

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2.1 Résumé

La réponse au stress est un mécanisme servant à faire face à des évènements imprévisibles et à minimiser les menaces immédiates à la survie. Cependant, l'accumulation de stress due à des stresseurs multiples peut avoir des effets délétères sur le fitness à long terme, en altérant la reproduction et la survie. Cet aspect de la physiologie du stress et ses conséquences sur les traits démographiques ont fait l'objet de peu d'attention dans l'étude des populations sauvages, et ces études sont principalement observationnelles. Ici, nous étudions les conséquences démographiques de stresseurs multiples (privation de nourriture et captivité prolongée) imposés expérimentalement à des grandes oies des neiges (*Anser caerulescens atlanticus*) pendant la migration printanière. En 2009, des femelles oie des neiges ont été capturées sur leur site d'escale printanière, et gardées en captivité jusqu'à 4 jours avec ou sans nourriture. Des échantillons de sang ont été prélevés à la capture, au baguage et à la relâche pour mesurer les niveaux de corticostérone (CORT), une hormone de la réponse au stress. La CORT a atteint son maximum dans les premières heures après la capture, puis a diminué dans les jours suivant la capture. Nous avons observé que les niveaux de CORT à la relâche des individus captifs dépendait de leur condition corporelle pré-expérience, mais pas la réponse maximale de CORT. Nous n'avons pas montré de lien avec le succès reproducteur subséquent, mais nous avons détecté un effet reporté négatif de la privation de nourriture sur la survie l'année suivante. La condition corporelle pré-expérience et la CORT induite par le stress avaient des effets marginaux sur la survie. Nous avons ainsi montré que l'accumulation de stresseurs peut avoir des effets reportés sur la survie, et que l'intensité de la réponse hormonale peut également affecter la survie.

2.2 Abstract

The stress response is a mechanism to cope with unpredictable events and minimize immediate threats to survival. However, cumulated stress due to multiple stressors can have long-term deleterious effects on fitness by impairing reproduction and survival. This aspect of stress physiology and its consequences on demographic traits have received little attention in wild populations, and such studies are mostly observational. Here, we investigate the demographic consequences of multiple stressors (fasting and prolonged captivity) experimentally imposed during spring migration on greater snow geese (*Anser caerulescens atlanticus*). In 2009, female snow geese were captured at a spring staging site and kept in captivity for up to 4 days with or without access to food. Blood samples were taken at capture, banding, and release to measure corticosterone (CORT) levels, a stress-response hormone, during the experiment. CORT response peaked within the first hours after capture and decreased during the following days in captivity. We observed that stress-induced CORT levels of captive individuals at release depended on their pre-experiment body condition, but not the stress-induced peak CORT response. We showed no link with subsequent reproductive success, but we detected a negative carry-over effect of food deprivation on survival in the following year. Pre-treatment spring body condition and stress-induced CORT levels had marginal effects on survival. We showed that cumulated stressors could have carry-over effects on survival and that the intensity of the hormonal response can ultimately affect survival.

2.3 Introduction

In preparation for energy-demanding periods of the life cycle, like winter, migration or reproduction, individual behaviour and physiology are adjusted accordingly (Jacobs & Wingfield, 2000; Wingfield, 2008). Individuals capable of optimally managing energy allocation prior to reproduction are predicted to maximize their fitness (Rowe et al., 1994; Bêty et al., 2003; Hennin et al., 2016). Effective resource allocation involves a dynamic balance between factors like initial body condition or individual quality (Galgani & Ravussin, 2008), resource availability and acquisition, fattening rate, and costs such as competition for resources and predation risk (Wingfield & Kitaysky, 2002).

The total energy requirements during predictable daily or seasonal activities is called the allostatic load (McEwen & Wingfield, 2003; Romero et al., 2009). The normal variations within the allostatic load can encompass temporary increases in energy demand in response to an unpredictable event that quickly return to homeostasis (Romero et al., 2009). However, the organism can experience an allostatic overload when the demand in energy exceeds the available resources or when the increased demand is prolonged in time (McEwen & Wingfield, 2003). For example, an accumulation of stressors can considerably increase the overall energy demand and extend the effects of stress over time, pushing individuals into a state of allostatic overload (McEwen & Wingfield, 2003; Landys et al., 2006). In long-lived species, a typical mechanism to cope with allostatic overload during the reproductive season is to reduce reproductive effort or even skip or abandon reproduction to save energy and allocate it to self-maintenance and subsequent reproductive events ('the prudent parent hypothesis', Stearns, 1992). Variation in breeding effort or breeding propensity is most pronounced when environmental conditions are unfavourable and limit resources required for breeding (Legagneux et al., 2012; Warren et al., 2014; Grandmont et al., 2023). However, a reduced breeding effort may not be sufficient to maintain survival when stressors are too intense or numerous (Romero & Wikelski, 2001). Under such environmental stress, individuals may remain under a state of allostatic overload, which is associated with detrimental changes in behaviour, body condition or immunity, and consequently, with an increased vulnerability to predation or disease (Harrison et al., 2011; O'Connor et al., 2014).

Among the physiological drivers of behavioural and physiological adjustments to the environment, variations in glucocorticoids (GCs) are key for the maintenance of metabolism and energy storage (Romero et al., 2009). GCs are highly conserved hormones across vertebrates, secreted by the adrenal glands through the activation of the hypothalamic–pituitary–adrenal axis (HPA axis). Their role depends on the amount secreted and the receptors activated (Romero, 2004; Sopinka et al., 2015). The baseline level of GCs mediates daily energetic balance, foraging behaviour, body mass and metabolism (Landys et al., 2006). However, when an unpredictable acute stress occurs, the organism enters an emergency state (Wingfield et al., 1998; Angelier & Wingfield, 2013) and secretes high amounts of GCs (Romero, 2004). This initiates strong behavioural and physiological changes (Wingfield & Kitaysky, 2002; Romero, 2004) that go beyond the predictable variations based on life history strategy and available energy (Schoenle et al., 2018). Eventually, the secretion of GCs is downregulated by negative feedback, which keeps GC levels from becoming toxic (Romero, 2004).

The role of GCs as a link between the state of the organism and its environment has been increasingly investigated in recent years, with several studies looking at the association between GC response to various stressors and fitness traits (Breuner et al., 2008; Schoenle et al., 2018, 2021). In addition, the role of GCs in mediating transitions between life-history stages has also received a growing interest (Wingfield, 2008). Recent meta-analyses have linked both baseline and stress-induced GC levels to variations in survival and reproductive success, but these relationships depend on the physiological and environmental context, the trait studied and the life history of the species (Bókony et al., 2009; Hau et al., 2010; Sorenson et al., 2017; Schoenle et al., 2018, 2021). Furthermore, sustained elevated GC concentrations can result in cumulative damage over time (McEwen & Seeman, 1999), causing long-lived animals to suffer greater costs of high GCs than short-lived ones (Schoenle et al., 2018). However, the effect of increased GCs in response to multiple stressors at the intra-specific level is less clear and varies with the energy-demanding period considered (Cornelius et al., 2011). In birds, the main GC is corticosterone (CORT), and variations in its baseline level have been shown to prepare individuals for demanding periods in their life cycles (Romero, 2002, 2004). For example, in migratory passerines, the increased energy period before migration (*Zugunruhe*) is linked with increased levels of CORT (Cornelius et al., 2013).

Body condition is also a flexible component of the phenotype that can act as a buffer for energetically demanding life history stages and can be modified through CORT-induced modifications in behaviour and diet (Angelier et al., 2007; Hoarau et al., 2022).

Allostatic overload caused by cumulative or prolonged stressors can have multiple effects, including an extended disruption of the GC response and feedback mechanisms (Romero & Wikelski, 2001; Romero, 2004) that can affect survival or reproduction at subsequent stages of the annual cycle (Harrison et al., 2011). Such carry-over effects have been neglected in the literature but are increasingly studied (Harrison et al., 2011; Grandmont et al., 2023). Energy has been hypothesized to be the major link between life-history stages affected by carry-over effects (Harrison et al., 2011; Legagneux et al., 2012). Because of the functional link between energy regulation and GCs, GCs also have the potential to be key mediators of carry-over effects (Legagneux et al., 2012; Schultner et al., 2014). Although carry-over effects have been reported in many taxa (O'Connor et al., 2014), migrating birds are highly relevant models to study them because migration acts as a clear separation between lifecycle stages (Harrison et al., 2011). Like classic stress responses, stress-induced carry-over effects are life-history dependent and their links to fitness traits are increasingly studied (Schoenle et al., 2018, 2021). For example, several studies have reported a reduction of reproductive output several months after exposure to stressors in wild vertebrates (Harrison et al., 2011; Grandmont et al., 2023). However, the consequences of prolonged or accumulated stressors on survival remain poorly known and difficult to study, especially in long-lived birds, because it requires following individuals over multiple years.

Field experiments are especially suitable to understand carry-over effects resulting from cumulative stressors and the associated physiological mechanisms (Harrison et al., 2011). We carried out such an experiment on the greater snow goose (*Anser caerulescens atlanticus*), a long-lived migratory bird for which we have extensive long-term monitoring data. We applied multiple stressors during spring staging by maintaining birds in captivity with or without access to food, and we previously reported their impacts on subsequent reproduction (Legagneux et al., 2012; Grandmont et al., 2023). In this paper, we use additional physiological and demographic data from the same experiment, which was not

included in prior analyses, to further investigate the impact of body condition and CORT stress-response to cumulative stressors on survival and reproduction.

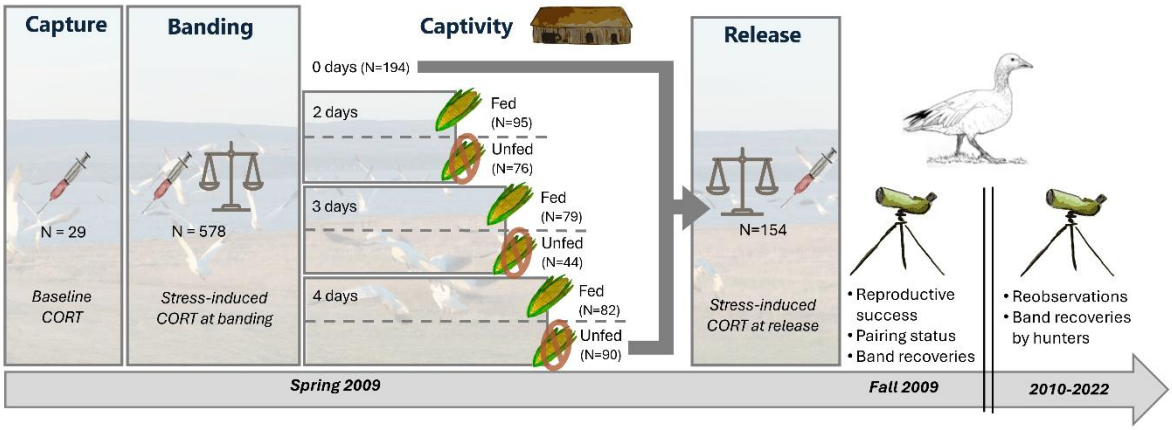

Figure 2.1 : Graphical representation of the experimental design with the sequence of operations on greater snow geese. A maximum of three blood samples were taken for a given individual: within 3 min after capture to measure basal CORT level, at banding to measure stress-response CORT and at release to measure prolonged stress response for experimental individuals. Control birds were released just after banding. Marked females were resighted in the field during the following autumn and the presence of young and mates were noted. Spring reobservations and recoveries of dead birds by hunters in subsequent years were used to model survival rates.

Body condition and stress level of staging geese were manipulated by capturing wild birds and maintaining individuals in captivity for 2–4 days, with or without access to food, before releasing them (Figure 2.1). Control individuals were released immediately after banding. Using physiological data (CORT levels and body condition), and reobservations and recoveries of marked individuals, we investigated potential effects of the experimental treatments and stress-response on reproduction and survival in the following year. Specifically, we (i) quantified the relationship between experimental treatments and CORT levels; (ii) tested whether individual variations in CORT stress response could explain individual variations in demographic parameters in the year following the experiment; (iii) tested whether the accumulation of stressors from our experimental treatment groups affected survival the year following the experiment. We hypothesized that higher CORT levels would mediate the previously observed reduction in reproductive success of manipulated individuals (Legagneux et al., 2012; Grandmont et al., 2023). Considering that greater snow geese are long-lived, we could expect major effects on reproduction but a negligible effect on survival, as the stressors were applied over a relatively short period, allowing for recovery

to a sustainable allostatic load. Alternatively, we could also anticipate some reduction in survival due to the experimentally induced cumulative stress (LeTourneux et al., 2022).

2.4 Materials and methods

2.4.1 Study Species and Study Site

The greater snow goose is a migratory species wintering along the Atlantic coast of the United States and breeding in the Canadian Arctic in Nunavut. This experiment took place in 2009 at Île-aux-Oies (Québec; 47°00′ N 70°33′ W) in the St. Lawrence estuary, the major staging site used by geese from late March to late May to accumulate reserves for migration and reproduction (Gauthier et al., 1992). They also come back to this region during the autumn migration from the breeding grounds 3000 km further north. Geese have permanent pair bonds and stay in family units (i.e., young with their parents) for up to a year (Prevett & MacInnes, 1980), so it is possible to quantify their reproductive success by determining the presence or absence of young with their parents from field observations during autumn staging in Québec. The proportion of young observed in the autumn flocks is a good proxy of reproductive success in this species and is highly variable from year to year, largely due to abiotic and biotic conditions encountered at the breeding site (Morrissette et al., 2010). The greater snow goose is a hunted species in autumn and spring in Quebec and in winter in the United States (LeTourneux et al., 2022). Adults can live on average for 10 years and up to 24 years (Chapter 3). Their life history strategy is on the slow side of the slow-fast continuum, meaning they tend to skip reproduction to optimize survival if their physiological condition and/or the environmental conditions are not favourable (Reed, 2004).

2.4.2 Capture, Measurements and Experimental Set-Up

Geese were captured with cannon-nets from late April to mid-May 2009, mostly near the end of spring staging (Legagneux et al., 2012). As spring is the period of family break-up (Gauthier & Tardif, 1991), juveniles were released immediately after capture. The number of adults caught in each capture event ranged from 40 to 105. Adults were retrieved from under the nets and transported to a barn for banding and experimental manipulations (graphical representation in Figure 2.1). When groups were captured late in the day, they were banded the following morning, which was the case for 12 out of 22 capture groups. All adults were banded, sexed by cloacal examination, and females were measured (tarsus,

culmen and head lengths to the nearest 0.1 mm), weighed (to the nearest gram) and blood samples were collected (see details below). Females were marked with yellow plastic neck collars to facilitate re-observation at a distance after release.

Each capture group was assigned to a different treatment in a randomly predefined order. The control group was composed of individuals released immediately after banding, and treatment groups were composed of individuals subject to two different manipulations (see sample sizes in Figure 2.1): kept in captivity for 2, 3 or 4 days (captivity duration treatment) and either provided with food (crushed corn) or not (food treatment). Treatment groups were large and randomly selected, so any biases should be equally represented in all of them. Only females were involved in the experimental treatments because reproductive success is primarily related to female body condition (Bêty et al., 2003). Males were kept in captivity for the same amount of time as females, in separate indoor enclosures, with possible visual and vocal contact and with food. At release, females were weighed again, and blood samples were taken (see below). All females from the same capture group were assigned to a single treatment, and both males and females were released at once to prevent pair bond disruption (details in Legagneux et al. 2012). The experiment was shown to decrease body condition by 8% and 1.2% for unfed and fed groups, respectively (Legagneux et al., 2012). Also, time spent in captivity negatively affected reproductive success (Legagneux et al., 2012) through a reduction in breeding propensity (Grandmont et al., 2023).

Marked individuals were reobserved by our team and bird watchers throughout the area used by staging geese along the St. Lawrence River every spring from 2009 to 2022. Individuals harvested by hunters in autumn, winter and spring in both Canada and the US were also reported to us. This information enabled the estimation of survival with capture–mark–recapture models (LeTourneux et al., 2024). Collared females were also reobserved in autumn 2009 to record the presence of young and a partner with them, which we used as a measure of reproductive success and pairing status, respectively.

2.4.3 Blood Sample Collection and Analysis

Up to three different blood samples were taken from females at three different times: upon capture, during banding, and just before release. The first sample came from randomly

chosen females sampled within 3 min at each capture, which reflects basal hormonal levels (Romero & Reed, 2005; Legagneux et al., 2011). Only a small number of females could be sampled at each capture due to the 3-minconstraint. The second sampling was taken from all females (control and treatment groups) during banding (on average 11.0 ± [SD] 4.9 h after capture). Finally, the last blood sample was collected just before release from a random sample of females kept in captivity for 2, 3 or 4 days depending on the treatment group (Figure 2.1). Complementary information on sample sizes for each group is detailed in Table S2.1 (Appendix S2.1). We used 23G needles and 1 mL syringes to collect 100 µL to 1 mL of blood from the tarsal vein. Blood samples were kept on ice for up to 4 h, then centrifuged at 8000 g for 8 minutes to separate the plasma from the red blood cells. These were then frozen separately at −20°C initially (max. 20 days) and at −80°C after field work. We dosed plasma samples (29 at capture, 578 at banding, 154 at release) for corticosterone concentration by radio-immuno-assays as described in Lormée et al., (2003) at the Centre d'Études Biologiques de Chizé (France). Two assays were performed for each sample, and the intra-assay coefficient of variation was 2.17% (N = 104).

2.4.4 Statistical Analyses

To obtain an index of endogenous reserves independent of individual size, we corrected the body mass of females by skeletal measurements. We ran a principal component analysis (PCA) on tarsus and culmen lengths, using individuals captured and measured at the same site from 2006 to 2009. We used the first component (PC1), explaining 71% of the variance, in a linear regression with body mass as the dependent variable and added the residuals of this model to the mean mass. We then added the residuals of this linear model to the mean mass corrected by size to correct for the date of capture (see LeTourneux et al., 2021 for details). We thus obtained an index of fat reserves independent of skeletal size and capture date, which we will henceforth refer to as ‘body condition’.

Because of the high variability in time elapsed between capture and blood sampling at banding, we tested for an effect of this variable on CORT levels. We used a linear model as the quadratic model was not preferred and less parsimonious (ΔAIC = 1.33 in favour of the linear regression). We found a significant negative relationship between CORT levels at banding and time between capture and sampling (β = −0.01,95% CI = [−0.03, −2.41e-03];

N = 578; Figure S2.1 in Appendix S2.2). We thus used the residuals of the previous model as the CORT levels at banding corrected for the influence of time elapsed since capture in subsequent analyses, similarly to our approach for body condition. No such relationship was found in individuals measured at release (β = −4.98e-0.4 [−0.01, 4.92e-03]; N = 154), possibly due to a smaller number of individuals processed. Nonetheless, we applied the same correction for samples collected at release as for those collected at banding, for consistency. However, this correction did not apply to samples at capture because they were all taken in less than 3 min and the exact time needed to take the sample was not recorded.

Because geese are fattening during spring staging and thus their physiological state may change over time, we also tested for an influence of capture date on all three types of blood samples. We found a significant relationship between CORT and capture date in samples at capture and at banding ($\beta_{capture}$ = −1.48[−2.84, −0.11], N = 29; $\beta_{banding}$ = −1.42 [−2.00, −0.84], N = 560; $\beta_{release}$ = −0.69 [−2.03, 0.64], N = 154; Figure S2.2 in Appendix S2.2). We used the residuals of these models to correct for capture date in all sample types, again for consistency. Corrected CORT values were the residuals of the regression added to the mean CORT value foreach sample type and these values were used for all following analyses.

We tested for a difference in CORT concentration between the three sample types (capture, banding and release) using a two-way mixed-effect ANOVA with individual as a random effect to account for measures on the same individuals (*lmer* function, *lme4* package, Bates et al., 2024). A significant difference was found (see results), and thus we used CORT concentrations during banding corrected by time since capture and date of capture (referred to as stress-induced CORT at banding) as an indicator of the intensity of the stress response in subsequent analyses.

We tested the repeatability of the stress-induced CORT concentration at banding on nine individuals caught and sampled at two distinct capture events during the staging period. Repeatability analyses were conducted using the intraclass correlation coefficient (ICC), which uses a one-way ANOVA to estimate intra-and inter-group variance (Wolak et al., 2012) (*single_fixed_raters* index in *ICC* function from *psych* package in R, Revelle, 2024)

and a Spearman correlation. Those nine recaptured individuals were removed from all subsequent analyses (N = 560 hereafter).

We tested for differences in stress-induced CORT levels at banding (i.e., before the start of the experiments) between treatment groups using an ANOVA and a Tukey post hoc test. Relationships between stress-induced CORT levels at banding or at release and food treatment, number of days spent in captivity, and body condition were investigated using linear models. Normality and homoscedasticity of the residuals were graphically confirmed for these linear models. We also examined the influence of stress-induced CORT at banding on reproductive success (presence vs. absence of young in autumn) using generalised linear models of the quasibinomial family. We included an interaction with time spent in captivity (random effects impossible with quasibinomial family) to control for the known effect of captivity on reproductive success (Grandmont et al., 2023). In all statistical analyses, we selected the best model explaining variations in our response variable using the corrected Akaike information criterion (AICc; Burnham & Anderson, 2004), and we used 95% confidence intervals (reported throughout) around the beta estimates to evaluate significance.

We used a multi-event, capture-mark-recapture model (Pradel, 2005) combining live reobservations with dead recoveries (bands reported by hunters) to analyse survival of collared females, while controlling for probability of encounter. Marking occurred in a single year (spring 2009), but reencounters extended over a 13-year period. Extending the period to collect reencounters increased the precision of the estimation of survival in the first year following the experiment, which was of primary interest. On each occasion, an individual could be observed alive (coded 1), recovered (2) or not encountered (0). Individuals recovered dead between spring t and $t+1$ were coded dead at spring $t+1$ (Gauthier & Lebreton, 2008). The model is a simplification of LeTourneux et al. (2022) and could estimate survival (S), live-encounter (p) and recovery (Seber's r) probabilities. We examined the influence of stress-induced CORT level at banding, pre-and post-treatment body condition, food treatment (fed vs. unfed during captivity) and number of days spent in captivity (0, 2, 3 or 4 days) on survival in the first year after release. Continuous variables like CORT levels and body condition were standardized. A maximum of two covariates were included in each model because of restrictions due to sample size. We did not include pre-

and post-treatment body condition, or food treatment and post-treatment body condition in the same models because these variables were highly correlated. We conducted model selection sequentially, first on recovery, then on reobservation and finally on survival probabilities using Akaike's Information Criterion corrected for overdispersion (QAICc; Lebreton et al., 1992; Burnham & Anderson, 2004). After a first selection of the best survival models, we tested again for more complex models on reobservation and recovery parameters. Models with a difference in QAICc of 2 or less from the best model were considered competitive. The capture-recapture analysis was conducted with program E-SURGE V.2.2.3 (Choquet, Rouan, et al., 2009). Prior to conducting the survival analysis, we tested the goodness-of-fit of the general model with the program U-CAREV2.3.5 (Choquet, Lebreton, et al., 2009). We found a χ^2 value of 71.4 for 63 degrees of freedom, which yielded an overdispersion coefficient $\hat{c}$ of 1.134. We used this value in the capture–recapture analysis, which explains our use of QAICc rather than AICc. Because we found that the food treatment affected survival (see results), we compared *a posteriori* the raw hunter recovery probability of fed, unfed and control in the first year after the experiment using a binomial linear model with a logit link.

2.5 Results

2.5.1 Stress Response and Factors Affecting CORT Levels

A total of 560 adult females, excluding females sampled twice, were captured, banded, and released in this study. Among them, 184 were control birds released immediately after banding, and 377 birds were kept in captivity for 2–4 days with or without access to food (203 and 174 individuals respectively).

CORT concentrations differed depending on when blood sampling occurred (Figure 2.2). Baseline level taken within 3 min of capture (31.88 ± [SD] 20.57 ng/µL; N = 29) was much lower than stress-induced level at banding measured 11 h after capture on average (152.56 ± 44.43 ng/µL; N = 560; $\beta_{Banding/Baseline}$ = 117.23 [101.75, 132.76] 95%CI). The stress-induced CORT level decreased after 2–4 days spent in captivity (104.85 ± 38.28 ng/µL; N = 154; $\beta_{Release/Banding}$ = −50.72 [−58.06, −43.28]) but remained higher than baseline level ($\beta_{Release/Baseline}$ = 66.50 [49.80, 83.34]) and did not vary between 2 and 4 days of captivity ($\beta_{Days\ in\ captivity}$ = −2.15 [−9.51, 5.22], Figure 2.2). Stress-induced CORT levels at banding

(before the experiment) were similar between individuals selected for different food and capture duration treatments (see Table S2.2 in Appendix S2.3). Intra-individual baseline and stress-induced CORT levels at banding were unrelated (β = 0.44 [−6.58, 7.45]; N = 29).

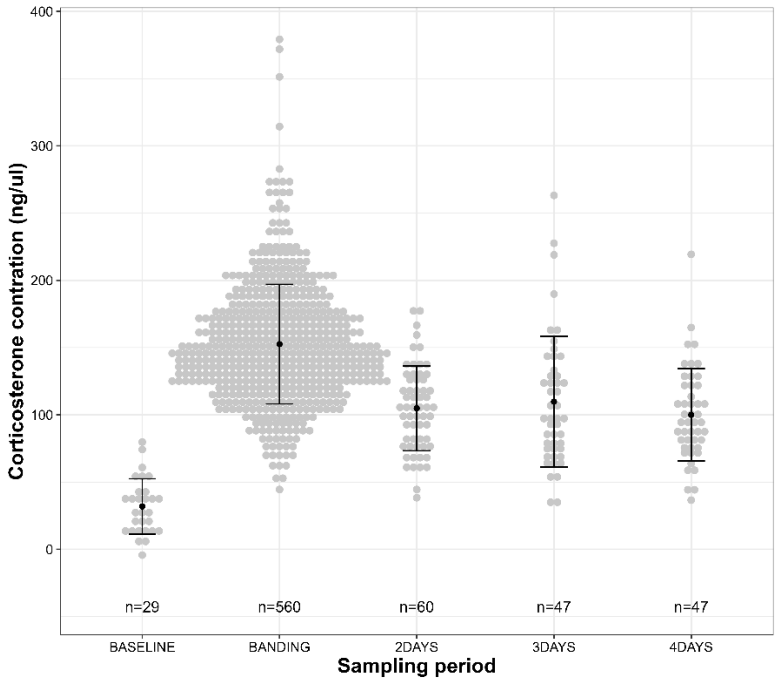

Figure 2.2 : Corticosterone level of greater snow geese measured within 3 min of capture (baseline level), at banding (11.0 ± [SD] 4.9 h after capture) and at release (2, 3 or 4 days after capture). Individual data points are represented in grey and mean and standard deviation in black. The CORT values are corrected for time before blood sampling and capture date. Individuals sampled twice were removed.

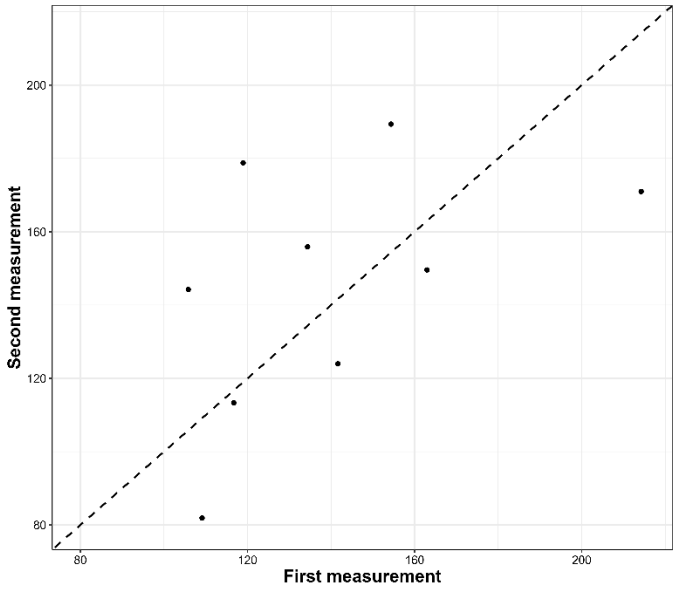

Figure 2.3 : Stress-induced corticosterone levels at banding (corrected by the time before sampling and capture date), for individuals with repeated measures. Dashed line represents 1:1 ratio for reference.

Repeatability of stress-induced CORT levels at banding for the nine individuals captured twice was relatively high but non-significant (ICC = 0.52 [−0.1, 0.9]; ρ = 0.55; p = 0.13; Figure 2.3).

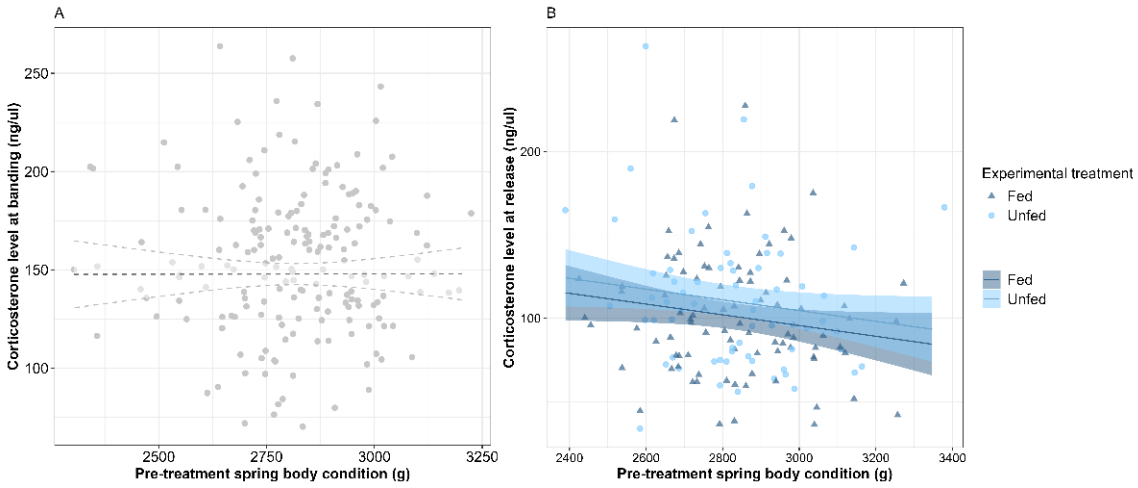

Figure 2.4 : Relationship between corticosterone level (corrected by the time before sampling and capture date) of greater snow geese at banding (A) or at release (B) and pre-treatment spring body condition (mass at banding corrected by size and date of capture). Mean predictions and 95% confidence intervals are presented [from model MC1, refer Table 1.1, for (B)].

Table 2.1 : Selection of linear models examining the influence of food treatment (fed or unfed), pre-treatment spring body condition (mass at banding corrected for size) and days spent in captivity (2, 3 or 4 days) on CORT level of greater snow geese at release. Only models with a AICc lower than the null model are reported here.

Model names	Factor included	K	Log Likelihood	Delta AICc
MC1	Condition + Food	4	-776.38	0.00
MC2	Condition	3	-777.44	0.02
MC3	Food	3	-778.24	1.62
MC.NULL	Intercept	2	-779.33	1.72
MC4	Food * Condition	5	-776.30	1.98

We found no significant relationship between stress-induced CORT level at banding and pre-treatment spring body condition ($\beta = -0.04$ [−5.32, 5.40]; Figure 2.4A) or post-treatment body condition ($\beta = -0.02$ [−0.04, 7.42e-03]). The stress-induced CORT level at release was marginally inversely related to pre-treatment spring body condition ($\beta = -5.96$ [−12.02, 0.10]; Figure 2.4B, MC2 in Table 2.1), and tended to be higher in the unfed than the fed group ($\beta_{fed/unfed} = 9.19$ [−3.13, 21.51]; Figure 2.4B, estimates from model MC3 in Table 2.1) but was not related to the number of days spent in captivity ($\beta_{Day3/Day2} = 4.97$ [−9.79, 19.72]; $\beta_{Day4/Day2} = -4.85$ [−19.60, 9.91]).

2.5.2 Influence of Stress Response on Fitness Parameters

Our proxy for reproductive success (presence of young with females in autumn) was not related to stress-induced CORT at banding or at release ($\beta_{CORTbanding} = -7.72e\text{-}03$ [−0.02, 7.50e-03] and $\beta_{CORTrelease} = -0.03$ [−0.11, 0.04]).

Table 2.2 : Results of model selection analysing survival of greater snow geese marked in spring 2009 in relation to several covariates. Covariates apply only to survival in the first year after release and include stress-induced corticosterone level at banding (CORT), spring body condition (COND1: condition at banding; COND2: condition at release), food treatment (FOOD: treatment fed/unfed/control), and number of days spent in captivity (DAYS: 0, 2, 3 and 4 days). Time (t, in years) was tested only on recovery and recapture probabilities. Survival was constant after the first year in all models. K: number of parameters, ΔQAICc: difference in QAICc between the current and the top-ranked model. See Table S2.3 in Appendix S2.4 for full model selection.

Model Name	1st year survival	Recapture	Recoveries	K	Deviance	ΔQAICc
MS24	FOOD	t	Constant	17	2845.63	0
MS27	FOOD + COND1	t	Constant	18	2844.50	1.08
MS5	FOOD	Constant	Constant	6	2872.35	1.10
MS30	CORT + FOOD	t	Constant	18	2845.18	1.67
MS6	FOOD + COND1	Constant	Constant	7	2871.17	2.09
MS7	CORT + FOOD	Constant	Constant	7	2871.85	2.69
MS35	COND2	t	Constant	14	2850.98	2.77
MS36	null	t	Constant	14	2855.77	2.77
MS8	COND2	Constant	Constant	5	2877.85	3.9

In the survival analysis, preferred models retained a yearly variation in recapture but constant recovery rates (see Table S2.3 in Appendix S2.4 for full details). Our top four models were all within two points of QAICc, suggesting a strong influence of the food treatment and a weaker influence of pre-treatment spring body condition and stress-induced CORT level at banding on survival during the first year after release (Table 2.2). However, there was no evidence for an influence of the number of days in captivity on survival (Table S2.3 in Appendix S2.4) and the survival rate was constant over time after the first year.

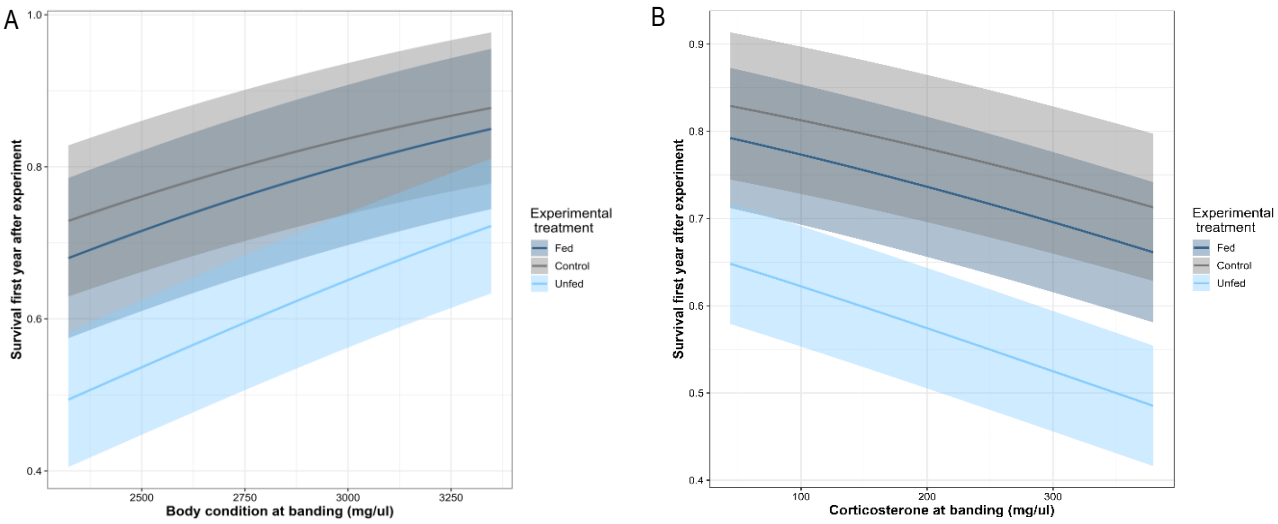

Figure 2.5 : Relationship between survival of greater snow geese in the first year after the experiment and pre-treatment spring body condition (A) or stress-induced corticosterone level (B) for birds kept in captivity with or without food for up to 4 days or released immediately after banding (control). Mean survival predictions with 95% confidence intervals are from parameters of models MS31 (A) and MS34 (B) in Table 2.2.

Survival in the first year after release was similar in control and fed birds but lower in unfed birds (effect of food treatments compared to the control group: βfed = −0.24 [−1.06, 0.57]; βunfed = −0.98 [−1.75, −0.21]; Model MS24 in Table 2.2; Figure 2.5A).Survival tended to be positively related to pre-treatment spring body condition (βcond = 0.16 [−0.16, 0.49]; Model MS27 in Table 2.2; Figure 2.5A), and negatively related to stress-induced CORT level at banding (βCORT = −0.09 [−0.36, 0.19]; Model MS30 in Table 2.2; Figure 2.5B). Although the 95% confidence intervals included zero, models with pre-treatment spring body condition and stress-induced CORT level at banding were retained as competitive models (Table 2.2).

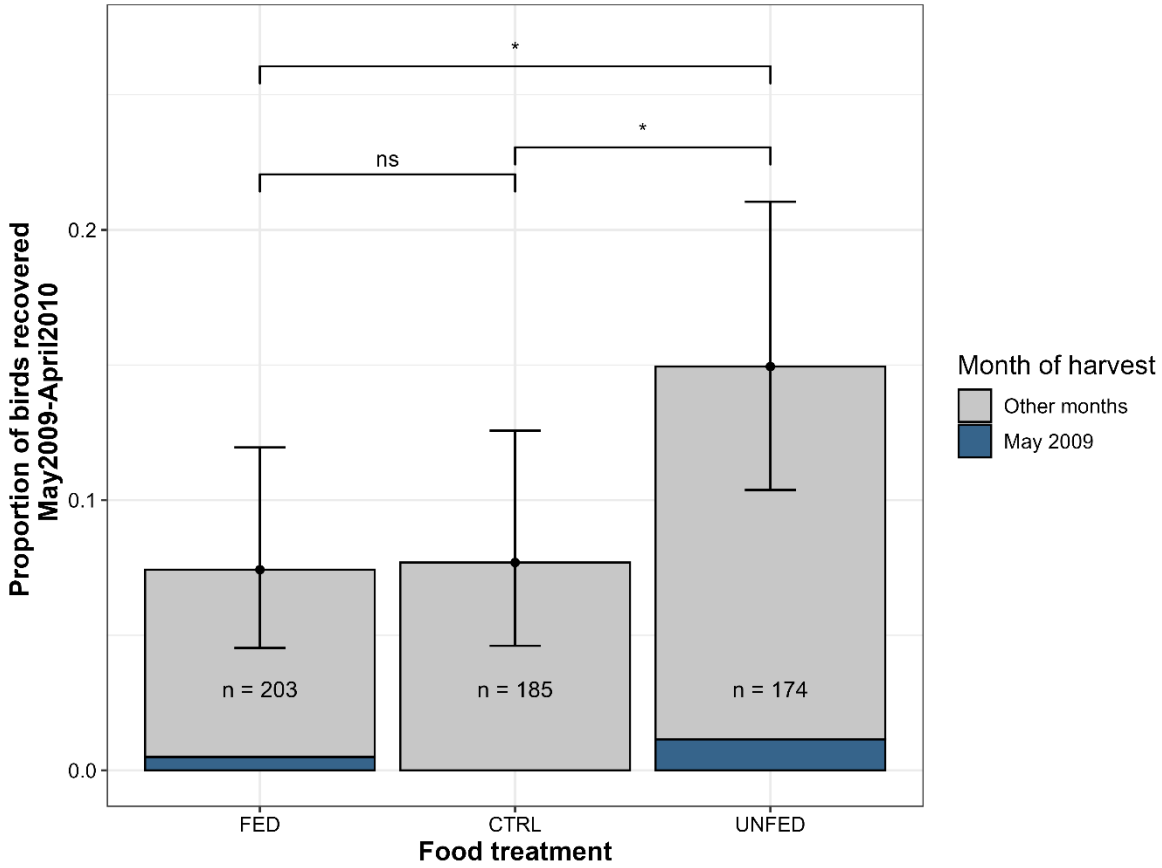

*Figure 2.6 : Recoveries of banded greater snow geese in the first year post-release as a function of the food treatment groups. Grey bars are the proportions from the raw data; the black points and error bars are the mean predictions and 95% confidence intervals from a binomial generalised linear model. * means the difference is significant. ns means the difference is non significant.*

When examining band recoveries between the food treatment groups, we found that a higher proportion of birds from the unfed treatment were recovered by hunters in the first year after release compared to birds in the control group ($\beta_{unfed/control}$ = 0.75 [0.07, 1.46]) or compared to the fed group ($\beta_{unfed/fed}$ = 0.78 [0.12, 1.48]; Figure 2.6). On the contrary, fed individuals were harvested at a similar rate ($\beta_{fed/control}$ = −0.04 [−0.80, 0.73]; Figure 2.6).

2.6 Discussion

Our study experimentally simulated an unpredictable stressful event during the spring staging of a long-distance migratory bird species to examine the consequences of the physiological response to multiple stressors and of a body condition reduction on reproductive success and survival. Our large sample size allowed us to describe the stress response of cumulative stressors and its consequences on survival despite the confounding environmental factors associated with any field experiment. The combination of physiological parameters and capture-recapture data enabled us to partition the effects of individual variation in body condition and intensity of stress response. Contrary to our first hypothesis, we found that the hormonal stress response did not explain individual variation in reproductive success. However, all geese exhibited a prolonged stress response during the 2–4 days spent in captivity, which likely explains the effect of captivity on breeding suppression found in a previous study (Grandmont et al., 2023). We found evidence for carry-over effects of the experiment on survival in the following year as it was reduced for unfed individuals, which cumulated the most stressors, thus supporting our alternative hypothesis regarding survival.

2.6.1 Proxy of Stress Response and Individual Variation

We measured three distinct CORT levels throughout our experiment: at capture (baseline), at banding (on average 11 h after capture, stress-induced) and at release from captivity (2–4 days later, also stress-induced). The maximal stress response was observed at banding, and this response appeared repeatable in individuals recaptured later in the same season. Though our repeatability analysis was non-significant, our effect size tended towards the same result as published data (0.52 vs. 0.38 according to a meta-analysis by Taff et al., 2018). Considering our very small sample size, the non-significance could be caused by a lack of statistical power, not by an absence of repeatability. Individuals may respond differently to the same situation, maintaining a diversity of coping styles within a given population (Cockrem, 2013; Taff et al., 2018), which is associated with distinct energy allocation strategies (Angelier et al., 2018). This heterogeneity in CORT response has been proposed as a mechanism to help explain individual heterogeneity in performance (Wada et al., 2008; Cockrem, 2013; Angelier et al., 2018). Interestingly, stress-induced CORT levels

were found to be more repeatable than baseline CORT levels in the meta-analysis of Taff et al. (2018). This may suggest that individual variation in CORT response reflects a personality-like trait, potentially indicating differing sensitivities to multiple environmental stressors. This result supports our exploration of the role of stress-induced CORT levels at banding in modulating reproduction and survival.

Most studies with comparable experimental designs have been conducted on other bird taxa. Nonetheless, experiments on wild passerines captured and kept in captivity have shown an immediate CORT increase in individuals deprived of food, but CORT levels of fed and unfed individuals were similar 1 day after the beginning of fasting (Lynn et al., 2003). In wild seabirds, naturally occurring food deprivation increased circulating CORT levels of individuals (Kitaysky et al., 2007; Angelier et al., 2015). After prolonged captivity, fasting and non-fasting individuals decreased their circulating CORT levels similarly (Angelier et al., 2015). Because our experimental design was built to minimally disturb the geese during captivity, we were not able to look for an initial increase of CORT immediately after the start of the food restriction for unfed individuals or to compare the pattern of CORT decrease between treatment groups. Stress-induced CORT levels at release tended to be higher for unfed than for fed individuals, which is coherent with the idea of an allostatic overload as the energy necessary to respond to the stress of captivity exceeded the energy available (McEwen & Wingfield, 2003). Moreover, the stress-induced CORT levels at release were higher than baseline values but lower than the peak response measured at banding, which is typical of the downregulation that occurs during chronic stress (Romero, 2004; Rich & Romero, 2005). Although the duration of our experiment is too short to fit the formal description of chronic stress (Romero, 2004), we believe that similar mechanisms occurred in our experiment. In fact, geese in our experiment were not habituated because they were not repeatedly handled during captivity, nor exhausted because they were able to fly away immediately after release, suggesting they downregulated their CORT response as described in Rich and Romero (2005). Under acute stress conditions, the stress response and its associated energy allocation pathways are saturated, making individuals unable to respond toa new stressor with the same intensity (Romero, 2004). This mechanism has also been observed in other bird taxa such as passerines (Cockrem, 2013; Angelier et al., 2015).

Interestingly, we had some evidence that stress-induced CORT levels at release could be modulated by pre-treatment spring body condition. This could indicate that individuals in better condition, with higher endogenous reserves, presented a lower stress response than those in lower condition, even under prolonged stressors. A similar relationship was found after reproduction in Adelie penguins (*Pygoscelis adeliae*; Cockrem et al., 2006). This suggests that variations in CORT levels after an acute or prolonged stressor can be modulated by the individual state and the amount of body reserves.

2.6.2 Carry-Over Effects of Multiple Stressors on Fitness Parameters

We investigated the role of stress-induced CORT level on fitness parameters, which has been less studied than the links with baseline CORT level (Breuner et al., 2008; Bonier et al., 2009; Sorenson et al., 2017). Results reported in the literature highly depend on the fitness trait being studied but seem to show a stronger association of baseline levels with reproductive success than with other demographic traits, especially in long-lived seabird species (Sorenson et al., 2017).

In our study, variations in stress-induced CORT levels, assumed to be an index of the intensity of the hormonal stress response, did not explain differences in reproductive success. On the other hand, Legagneux et al. (2012) and Grandmont et al. (2023) found that time spent in captivity was the main driver of a reduction in reproductive success in our experiment. The effect was stronger in 2009, when conditions were unfavourable for migration and breeding, offering fewer opportunities to recover from stress experienced during the spring (Legagneux et al., 2012; Grandmont et al., 2023). This was also the year we collected blood samples. We thus cannot exclude that covariations between CORT response and reproductive success might have occurred in years with better environmental conditions. However, a longer time spent in captivity will translate into more time spent in the state of hormonal saturation, as described above, and we suggest that it may explain the decrease in breeding propensity reported in Grandmont et al. (2023).

The effects of stress-induced or acute GC levels on survival are not fully understood, nor are the patterns clear (Breuner et al., 2008; Schoenle et al., 2018) as it is generally context-dependent (Cockrem et al., 2006; Bókony et al., 2009; Crespi et al., 2013). For

example, a positive relationship between survival and CORT levels was found in poor-quality habitats but not in high-quality habitats for a passerine species (Angelier et al., 2009) and an inverse tendency was shown in a group of long-lived species (Schoenle et al., 2021). In this context, our results provide evidence that stress-induced CORT alone does not explain variations in survival but may reduce survival in addition to food deprivation, which supports the results of LeTourneux et al. (2022) regarding the impact of cumulated stress on survival. Unfed individuals lost up to 8% of their endogenous reserves during captivity (Legagneux et al., 2012) and were less likely to survive the following year compared to controls and fed individuals. Reduced energy reserves and the accumulation of stressors during spring staging therefore appear to be potential predictors of subsequent survival. Considering that baseline and stress-induced CORT were not predictive of one another, we also suggest that they have complementary roles in the regulation of fitness traits, as previously suggested (Romero, 2004; Landys et al., 2006; Angelier & Wingfield, 2013).

It is important to note that all captured females (experimental and control groups) were equipped with plastic collars, which were shown to interact with hunting in reducing survival, likely by inducing a negative effect on body condition (LeTourneux et al., 2022). As stated earlier, 2009 had rather unfavourable environmental conditions. It is thus possible that under better conditions, the stress-induced effects that we observe here might have been mitigated (Grandmont et al., 2023). On the other hand, we can argue that these unfavourable environmental conditions were a supplementary stressor on top of those imposed by our experimental conditions. As a result, geese had to face the cumulated stress generated by capture, time spent in captivity, food restriction, wearing a neck collar, and unfavourable environmental conditions in 2009. This could heighten the impact on survival, as multiple stressors can have consequences more severe than the sum of each due to synergistic effects (LeTourneux et al., 2022). Nonetheless, the differences found between the food treatment groups can only be attributed to this treatment because all individuals were equipped with the same collars. Similarly, pre-experiment body condition, baseline, and stress-induced CORT levels cannot be affected by the collar for the same reason, so our interpretations of the links between these physiological parameters and survival remain valid.

Hunting is a major source of mortality in our population (LeTourneux et al., 2022), potentially affecting individuals differently depending on their individual allostatic state (Dehorter & Tamisier, 1998). We consequently looked at recovery rates in the year following the experiment, which can be an index of hunting mortality. This could provide insights into the possible role of this factor in the differences in survival found between our experimental groups. Recovery rates by hunter were higher for unfed individuals than for control and fed individuals, and most mortalities occurred in subsequent autumn and winter, not immediately after release. This suggests a carry-over effect of the food treatment on survival, at least partly through hunting mortality. Captivity but not food treatment was shown to decrease reproductive success (Legagneux et al., 2012; Grandmont et al., 2023), so fed and unfed individuals had comparably low reproductive success. Therefore, it is unlikely that differences in hunting vulnerability related to reproductive success (e.g., due the presence of naive juveniles accompanying the parents, Calvert and Gauthier, 2005) can explain the high hunting mortality of the unfed group. We offer three mutually non-exclusive hypotheses to explain our result.

1. *The body condition hypothesis*: Geese exposed to prolonged stressors may decrease their foraging behaviour for an extended period of time after the event. The reduction in body condition incurred might thus still be present in autumn, forcing individuals to forage in higher quality but riskier habitats, namely crop lands (LeTourneux et al., 2021), where most of the hunting activity takes place. A prolonged reduction in body condition also underlies the other hypotheses through different mechanisms.
2. *The moult hypothesis*: A reduced body condition could hinder moulting, producing lower quality feathers. Since geese moult their flight feathers only once a year during the summer (Marmillot et al., 2016), low quality flight feathers can have long-lasting carry-over effects by increasing allostatic load, thereby impacting body condition and survival (Johns et al., 2018). This could translate into reduced abilities to escape from hunters or increased energy costs for flight, generating more risk-taking behaviours to forage (see Hypothesis 1), thus increasing vulnerability to hunting.
3. *The pair-bond disruption hypothesis*: Chronic stress imposed by marking with radio-transmitters was shown to increase pair-bond disruption by 25% (Demers et al., 2003). Although males and females were released at the same time to avoid pair break

up, a change in behaviour or reduced body condition in unfed females after release could have increased the pair-bond disruption rate in this group (Choudhury, 1995), which could be supported by the decrease in breeding propensity found in Grandmont et al. (2023). Unpaired geese have a subordinate status in flocks (Stahl et al., 2001), leading to lower body condition, increased vulnerability to hunters (see Hypothesis 1) and reduced survival (Leach et al., 2020). We were able to indirectly test this hypothesis using resightings of birds with or without a mate during autumn 2009 in Québec (Legagneux et al., 2012). In a posteriori analysis (Figure S2.3 in Appendix S2.5), we found evidence for a negative effect of days spent in captivity on the pairing status in autumn (Figure S2.3A in Appendix S2.5) but not of the food treatment (Figure S2.3B in Appendix S2.5). It was not possible to directly test for the effect of pairing status on survival because of low sample size, but by using the probability of band recovery, we found that unpaired birds, especially those in low post-treatment body condition, had a higher recovery rate than paired ones (Figure S2.3C,D in Appendix S2.5). Although these analyses offer ambiguous support for this hypothesis, investigation of the overall effect of pair-bond disruption on vital rates appears promising for future work.

Even if we cannot conclude with certainty which mechanisms could be the main drivers of the observed carry-over effect on survival, a sustained lower body condition, possibly mediated by reduced-quality feathers and pair-bond disruption, could explain the prolonged effects of cumulative stressors lasting up to 10 months after the experiment.

2.7 Conclusion

Our experiment simulated unpredictable and cumulative stress conditions during an important energy demanding period of the life cycle. Cumulative stressors can lead to an allostatic overload that results not only from the hormonal reactivity of individuals but also from the limited energy available that can either be allocated to maintenance or reproduction (McEwen & Wingfield, 2003; Romero et al., 2009). This response to stressors is modulated by individual heterogeneity in the initial energy state (i.e., pre-treatment spring body condition) and the intensity of the hormonal response (Cockrem, 2013). Manipulating body condition provided evidence that individual state is key to coping with unpredictable

stressors. This was found in a partial capital breeder that can accumulate substantial endogenous reserves to adjust to environmental stochasticity (Gauthier et al., 2003). We could thus expect an even stronger response in species that cannot accumulate large body reserves. The carry-over effects observed several months after imposing an unpredictable stress also highlight how long-lived individuals can adjust life history decisions (e.g., by reducing reproductive investment) based on their state, which can take along time to recover from some perturbations. However, when stressors were accumulated (e.g., kept in captivity without food after a stressful capture event), individuals were less able to cope with the increased allostatic (McEwen & Wingfield, 2003; Romero et al., 2009), which ultimately reduced survival. Even if the pair-bond disruption hypothesis remains a possible mechanism to explain our results, further studies should be dedicated to quantifying more directly the effect of pair-bond disruption on survival in long-lived monogamous species. Experimental designs in wild populations are difficult due to the many potentially confounding factors involved, necessitating large sample sizes to compensate for them, which can explain their low occurrence in the literature. Nonetheless, experimental studies such as ours remain a promising avenue to understand the mechanisms linking physiology and fitness to multiple environmental stressors that can interact with each other (Crespi et al., 2013).

2.8 Acknowledgements

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Data Availability Statement

All data and codes are available from the Dryad Digital Repository:

https:// doi. org/ 10. 5061/ dryad. c2fqz 61jr (Grentzmann et al. 2025).

2.9 Supplementary Materials

- Annexe S2.1: Details of the number of samples and observations collected for our analyses
- Annexe S2.2: Correction of corticosterone values by date and time since capture
- Annexe S2.3: Validation of random sampling
- Annexe S2.4: Complete model selection for CMR survival models
- Annexe S2.5 : Exploration of pairing status as a mechanism for the observed carry-over effect

Chapitre 3. Telomere length decreases with age and reflects early-life environment but not adult condition in a long-lived migratory bird

Île Bylot, Août 2021

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3.1 Résumé

Au sein des espèces et des populations, les individus diffèrent souvent dans leurs stratégies d'ajustements et plus généralement dans leurs stratégies d'histoire de vie. Cette hétérogénéité de stratégie individuelle est classiquement reflétée par des variations de traits physiologique, de probabilité de survie et de succès reproducteur. Chez de nombreuses espèces de vertébrés, la performance physiologique est partiellement médiée par les glucocorticoïdes et décline avec l'âge due à la sénescence, ce qui peut être reflétée par des variations de longueur des télomères. Les télomères sont des séquences d'ADN non codantes qui raccourcissent avec l'âge. Leur dynamique a été reliée au stress oxydatif et à l'allocation d'énergie aux mécanismes de réparation de l'ADN. Dans cette étude, nous avons exploré le lien entre longueur des télomères et quelques proxys de la qualité individuelle chez les femelles grandes oies des neiges (*Anser caerulescens atlanticus*), une espèce migratrice longévive. Plus spécifiquement, nous avons examiné les relations entre la longueur des télomères, les niveaux de corticostérone sous stress (principal glucocorticoïde chez les oiseaux), la taille corporelle et la condition corporelle post-reproduction. Nos résultats montrent la diminution attendue de la longueur des télomères avec l'âge. La longueur des télomères corrigée pour l'âge augmente avec la taille, mais ne varie ni avec la condition corporelle, ni avec la concentration de corticostérone sous stress. Par ailleurs, nous montrons que la condition post-reproduction des femelles varie avec l'âge, en augmentant jusqu'à l'âge de 10 ans et en diminuant ensuite. Nos résultats appuient l'idée que la longueur des télomères pourrait être un indicateur fiable des conditions de début de vie, et potentiellement de la qualité individuelle. En effet, la taille corporelle et l'intensité de la réponse hormonale au stress sont principalement déterminées pendant le développement, et la longueur des télomères ne semble pas liée aux réserves endogènes au stade adulte.

3.2 Abstract

Within species and populations, individuals often differ in their coping styles and more generally in their life history strategies. This individual heterogeneity in individual strategy is classically reflected by variations in physiological traits, survival probabilities, and reproductive success. In many vertebrate species, physiological performance is partially mediated by glucocorticoids and declines with age through senescence, which may be reflected by variation in telomere length. Telomeres are non-coding DNA sequences that shorten with age. Their dynamics have been related to oxidative stress and energy allocated to DNA repair mechanisms. In this study, the link between telomere length and several proxies of individual quality was investigated in female greater snow geese, a long-lived migratory species. More specifically, we examined the relationships between telomere length, stress-induced corticosterone (the main avian glucocorticoid), body size and post-breeding body condition. Our results showed the expected decline of telomere length with age. Telomere length adjusted for age increased with body size, but did not vary with body condition or stress-induced corticosterone levels. On the other hand, we show that female post-breeding body condition varies with age, increasing until 8 years old and decreasing at a later age. Because body size and corticosterone stress response sensitivity are primarily determined during development, our results point to telomeres being a good indicator of the conditions experienced during early life. In contrast, it does not seem to be related to the amount of endogenous reserve during adult life. This supports the idea that telomere length could be a reliable marker of early developmental conditions, and potentially of individual quality.

3.3 Introduction

Individual heterogeneity in performance can be observed across all life-history stages, revealing itself in uneven physiological performance, reproductive success or survival probabilities, or in the unequal mitigation of reproductive costs (Wilson & Nussey, 2010; Hamel, Gaillard, & Yoccoz, 2018; Vedder & Bouwhuis, 2018). This inter-individual variability can arise from genetic factors, developmental conditions in early life, or the accumulation of adverse life experiences (Wilson & Nussey, 2010; Hau et al., 2015). Individual quality describes this heterogeneity in relation to important fitness traits (Wilson & Nussey, 2010; Hamel, Gaillard, & Yoccoz, 2018), such as the age at first reproduction, breeding frequency, the number of offspring produced per breeding event and longevity (Hamel, Côté, et al., 2009; Hamel, Gaillard, & Yoccoz, 2018; Angelier et al., 2019). Individual quality is often interpreted as a fixed inherent characteristic, but an increasing number of authors suggest that individual heterogeneity in quality may also be dynamic, and the characteristics interpreted as intrinsic quality, usually inherited via genetics, epigenetics or parental effects, could be only part of the observed variability (Wilson & Nussey, 2010; Hamel, Gaillard, & Yoccoz, 2018).

One example of dynamic individual quality in fitness is senescence, which is the deterioration of an organism with age, through the accumulation of damage in the body (Kirkwood, 2002). The internal organs become progressively less efficient, resulting in reduced performance of older individuals compared to younger ones (Kirkwood, 2002), as revealed by a decrease in survival probability or reproductive success (Bouwhuis et al., 2012). Consequently, high-quality individuals may be more likely to reach older ages as their performance will probably decrease at a slower senescence rate than lower quality individuals (Angelier et al., 2007; Hamel, Gaillard, & Yoccoz, 2018). In this context, approaching individual quality as a dynamic characteristic changing throughout life helps to conceptualize the heterogeneity in performance observed within a population and highlights the importance of integrating age into studies of individual quality (Wilson & Nussey, 2010). Individual quality can therefore be measured using labile traits, which stay positively correlated with fitness though they vary throughout the individual's life (Hamel, Côté, et al., 2009; Wilson & Nussey, 2010).

Recent studies have looked at telomeres as markers of this dynamic heterogeneity (Bauch et al., 2013; Le Vaillant et al., 2015; Angelier et al., 2019). Telomeres are repetitive non-coding DNA sequences located at the extremities of chromosomes that protect coding DNA from loss of information due to DNA attrition during replication (Blackburn, 2005). Their length is regulated by multiple mechanisms including mechanical attrition and damages due to oxidative stress, and potential repair by telomerase enzymes (Blackburn, 2005; Angelier et al., 2018). As such, telomere length is a combination of genetically inherited telomere length at birth (Asghar, Bensch, et al., 2015; Froy et al., 2021; Bauch et al., 2022; Vedder et al., 2022), maternal effects (Asghar, Bensch, et al., 2015), energetically demanding life-stages (Bauch et al., 2013; Criscuolo et al., 2018) and cumulated exposure to stress (Hau et al., 2015; Angelier et al., 2018). Several studies have found a relationship between telomere length and individual quality, with positive links between telomere length and reproductive output (Criscuolo et al., 2018; Eastwood et al., 2019; Pepke, Kvalnes, Ranke, et al., 2022) or with lifespan (Wilbourn et al., 2018), as formulated in the 'telomere-quality hypothesis' (Angelier et al., 2019). The high inter-individual variability in telomere length typically observed regardless of age (Zhao et al., 2019; Remot et al., 2022) further supports telomere length as a potential indicator of individual quality (Angelier et al., 2019).

Coupling telomere length with other phenotypic traits that reflect individual quality is essential to uncover potential coping mechanisms to changing environmental conditions. Glucocorticoids are a good proxy of the ability of individuals to cope with their environment (Wingfield et al., 1998) because they are involved in general mediation of energy allocation, metabolic activity and stress response (Romero et al., 2009). Variations in baseline glucocorticoids have been linked to variations in overall individual performance (Bonier et al., 2009; Schoenle et al., 2021). When exposed to an unexpected stressor, glucocorticoid levels increase outside the baseline range (Wingfield et al., 1998), mediating a series of short-term behavioral and physiological changes that aim to promote immediate survival (Wingfield et al., 1998; Angelier & Wingfield, 2013). The magnitude of the stress response is classically associated with life-history decisions with immediate or long-term consequences on reproduction and survival (Breuner et al., 2008; Schoenle et al., 2021). The shortening effect of stressors on telomeres (Chatelain et al., 2020) is thought to be mediated, at least partly, by the detrimental effect of elevated glucocorticoid levels on telomere length

(Monaghan, 2014; Hau et al., 2015; Angelier et al., 2018). This link has notably been experimentally shown in early life (Haussmann et al., 2012; Casagrande et al., 2020).

Glucocorticoid levels ultimately relate to an efficient management of current allostatic load by individuals and is thus linked to body condition (McEwen & Wingfield, 2003; Romero et al., 2009; Angelier et al., 2018). They might thus reflect heterogeneity in energy allocation strategies or resource acquisition capacity (McEwen & Wingfield, 2003), which could be important drivers of individual quality (Vedder & Bouwhuis, 2018). As glucocorticoids and body condition give insight into the physiological state and energy allocation in the present or recent past (Young et al., 2015), body size can reflect the impacts of environmental conditions and energy allocation in early life (Boonekamp, Mulder, et al., 2014; Angelier et al., 2019). An unfavourable environment can indeed induce a direct decrease in growth through diminished nutrition (Gauthier et al., 2006; Doiron et al., 2015; Vedder & Beccardi, 2025). It can also provoke a high hormonal stress response which itself has repercussions on development (Blas et al., 2007; Crino & Breuner, 2015). The correlation between early-life development and adult performance warrants the inclusion of data from both adults and juveniles in integrative studies (Nussey et al., 2007; Cam & Aubry, 2011; Vedder & Beccardi, 2025).

The aim of the current study is to investigate the link between telomere length and physiological traits previously associated with individual quality and how these traits vary with age in a long-lived bird, the greater snow goose (*Anser caerulescens atlanticus*). We first examined how telomere length varies with age as telomere length is supposed to decrease with advancing age (Remot et al., 2022). We then investigated how stress-induced variations in corticosterone (CORT) levels, the main glucocorticoid in birds (Romero, 2004) relate to telomere length and age. Stress-induced CORT has been shown to be heritable (Stedman et al., 2017), repeatable (Taff et al., 2018; Grentzmann et al., 2025), and related to coping strategies (Angelier et al., 2018). For example, a phenotype that allocates more energy in the short-term response to stressful events, would also increase metabolism and oxidative stress (Buchanan, 2000). The increased oxidative stress in turn accelerates telomere attrition (Hau et al., 2015; Boonekamp et al., 2017) and reduces performance (Angelier et al., 2018). We thus expected the intensity of the CORT stress response to vary negatively with telomere

length. Finally, we used body size as a proxy for the influence of developmental conditions on fitness as it highly depends on environmental conditions during early growth and is linked to performance (Gauthier et al., 2006). Unfavorable developmental conditions can accelerate telomere shortening and result in shorter telomere length in adult life (Asghar, Bensch, et al., 2015), and a smaller adult body size. We also looked at the relationship between telomere length and body condition as it represents the capacity of individuals to accumulate energy reserves and has been repeatedly linked to reproduction (Rowe et al., 1994; Bêty et al., 2003; Angelier et al., 2020) and shown to mitigate the effects of stress, increasing the capacity to cope with a stress event (Angelier et al., 2020). We therefore predicted a positive correlation between telomere length and body condition. To attempt to decipher the role of early life developmental conditions, we also tested the relationships between these variables in juveniles.

3.4 Materials and methods

3.4.1 Study species, data and sample collection

The greater snow goose is a long-distance migratory species wintering along the Atlantic coast of the United States, staging in spring and fall in Québec along the St. Lawrence River and breeding in the Canadian High Arctic (Lefebvre et al., 2017). Geese are precocial birds that form permanent, monogamous pair bonds, and females return to their birth colony to breed with their mate (Lecomte et al., 2009). Bylot Island, Nunavut (73° N, 80° W) is the largest known breeding colony of greater snow geese (Reséndiz-Infante et al., 2020). The arrival of geese on the breeding site, and the subsequent laying of eggs occurs in June. The eggs are then incubated in late June and early July, after which the young are raised until the end of August, when they depart the island for their fall migration.

Nesting geese are monitored annually on Bylot Island since 1990 (Reséndiz-Infante et al., 2020). Between 300 and 1 500 goslings are annually marked in the nest with uniquely numbered web-tags within 24 hours of hatching. Near the end of brood-rearing period and while adults moult their flight feathers and are flightless, mass captures take place for banding. All captured birds (young and adults) are sexed by cloacal examination and banded,

and we checked for presence of web-tags in young (Reséndiz-Infante et al., 2020). All web-tagged hatching birds, all known-age females and a random sample of adults were weighed (nearest g) and culmen, head and tarsus length were measured (nearest 0.1 mm). Our marking process does not enable us to collect any data on relatedness.

Blood samples were taken between 2018 and 2023 during banding operations. We only sampled females because their return rate is higher than males due to their fidelity to their natal breeding grounds, as pairs form on the wintering grounds and males disperse to follow their mate (Lecomte et al., 2009). Blood samples were drawn from 92 known-age adult females (i.e. recaptured birds originally banded in their hatching year), ranging from 1 to 24 years old, and from 23 goslings (15 females and 8 males), web-tagged during hatching, ranging from 25 to 41 days old and unable to fly. Samples on goslings and adults were taken ca. one and two hours after the initiation of the capture process, respectively. We collected 1ml of blood from the tarsal vein using 23G needles and syringes, kept the samples on ice for up to eight hours in a tightly closed cooler, and then centrifuged at 8000G for 8min to separate the plasma from the red blood cells. These fractions were then frozen separately at -20°C initially (max. 20 days) and at -80°C after field work.

3.4.2 Laboratory analyses

DNA was extracted from red blood cells using NucleoSpin 96 Blood Kit from Macherey-Nagel (Macherey-Nagel, 2023), following the manufacturer's instructions. The quantity and quality of the extracted DNA were established using UV absorbance spectroscopy and all samples used had 260/280 absorbance ratio between 1.8 and 2.0 and 230/260 ratios between 2.0 and 2.2, indicating good DNA purity and low contamination (Shim et al., 2010). Relative telomere length (RTL) was quantified using quantitative PCR (qPCR) (Cawthon, 2002) on the total genomic DNA, a technique validated in birds (Criscuolo et al., 2009). The control single-copy gene (Recombination activating gene 1 - RAG1) was detected with the following primers: RAG1-F (5'-TGTACGGGAAGTGGAAGGG-3') and RAG1-R (5'-GGTGATGGAGTGGAAGACC-3'). The telomere primers used were: Tel1b-F (5′-CGGTTTGTTTGGGTTTGGGTTTGGGTTTGGGTTTGGGTT-3′) and Tel 2b-R (5′-GGCTTGCCTTACCCTTACCCTTACCCTTACCCTTACCCT-3′). All primers were

obtained from Sigma Aldrich. qPCR for both RAG1 and telomeres was performed with iQ™ SYBR® Green Supermix containing iTaq™ DNA Polymerase (Bio-Rad) using 7.5 ng of DNA and primers at a concentration of 500nM and followed the same amplification protocol, which consisted of 2min at 95°C followed by 40 cycles of 5 seconds at 95°C and 30 seconds at 55°C on a CFX Opus 384-well real time PCR detection system (Biorad). All samples were run in triplicates on one 384-well-plate for each gene. All samples were run on a single plate and were randomized over the plate. The plate included a standard curve and negative control wells. To create the standard curve, we pooled 10µL of each sample and diluted this mix by a factor of 2 from 40nmol/µL to 0.3nmol/µL, which was used to control for the amplification efficiency (Cawthon, 2002). The efficiency for telomere amplification was 101.5% (R^2=0.99) and for RAG1 was 108.5% (R^2=0.96). We used our first sample as a reference, after controlling for its quality. We used the CFX Maestro application (Biorad) to visualize our results and calculate RTL (Biorad, 2017). RTL is a ratio of quantification cycles of telomere in the sample compared to the reference gene RAG1 for the same sample, relative to a reference sample present on each plate (Pfaffl, 2001).

$$RTL = \frac{(E+1)^{Cq_{reference\ sample} - Cq_{sample}}\ for\ telomeres}{(E+1)^{Cq_{reference\ sample} - Cq_{sample}}\ for\ reference\ gene}$$

where E: efficiency of the amplification calculated for each plate using our standard curve and Cq: quantification cycles.

Corticosterone was quantified by radio-immuno assay on 74 plasma samples (60 adult and 12 hatching year females for which we also had RTL) (Lormée et al., 2003). Samples were run in duplicates, intra-assay precision was 10.8%, inter-assay precision was 13.1%. The CORT level measured was a stress-induced level because measurements was taken ≥ 1 hour after capture. To test whether the time elapsed before sampling affected CORT values, we used the order of sampling in a linear model, but no significant influence was found (β_{Juv} = -1.51 [-5.55, 2.53]; β_{Ad} = 1.88 [-1.43, 5.18]).

3.4.3 Statistical analyses

We analyzed goslings and adult females separately. For each analysis, we checked whether our data was structured by year of sampling or year of birth using linear models. Since these variables never explained the variance in our data, we did not include any random effects in our analyses. For all linear models, the normality and homoscedasticity of residuals were confirmed graphically. We identified the best fitted models based on the corrected Akaike information criterion (AICc) model selection (Burnham & Anderson, 2004). We then used estimates (β) and 95% confidence intervals to assess significance of relationships with the variables of interest. Sample sizes for adult and juvenile females vary between models due to differences in data availability across individuals.

The body size and body condition indices were calculated separately for adult females and goslings. To calculate our body size index, we did a principal component analysis (PCA) including head, culmen and tarsus length measurements (see PCA results in Figure S3.1 in Appendix S3.1). We used the scores on the first principal component (PC1) of the PCA as the body size index. The body condition index was calculated as the body mass corrected by the body size index and capture date. This was done by adding the residuals of the regression between body mass and body size of all adult females captured between 1990 and 2023 to the population mean. We used the same method to correct for date because females gain mass during the summer to recover from the energetic cost of reproduction (Marmillot et al., 2016). Body condition of goslings was estimated in the same way as adults in a separate analysis using all individuals captured between 2018 and 2023.

In goslings, we tested for a fixed effect of year of birth, sex and age (in days) on RTL in goslings using linear models. We performed a model selection using AICc on models with additive effects, but could not test the interactions between these variables due to our limited sample size for goslings (N=23). As we did not have the data about the parents, the effect of family could not be tested. We tested for linear relationships between RTL, age (in years for adults and in days for goslings), stress-induced CORT, body size, and body condition. Since goslings are growing, we included age as a fixed effect in our models for body size. We considered the relationship between age and body size to be linear, because ages 20 to 41 days belong to the linear section of the growth curve (Lesage & Gauthier, 1997).

Relationships between RTL, age (in years for adults and in days for goslings), stress-induced CORT, body size, and body condition were tested using linear models. Since RTL and age were related in adult females (see results), we corrected RTL values for this age effect in subsequent analyses by including an additive fixed effect of age in the models. We ensured the variance inflation factor (VIF) remained below 5 for all models, indicating that the correlation between RTL and age did not interfere with model estimations (Zuur et al., 2010). For visualization purposes, we represented model predictions for a fixed age (set to sample mean: 7.66 years). To investigate differences in age-corrected RTL between years of birth, we included year of birth as a fixed effect in a linear model. Because our samples were unevenly distributed between years of birth, we removed all years where we only had one or two samples. Consequently, 73 samples were included in this analysis distributed across 12 years of birth.

Stress-induced CORT level was related to date of capture (β_{adults}= -5.72 [-10.74; -0.69], $\beta_{goslings}$= -4.68 [-10.74; 1.38]). We corrected CORT values by adding the linear regression residuals to the population mean in each group to account for the variation of CORT in time.

We tested for a senescence effect on body condition with either a linear or a quadratic model relating body condition and age in the 1990-2023 and the 2018-2023 datasets (N=1084 and N=92 known-age adult females respectively). Because some individuals were measured more than once between 1990 and 2023, but too few to include a random effect for ID, we only kept the latest value to eliminate pseudo-replication, while increasing sample size for older age individuals.

3.5 Results

RTL did not differ between goslings and adults (β = 0.07 [-0.20, 0.34]). Among adult females, we found evidence for a linear decrease in telomere length with age (in years; β = −0.04 [−0.06, −0.01]; Figure 3.1). RTL in adult females varied significantly with year of birth (F-value = 1.38 on 12 and 60 df; p-value = 0.05; Figure S3.2 in Appendix S3.2) in a model taking age into account.

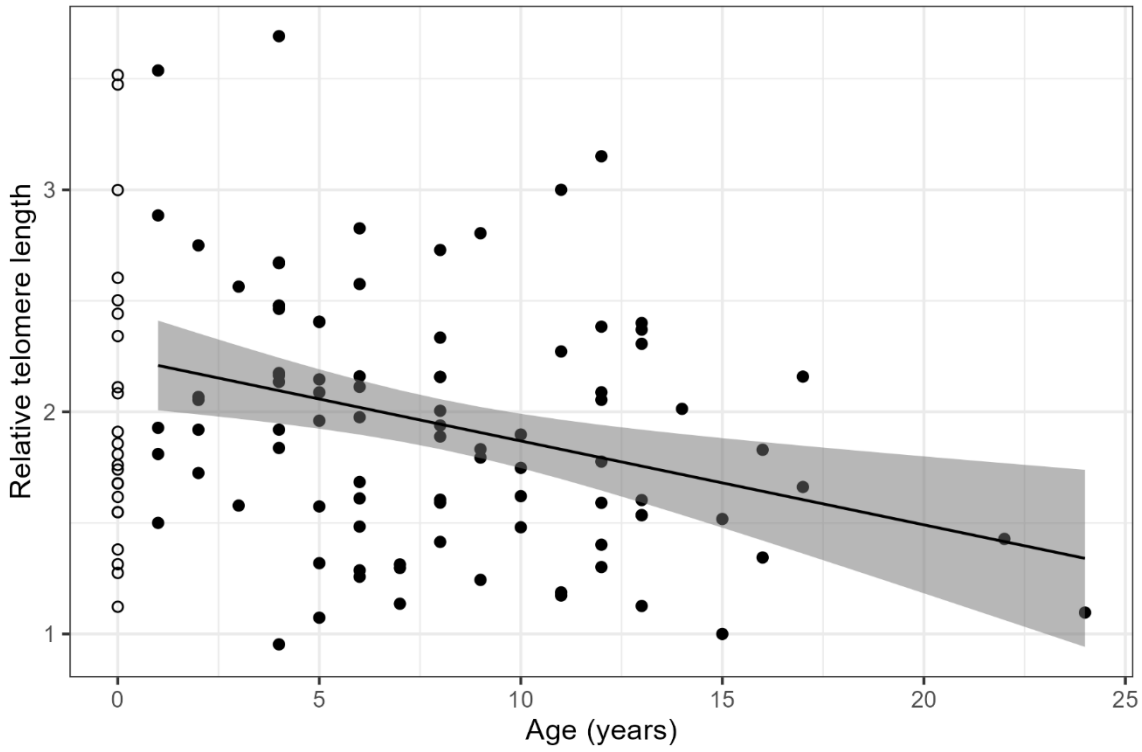

Figure 3.1 : Relationship between relative telomere length (RTL) and age in 92 adult female (≥ 1 year old) greater snow geese and 23 goslings (R^2=8%). The estimated linear regression with 95% confidence interval is provided. RTL of goslings were included in the figure (empty circles) but not in the regression.

Table 3.1 : Model selection for the influence of hatching year (2019, 2022, 2023), age (ageDAYS = number of days since hatching) and sex on RTL in 22 goslings (one outlier was removed). Only models with a ΔAIC<4 were included in the table.

Explanatory variables	Number of parameters	Log-likelihood	ΔAIC
Year	4	-17.57	0.0
Null model	2	-21.32	1.77
Year + ageDAYS	5	-17.14	2.54
Year + sex	5	-17.36	2.98
Sex	3	-20.90	3.63
ageDAYS	3	-21.01	3.86
ageDAYS + sex	4	-20.46	5.78

Sex and number of days since hatching did not explain the RTL variation in goslings (β_{Male} = -0.34 [-0.93, 0.25]; $\beta_{ageDAYS}$ = 0.02 [-0.04, 0.07]; Table 3.1; Figure 3.2A). RTL of goslings varied among years of birth, being higher in 2022 than 2023 ($\beta_{2023/2022}$ = 0.85 [0.21, 1.48]; best model of Table 3.1; Figure 3.2B).

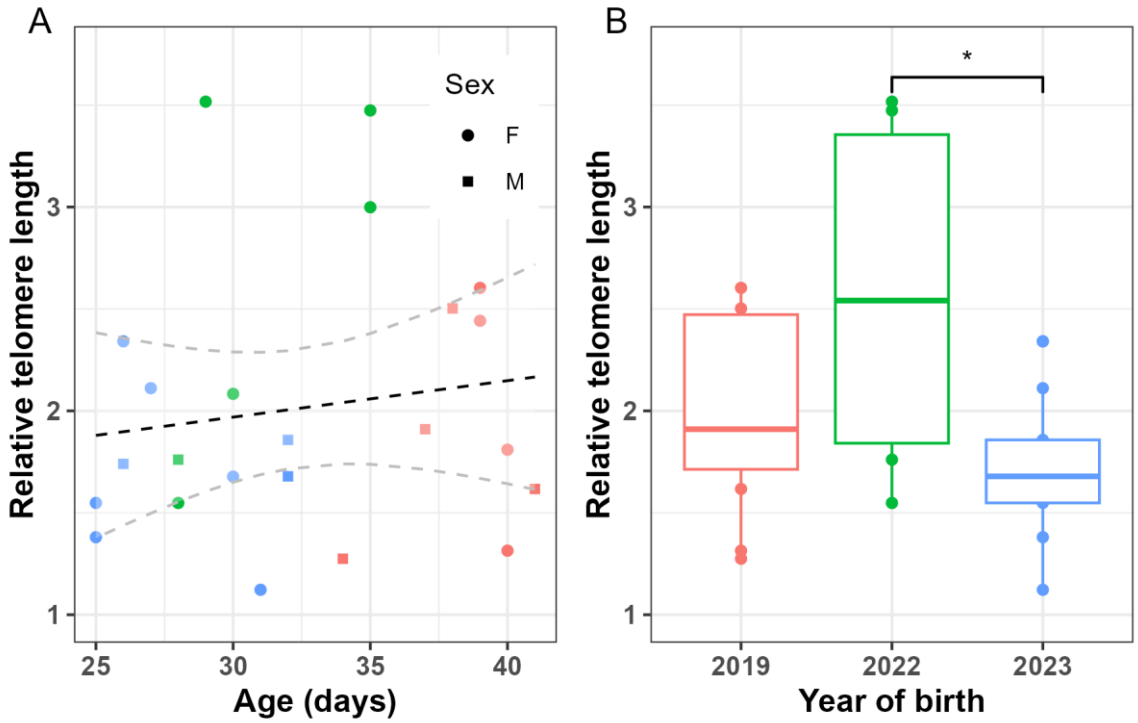

Figure 3.2 : Variation of RTL in 23 goslings (15 females and 8 males). A: Relationship between RTL and age in days. The regression is represented by a dashed line because it is non-significant. B: Boxplot (mean and SD) of the distribution of RTL for each year of birth. The star represents a significant different in RTL value. In both panels, 2019 samples are represented in red, 2022 in green and 2023 in blue.

Stress-induced CORT levels were lower in goslings than in adults (β = -63.14 [-95.09, -31.19]; Figure 3.3C). However, CORT levels were not explained by age in days in goslings (age in days; β = 0.24 [-2.17, 2.65]; Figure S3.3 in Appendix S3.3), nor in adults (age in years; β = 0.10 [-3.15; 3.35]; Figure S3.3 in Appendix S3.3) and was not correlated with body condition (adults: β = 6.20e-04 [-0.08, 0.09]; goslings: β = -0.03 [-0.12, 0.05]). In goslings, stress-induced CORT did not explain RTL (β = 3.84e-03 [-0.03, 0.03]; Figure 3A). Similarly, RTL was not related to stress-induced CORT levels in adults (β = -3.68e-04 [-2.76e-03, 2.02e-03]; Figure 3.3B).

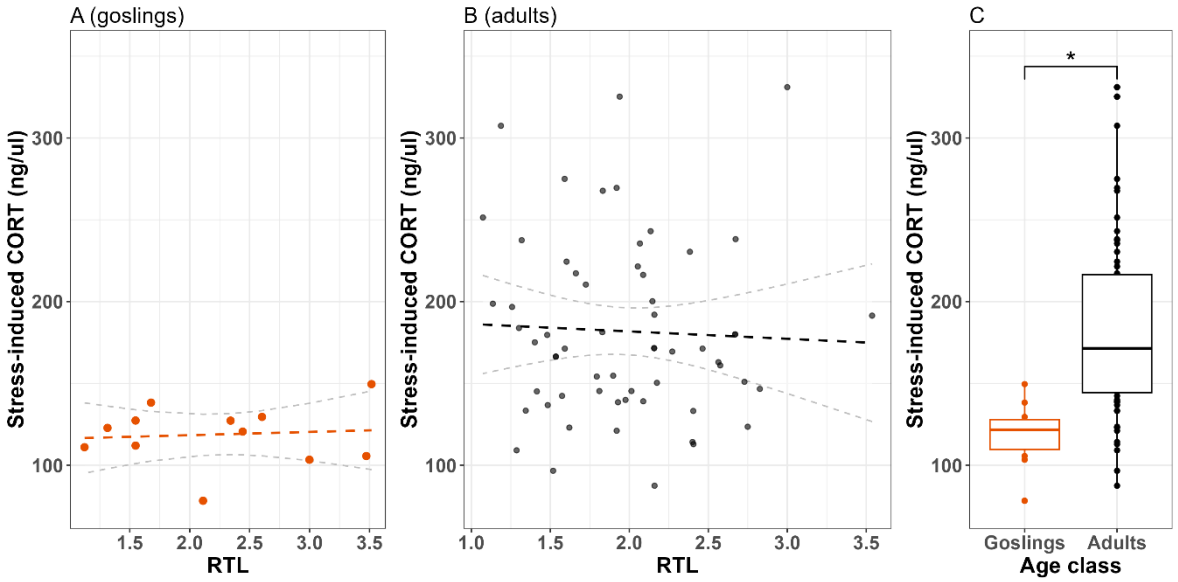

Figure 3.3 : Relationship between RTL and stress-induced CORT levels corrected for date of capture in 12 goslings (A) and 60 adult female greater snow geese (B; controlled for age). Panel C represents the mean and standard error of stress-induced CORT values in goslings and adults. The star represents a significant difference in stress-induced CORT levels. Linear regression estimates and 95% confidence intervals are represented by dashed lines because they are not significant (A and B).

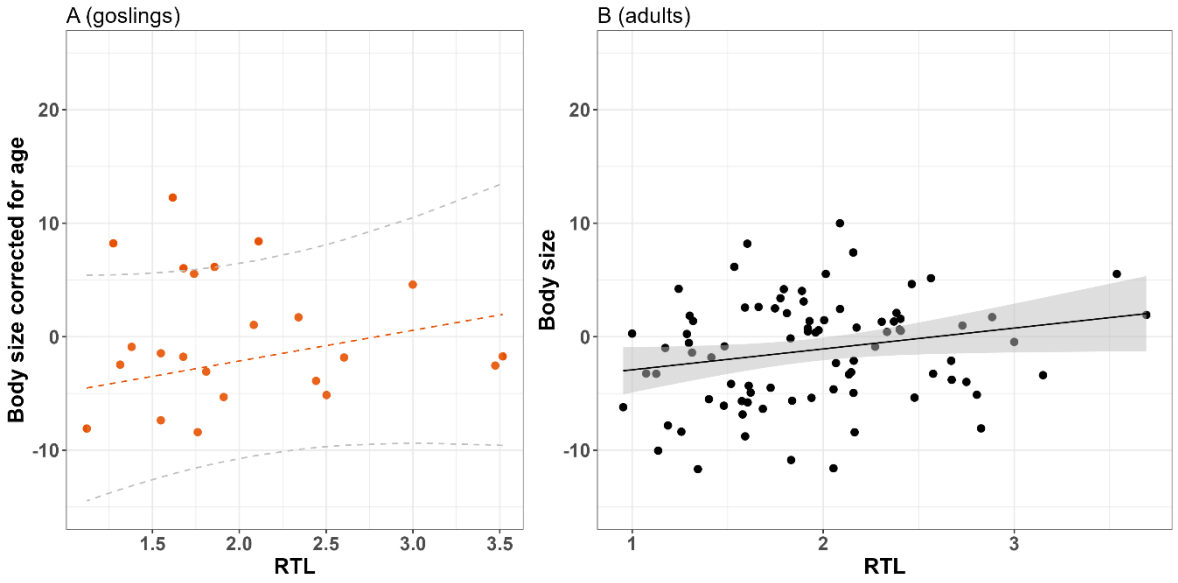

Figure 3.4 : Relationship between body size (-PC1 scores) and RTL. A: model in 23 goslings included age as a fixed effect. B: model controlling for age in 92 adult female greater snow geese (R^2=6%). The estimates and 95% confidence interval of the linear regressions are represented by dashed lines when non-significant (A) and in solid lines when significant (B).

In goslings, size did not correlate with RTL in our model including age (β = -0.38 [-4.46, 3.70]; $\beta_{shortRTL}$ = -2.09 [-8.69, 4.74]; Figure 3.4A). In adults however, body size increased significantly with RTL (β = 1.84 [0.22, 3.65]; Figure 3.4B).

Table 3.2 : Model selection for the relationship between post-breeding body condition (condition) and age (in years) in 1084 known-age adult female greater snow geese measured between 1990 and 2023. Year of capture is included as a random effect. Age ranges from 1 to 24 years old.

Model name	Explanatory variables	Number of parameters	Log-likelihood	ΔAICc
Cond1.1	Age + Age2	4	-6938.12	0.0
Cond1.2	Age + Age2+ Age3	6	-6938.04	1.86
Cond1.3	Age	3	-6945.80	13.35
Cond1.4	Null model	2	-6951.03	21.79

Table 3.3 : Model selection for the relationship between post-breeding body condition, age and relative telomere length (RTL) in 92 known-age adult female greater snow geese from 1 to 24 years old sampled between 2018 and 2023.

Model name	Explanatory variable	Number of parameters	Log-likelihood	ΔAIC
Cond2.1	Age + Age^2	4	-592.91	0.00
Cond2.2	Null model	2	-595.27	0.38
Cond2.3	Age	3	-594.79	1.57
Cond2.4	Age + Age^2 + Age^3	5	-593.39	3.21
Cond2.5	RTL	3	-594.74	3.68
Cond2.6	RTL*Age	5	-594.22	4.87
Cond2.7	RTL + RTL2	4	-594.74	5.91

When using all 1084 known-age females measured between 1990 and 2023, we found that body condition of post-breeding females varied with age following a quadratic pattern (β_{Age} = 509.03 [212.82; 805.33]; β_{Age2} = -575.74 [-862.65, -288.72]; Model Cond1.1 in Table 3.2; Figure 3.5). The cubic model was also within an AICc difference of two points, but the quadratic model was preferred and more parcimonious, it is thus the one we retained.

According to this quadratic model, body condition reached its maximum at 10 years (Figure 3.5). These results were also supported when using only the adult females captured in 2018-2023 for which we have blood samples. Models including a linear or a quadratic effect of age on body condition were equivalent to the null model (β_{Age} = 163.42 [-166.91, 493.74]; β_{Age2}= -320.24 [-650.56, 10.09]; Model Cond2.1 in Table 3.3). Body condition was not related to RTL (β_{RTL} = 9.26 [-56.84; 75.35]; Model Cond2.5 in Table 3.3) and neither to RTL in goslings ($\Delta AICc_{linear-null}$ = 2.15; β_{RTL} = 29.17 [-112.80; 171.14]). No difference in body condition was found between sexes in goslings (β_{Male} = -114.75 [-300.13, 70.63]).

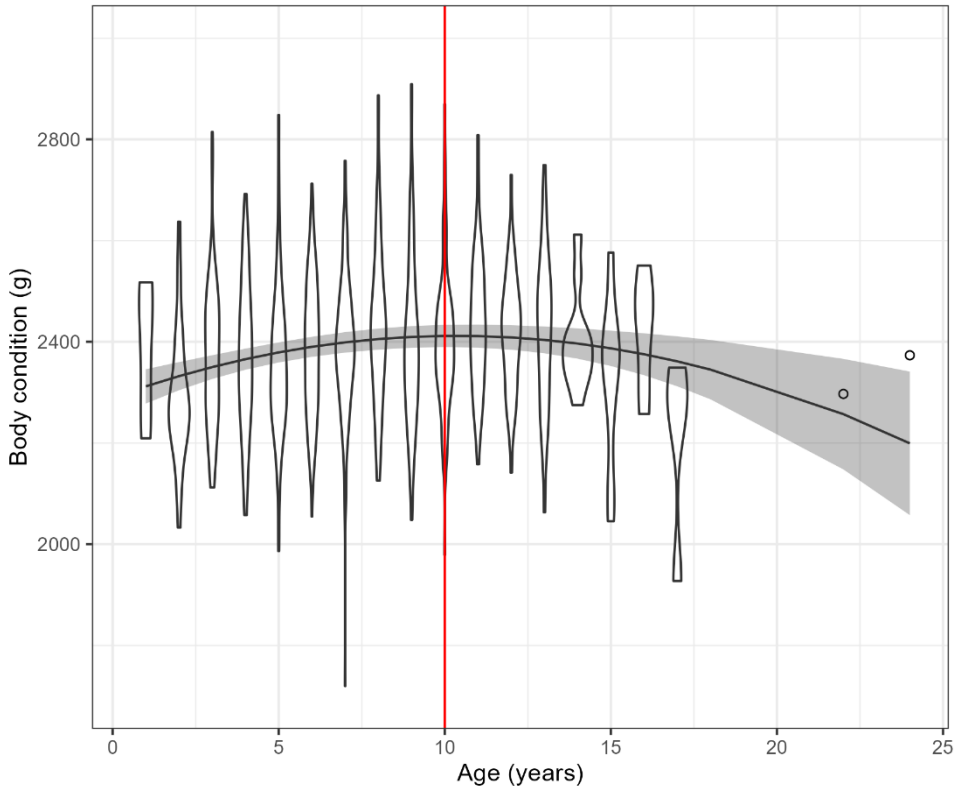

Figure 3.5 : Relationship between post-breeding body condition (weight corrected for body size and date of capture) and age in 1084 known-age adult female greater snow geese measured between 1990 and 2023. The gray line is the estimated quadratic regression with its 95% confidence interval (gray ribbon) based on the preferred model in Table 2. The vertical red line is the age of maximal predicted body condition (10 years). Black violons depicts the density distribution of annual data points. Two points were removed from the graph but not from the analysis for better graphical representation: one under 1700g and one above 3000g.

3.6 Discussion

As hypothesized, we observed a decrease in telomere length with age in snow geese breeding in the colony of Bylot Island. However, further analysis indicated that both age and telomere length may provide complementary information to explain individual heterogeneity and its dynamics throughout individual lifespan. The high variability in RTL observed in adults, and especially in goslings, is consistent with the observations reported in the literature in birds (Monaghan & Haussmann, 2006) and in numerous other vertebrate species (Remot et al., 2022). In adult females, there was some evidence that variability in telomere length could partly be explained by the intensity of the stress response and was linked to body size but not to body condition. Instead, variation in body condition seemed to be better explained by age alone.

Interestingly, neither RTL mean or variance varied between adults and goslings. The variability in RTL observed in goslings aligns with several mechanisms that have been suggested in the literature to explain inter-individual variations in telomere length at birth and during early life, when telomere length is most dynamic (Asghar, Bensch, et al., 2015; Angelier et al., 2018). Hatching year seemed to explain this high variation best, highlighting the importance of environmental conditions experienced in early-life on telomere dynamics (Angelier et al., 2018; Giraudeau et al., 2019; Pepke, Kvalnes, Ranke, et al., 2022). In our study, RTL on goslings were measured between 25 and 41 days after hatching, towards the end of growth (Gauthier et al., 2006). With this in mind, we note that RTL of goslings were highest in 2022, which was a particularly poor reproduction year, in part due to a very late snow melt (Cadieux et al., 2023). The low number of individuals that attempted to breed and had a successful reproduction were more likely high-quality parents. Since telomere length and longevity share a strong genetic basis (Vedder et al., 2022), goslings with very short telomeres are likely to experience reduced survival and thus disappear more rapidly from the population. Then, both genetic and parental effects could thus explain the long telomere lengths observed in goslings in that year (Asghar, Bensch, et al., 2015; Bauch et al., 2022). Combined with the natural within-individual shortening of telomeres with age, the selective disappearance of goslings with short telomeres would explain why older individuals no longer show long telomeres. In fact, telomere length decreased with age in adults, as in most

vertebrate species that have been studied (Remot et al., 2022), in particular in other species of similar lifespans, as in jackdaws (*Corvus monedula*; Salomons et al., 2009). This is consistent with the hypothesis of a mechanical degradation of telomeres with age (Blackburn, 2005; Angelier et al., 2018). The high variability observed at each age in snow geese (R^2=8%) also aligns with previous findings (Remot et al., 2022). This variability underpins current investigations into the relationship between telomere dynamics and individual quality (Monaghan & Haussmann, 2006).

We expected RTL to vary with body size, body condition or number of days since hatching, because telomeres shorten at a rapid rate during early development (Boonekamp, Mulder, et al., 2014; Angelier et al., 2018) and harsh environmental conditions can accelerate this attrition (Chatelain et al., 2020; Pepke, Kvalnes, Ranke, et al., 2022), increase CORT levels (Monaghan, 2014) and hinder growth (Gauthier et al., 2006; Monaghan, 2014). The absence of a relationship between RTL and the mentioned variables in goslings could partially be attributed to the fact that goslings have not reached their final size at the time of sampling. Alternatively, the timing of hatching because older individuals at banding tend to be born earlier in the season than younger individuals. Previous studies have in fact shown the strong influence of timing of hatching on gosling growth (Lepage et al., 1999; Doiron et al., 2015) and both seasonal and inter-annual variations in environmental conditions on their survival (Lepage et al., 1999; LeTourneux, 2024). However, adult body size was positively related to RTL, as previously observed in the black-browed albatross (*Thalassarche melanophris;* Angelier et al., 2019) and other passerine species (Angelier et al., 2015; Parolini et al., 2017). Body size has been shown to be genetically heritable in mammals (Bouwman et al., 2018), and in birds (Jensen et al., 2003; Luo et al., 2025), suggesting that the correlation we observe could stem from a shared genetic basis. However, given the importance of environmental conditions for growth in our species (Lepage et al., 1999; Gauthier et al., 2006; Doiron et al., 2015; LeTourneux, 2024), we hypothesize that the positive association between large body size and telomere length in adults may reflect environmental conditions experienced early in life (Boonekamp, Mulder, et al., 2014; Angelier et al., 2018, 2019).

Telomeres and CORT did not vary with the number of days since hatching, but we also found that goslings had a lower CORT stress response than adults. Similarly, Magellanic penguin (*Spheniscus magellanicus*) chicks had lower stress-induced CORT levels than adults when facing similar stressors but were physiologically capable of excreting similar CORT levels (Walker et al., 2005). A possible explanation for this difference may be the need for young birds to avoid deleterious effects of high CORT values on development, as CORT can interfere with growth hormones (Mazziotti & Giustina, 2013). It is also possible that goslings may preferentially allocate their energy to growth rather than the HPA-axis and associated systems function (Crino & Breuner, 2015) as their survival could be more sensitive to growth than to their ability to respond to stress in presence of parents (Gauthier et al., 2006; Richman et al., 2015). In this study, stress-induced CORT level of goslings was not related to telomere length. Other studies have experimentally shown that increased levels of CORT during development shortens juveniles' telomeres (Haussmann et al., 2012; Casagrande et al., 2020). However, most studies on wild nestlings measure baseline CORT (e.g. Quirici et al., 2016) and studies looking at the link between stress-induced CORT and telomere length in wild animals are still rare. The absence of relationship in our results would be consistent with the idea that CORT is downregulated in growing young and thus is not an important driver of metabolism and physiology for them.

Telomere dynamics is influenced by various forms of stress, as measured by CORT level, including environmental and physiological challenges, which affect cellular aging and organismal performance (Chatelain et al., 2020). Therefore, CORT could also be a good candidate to explain variability of telomere length in adults, as telomeres and CORT have both been linked to the capacity of individuals to respond to their environment by mediating energy allocation and life-history trade-offs (Angelier et al., 2018). Given our capture method, we were unable collect baseline samples or to account for duration of handling. The CORT levels in our data are thus impacted by the stress response and the subsequent negative feedback loop (Romero, 2004). CORT has been mechanistically linked to telomere length (Lin & Epel, 2022) and the shortening effects of oxidative stress on telomere length have also been shown in wild populations (Monaghan, 2014; Boonekamp et al., 2017). We had therefore hypothesized that stress-induced CORT and telomere length would be negatively correlated, which we do not observe in our data. This could either be due to the very high

variability in both CORT values and RTL in our data, or to the season at which the samples were collected. The stress-induced CORT levels measured at the end of the reproductive season in the current study were similar to those measured during spring migration (Chapter 2) so we cannot confirm a potential seasonal effect. Finally, a negative correlation between stress-induced CORT and RTL would have been evidence for a trade-off between immediate and long-term survival (Angelier et al., 2018). It is entirely possible that we cannot observe this trade-off in our sampled population, composed of reproducing adults. The sampled population could be biased towards higher-quality individuals, because non-reproducing pairs do not travel to the Bylot Island colony.

To test whether differences in telomere length could be linked to heterogeneity in individual performance according to the telomere-quality hypothesis (Angelier et al., 2019), we used the body condition near the end of brood rearing, which should be indicative of an individual's capacity to mitigate the costs of reproduction. Females invest a significant amount of their endogenous reserves in the eggs (Gauthier et al. 2003) and they lose an additional 17% of body mass during incubation due to high nest attentiveness (Reed et al., 1995). Studies on individual heterogeneity in chick-rearing cost mitigation are still rare, but high energy requirements have been shown to lead to a loss in body condition (Leclaire et al., 2011). We captured females in late summer when they were still molting, another energy-demanding process (Marmillot et al., 2016), and at a time when they need to start accumulating body reserves for the upcoming fall migration. Consequently, the late summer body condition could indicate their capacity to manage energy reserves efficiently. This capacity to optimize allocation to several energy-demanding activities may ultimately impact their survival or future reproductive success. Their post-breeding body condition was linked to age, increasing until about 10 years and decreasing at a later age. However, body condition was not associated with age-corrected RTL. This could be explained by the relatively low sample size, as uncovering a significant relationship with a highly variable parameter such as RTL may necessitate large sample sizes. Alternatively, compensation mechanisms may be involved to attenuate the consequences of telomere attrition until a point when it becomes too energy-demanding compared to expected benefits (Monaghan, 2014; Angelier et al., 2018; Giraudeau et al., 2019). Lastly, post-breeding body condition could remain fairly conserved despite the multiple energy-demanding activities present at that time, meaning

females recuperate from mass loss at a similar rate regardless of individual quality to minimize potential negative effects on survival. This could also be explained by the selective disappearance of heavier individuals, but another study found a trend of higher body condition increasing survival in this species (Chapter 2). Consequently, senescence remains a solid hypothesis to explain the quadratic effect of age on body condition (Kirkwood, 2002). This indicates that individual heterogeneity may be dynamic during life. However, additional data on reproductive output and subsequent survival would be needed to deepen this aspect and evaluate which traits are mostly affected depending on the life-history strategy.

Recent studies in common terns (*Sterna hirundo;* Vedder et al., 2022) and Soay sheep (*Ovis aries*; Froy et al., 2021) have shown that the inherited component explained a large portion of an individual's telomere length. This genetically inherited portion of telomere length also covaried with lifespan (Froy et al., 2021; Vedder et al., 2022). As such, our correlation between RTL and body size could result from a common genetic basis. Given we have no information on pedigrees, our data does not allow us to test for the role of genetics in the observed relationships. Genetics probably explains the large residual variability we observe in all our analyses. However, our goal in this study was to explore the physiological implications of heterogeneity in telomere length. Controlling for the genetic component of all metrics would probably have improved the clarity of our results, but the relationships we highlight remain precious evidence of telomere length serving as a marker of individual quality. Moreover, in our system, early-life development has a stronger effect on body size (Gauthier et al., 2006), which supports our interpretation of this metric as a proxy of a cohort effect.

3.7 Conclusion

To conclude, we found that early-life development seems to impact telomere length throughout life, and we provide evidence conditions during growth could be one of the factors explaining heterogeneity in telomere length in adults. More generally, we suggest that differences in resource allocation at different points in life might explain part of the heterogeneity in individual quality, as well as its variation with age, as suggested in Vedder and Bouwhuis (2018). We found no direct link between telomere length and body condition of adults during brood rearing but further research including data on reproductive output and

survival is necessary. Finally, we show that age is essential when looking at individual heterogeneity in performance, which is coherent with previous studies (Moe et al., 2007; Elliott et al., 2015) and that heterogeneity in telomere length can be a useful tool to understand differences in strategies present within a population. It is possible that telomere length and age interact in the decrease in performance associated with senescence. In this respect, longitudinal studies would offer valuable opportunities to deepen our understanding of how individual performance varies over the lifespan and to uncover the diversity of life-history strategies within populations.

3.8 Aknowledgements and data availability

We are grateful to Gérald Picard, Marie-Christine Cadieux and Christian Marcotte for assistance in the field and Marie-Christine Cadieux for managing the database and the Bylot Island field research station. We thank Parks Canada for the access to Sirmilik National Park and support in the field. We are grateful to the Mittimatalik community for their welcome. We further thank all the Inuit, students and field assistants who contributed to goose banding and colony monitoring. We thank the Centre d'Études Biologique de Chizé (CEBC), especially Cécile Ribout, for their welcome and for providing training in laboratory techniques. We thank the Canada Excellence Research Chair on the Microbiome-Endocannabinoidome Axis in Metabolic Health (CERC-MEND) group for the access to their laboratory and their welcome in their team, in particular Giada Giordini for her input and support. We acknowledge funding from the Sentinelle North Research Chair from the Canada First Research Excellence Fund; the Fonds de Recherche du Québec—Nature et Technologies (FRQNT) [2020-NC- 271544]; the Natural Sciences and Engineering Research Council of Canada (NSERC); The Arctic Goose Joint Venture (Environment Canada); ArcticNet from the Networks of Centres of Excellence of Canada; Polar Continental Shelf Program. This research was conducted according to the relevant national and institutional regulations on animal welfare and was approved by the Comité de Protection des Animaux de l'Université Laval (CPAUL), the Canadian Bird Banding Office, the Mittimatalik Hunter and Trapper Organization and Parks Canada.

Data availability statement

All data and codes are available from the Borealis Repository

doi:10.5683/SP3/GA4I5G (Grentzmann et al., 2025)

Access is possible via this link until final publication:

https://borealisdata.ca/privateurl.xhtml?token=89681273-0804-458e-8bfa-5bdc19cd829b.

3.9. Supplementary Materials

- Annexe S3.1: PCA results for body condition index
- Annexe S3.2: Cohort effect on relative telomere length
- Annexe S3.3: Relationship between stress-induced CORT level and age

Chapitre 4. Variations in survival in relation to markers of different life stages in a long-lived bird

Montmagny, Avril 2021

En préparation:
Grentzmann I., Gauthier G., Angelier F., Yannic G., Silvestri C., Legagneux P. In prep. Variations in survival in relation to markers of different life stages in a long-lived bird

4.1 Résumé

Chez les espèces longévives, la survie est une composante clé du fitness qui est généralement sous forte sélection. Les variations de survie adulte, qu'elles soient liées à des facteurs environnementaux ou à des caractéristiques individuelles, peuvent avoir des impacts importants sur la dynamique de population. Comprendre ces variations est ainsi crucial pour éclairer les stratégies d'histoire de vie dans les populations sauvages. Dans cette étude, nous avons étudié les potentiels déterminants de la survie chez la grande oie des neiges (*Anser caerulescens atlanticus*), une espèce d'oiseaux migratrice et longévive, en utilisant des données démographiques, morphométriques et physiologiques riches. Avec des modèles de capture-marquage-recapture, nous avons évalué la relation entre la survie et trois traits : la taille corporelle, la condition corporelle et la longueur des télomères. La taille reflète les conditions de développement en début de vie, la condition les réserves endogènes présentes, et la longueur des télomères peut être utilisé comme un marqueur du vieillissement et de la qualité individuelle. Seulement la taille expliquait marginalement les variations de la survie annuelle globale, pointant vers des effets à long terme des conditions de développement sur le fitness adulte. Nous avons par ailleurs exploré le lien entre longueur des télomères et une mortalité cause-dépendante, comme cela a été reporté dans la littérature. Nous avons observé une covariation positive de la mortalité à la chasse avec la longueur des télomères chez les individus libérés alors qu'elle est négative chez les individus captifs. Nous suggérons que les réponses contrastées de la mortalité à la chasse en fonction de la longueur des télomères témoignent d'une interaction entre qualité individuelle et décisions reproductives. Dans l'ensemble, nos résultats soulignent la complexité des dynamiques de survie, qui résultent d'interactions entre des traits individuels intrinsèques et des facteurs stochastiques externes.

4.2 Abstract

In long-lived species, survival is a key fitness component that is typically under strong selection. Variations in adult survival, whether driven by environmental factors or individual characteristics, can have significant impacts on population dynamics. Understanding these variations is therefore crucial to elucidate life history strategies in wild populations. In this study, we investigated potential determinants of survival in the greater snow goose (*Anser caerulescens atlanticus*), a long-lived migratory bird, using extensive demographic, morphometric and physiological data. Using capture-mark-recapture models, we tested the relationships between survival and three traits: body size, body condition and telomere length. Body size reflects early-life developmental conditions, body condition current endogenous reserves and telomere length can serve as a marker of both ageing and individual quality. Only body size marginally explained variation in overall survival, suggesting long-lasting effects of developmental conditions on adult fitness. We further investigated the link between telomere length and cause-specific mortality, as it has been reported in the literature. We detected a positive covariation of hunting mortality with telomere length in individuals from the control group whereas the covariation was negative in captive individuals. We suggest that the contrasted relationships between hunting mortality and telomere length indicate an interaction between individual quality and reproductive decisions. Overall, our findings highlight the complexity of survival dynamics, which arise from intricate interplay between individual intrinsic traits and external stochastic factors.

4.3 Introduction

In wild animal populations, some individuals have better fitness outcomes than others, in terms of survival or reproduction (Hamel, Gaillard, et al., 2009; Péron et al., 2016; Hamel, Gaillard, & Yoccoz, 2018). This individual heterogeneity when relating to fitness components is called individual quality (Wilson & Nussey, 2010) and results from genetics, early-life conditions as well as cumulative life experiences (Wilson & Nussey, 2010; Bauch et al., 2022). For instance, environmental conditions during growth affects adult body size (Gomez-Mestre & Buchholz, 2006; Moore & Martin, 2019), which is closely associated with survival and longevity, both at the interspecific and intraspecific levels (Austad, 2010).

Models analyzing patterns of mortality have long incorporated individual heterogeneity, often characterized by contrasting biological states, but quantifying this variation remains a major challenge (McNamara & Houston, 1996; Monaghan & Haussmann, 2006). The number of traits that may vary with individual quality and age can be overwhelming, which has prompted the development of methods to synthesize heterogeneity across traits. For instance, multivariate analyses can generate composite variables that can subsequently be used as fixed effects in survival models (Hamel, Gaillard, & Yoccoz, 2018). Alternatively, individuals can be clustered using latent, unmeasured variables in mixture models (Péron et al., 2016; Gimenez, Cam, et al., 2018; Hamel, Gaillard, Douhard, et al., 2018). These approaches are powerful, but they cannot replace the direct investigation of the relationships between survival and individual physiological covariates. Examining such covariates may help decipher differences in intrinsic quality due to early-life developmental conditions, or arising from stochastic variation in life trajectories (Cohen et al., 2020).

For example, when encountering a stressful event, physiological and behavioral adjustments, called the stress response, occur to mitigate the immediate deleterious effects of the stressor (Wingfield et al., 1998). However, if the stressors are particularly intense, prolonged or cumulative, they can lead to negative consequences and affect individual quality (Schoenle et al., 2021), notably by decreasing survival (Romero & Wikelski, 2001; Breuner et al., 2008; Schoenle et al., 2018; Chapter 2). One labile trait that modulates the stress response and is a key determinant of reproduction and survival is body condition, often

considered as a proxy for energy reserves (Anderson, 2018). Individuals that are more efficient at acquiring and storing energy are likely to achieve higher fitness outputs, through both better reproduction and better survival (Bourg et al., 2019). Finally, as it is attributed to labile traits, individual quality can also vary with age (Wilson & Nussey, 2010; Hamel, Gaillard, Douhard, et al., 2018; Cohen et al., 2020). As such, the recognition that wild animals can experience senescence before death (Monaghan et al., 2008) has significantly refined our understanding of age-related variations in survival over the past two decades (Kirkwood, 2002; Roper et al., 2021). The relationship between age and survival is not always linear, and using a proxy of biological age – i.e. an organism state of ageing (Benjamin, 1947; Zhao et al., 2019) – may provide additional insights beyond chronological age. Such proxies offer a promising avenue to account for individual heterogeneity in survival (Monaghan, 2010; Barrett et al., 2013; Wilbourn et al., 2018).

Telomere length has been studied for more than a decade and is considered a valuable integrative biomarker of cumulative life experience (Haussmann et al., 2005; Haussmann & Marchetto, 2010; Monaghan, 2010). Telomeres are non-coding sequences at the extremities of chromosomes, mechanically shortened with each cell division, damaged by oxidative stress, and repaired by the enzyme telomerase (Blackburn, 2005). Telomere length was initially offered as a proxy for chronological age, and then for biological age, but correlation with age has proven to be highly variable among studies (Monaghan, 2010; Zhao et al., 2019; Remot et al., 2022). There is also evidence that telomere length is partly heritable (Asghar, Hasselquist, et al., 2015) and that telomere dynamics are sensitive to stressful events throughout life (Bateson, 2016; Angelier et al., 2018; Chatelain et al., 2020). Telomere length may reflect physiological state, with demonstrated links to mortality, particularly in mammals and birds (Wilbourn et al., 2018; Eastwood et al., 2019). It has also been found to correlate with other individual traits such as those determined by environmental conditions experienced during growth like body size (Pepke et al., 2022; Chapter 3). As such, telomere length and attrition rate are now viewed as indicators of an individual's cumulative life experience, integrating the effects of age, early-life heterogeneity, and stochastic environmental events occurring during adulthood (Marasco et al., 2022). Although the number of studies linking telomere length and survival remains limited, recent meta-analyses indicate that longer telomeres in both early life and adulthood are generally associated with

higher return rates, longer remaining lifespan and lower mortality (Wilbourn et al., 2018; Eastwood et al., 2019, 2023).

Most studies on wild animals linking telomere length to survival have been conducted in intensively monitored populations (i.e., colonial species), where the fate of individuals is precisely known until death (Wilbourn et al., 2018; Chik et al., 2024), or have focused on the survival up to fledging (Eastwood et al., 2019; Wood & Young, 2019; Kärkkäinen et al., 2022; Pepke, Kvalnes, Rønning, et al., 2022). However, such studies are typically limited to a small number of species, individuals or life history stages. In most wild populations, detection of marked individuals is incomplete and, in birds in particular, age is often unknown or imprecise (Zhao et al., 2019). In such cases, capture-mark-recapture (CMR) techniques remain the method of choice to study survival, especially when exploring senescence in wild populations (Gaillard et al., 2004). However, CMR analyses require extensive, long-term datasets. To our knowledge, only one study has investigated the relationship between telomere length and survival using CMR in black-browed albatrosses (*Thalassarche melanophrys;* Angelier et al., 2019). This study found no evidence for a significant relationship between telomere length and survival, but it was based on a relatively small sample size.

In this study, we investigated three individual traits, body size, body condition and telomere length, reflecting different aspects of individual heterogeneity and their link to survival in the greater snow goose (*Anser caerulescens atlantica*). Their life history strategy is on the slow side of the slow-fast continuum, meaning they tend to skip reproduction to optimize survival if their physiological condition and/or environmental conditions are not favourable (Reed, 2004; Souchay et al., 2014). This is a typical strategy in long-lived species that have a high sensitivity to variations in adult survival, which makes the study of survival variability essential (Stearns, 1992; Gaillard & Yoccoz, 2003). We used body condition as a proxy for current physiological state, which has been shown to vary with age in this species (Chapter 3). Body size was used as a proxy for early life conditions (Gauthier et al., 2006; Doiron et al., 2015), and telomere length as an integrative marker of cumulative life experience. Telomere length has previously been shown to be highly variable but to correlate with both age and body size in this population (Chapter 3). Together, these three variables

offer complementary insights into traits related to individual quality and stochasticity across different life stages: past (body size), present (body condition), and cumulative (telomere length). We tested whether these covariates could explain variations in survival probabilities using capture-mark-recapture data. Given that previous studies have assessed the impact of telomere length on lifespan through its association with mortality risk (Vedder et al., 2017; Marasco et al., 2021; Pepke, Kvalnes, Rønning, et al., 2022; McCollum et al., 2024), we also examined the relationship between telomere length and a major source of mortality in geese, the probability of being harvested by hunters. The greater snow goose provides a valuable study model for such investigations due to its long lifespan and the availability of long-term, individual-based data on a large number of individuals (Gauthier et al., 2001; LeTourneux et al., 2024). We used data from a previous experiment on a wild, hunted greater snow goose population, where some groups were kept in captivity with or without access to food (Legagneux et al., 2012; Grandmont et al., 2023). This presented a rare occasion to look at the interaction between telomere length, cause-specific mortality and stressors. Following previous studies, including on this species, we expected second year survival to vary positively with telomere length, body condition and body size (Van De Pol et al., 2006:200; Briga et al., 2017; Wilbourn et al., 2018; Chapter 2). We also expected telomere length to interact with experimental group in explaining survival in the first year after the experiment, as it is known to modulate responses to stress events (Chatelain et al., 2020).

4.4 Materials and methods

4.4.1 Study species and study site

The greater snow goose is a long distant migratory species wintering along the Atlantic coast of the United States and breeding in the Canadian Arctic in Nunavut, with stopovers in Southern Québec (Gauthier et al., 1992; Lefebvre et al., 2017). During these stopovers, but particularly in spring, geese fatten to accumulate reserves for migration and reproduction (Gauthier et al., 1992; Bêty et al., 2003). The snow goose is a species hunted year-round including in spring because it is considered overabundant (Lefebvre et al., 2017). Harvest during both autumn and spring in Québec and during winter in the United States provides substantial data through band recoveries reported by hunters. Lifespan is on average 10 years with some individuals known to live up to 24 years (Chapter 3).

4.4.2 Capture, measurements and blood sampling

Captures occurred in April-May 2009 at Île-aux-Oies (Québec; 47°00′N 70°33′W) in the St. Lawrence estuary (Legagneux et al., 2012; Chapter 2), a key spring staging area. Geese were captured with baited cannon-nets; the number of adults per capture ranged from 40 to 105, for a total of 22 captures events. All adults were banded, sexed by cloacal examination, and females were measured (tarsus, culmen and head lengths to the nearest 0.1 mm), weighed (to the nearest gram) and blood samples were collected (see details below). Females were marked with neck collars to facilitate re-observation at a distance after release (LeTourneux et al., 2022). Such collars are known to affect survival (LeTourneux et al., 2022), but since all females were equipped the same way, investigating individual covariate among these individuals remains valid.

Samples and data used in this study come from adult females (N=570) that were included in a carry-over experiment (Legagneux et al., 2012; Grandmont et al., 2023; Chapter 2). Each capture group was assigned to a different treatment in a predefined order. The control group was composed of individuals released immediately after banding and treatment groups were composed of individuals subject to two different manipulations: kept in captivity for 2, 3 or 4 days (captivity duration treatment), and either provided with food (crushed corn) or not (food treatment). Marked individuals were reobserved by our team and birdwatchers along the St. Lawrence River every spring from 2009 to 2022, with a relatively constant effort. We also included dead recoveries reported by hunters both from Canada and the USA in the reencounters (see below).

We collected 570 blood samples of up to 1 mL from the tarsal vein on females during banding operations using 23G needles. Blood samples were kept on ice for up to four hours, then centrifuged at 8000 G for 8 min to separate the plasma from the red blood cells. Samples were frozen separately and stored at -20°C until lab analyses.

4.4.3 Blood analyses for telomere length quantification

Relative telomere length (RTL) was quantified on 570 samples using quantitative PCR (qPCR; Cawthon, 2002) on the total genomic DNA, a technique validated in birds (Criscuolo et al., 2009), including this species (see detailed protocol in Chapter 3). Briefly,

DNA was extracted from red blood cells using the NucleoSpin 96 Blood Kit (Macherey-Nagel, 2023), and quality was confirmed via UV absorbance spectroscopy (260/280 absorbance ratio being between 1.8–2.0; and 260/230 absorbance ratio being between 2.0–2.2). Quality of the samples was also tested by electrophoresis on a random subset of samples (Figure S4.1 in Appendix S4.1). The control single-copy gene (Recombination activating gene 1 - RAG1) was detected with the following primers: RAG1-F (5'-TGTACGGGAAGTGGAAGGG-3') and RAG1-R (5'-GGTGATGGAGTGGAAGACC-3'). The telomere primers used were: Tel1b-F (5′-CGGTTTGTTTGGGTTTGGGTTTGGGTTTGGGTTTGGGTT-3′) and Tel 2b-R (5′-GGCTTGCCTTACCCTTACCCTTACCCTTACCCTTACCCT-3′). All reactions were run in triplicates, and an inter-plate calibration was then executed by the CFX Maestro software (Biorad, 2017). Amplification efficiency was estimated from a standard curve on each plate (see Table S4.1 in Appendix S4.1 for plate-specific efficiencies and R^2 values before calibration). Overall efficiency used for the calculations was corrected for the previously mentioned inter-plate calibration: 94.3% for telomere amplification and 97.5% for RAG1. RTL was calculated using efficiency-corrected Cq ratios relative to a common reference sample on each plate. RTL is therefore a ratio of quantification cycles of telomere in the sample compared to the reference gene RAG1 for the same sample, relative to a reference sample present on each plate, hence the name "relative telomere length". Details on RTL calculations are all available in Chapter 3.

4.4.4 Statistical analyses

Body condition estimate

To obtain an index of endogenous reserves independent of body size, we corrected female body mass by skeletal measurements. We used data from 2 511 adult females captured at the same site and with the same method between 2006 and 2009 and conducted a principal component analysis (PCA) on tarsus, culmen and head lengths. The first principal component (PC1), which explained 73.8% of the variance (Figure S4.2 in Appendix S4.2), was used as a predictor in a linear regression with body mass. Residuals from this model were added to the mean mass to obtain a size-corrected mass. Because condition can change over time during staging, we further corrected for the capture date with a second regression between

this index against the date of capture (in julian days) and added the residuals of this model to the mean body mass (see LeTourneux *et al.* (2021) for more details). This body condition index reflects individual fat reserves independently of structural size and capture date (Féret et al., 2003).

We tested for relationships between RTL, body condition and body size using linear models. The relationship between these variables were not affected by the treatment groups because all sampling and measurements occurred before the experiment. Normality and homoscedasticity of residuals were confirmed graphically. For all analyses, we report the parameter estimates with 95% confidence intervals (95% CI) and conclude to a significant result when the 95% CI do not overlap zero.

Capture-mark-recapture analysis

To investigate the influence of RTL, body condition and body size on survival, we used a multi-event, capture-mark-recapture model (Pradel, 2005) combining live reobservations with dead recoveries (bands reported by hunters) to analyse survival of 559 collared females. Marking occurred in a single year (spring 2009), but reencounters extended over a 13-year period. Extending the period to collect reencounters increased the precision of the survival estimates in the first two year following the experiment, which were the year of primary interest to examine covariate effects. On each occasion, an individual could be observed alive (coded 1), recovered (2) or not encountered (0). Individuals recovered dead between spring t and $t+1$ were coded dead at spring $t+1$ (Gauthier and Lebreton, 2008). The model is a simplification of LeTourneux et al. (2022) and could estimate survival (S), live-encounter (p) and recovery (Seber's r) probabilities. We estimated survival in the first and second year separately because the experiment that took place in 2009. We thus tested the influence of RTL, body condition and body size on survival in combination with the experimental treatments (food restriction) in the first year and without them in the second year. Only the food treatment was included as a grouping variable in the survival models because it was found to affect survival unlike captivity duration (Grandmont et al., 2023; Chapter 2). Continuous variables like RTL and body condition were standardised. Survival for all subsequent years was considered constant in the model, as in Chapter 2. We conducted model selection sequentially, first on recovery, then on reobservation and finally on survival

probabilities using Akaike's Information Criterion corrected for overdispersion (QAICc; Lebreton et al., 1992; Burnham & Anderson, 2004). Models with a difference in QAICc of 2 or less from the best model were considered competitive. The capture-recapture analysis was conducted with program E-SURGE V.2.2.3 (Choquet, Rouan, et al., 2009). Prior to conducting this analysis, we tested the goodness-of-fit of the general model with the program U-CARE V2.3.5 (Choquet, Lebreton, et al., 2009). We found a χ^2 value of 80.54 for 67 degrees of freedom, which yielded an overdispersion coefficient ($\hat{c}$) of 1.20. We used this value in the capture-recapture analysis, which explains our use of QAICc rather than AICc (corrected Akaike's Information Criterion; Burnham & Anderson, 2004).

We looked at the relationship between RTL and the probability of being harvested using the direct band recovery rate. We considered all individuals recovered between May 2009 and May 2010 as recovered in the first year (1) and individuals recovered or reobserved in any of the following 13 years as not recovered in the first year (0). All other individuals for which we had no information were excluded. We used a binomial model relating direct recovery probability with RTL and the experimental treatments and relied on AICc for model selection.

4.5 Results

Body size and RTL were positively related (β = 1.02 [0.16, 1.88]; Figure 4.1A) whereas body condition was not associated with RTL (β = -8.24 [-40.48, 24.01]; Figure 4.1B). By design, body size and condition were uncorrelated.

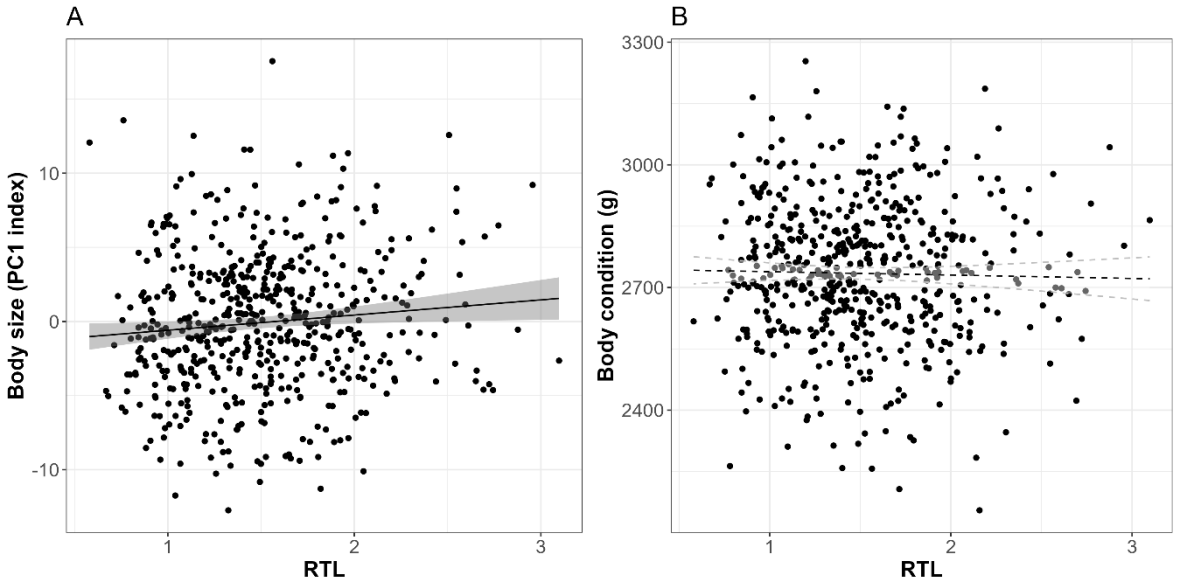

Figure 4.1 : Relationship between body size (A) or body condition (B) and relative telomere length (RTL). The black or dashed line (respectively significant or non-significant results) shows model prediction, and the grey ribbon represents the 95% confidence interval estimated by bootstrapping.

Table 4.1 : Results of model selection for survival in the first year (A) and second year (B) after capture of greater snow geese marked in spring 2009, in relation to relative telomere length (RTL), body size (SIZE), body condition (COND) and experimental treatment (food addition; TRT, first year only). Survival in subsequent years (years 3 to 13) was considered constant. For all models, recapture probabilities included a time effect, whereas recovery probabilities were held constant over time. K: number of parameters, ΔQAICc: difference in QAICc between the current and the top-ranked model (N = 559). See Table S4.2 in Appendix S4.3 for full model selection results.

	Model	K	Deviance	ΔQAICc
A) 1st year survival				
	Null	14	2850.26	0.00
	TRT	17	2843.99	1.10
	Cond	16	2847.30	1.73
	RTL	16	2847.44	1.83
	Size	16	2847.58	1.95
	TRT+COND	18	2843.838	3.05
	TRT+SIZE	18	2843.8934	3.10
	TRT*SIZE	20	2839.77	3.91
	TRT*COND	20	2842.20	5.88
B) 2nd year survival				
	Null	14	2850.26	0.00
	Size	17	2843.01	0.31
	Cond	17	2845.20	2.08
	RTL	17	2845.39	2.24

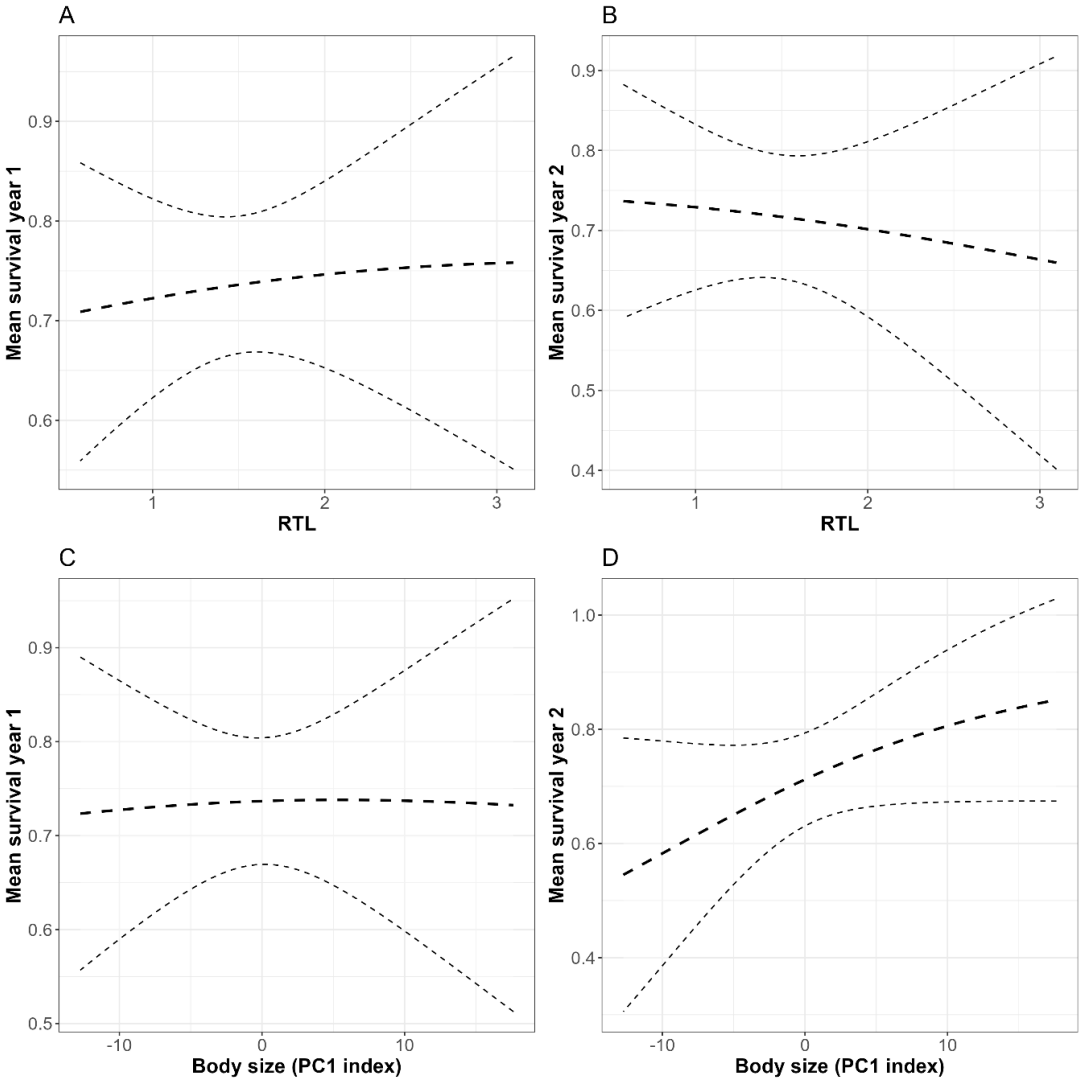

Figure 4.2 : Relationship between annual survival and relative telomere length (RTL; A, B) or body size (C, D) in the first (A, C) second (B, D) year after capture. Model prediction and 95% confidence intervals computed by bootstrapping are represented by dashed lines because results were not significant (N = 559).

The null model was the best-supported model to explain variations in survival during the first year after capture although models including RTL, body size or body condition were competitive (Table 4.1). However, none of the estimates for these covariates were significant (β_{Cond} = -0.08 [-0.42, 0.25]; β_{RTL} = 0.06 [-0.25, 0.37]; β_{size} = 0.02 [-0.27, 0.32]; Figure 4.2A and C respectively). Regarding second year survival, both the null model and the model including body size as a covariate were competitive. Survival tended to increase with increasing body size (β_{size} = 0.25 [-0.10, 0.60]; Figure 4.2D). RTL did not explain variation in second year survival (β_{RTL} =-0.06 [-0.40, 0.28]; Figure 4.2B).

Table 0.1: Model selection for the influence of relative telomere length (RTL) and experimental treatment (food availability; TRT) on the probability of being harvested in the first year (N = 206).

Explanatory variables	Number of parameters	Log-likelihood	ΔAIC
RTL*TRT	6	-97.09	0.00
Null	1	-102.81	1.03
RTL	2	-102.80	3.06
RTL+TRT	4	-102.08	5.75

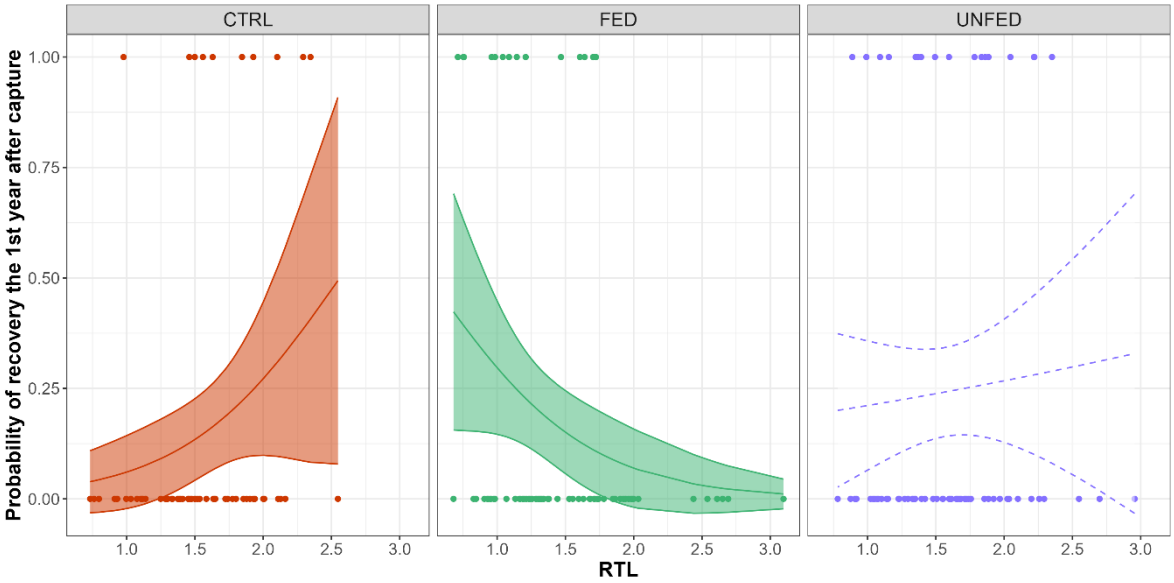

Figure 4.3 : Relationship between relative telomere length (RTL) and the probability of being harvested during the first year after capture shown separately for each treatment group. Predicted values and 95% confidence intervals were obtained from the preferred model in Table 4.2 and are represented by solid lines and shading when significant and dashed lines when not (N = 206).

The model linking probability of being recovered to RTL in interaction with treatment group was preferred over the null model, though the AICc difference is less than 2.0 (Table 4.2). In the control group, RTL had a positive influence on the probability of being harvested ($\beta_{Control}$ = 1.76 [0.04, 3.70]; Figure 4.3), whereas RTL had a negative influence in fed individuals (β_{Fed} = -1.73 [-3.55, -0.28]; Figure 4.3). No relationship was found in the unfed group (β_{Unfed} = 0.31 [-0.86, 1.46]; Figure 4.3).

4.6 Discussion

Contrary to our expectations, survival was not related to telomere length or body condition. Only body size tended to have a positive impact on survival in the second year

after capture. In another study on the same population, we found evidence for a slight positive influence of body condition on survival, especially modulating the effects of stressful events (Chapter 2). The analyzed samples differed slightly between the two studies because of plasma vs. red blood cells availability. Since the effect of the experimental treatment is weaker in this study than in the previous one, the mediating effect of body condition might be diminished as well. The absence of an influence of body condition on survival in the second year after capture is consistent with Chapter 2 and with the fact that body condition varies between years (Bêty et al., 2003).

4.6.1 Evidence of long-lasting effects of developmental conditions on adult survival

The marginally positive relationship between second-year survival and body size points to a possible long-lasting effect of developmental conditions on adult fitness, as has already been found in the literature (Nussey et al., 2007; Hamel, Gaillard, et al., 2009; Cam & Aubry, 2011). In our species, the greater snow goose, individuals reach their adult size within the first three months of life (Lesage & Gauthier, 1997). Gosling body size near fledging is linked to environmental conditions experienced during growth (Gauthier et al., 2006; Doiron et al., 2015). Post-fledging and pre-reproduction survival is also related to body size in other goose species (Jeugd & Larsson, 1998; Richman et al., 2015). Finally, the link between growth conditions and adult survival has been shown in several other bird species such as the oystercatcher (*Haematopus ostralegus*; Van De Pol et al., 2006) and the zebra finch (*Taeniopygia guttata;* Briga et al., 2017), as well as in ungulates (Douhard et al., 2014). The complete chain of events starting with unfavourable environmental conditions leading to reduced growth and ultimately to negative consequences on adult survival has been demonstrated in Japanese quail (*Coturnix japonica*; Vedder and Beccardi, 2025).

This is further supported by the positive relationship we found between body size and telomere length, a pattern that has already been established in this species using different samples (Chapter 3), although the relationship in the current chapter explains a very small proportion of variance (1%). This relationship is contrary to the hypothesis of a trade-off between growth and telomere length, if we consider telomere length as a marker of an underlying trade-off between immediate survival and long-term survival or lifespan (Caprioli et al., 2013; Pepke, Kvalnes, Rønning, et al., 2022). However, other studies on the black-

browed albatross (*Thalassarche melanophris;* Angelier et al., 2019) and other passerine species (Angelier et al., 2015; Parolini et al., 2017) have also found a positive association between telomere length and body size. Without excluding that this relationship could partially be explained by genetic factors (Jensen et al., 2003; Vedder et al., 2022; Luo et al., 2025), we hypothesize that body size could be a relevant indicator of good developmental conditions in species with finite growth, as in Chapter 3.

4.6.2 Cause-specific mortality is linked to telomere length, but not overall survival

Though we did not detect any association between survival and telomere length, we did find that telomere length was related to a specific source of mortality, namely the probability of being killed by hunters. It is important to note that individuals in our study were subjected to experimental treatments designed for different research objectives, which were not directly related to the questions addressed in this study (Legagneux et al., 2012; Grandmont et al., 2023; Chapter 2). Here, we observed contrasting relationships between telomere length and the probability of being harvested according to the food treatment group. Telomere length can either be interpreted as a proxy for age or individual quality (Monaghan & Haussmann, 2006), and was negatively related with age in this species (Chapter 3; R^2=8%). The age of the individuals in this study was unknown, so we could not test for an effect of age. However, if age were the main driver of the relationships despite the low R^2 found in the previous chapter, individuals with longer telomeres, being younger and less experienced, would be expected to be more harvested in all groups, which is not the case.

We offer an alternative interpretation of these results by considering telomere length as a marker of individual quality. In the captive unfed group, mortality was unrelated to telomere length, which may be explained by this group's pronounced decline in body condition (Legagneux et al., 2012) and its high overall mortality (Chapter 2). Under such conditions, all individuals may have become uniformly vulnerable to hunting. In contrast, individuals in the captive fed group exhibited overall survival rates comparable to the control group (Chapter 2), but their reproductive output in 2009 was markedly reduced (Legagneux et al., 2012; Grandmont et al., 2023). This situation offers a unique opportunity to examine the relationship between telomere length and mortality risk under contrasting levels of reproduction. In the fed group, where reproduction was lower, individuals with longer

telomeres were less likely to be harvested, possibly because they were higher quality individuals. Telomere has been shown to reflect individual quality (Angelier et al., 2019), and has been linked to risk-taking behaviour. For example, experimental studies have shown that telomere length correlates with risk-taking behavior in species such as the European starling (*Sturnus vulgaris;* Bateson et al., 2015; Andrews et al., 2018) and the zebra finch (*Taeniopygia guttata;* Marasco et al., 2021). Conversely, in the control group, where reproductive success was higher, longer telomeres were associated with an increased probability of being harvested. If telomere length reflects higher individual quality, these individuals would be more likely to breed successfully. However, the presence of naïve young has been shown to increase hunting mortality risk for attending adults, particularly females (Giroux et al., 1986; Calvert et al., 2005), which could explain the observed pattern. Our results do not allow us to conclude on the role of reproduction in the relationship between telomere length and hunting mortality, but they open an interesting research avenue. These findings highlight the importance of considering interactions between intrinsic and extrinsic sources of mortality when examining survival variations (Koopman et al., 2015).

We found a significant association between telomere length and a specific cause of mortality, i.e. hunting mortality, but not with overall survival, similarly to the only other study examining overall survival with CMR did not find a significant relationship either (Angelier et al., 2019). A possible reason is that CMR only estimates overall survival, which combines various sources of mortality potentially not equally affected by telomere length. This could be the case for hunting mortality in our system. Alternatively, this could be linked to the methodology used: in studies reporting a positive association between survival and telomere length, survival was estimated using known age individuals with a perfect encounter probability (reviewed in Wilbourn *et al.*, 2018; Eastwood *et al.*, 2023). In contrast, in CMR analyses imperfect detection is present and must be estimated by the model. This could make the telomere length signal on survival more difficult to detect. Finally, covariates reflecting long-lasting consequences of developmental conditions, such as body size, may provide a stronger signal, which can be easier to detect even when we account for imperfect detection of individuals. Looking at cause-specific mortality might thus be a better route to understand the links between telomere length and individual fitness.

4.6.3 The importance of age in establishing links between survival and telomere length

Chronological age may also be an important factor to account for when examining links between telomere length and survival. In this study, age was unknown, which could partly explain the absence of relationship between telomere length and survival. A majority of studies addressing this question relies on known-age individuals (reviewed in Wilbourn *et al.*, 2018; Eastwood *et al.*, 2023; and see Chik *et al.*, 2024; McCollum *et al.*, 2024). Telomeres are correlated to age in many species, but large variability is always associated to this relationship (Remot et al. 2022; Chapter 3). While age did not interact with telomere length in the survival analyses of most studies (Bize et al., 2009; Foote et al., 2011; Heidinger et al., 2012; Asghar, Hasselquist, et al., 2015; Young et al., 2021; Chik et al., 2024), some have reported that telomere attrition rate, rather than telomere length, explained variations in survival (Salomons et al., 2009; Tricola et al., 2018). In addition, meta-analyses provided evidence that age or age group influenced the relationship between telomere length and survival (Bichet et al., 2020; Marasco et al., 2021; Eastwood et al., 2023). Finally, a strong publication bias in favor of confirmative results has been highlighted by two meta-analyses (Wilbourn et al., 2018; Eastwood et al., 2023). Publishing more non-confirmative results might help disentangle the respective role of telomere length and age in shaping survival patterns, as these two variables may play distinct roles (Bichet et al., 2020; Marasco et al., 2021; Eastwood et al., 2023).

4.7 Conclusion

By combining individual-level data on physiology, morphology, and long-term reobservations from over 500 greater snow geese, we found a slight positive relationship between telomere length and body size, and between body size and survival. However, we did not detect a direct effect of telomere length or body condition on survival, though body condition did relate to survival in another study on the same species (Chapter 2). These results suggest that body size may serve as a key link between early-life conditions and adult fitness (Hamel, Gaillard, et al., 2009; Cam & Aubry, 2011), whereas body condition might be a secondary determinant, mediating the response to stress events (Chapter 2). Although telomere length did not predict overall survival, it was associated with the probability of

being harvested, in interaction with captivity. We suggest that reproductive success might be a missing link to explain the contrasting responses we observe in our study, since it may indirectly increase hunting vulnerability in this species (Giroux et al., 1986; Calvert et al., 2005). Our findings show that the relationships between telomere length and mortality might be complex and context dependent. They also highlight the interplay among life-history traits that may involve compensatory mechanisms, a pattern commonly observed in long-lived species (Gaillard & Yoccoz, 2003).

4.8 Aknowledgements and data availability

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Data availability statement

Data will be available on the Borealis repository upon publication.

4.9 Supplementary Materials

- Annexe S4.1 : Complementary information about relative telomere length extractions
- Annexe S4.2: PCA results for body condition index
- Annexe S4.3: Complete model selection for CMR survival models

Conclusion

L'intégration des variations individuelles dans les modèles démographiques et écologiques a pris de l'essor pendant la dernière décennie. Cette thèse a approché ces questions en tentant d'établir des liens explicites entre certains traits individuels physiologiques ou morphométriques et la survie. Nous avons étudié la survie car c'est le paramètre démographique le plus élastique de la dynamique de population de la grande oie des neiges. Nous avons émis l'hypothèse que les variations de survie suivraient les variations de qualité individuelle, c'est-à-dire la part de l'hétérogénéité individuelle corrélée positivement au fitness. Nous nous sommes donc appuyés sur la littérature pour sélectionner des variables physiologiques ou morphométriques qui pourraient faire varier la qualité individuelle : l'âge, la réponse au stress, la condition, la taille et la longueur des télomères.

De manière générale, nous avons été capables d'observer des variations de survie adulte, malgré la stabilité de ce paramètre démographique dans le temps et la forte pression de chasse, à l'échelle de la population. Dans le premier chapitre, nous trouvons que la survie varie avec l'âge selon le patron de sénescence attendu pour les oiseaux longévifs (Anderson & Apanius, 2003; Péron et al., 2010). La pression de chasse n'a pas d'impact sur ce patron de sénescence, qui s'amorce autour de 11 ans. Par ailleurs, la survie est fortement affectée par l'accumulation de stresseurs et peut varier avec l'intensité de la réponse hormonale au stress, la condition corporelle (Chapitre 2), ou la taille corporelle (Chapitre 4). Pour approfondir notre compréhension de la qualité individuelle et son lien étroit avec l'âge, nous avons exploré les corrélations entre nos principaux traits, la longueur des télomères et l'âge, dans le Chapitre 3. Nous trouvons que la longueur des télomères varie principalement avec l'âge et la taille corporelle, ainsi que marginalement avec les niveaux de corticostérone sous stress. La condition corporelle varie elle aussi avec l'âge en suivant un patron quadratique avec une diminution de la condition pour les individus les plus âgés. Trois grands déterminants communs à l'hétérogénéité de survie et de qualité individuelle chez la grande oie des neiges se dégagent : les conditions de développement, l'effet de la longueur des télomères et de la condition corporelle sur la vulnérabilité à la chasse, et l'âge.

Présence de sénescence actuarielle

Tout au long de cette thèse, nous avons montré que des traits tels que la condition corporelle, la longueur des télomères et la survie varient avec l'âge, démontrant la présence de sénescence chez cette espèce longévive malgré une forte pression de chasse. Ce résultat est important car les études de la sénescence sont principalement effectuées sur des oiseaux longévifs non sujets à la chasse (Bouwhuis & Vedder, 2017), et les oiseaux chassés sont rarement étudiés pour la sénescence (Grzegorczyk et al., 2024). Au contraire, les études sur les mammifères regardent plus souvent des espèces modèles chassées (Gaillard et al., 2017). La chasse au trophée est par ailleurs un enjeu évolutif chez les mammifères qu'il y a moins chez les oiseaux (Festa-Bianchet & Mysterud, 2018; Grzegorczyk et al., 2022). Or, la chasse est souvent utilisée comme outil de conservation (Grzegorczyk et al., 2024). Cependant, l'influence de la chasse sur la sénescence reste peu étudiée ou discutée (Gaillard et al., 2017, mais voir Gamelon et al., 2020). L'absence d'interaction entre la sénescence et la pression de chasse que nous trouvons ici (Chapitre 1) est un résultat intéressant et innovant. La vulnérabilité à la chasse semble donc dépendre d'autres variables individuelles présentant une forte hétérogénéité.

Les conditions de développement

L'influence des conditions de croissance sur le fitness des individus à l'âge adulte a été démontré dans de nombreux contextes et largement discuté dans la littérature (Nussey et al., 2007; Hamel, Gaillard, et al., 2009; Cam & Aubry, 2011; Vedder & Bouwhuis, 2018). La corrélation que nous trouvons entre taille corporelle et longueur des télomères (Chapitres 3 et 4) et de la taille avec la survie (Chapitre 4) semble appuyer cette hypothèse. En effet, la taille corporelle reflète en grande partie les conditions environnementales lors de la croissance (Doiron et al., 2015).

Cet effet des conditions de croissance est souvent inclus dans le '*silver spoon effect*' (effet de la cuillère en argent), qui traduit la composante fixe de l'hétérogénéité présentée en introduction (Figure 0.1). Le Chapitre 3 de cette thèse montre que les conséquences des conditions en début de vie sur la longueur des télomères (Chatelain et al., 2020; Pepke, Kvalnes, Ranke, et al., 2022) perdurent avec l'âge. Grâce au suivi systématique de la colonie

et de son écosystème depuis 1990, nous pourrions dégager les facteurs importants régissant la croissance des jeunes. Il serait ensuite possible de tester l'influence de ces conditions en début de vie sur la longueur des télomères chez les jeunes et les adultes. Par exemple, un résultat préliminaire montre que la densité de nids à l'année de naissance est négativement corrélée à la longueur des télomères, chez les jeunes et les adultes ($\beta_{densité}$ = -0.18 [-0.31; -0.06]; Figure 5.1).

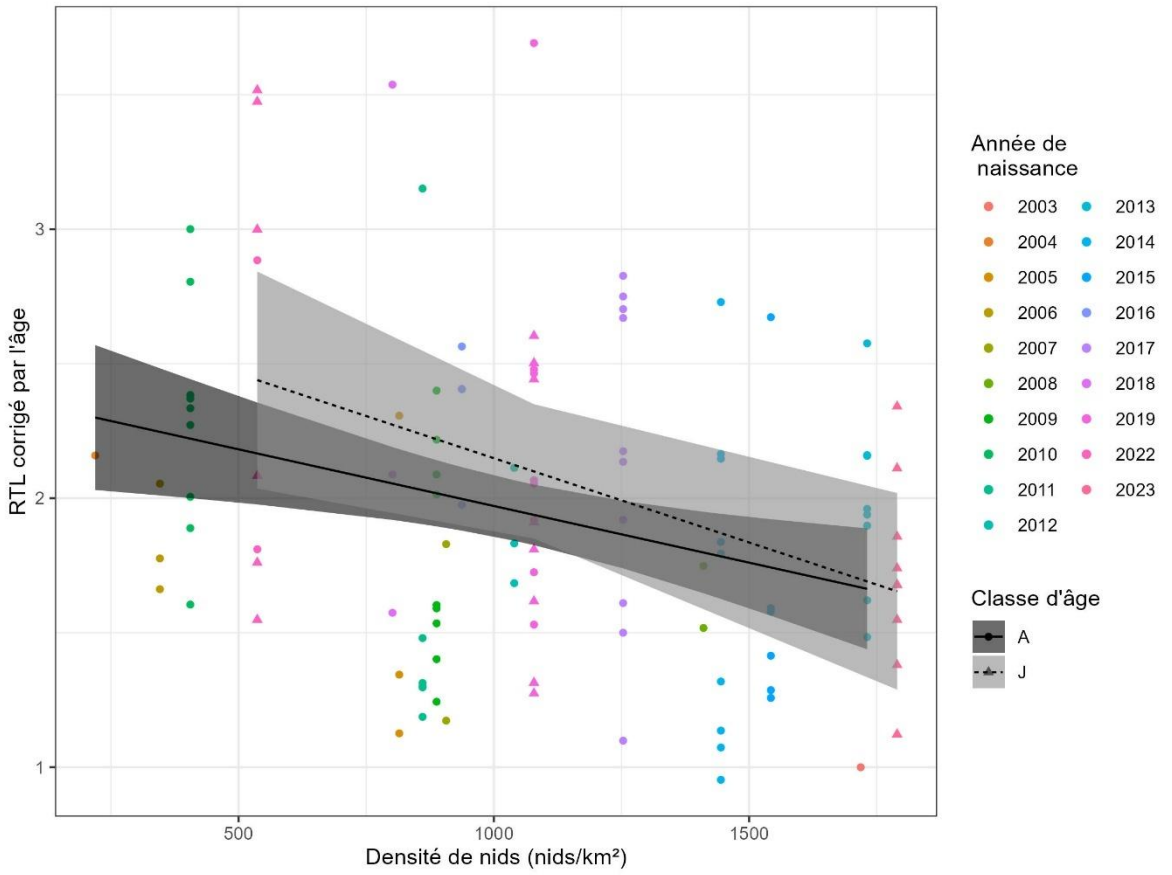

Figure 5.1: Relation entre la longueur relative des télomères (RTL) corrigée par l'âge et la densité de nids au km2 au cœur de la colonie de l'Île Bylot. Les données de densité de nids proviennent du suivi annuel de la nidification du cœur de la colonie. Les longueurs de télomères ont été extraites à partir de prises de sang collectées lors du baguage (voir Chapitre 3). Les relations pour les jeunes (ligne pointillée et gris clair, J) et les adultes (ligne continue et gris foncé, A) ont été estimées séparément. Les échantillons sur les jeunes ne couvrent que 3 valeurs de densité, celles pour les adultes couvrent 18 valeurs de densité. Le modèle pour les adultes comprend l'âge en effet additif pour contrôler pour la relation âge-RTL (voir Chapitre 3). L'année de naissance est incluse en effet aléatoire dans les deux modèles. Nous avons représenté les estimations et intervalles de confiance à 95%.

Dans notre étude, aucune corrélation directe n'a été trouvée entre la survie globale et la longueur des télomères, ce qui a également été rapporté dans quelques études sur des oiseaux (Beaulieu et al., 2011; Ouyang et al., 2016), notamment dans une autre étude de capture-marquage-recapture (Angelier et al., 2019). Le lien entre longueur des télomères et mortalité a plutôt été établi dans des suivis où le destin des individus est connu (Wilbourn et al., 2018), notamment avec des sources de mortalité spécifiques (Asghar, Hasselquist, et al., 2015) et à court terme (Bize et al., 2009; Barrett et al., 2013). La longueur des télomères est également corrélée à la taille (Chapitres 3 et 4), et de nombreuses études montrent le lien de la longueur des télomères avec les conditions en début de vie (Asghar, Bensch, et al., 2015; Angelier et al., 2018; Giraudeau et al., 2019; Pepke, Kvalnes, Ranke, et al., 2022). Le lien entre les conditions de développement et la survie globale passerait ainsi par la taille, similairement au lien expérimentalement démontré chez la caille japonaise (*Coturnix japonica*) entre effets maternels, croissance, taille et survie adultes (Vedder & Beccardi, 2025). Les conséquences à long terme de conditions développementales défavorables peuvent ensuite affecter la physiologie des individus (Vedder & Beccardi, 2025). Ces baisses de performance physiologiques pourraient ultimement rendre ces individus plus vulnérables à la chasse (Chapitre 2), ce qui est appuyé par le lien entre longueur des télomères et prise de risque (Bateson et al., 2015; Andrews et al., 2018).

La vulnérabilité à la chasse

Les résultats des Chapitres 2 et 4 suggèrent que la vulnérabilité à la chasse est un mécanisme important des variations de mortalité individuelles, mais que l'impact sur la survie globale peut être modulé par la qualité individuelle. En effet, malgré une pression de chasse importante (Chapitre 1) ou une vulnérabilité à la chasse accrue chez certains individus (Chapitre 4), la survie globale ne varie pas nécessairement. Cela semble indiquer que les individus tués à la chasse seraient peut-être morts d'une autre cause la même année (Sandercock et al., 2011; LeTourneux et al., 2024). Cependant, des individus expérimentalement stressés et amaigris sont plus vulnérables à la chasse et cette différence se répercute sur la survie globale (Chapitre 2). Ainsi, l'accumulation de stresseurs aléatoires augmente assez la vulnérabilité à la chasse pour que la mortalité devienne additive. Cela démontre que la mortalité intrinsèque et extrinsèque interagissent différemment selon les

circonstances (Koopman et al., 2015). En effet, selon les caractéristiques intrinsèques telles que la condition corporelle, l'intensité de réponse hormonale au stress ou la longueur des télomères, les individus vont être plus ou moins vulnérables à la chasse (Morez et al., 2000; Guillemain et al., 2007; Fowler et al., 2020) et la mortalité à la chasse se répercutera différemment sur la survie annuelle.

Les résultats du Chapitre 2 montrent que les effets d'une accumulation de stress, provoquant entre autres une diminution de condition corporelle, peuvent être reportés sur plusieurs mois. Les individus concernés, ayant subi une perte de masse corporelle importante au printemps, présentaient une réponse au stress saturée et ces individus étaient plus vulnérables à la chasse. Ainsi, nos résultats montrent que la condition corporelle n'est pas suffisante pour expliquer la variation de vulnérabilité à la chasse. L'interaction avec un stress chronique important (entrainant une saturation de la cascade de réponse) serait assez forte pour entrainer un déséquilibre allostatique et ainsi augmenter une prise de risque en s'alimentant dans les champs, même si le risque de mortalité à la chasse y est élevé (LeTourneux et al., 2023). En considérant la baisse de reproduction observée chez les individus captifs (Legagneux et al., 2012; Grandmont et al., 2023), les résultats du Chapitre 4 semblent suggérer qu'en absence de reproduction, les individus de moindre qualité individuelle, avec de plus courts télomères, sont également plus vulnérables à la chasse. Nous émettons l'hypothèse que ce résultat s'explique par une plus grande prise de risque des individus de moindre qualité (Bateson et al., 2015; Andrews et al., 2018) et nous insistons sur l'intérêt d'explorer le rôle des décisions reproductives dans la modulation de la mortalité à la chasse.

En effet, chez les oies, l'habitat où l'alimentation est la plus riche est également celui où elles sont le plus vulnérables à la chasse. Lors de la forte réduction de la chasse en 2020, les oies se sont engraissées plus rapidement, probablement en utilisant les ressources disponibles dans les champs agricoles (maïs). Puisque leur engraissement a été plus efficace, elles ont arrêté d'aller dans les champs plus tôt dans la saison 2020 comparativement aux années où le dérangement était plus important (LeTourneux et al., 2023). On peut ainsi faire le lien avec l'hétérogénéité d'acquisition de l'énergie qui pourrait être à la base de toute hétérogénéité de qualité individuelle (Vedder & Bouwhuis, 2018). Si certains individus sont

plus efficaces pour acquérir et accumuler des réserves énergétiques et peuvent ainsi échapper à certains compromis, les individus de moindre qualité doivent compenser en se nourrissant d'aliments plus riches mais en prenant des risques supplémentaires.

Limites et perspectives

Cette thèse nous éclaire sur les déterminants de la variabilité individuelle expliquant la survie adulte chez une espèce longévive. Toutefois, un suivi longitudinal est préférable pour étudier ces variations individuelles (Nussey et al., 2008). À cause du faible taux de recapture, notre système d'étude ne nous permet pas d'avoir suffisamment d'échantillons répétés sur un même individu. Nous avons donc dû étudier différents individus présentant des différences marquées en termes de condition physiologiques et d'âge pour répondre à nos objectifs. Le fait d'avoir une taille d'échantillons relativement conséquente vient compenser pour ce biais potentiel, et permet tout de même d'observer les tendances dans la population. Les espèces longévives telles que les pétrels ou les albatros sont plus propices à des études longitudinales car elles nichent en colonies serrées ce qui augmente la probabilité de recapture d'un même individu (Bouwhuis & Vedder, 2017; Angelier et al., 2019). Ces espèces sont donc les modèles privilégiés pour comprendre la sénescence et l'hétérogénéité de qualité individuelle. Cependant, ces espèces ne sont pas soumises à une pression de chasse importante comme la grande oie des neiges. Plus largement, la diversification des modèles d'études permet l'approfondissement des connaissances et la généralisation des patrons et mécanismes observés. Ainsi, la collecte d'échantillons de sang est maintenant intégrée au suivi à long terme de la grande oie des neiges à l'Île Bylot et il devrait être possible dans un horizon d'une dizaine d'années de bénéficier d'échantillons répétés sur un nombre significatif d'individus.

Pour s'affranchir de la nécessité de recapturer physiquement des individus de manière répétée, de plus en plus d'études utilisent des émetteurs qui donnent accès à de riches données sur la localisation et la performance des individus, à fine échelle, et sur plusieurs saisons. Une étude récente relie par exemple les stratégies migratrices au taux de sénescence chez le flamand rose (*Phoenicopterus roseus;* Cayuela et al., 2025). Les différentes stratégies individuelles peuvent ensuite être corrélées à des marqueurs physiologiques extraits d'échantillons prélevés lors de la pose de l'émetteur. Pour s'affranchir des recaptures et

affiner les données physiologiques, des émetteurs pouvant lire le rythme cardiaque et des molécules du sang commencent également à être développés (Riccio et al., 2024).

Le type de mesure de notre indicateur physiologique principal, la longueur des télomères, est également une limitation de l'étude. Nous avons choisi la méthode de quantification relative par qPCR pour des questions de budget et d'expertise. Cependant, cela ne permet pas la comparaison des échantillons entre projets, puisque tous les échantillons de référence doivent être communs et que la méthode est sensible à l'espacement des analyses (Cawthon, 2002). La méthode de qPCR peut également ajouter de la variabilité dans les résultats (Lindrose et al., 2021). Même si nos tailles d'échantillons importantes nous permettent d'avoir confiance en nos résultats, l'utilisation de méthodes absolues comme la migration sur gel d'agarose semble ouvrir plus d'avenues de recherche. L'estimation de l'attrition des télomères lors de potentielles études longitudinales serait ainsi possible et précise, et cela permettrait la comparaison entre populations ou espèces. Ces recherches prennent récemment de l'importance pour comprendre le lien entre télomères et compromis d'histoire de vie (Pepke et al., 2023). La comparaison entre grande et petite oie des neiges pourrait par ailleurs nous permettre de confirmer le lien entre les conditions environnementales pendant la croissance (notamment densité de nid) et la longueur des télomères, puisque la petite oie des neiges niche dans des colonies moins denses (Reiter & Andersen, 2013).

D'autres marqueurs physiologiques pourraient également être considérés. Dans cette étude, comme dans une importante méta-analyse (Remot et al., 2022), la corrélation de la longueur des télomères avec l'âge est très variable et explique peu de variabilité (8%). Son utilisation comme proxy de l'âge est en voie d'être délaissée (Pepke, 2024), au profit de la méthylation de l'ADN qui parait être un prédicteur plus fiable de l'âge (Zhao et al., 2019; Tangili et al., 2023). Additionnellement à cette utilisation comme proxy de l'âge, la progression de la recherche en épigénétique ouvre les portes vers des recherches approfondies des effets maternels reflétés par la méthylation de l'ADN (Chapelle & Silvestre, 2022; Hukkanen et al., 2023).

Étant donné le rôle central que semblent jouer l'acquisition et l'accumulation de réserves énergétiques dans la qualité individuelle, des marqueurs physiologiques de

l'allocation des réserves à la consommation ou à l'accumulation semblent essentiels. La corticostérone (CORT) est un médiateur important de l'engraissement au niveau basal (Hoarau et al., 2022; Colin, 2025). Sa valeur basale reflète l'état allostatique d'un individu, mais elle peut être difficile à obtenir car c'est également l'hormone principale de réponse au stress. Dans cette thèse, seules 31 valeurs basales étaient disponibles dans le Chapitre 2 (échantillons de l'Île-Aux-Oies), et aucune dans le Chapitre 3 (échantillons de l'Île Bylot). En effet, il est très difficile d'obtenir des échantillons sanguins en moins de trois minutes (Romero & Reed, 2005), surtout sur notre espèce où les captures s'effectuent en grands groupes, souvent d'une centaine d'individus. Avec les avancées récentes en lipidomique, de nouveaux marqueurs potentiels apparaissent. Pour l'instant, les études sont très descriptives, mais les variations en composition lipidique des réserves graisseuses et du plasma (Akram et al., 2025; Colin, 2025) semblent répondre aux conditions environnementales (Price et al., 2008) et à l'état physiologique des individus (Colin, 2025). Chez l'humain, et progressivement chez d'autres espèces, les médiateurs lipidiques de l'endocannabinoïdome, système complexe que l'on suppose jouer un rôle dans le comportement alimentaire, semblent réguler l'appétit, la glucogénèse, la glycolyse et plus largement impliqué dans le maintien de l'homéostasie (Silvestri & Di Marzo, 2013). Certaines molécules de ce système réagissent au stress, mais d'autres ne semblent pas varier lors de la capture (Colin, 2025). De nombreuses questions restent en suspens pour identifier le rôle des différents lipides et médiateurs associés, mais cette recherche semble prometteuse et pourrait potentiellement mener à définir des nouveaux marqueurs de l'état physiologique et de la stratégie d'allocation d'énergie d'un individu.

Finalement, une grande limitation des études alliant physiologie et démographie est la rareté des données physiologiques, en opposition avec le besoin conséquent de données pour estimer les paramètres démographiques. Ceci aboutit souvent à la non-exploitation des données physiologiques. Par exemple, il n'a pas été possible d'utiliser les données de longueur de télomères dans des modèles de survie de l'Île Bylot car elles sont trop lacunaires et trop récentes. De même, l'effet de la condition corporelle sur la survie n'a jamais été estimée avec le jeu de données de l'Île Bylot car les données sont disponibles sur peu d'individus chaque année. Une des forces de cette thèse est la complémentarité de nos jeux de données, avec la capture à l'Île-Aux-Oies qui permet l'utilisation de ces données

individuelles. Cependant, les 13 ans et 560 individus du jeu de données de l'Île-Aux-Oies sont loin de la puissance statistique des 32 ans et 100 000 individus bagués à l'Île Bylot. Des modèles de capture-marquage-recapture avec données manquantes (Worthington et al., 2015) et des modèles de population intégrés (Plard et al., 2019) ont été développés, mais il faut encore optimiser ces approches au cas des données physiologiques particulièrement lacunaires. L'intégration de variables physiologiques dans les modèles démographiques permettrait l'exploitation de beaucoup de données jusqu'alors réservées aux études physiologiques. Cela permettrait d'explorer plus en profondeur l'influence des différents traits sur le fitness et de la contribution de l'hétérogénéité individuelle à la dynamique de population.

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Annexe S0 – Matériel supplémentaire pour l'introduction

Annexe S0.1: Illustration simplifiée des composantes de la qualité individuelle

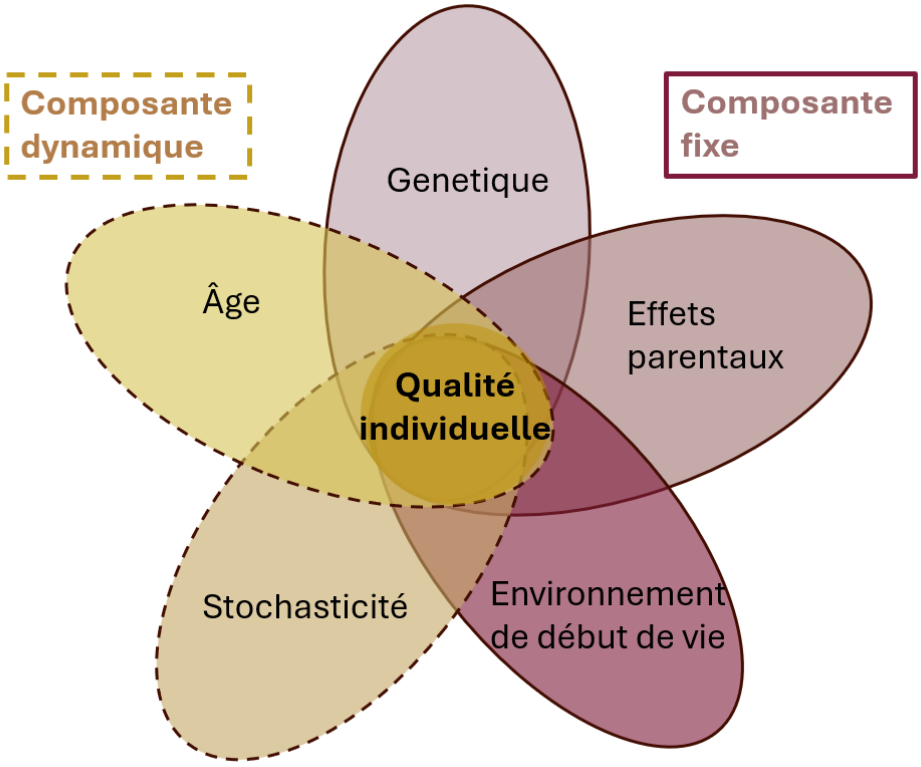

Figure S0.1 : Illustration simplifiée des différentes composantes de la qualité individuelle

Annexe S1 – Matériel supplémentaire pour le Chapitre 1

Annexe S1.1: Harvest rate data

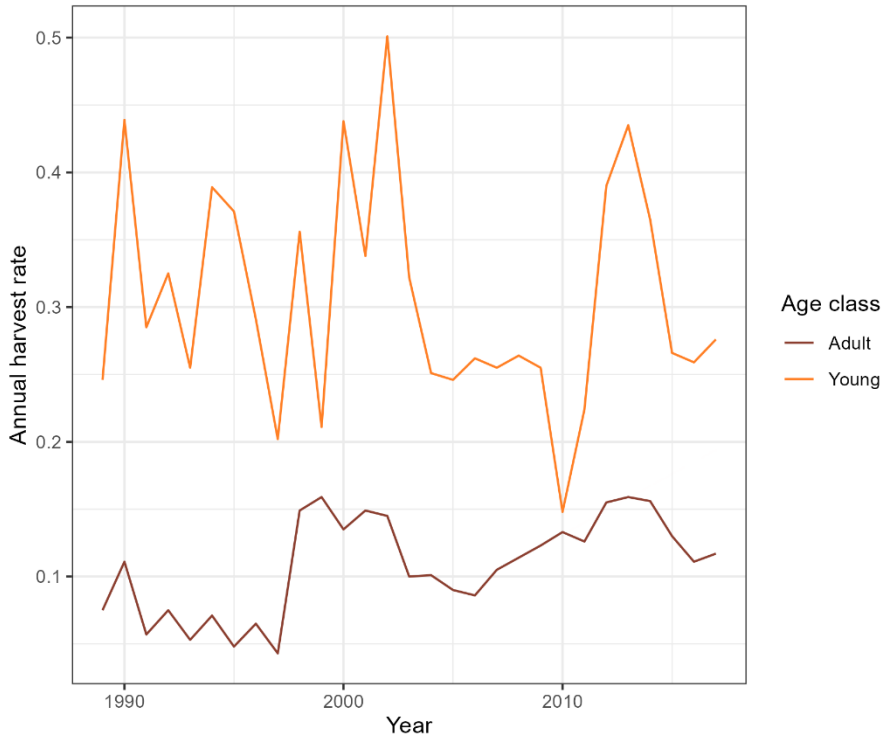

Figure S1.1: Annual harvest rate data from 1989 to 2017 for young (in orange) and adult (in brown) greater snow geese

Annexe S1.2: Observation matrix

	Not Observed	Observed alive	Observed dead
Alive Young	0	1	0
Alive Adult Bylot	$1 - p_{s;t}$	$p_{s;t}$	0
Alive Adult Emigrated	1	0	0
Newly Dead	$1 - r_{AC;t}$	0	$r_{AC;t}$
Dead	1	0	0

Detection probability ($p_{s;t}$) is defined for each sex (s) and includes a random effect of time (t). Recovery probability ($r_{AC;t}$) is defined separately for young and adults (age class: AC) and includes a random effect of time (t).

Annexe S1.3: List of priors used in the Bayesian capture-mark-recapture model.

We list priors used in the most general model, including an interaction between the effects of age and hunting pressure.

It is important to note that survival probabilities must be transformed using the inverse logit function. As such, prior distribution $\mathrm{N}(0, 1.6)$ is vague, although it might appear informative.

Survival priors

$\mu^Y \sim N(0, 1.6)$

$\beta_{HR}^Y \sim N(0, 1.6)$

$\varepsilon_t \sim N(0, \sigma_\varphi)$, *with* $\sigma_\varphi \sim U(0,10)$

$\mu^A \sim N(0, 1.6)$

$\beta_{age} \sim N(0, 1.6)$

$\beta_{HR}^A \sim N(0, 1.6)$

Recapture priors

$\mu_p \sim N(0, 1.6)$

$\beta_{p.male} \sim N(0, 1.6)$

$\varepsilon_t^p \sim N(0, \sigma_p)$, *with* $\sigma_p \sim U(0,10)$

Recovery priors

$\mu_r \sim N(0, 1.6)$

$\beta_{r.adult} \sim N(0, 1.6)$

$\varepsilon_t^{r,Y} \sim N(0, \sigma_r^Y)$, *with* $\sigma_r^Y \sim U(0,10)$

$\varepsilon_t^{r,A} \sim N(0, \sigma_r^A)$, *with* $\sigma_r^A \sim U(0,10)$

Emigration priors

$E_F^Y \sim truncated\ positive\ N(0.06, 0.03)$

$E_M^Y \sim truncated\ positive\ N(0.78, 0.04)$

$E_F^A \sim truncated\ positive\ N(0.01, 0.01)$

$E_M^A \sim truncated\ positive\ N(0.13, 0.01)$

Priors for missing harvest rate values

$HR_t^Y \sim N(\mu_{HR}^Y, \sigma_{HR}^Y)$, *with* $\mu_{HR}^Y \sim N(0,1)$ *and* $\sigma_{HR}^Y \sim N(0,2)$

$HR_t^A \sim N(\mu_{HR}^A, \sigma_{HR}^A)$, *with* $\mu_{HR}^A \sim N(0,1)$ *and* $\sigma_{HR}^A \sim N(0,2)$

Annexe S1.4: Complementary information on parameter estimations

R-hat ≤ 1.1 indicates that chains have mixed well. *N.eff* is the effective sample size, it reflects the number of independent samples, accounting for autocorrelation.

Table S1.1: Parameter estimation, SD, credible intervals, R-hat and N.eff values for model evaluation purposes.

Parameter	Mean	SD	25%	97.5%	Rhat	N.eff
int.phi	1.26	0.36	0.65	2.12	1.06	42.00
beta_age[1]	-2.04	0.35	-2.88	-1.43	1.08	52.00
beta_age[2]	0.14	0.34	-0.68	0.73	1.07	50.00
beta_age[3]	0.56	0.34	-0.26	1.16	1.07	50.00
beta_age[4]	0.64	0.34	-0.16	1.25	1.07	54.00
beta_age[5]	0.67	0.34	-0.15	1.28	1.07	52.00
beta_age[6]	0.70	0.35	-0.12	1.31	1.07	52.00
beta_age[7]	0.55	0.34	-0.27	1.16	1.07	52.00
beta_age[8]	0.45	0.35	-0.37	1.06	1.06	53.00
beta_age[9]	0.49	0.35	-0.34	1.10	1.07	53.00
beta_age[10]	0.44	0.35	-0.38	1.06	1.06	57.00
beta_age[11]	0.45	0.35	-0.37	1.08	1.06	56.00
beta_age[12]	0.25	0.36	-0.57	0.89	1.06	58.00
beta_age[13]	0.38	0.37	-0.45	1.05	1.06	65.00
beta_age[14]	0.13	0.37	-0.70	0.79	1.05	71.00
beta_age[15]	-0.15	0.37	-0.99	0.52	1.05	70.00
beta_age[16]	-0.47	0.39	-1.32	0.25	1.05	97.00
beta_age[17]	0.23	0.45	-0.70	1.07	1.04	146.00
beta_age[18]	-0.34	0.47	-1.31	0.56	1.03	210.00
beta_age[19]	-0.79	0.44	-1.71	0.02	1.04	133.00
beta_harvHY	-0.10	0.07	-0.24	0.04	1.01	923.00
beta_harvAHY	-0.23	0.08	-0.39	-0.07	1.00	1079.00
eps.phi[1]	1.07	0.31	0.49	1.70	1.01	1869.00
eps.phi[2]	0.78	0.24	0.34	1.27	1.02	1170.00
eps.phi[3]	-0.30	0.21	-0.72	0.12	1.01	849.00
eps.phi[4]	0.38	0.20	0.00	0.78	1.02	801.00
eps.phi[5]	-0.28	0.20	-0.67	0.11	1.01	632.00
eps.phi[6]	0.79	0.20	0.41	1.19	1.01	728.00
eps.phi[7]	0.74	0.21	0.34	1.17	1.02	895.00
eps.phi[8]	-0.90	0.20	-1.30	-0.52	1.02	736.00
eps.phi[9]	0.40	0.22	-0.01	0.85	1.00	799.00
eps.phi[10]	-0.72	0.23	-1.18	-0.27	1.02	919.00
eps.phi[11]	0.83	0.19	0.45	1.21	1.02	571.00
eps.phi[12]	0.66	0.19	0.28	1.05	1.01	738.00

eps.phi[13]	-0.45	0.25	-0.94	0.05	1.01	705.00
eps.phi[14]	0.27	0.17	-0.07	0.61	1.02	463.00
eps.phi[15]	0.17	0.18	-0.20	0.52	1.02	657.00
eps.phi[16]	0.03	0.17	-0.32	0.37	1.02	522.00
eps.phi[17]	0.03	0.17	-0.31	0.36	1.02	491.00
eps.phi[18]	0.56	0.18	0.22	0.91	1.02	571.00
eps.phi[19]	-0.13	0.18	-0.48	0.21	1.02	622.00
eps.phi[20]	0.44	0.17	0.09	0.77	1.03	537.00
eps.phi[21]	0.48	0.21	0.07	0.89	1.02	809.00
eps.phi[22]	0.29	0.20	-0.11	0.69	1.03	784.00
eps.phi[23]	0.08	0.21	-0.34	0.50	1.01	781.00
eps.phi[24]	-0.09	0.21	-0.50	0.32	1.01	695.00
eps.phi[25]	-0.34	0.21	-0.76	0.07	1.01	844.00
eps.phi[26]	-0.89	0.19	-1.27	-0.52	1.02	746.00
eps.phi[27]	-0.83	0.19	-1.22	-0.46	1.02	742.00
eps.phi[28]	-0.44	0.22	-0.87	0.01	1.01	1083.00
eps.phi[29]	-1.21	0.27	-1.74	-0.66	1.01	1653.00
eps.phi[30]	-0.50	0.30	-1.09	0.07	1.01	1785.00
eps.phi[31]	-0.93	0.45	-1.84	-0.10	1.00	1474.00
eps.phi[32]	0.00	0.67	-1.33	1.33	1.00	55963.00
E1[1]	0.56	0.03	0.50	0.61	1.00	2882.00
E1[2]	0.96	0.01	0.95	0.97	1.00	7508.00
E2[1]	0.00	0.00	0.00	0.00	1.00	12910.00
E2[2]	0.01	0.01	0.00	0.04	1.00	19451.00
int.r	-2.22	0.12	-2.45	-1.97	1.04	261.00
beta.r.ad	1.04	0.20	0.64	1.43	1.03	266.00
eps.r.j[1]	0.34	0.27	-0.19	0.88	1.00	3435.00
eps.r.j[2]	0.50	0.20	0.13	0.90	1.01	1481.00
eps.r.j[3]	0.16	0.17	-0.18	0.49	1.02	649.00
eps.r.j[4]	0.51	0.16	0.20	0.82	1.02	747.00
eps.r.j[5]	-1.15	0.19	-1.53	-0.79	1.02	819.00
eps.r.j[6]	0.12	0.16	-0.20	0.44	1.02	906.00
eps.r.j[7]	0.04	0.18	-0.32	0.40	1.01	1147.00
eps.r.j[8]	-1.64	0.21	-2.07	-1.24	1.01	1012.00
eps.r.j[9]	0.50	0.15	0.20	0.81	1.03	617.00
eps.r.j[10]	-0.87	0.25	-1.38	-0.41	1.01	2081.00
eps.r.j[11]	0.28	0.15	-0.02	0.58	1.02	692.00
eps.r.j[12]	0.83	0.16	0.52	1.15	1.01	785.00
eps.r.j[13]	-0.86	0.21	-1.27	-0.46	1.01	1053.00
eps.r.j[14]	-0.26	0.16	-0.57	0.04	1.02	563.00
eps.r.j[15]	-0.07	0.17	-0.41	0.27	1.01	842.00
eps.r.j[16]	-0.26	0.16	-0.57	0.04	1.02	551.00
eps.r.j[17]	-0.35	0.16	-0.67	-0.04	1.02	571.00
eps.r.j[18]	0.32	0.15	0.02	0.63	1.02	712.00
eps.r.j[19]	0.01	0.15	-0.31	0.30	1.02	516.00

eps.r.j[20]	0.64	0.14	0.36	0.92	1.02	521.00
eps.r.j[21]	0.82	0.15	0.51	1.12	1.02	657.00
eps.r.j[22]	0.40	0.15	0.11	0.70	1.02	680.00
eps.r.j[23]	-0.21	0.17	-0.55	0.13	1.02	771.00
eps.r.j[24]	-0.26	0.15	-0.56	0.03	1.03	517.00
eps.r.j[25]	-0.10	0.17	-0.43	0.22	1.01	688.00
eps.r.j[26]	-0.44	0.16	-0.76	-0.14	1.02	530.00
eps.r.j[27]	-0.20	0.15	-0.49	0.09	1.02	404.00
eps.r.j[28]	0.17	0.15	-0.14	0.46	1.02	506.00
eps.r.j[29]	-0.36	0.16	-0.67	-0.04	1.02	481.00
eps.r.j[30]	0.24	0.16	-0.08	0.56	1.02	519.00
eps.r.j[31]	0.00	0.61	-1.23	1.21	1.00	32457.00
eps.r.j[32]	0.00	0.62	-1.22	1.21	1.00	54590.00
eps.r.ad[1]	0.01	0.65	-1.27	1.29	1.00	33668.00
eps.r.ad[2]	0.07	0.50	-0.89	1.08	1.00	21671.00
eps.r.ad[3]	-0.44	0.32	-1.07	0.18	1.00	4629.00
eps.r.ad[4]	0.44	0.36	-0.24	1.19	1.00	6334.00
eps.r.ad[5]	-0.69	0.29	-1.26	-0.13	1.00	2353.00
eps.r.ad[6]	0.22	0.36	-0.46	0.94	1.00	3095.00
eps.r.ad[7]	0.08	0.30	-0.49	0.68	1.00	4008.00
eps.r.ad[8]	-1.24	0.26	-1.75	-0.73	1.00	2282.00
eps.r.ad[9]	0.40	0.27	-0.11	0.95	1.00	2061.00
eps.r.ad[10]	-0.99	0.27	-1.51	-0.46	1.01	1432.00
eps.r.ad[11]	0.96	0.31	0.38	1.61	1.00	2485.00
eps.r.ad[12]	0.86	0.29	0.32	1.48	1.00	1971.00
eps.r.ad[13]	-0.86	0.27	-1.40	-0.33	1.00	1180.00
eps.r.ad[14]	0.49	0.24	0.04	0.97	1.00	2511.00
eps.r.ad[15]	0.32	0.24	-0.14	0.80	1.01	2541.00
eps.r.ad[16]	0.18	0.23	-0.26	0.63	1.00	2668.00
eps.r.ad[17]	0.05	0.22	-0.38	0.49	1.00	2695.00
eps.r.ad[18]	0.73	0.25	0.26	1.25	1.01	2497.00
eps.r.ad[19]	-0.07	0.21	-0.48	0.34	1.00	1779.00
eps.r.ad[20]	0.38	0.21	-0.04	0.81	1.00	1875.00
eps.r.ad[21]	0.45	0.28	-0.09	1.03	1.01	1218.00
eps.r.ad[22]	0.04	0.24	-0.41	0.52	1.01	1363.00
eps.r.ad[23]	-0.32	0.24	-0.80	0.15	1.00	1491.00
eps.r.ad[24]	-0.39	0.23	-0.85	0.08	1.01	1177.00
eps.r.ad[25]	-0.36	0.23	-0.82	0.10	1.00	1533.00
eps.r.ad[26]	-0.70	0.21	-1.10	-0.29	1.01	1480.00
eps.r.ad[27]	-0.22	0.21	-0.62	0.20	1.00	1956.00
eps.r.ad[28]	0.29	0.25	-0.19	0.81	1.00	2401.00
eps.r.ad[29]	-0.26	0.31	-0.83	0.38	1.00	2045.00
eps.r.ad[30]	0.50	0.39	-0.23	1.29	1.00	2167.00
eps.r.ad[31]	0.64	0.44	-0.14	1.59	1.01	1657.00
eps.r.ad[32]	0.00	0.65	-1.28	1.30	1.00	57062.00

int.p	-2.78	0.14	-3.06	-2.50	1.00	832.00
beta.p.m	0.34	0.13	0.09	0.59	1.00	12125.00
eps.p[1]	0.00	0.75	-1.48	1.49	1.00	30638.00
eps.p[2]	-0.19	0.47	-1.17	0.68	1.00	17643.00
eps.p[3]	-0.53	0.36	-1.27	0.13	1.00	6920.00
eps.p[4]	-0.07	0.30	-0.68	0.49	1.00	4730.00
eps.p[5]	-0.62	0.29	-1.21	-0.08	1.00	4491.00
eps.p[6]	-0.87	0.30	-1.49	-0.31	1.00	4909.00
eps.p[7]	0.05	0.20	-0.35	0.45	1.00	1778.00
eps.p[8]	-0.32	0.21	-0.74	0.08	1.00	2088.00
eps.p[9]	0.33	0.19	-0.03	0.70	1.00	1672.00
eps.p[10]	-1.49	0.30	-2.10	-0.94	1.00	4459.00
eps.p[11]	0.63	0.18	0.26	0.99	1.00	1462.00
eps.p[12]	0.08	0.19	-0.29	0.45	1.00	1565.00
eps.p[13]	-0.73	0.22	-1.18	-0.30	1.00	2337.00
eps.p[14]	0.57	0.18	0.22	0.92	1.00	1306.00
eps.p[15]	0.58	0.17	0.24	0.93	1.00	1155.00
eps.p[16]	0.59	0.17	0.25	0.93	1.00	1220.00
eps.p[17]	0.24	0.18	-0.12	0.59	1.00	1295.00
eps.p[18]	0.38	0.17	0.03	0.72	1.00	1297.00
eps.p[19]	-0.67	0.21	-1.10	-0.27	1.00	1900.00
eps.p[20]	0.22	0.18	-0.13	0.57	1.00	1334.00
eps.p[21]	-0.10	0.18	-0.46	0.26	1.00	1345.00
eps.p[22]	-0.11	0.18	-0.46	0.24	1.00	1240.00
eps.p[23]	-0.48	0.19	-0.86	-0.11	1.00	1519.00
eps.p[24]	0.11	0.18	-0.24	0.45	1.00	1294.00
eps.p[25]	-0.46	0.20	-0.86	-0.07	1.00	1734.00
eps.p[26]	0.26	0.19	-0.11	0.62	1.00	1474.00
eps.p[27]	0.80	0.18	0.44	1.16	1.00	1392.00
eps.p[28]	0.80	0.19	0.42	1.17	1.00	1719.00
eps.p[29]	0.61	0.21	0.20	1.03	1.00	1848.00
eps.p[30]	0.84	0.23	0.40	1.29	1.00	2418.00
eps.p[31]	-2.00	0.52	-3.12	-1.10	1.00	12645.00
eps.p[32]	0.72	0.35	0.07	1.44	1.00	2683.00
mu.huntHY	0.04	0.19	-0.34	0.40	1.00	26711.00
sd.huntHY	1.05	0.27	0.57	1.64	1.00	25961.00
mu.huntAHY	0.05	0.18	-0.32	0.41	1.00	23721.00
sd.huntAHY	1.06	0.28	0.59	1.67	1.00	21273.00

Annexe S1.5: Emigration and detection probabilities

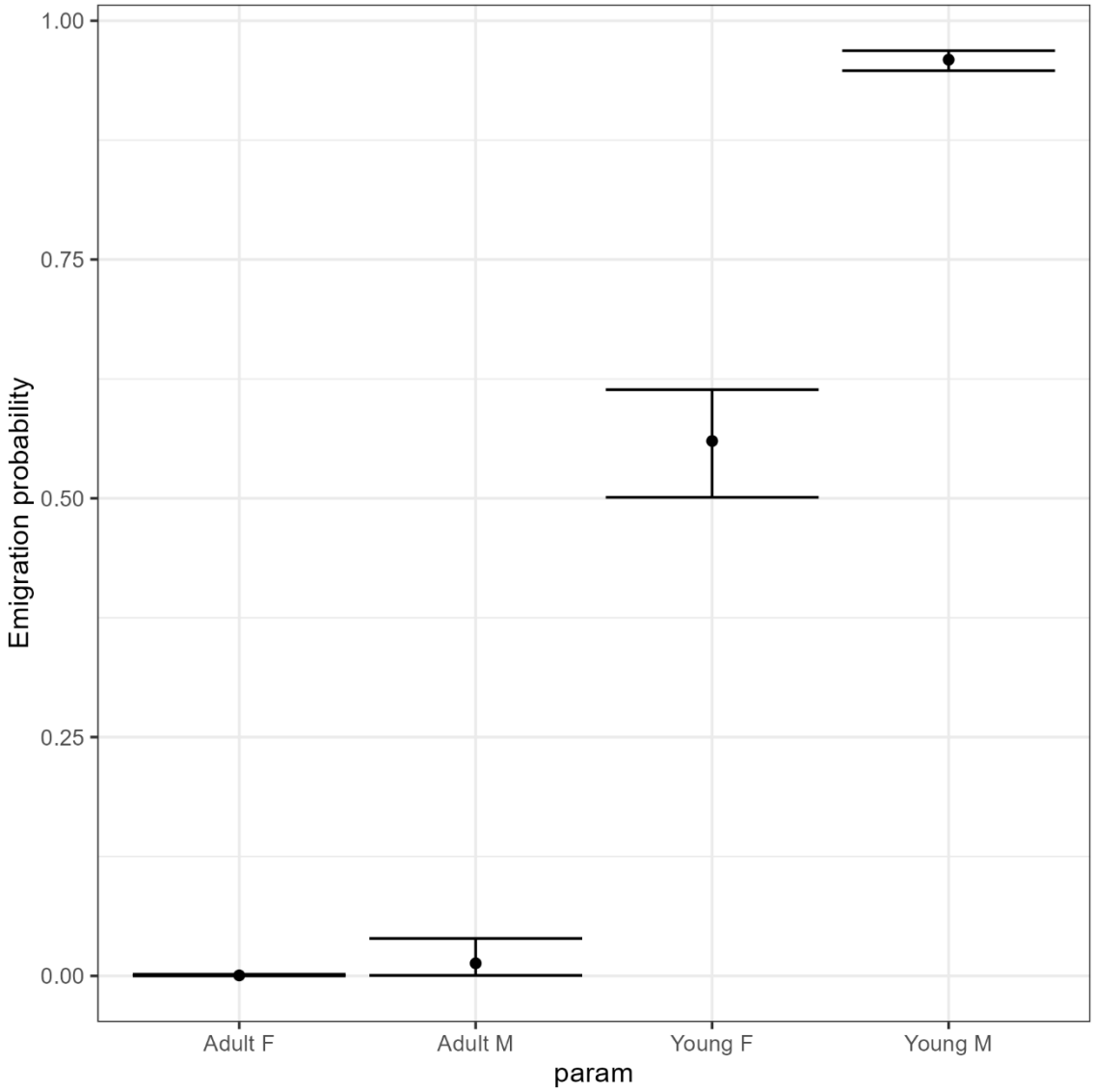

Figure S1.2: Permanent emigration probabilities for adult females, adult males, young females, amd young males.

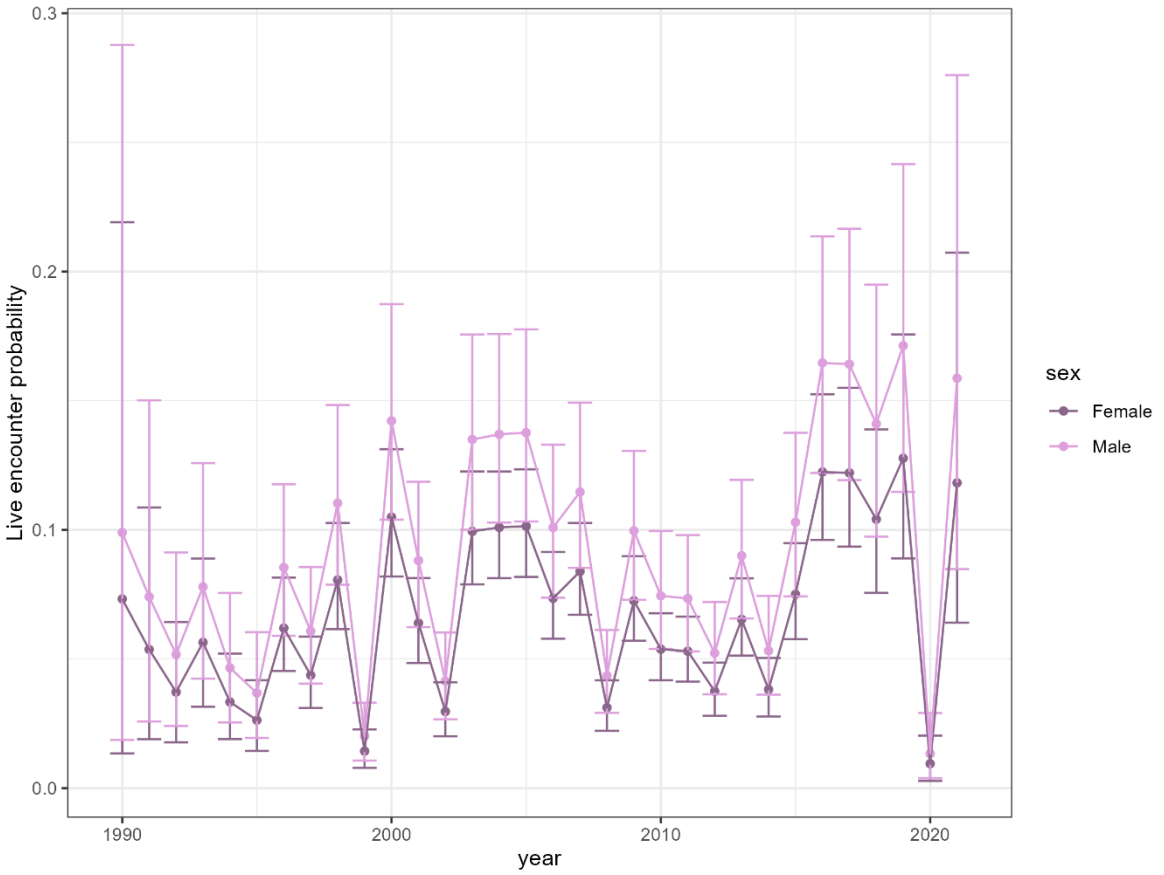

Figure S1.3: Live encounter (capture) probabilities for adult female (dark) and male (light) greater snow geese captured at Bylot Island from 1990 to 2021.

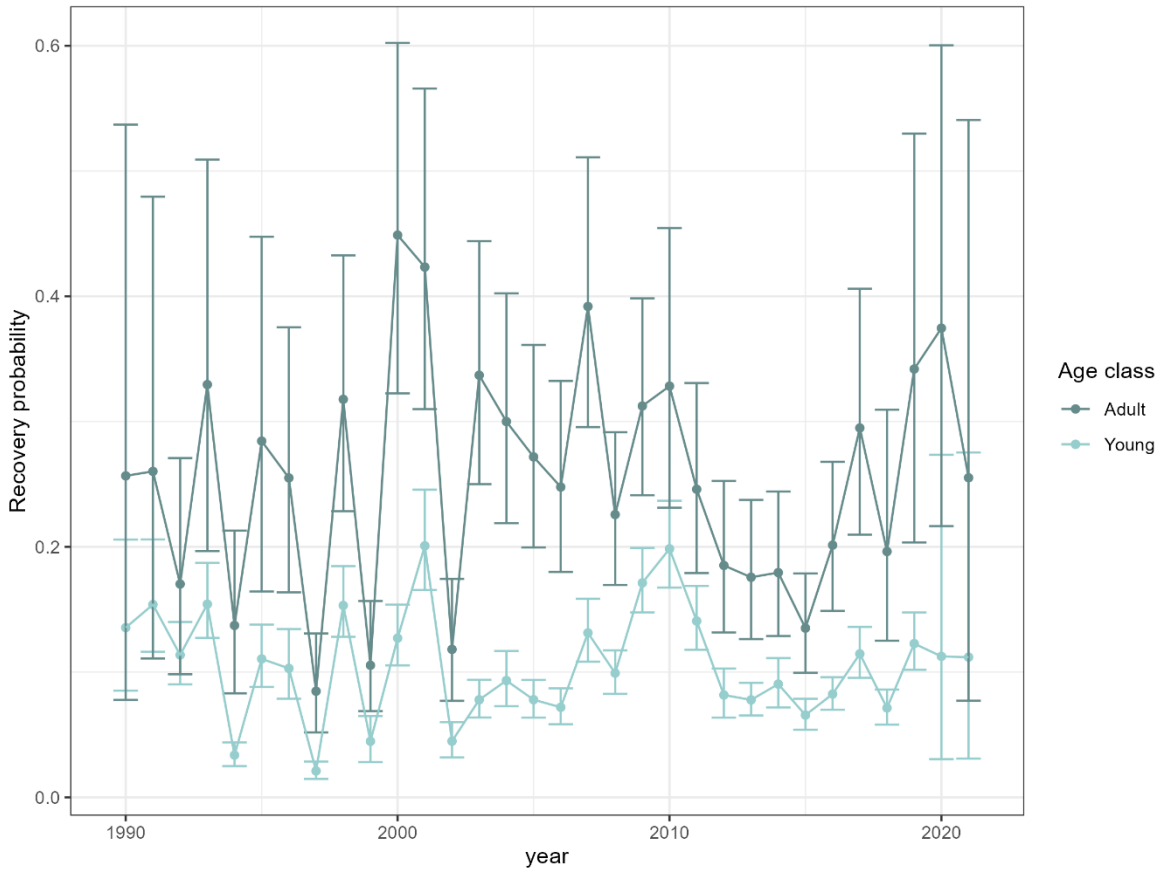

Figure S1.4: Recovery probabilities for juveniles (light) and adult (dark) greater snow geese from 1990 to 2021.

Annexe S2 – Matériel supplémentaire pour le Chapitre 2

Annexe S2.1: Details of the number of samples and observations collected for our analyses

Table S2.1 : Details of the number of samples and observations collected for our analyses

Data or sample type	Number of samples or observations
Baseline CORT	29
Stress-induced CORT at banding	578
Stress-induced CORT at release	154
Body condition at banding	560
Body condition at release	154
Total number of birds recovered in 2009-2010	56
Total number of birds reobserved in autumn 2009	37 unpaired; 131 paired

Annexe S2.2: Correction of corticosterone values by date and time since capture

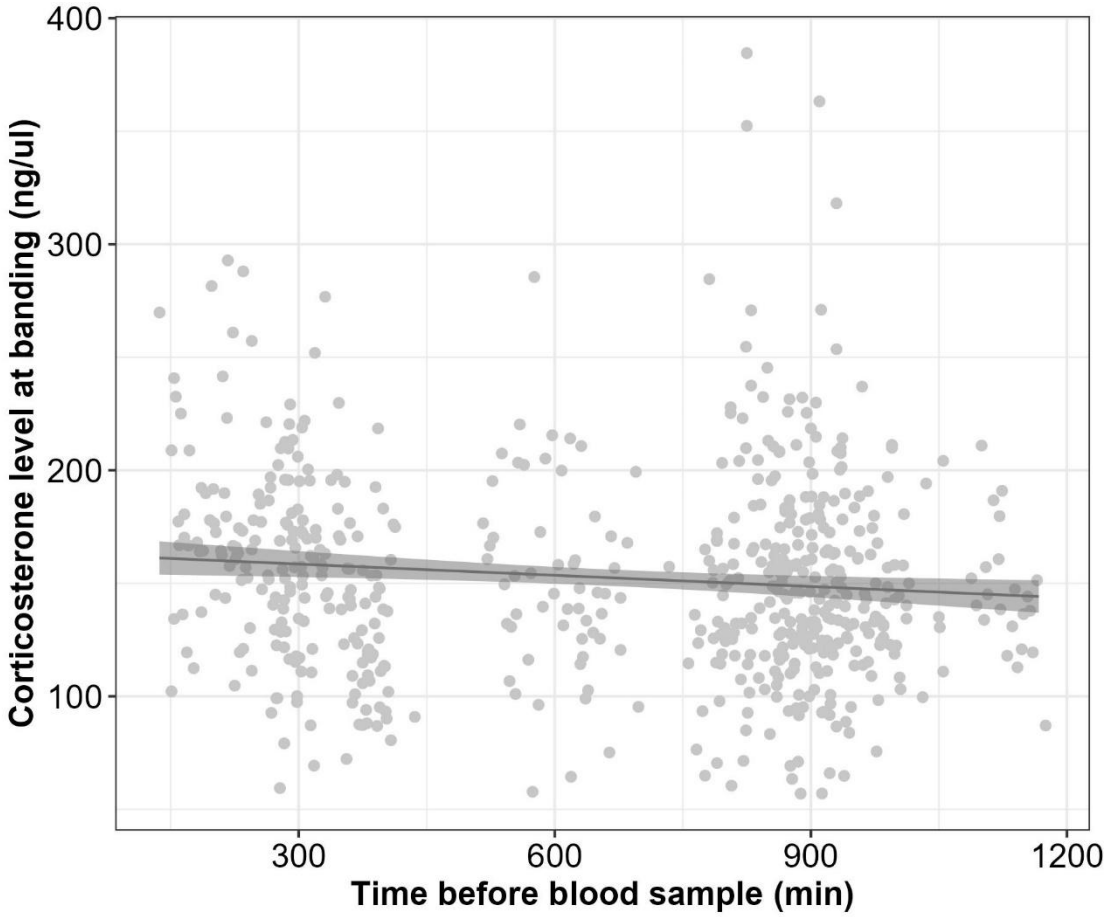

Figure S2.1 : Relationship between corticosterone level measured at banding (stress-induced CORT) and time elapsed between capture and blood sampling during banding. Regression with and 95% confidence intervals (shading).

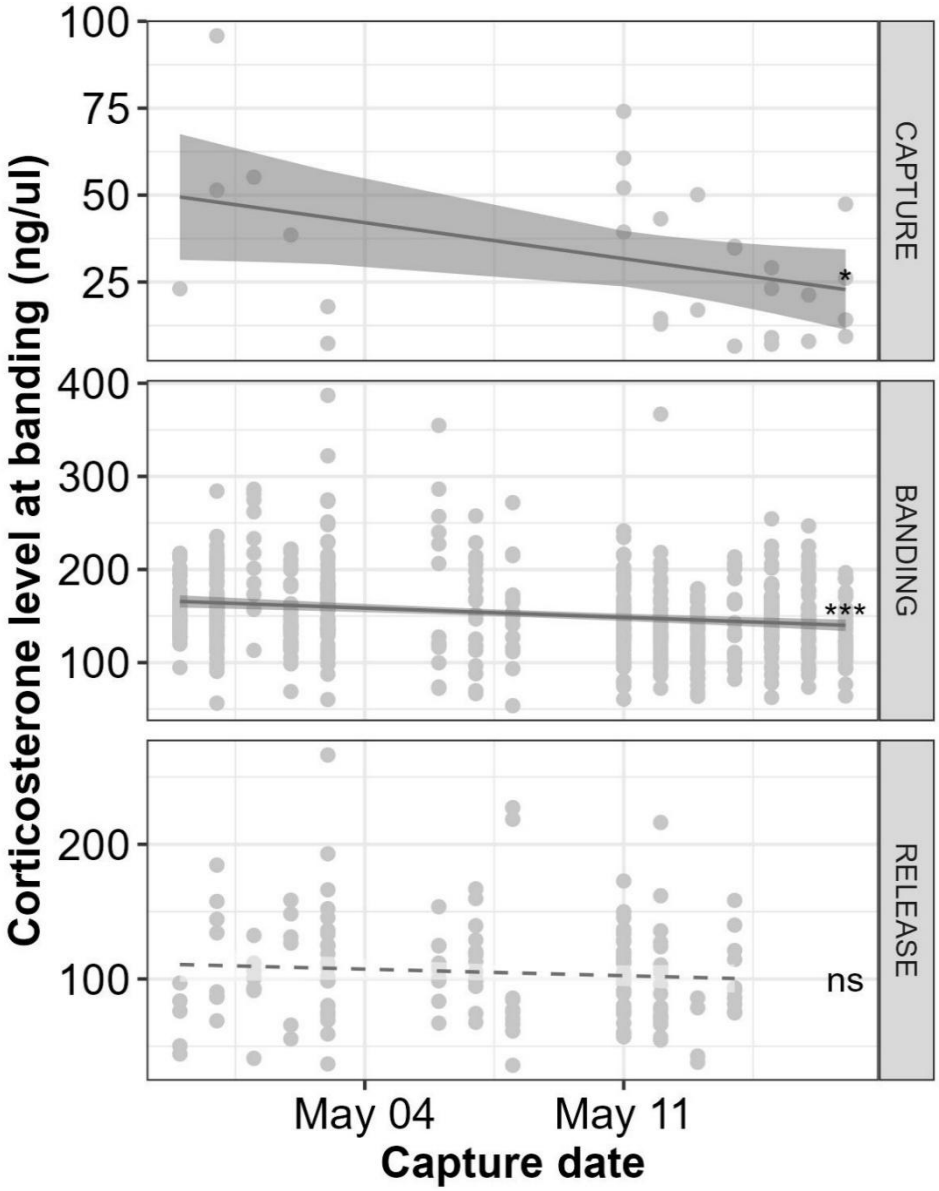

Figure S2.2: Relationship between corticosterone level and capture date for each sample type. Regression with and 95% confidence intervals (shading) when the relationship was significant.

Annexe S2.3: Validation of random sampling

Table S2.2: Validation that CORT levels were similar between individuals before they were randomly assigned to treatment groups. Estimates and 95% confidence intervals from TukeyHSD post-hoc tests on ANOVAs.

Two by two comparisons	Beta
Fed-Control	-3.14 [-13.80; 7.52]
Unfed - Control	-0.38 [-11.43; 10.68]
Unfed - Fed	2.76 [-8.05; 13.57]
2 days - Control	-2.75 [-15.63; 10.14]
3 days - Control	2.16 [-11.91; 16.22]
4 days - Control	-4.07 [-17.12; 8.97]
3 days - 2 days	4.90 [-9.96; 19.77]
4 days - 2 days	-1.33 [-15.23; 12.58]
4 days - 3 days	-6.23 [-21.24; 8.78]

Annexe S2.4: Complete model selection for CMR survival models

Table S2.3: Complete model selection analysing survival of greater snow geese marked in spring 2009 in relation to several covariates.

Covariates include stress-induced corticosterone level at banding (CORT, continuous individual covariate), body condition (COND1 : condition at banding; COND2 : condition at release; both as continuous individual covariate), food treatment (FOOD: treatment fed/unfed/control), number of days spent in captivity (DAYS : 0, 2, 3 and 4 days), and time (t, in years; t(1): first year after release; t(2:13):constant for years 2 to 13; t(2_13): time varying for years 2 to 13). K = number of parameters, ΔQAICc = difference in QAICc between the current and the top-ranked model within each model selection. Three separate model selection done sequentially are presented and are separated by the grey lines. Models in bold were preferred and were used to go to the next model selection.

Model name	Survival	Recapture	Recovery	K	Deviance	QAICc	ΔQAICc
Initial model selection for recovery and reobservation probabilities (event matrix)							
MS1	**General model**	**Constant**	**Constant**	**53**	**2826.46**	**2613.40**	**0**
MS2	General model	t	Constant	64	2800.65	2615.42	2.03
MS3	General model	Constant	t	64	2801.07	2615.79	2.40
MS4	General model	t	t	76	2777.90	2623.11	9.71
Model selection for survival probability (transition matrix)							
MS5	**t(1).FOOD+t(2:13)**	**Constant**	**Constant**	**6**	**2872.35**	**2553.99**	**0**
MS6	**t(1).[FOOD+COND1]+t(2:13)**	**Constant**	**Constant**	**7**	**2871.17**	**2554.98**	**0.99**
MS7	**t(1).[CORT+FOOD]+t(2:13)**	**Constant**	**Constant**	**7**	**2871.85**	**2555.65**	**1.58**
MS8	t(1).COND2+t(2:13)	Constant	Constant	5	2877.85	2556.83	2.84
MS9	t(1).[CORT+COND2]+t(2:13)	Constant	Constant	6	2877.54	2558.58	4.59
MS10	t(1).COND1+t(2:13)	Constant	Constant	5	2880.03	2558.76	4.77
MS11	t(1).CORT+t(2:13)	Constant	Constant	5	2880.16	2558.88	4.89
MS12	t(1).[FOOD.COND2]+t(2:13)	Constant	Constant	7	2876.46	2559.65	5.66
MS13	t(1).[CORT.DAYS]+t(2:13)	Constant	Constant	8	2874.48	2559.94	5.94
MS14	t(1).[CORT+COND1]+t(2:13)	Constant	Constant	6	2879.57	2560.38	6.39
MS15	t(1).[FOOD.COND1]+t(2:13)	Constant	Constant	7	2877.34	2560.43	6.44

MS16	t(1).[CORT+DAYS]+t(2:13)	Constant	Constant	8	2876.41	2561.64	7.65
MS17	Constant	Constant	Constant	3	2889.10	2562.75	8.76
MS18	t(1).[CORT.FOOD]+t(2:13)	Constant	Constant	7	2880.33	2563.10	9.09
MS19	t(1).FOOD+t(2_13)	Constant	Constant	16	2860.48	2563.96	9.97
MS20	t(1).[CORT+FOOD]+t(2_13)	Constant	Constant	17	2860.00	2565.60	12.71
MS21	t	Constant	Constant	14	2869.23	2567.57	13.58
MS22	t(1).CORT+t(2_13)	Constant	Constant	15	2868.27	2568.78	14.79
MS23	t(1).[CORT.FOOD]+t(2_13)	Constant	Constant	17	28678.43	2573.07	20.18
Final model selection on best models							
MS24	**t(1).FOOD+t(2:13)**	**t**	**Constant**	**17**	**2845.63**	**2552.89**	**0**
MS25	t(1).FOOD+t(2:13)	t	t	24	2835.31	2558.36	5.48
MS26	t(1).FOOD+t(2:13)	Constant	t	17	2858.42	2564.21	11.32
MS27	**t(1).[FOOD+COND1]+t(2:13)**	**t**	**Constant**	**18**	**2844.50**	**2553.96**	**1.07**
MS28	t(1).[FOOD+COND1]+t(2:13)	Constant	t	18	2857.23	2565.22	12.34
MS29	t(1).[FOOD+COND1]+t(2:13)	t	t	29	2835.03	2568.69	15.80
MS30	**t(1).[CORT+FOOD]+t(2:13)**	**t**	**Constant**	**18**	**2844.50**	**2553.96**	**1.08**
MS31	t(1).[CORT+FOOD]+t(2:13)	Constant	t	18	2857.91	2565.82	12.94
MS32	t(1).[CORT+FOOD]+t(2:13)	t	t	29	2835.03	2568.69	15.80
MS33	i+t(1).CORT+t(2:13)	t	Constant	16	2853.54	2557.82	4.93
MS34	t(1).COND1+t(2:13)	t	Constant	16	2853.18	2557.49	4.61
MS35	t(1).COND2+t(2:13)	t	Constant	16	2850.98	2555.55	2.66
MS36	Constant	t	Constant	14	2855.77	2555.66	2.77

Annexe S2.5 : Exploration of pairing status as a mechanism for the observed carry-over effect

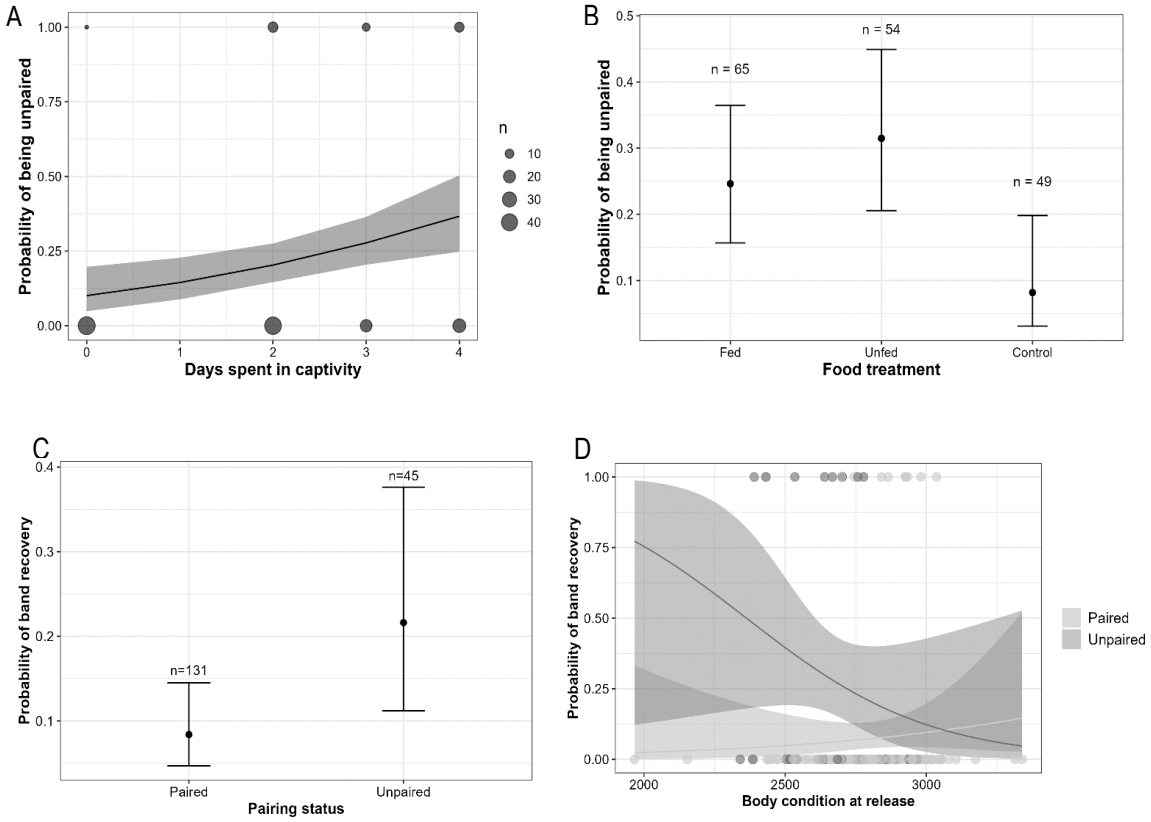

Figure S2.3: A: Probability of observing a marked females without a mate (i.e. unpaired) in autumn depending on the number of days spent in captivity in spring (β = 0.33 [0.13, 0.54]). B: Probability of observing a marked and unpaired female in autumn as a function of the food treatment in spring ($\beta_{fed/unfed}$ = 0.34 [-0.46, 1.15]). C: Probability of band recovery in the first year after marking of paired and unpaired females in autumn (β_{status} = 13.9 [-1.2, 29.9]) D: Relationship between the probability of band recovery in the first year after marking and body condition at release for paired and unpaired females observed in autumn ($\beta_{status:condition}$ = -0.005 [-0.01, 9.80e-04]). The parameters estimates are presented with 95% confidence intervals in all four panels.

Annexe S3 – Matériel supplémentaire pour le Chapitre 3

Annexe S3.1 : PCA results for body condition index

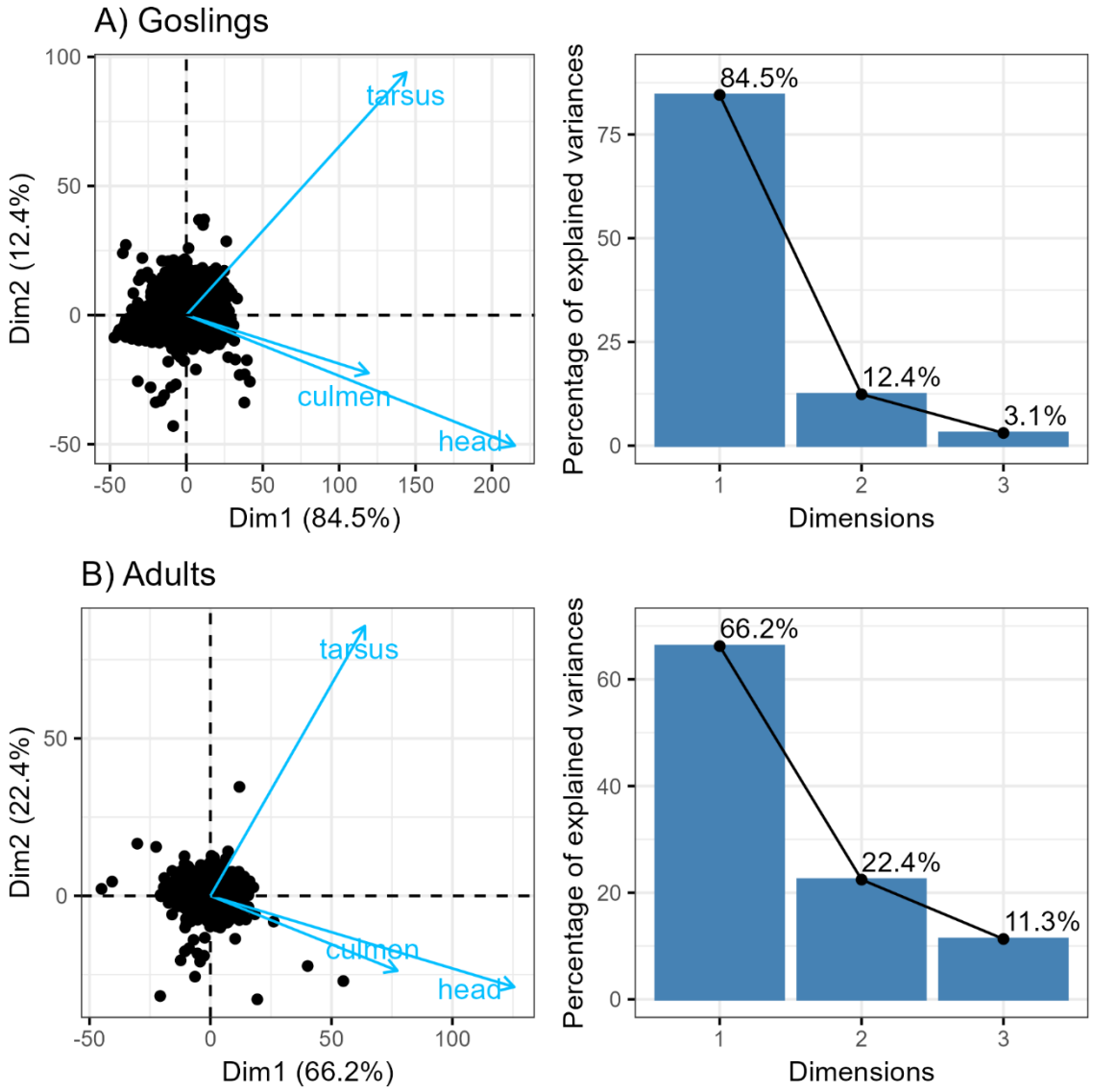

Figure S3.1: Results from the principal component analysis (PCA) used to get the body size index and correct for size in the body condition calculation. PCAs were done separately for goslings (A) and adult females (B).

Annexe S3.2: Cohort effect on relative telomere length

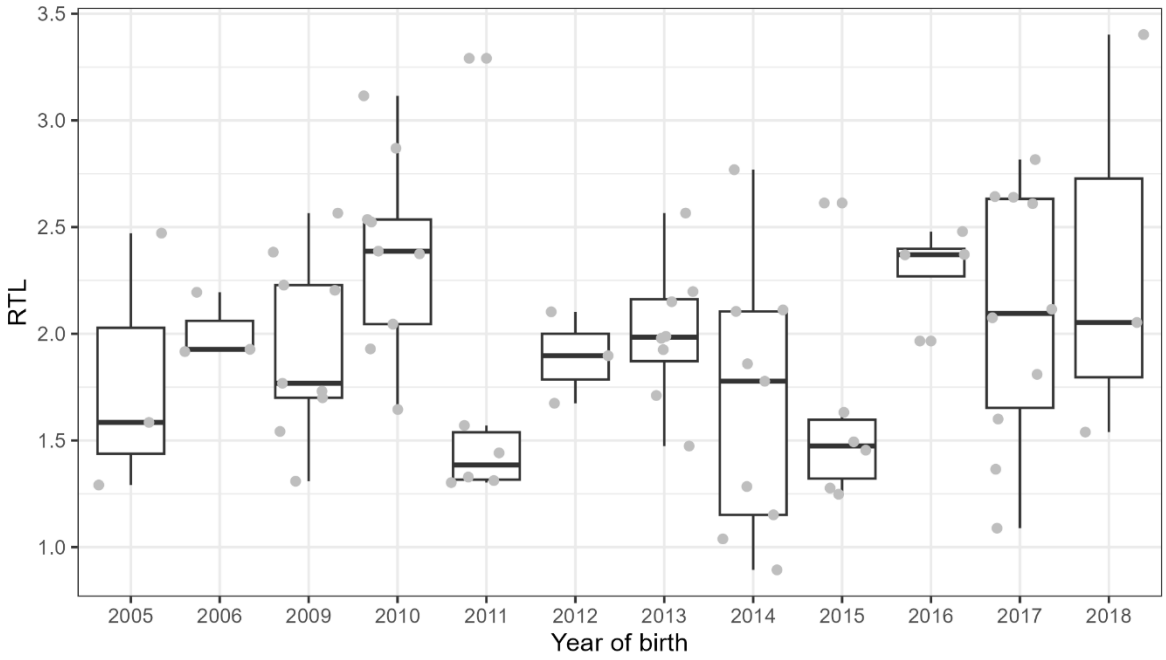

Figure S3.2 : Variation in age-corrected RTL with year of birth within 92 female adult (>1 year) greater snow geese. All data points are represented in gray and boxplots show median,1st and 3rd quartiles and 95% CI.

For this plot, we decided to represent RTL corrected by age. For this, we extracted the residuals of the linear model explaining RTL by age and added them to the average RTL. This enabled us to represent raw data but still show an age-independent effect. We used only years for which we had a minimum of 3 data points.

Annexe S3.3: Relationship between stress-induced CORT level and age

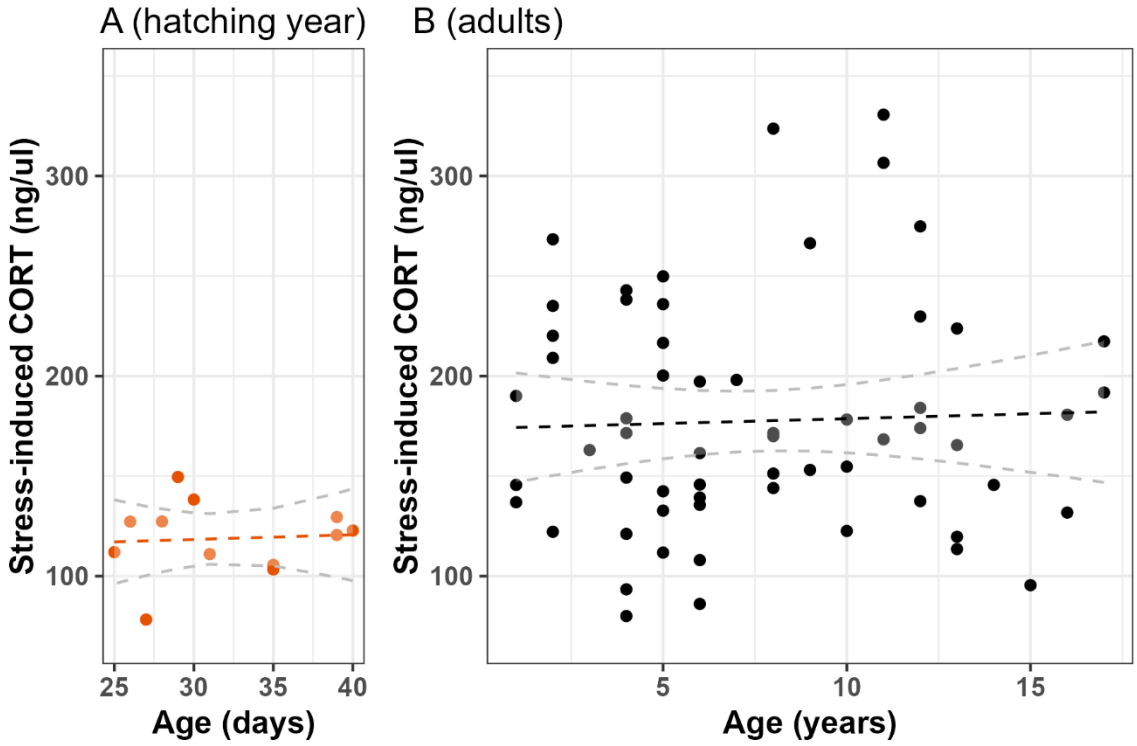

Figure S3.3: Relationship between stress-induced CORT level corrected by date of capture and age in A) hatching year females (orange; left panel) and B) adult females (black; right panel) greater snow geese. The estimates and 95% confidence interval are represented by dashed lines because they are non-significant. The distribution of stress-induced CORT values is represented for each age class (orange: hatching year females and black: adult females; right panel)

Annexe S4 – Matériel supplémentaire pour le Chapitre 4

Annexe S4.1 : Complementary information about relative telomere length extractions

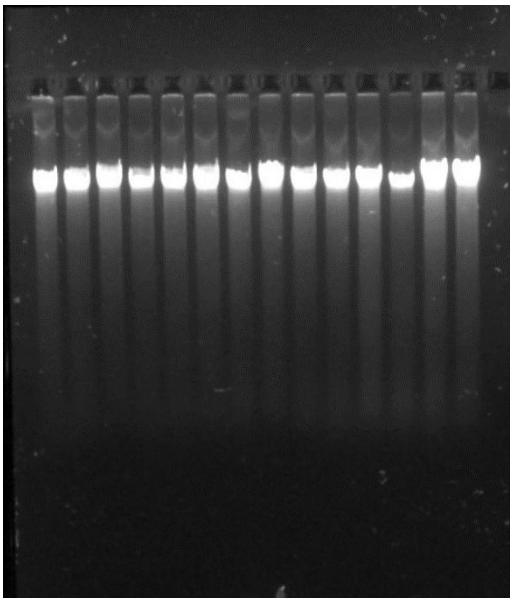

Figure S4.1 : Photograph from DNA migration of extracted and amplified telomere sequences on a subset of samples.

Table S4.1: Efficiencies and R2 of each qPCR plate. These values were obtained using the standard curve ranging from 0.3 to 40nmol/µL.

Plate	Telomeres		RAG1	
	Efficiency	R^2	Efficiency	R^2
P1	100.6%	0.987	104.4%	0.995
P2	95.4%	0.986	116.5%	0.971
P3	94.0%	0.986	110.5%	0.966
P4	88.8%	0.978	112.2%	0.947
P5	82.0%	0.983	99.1%	0.975

Annexe S4.2: PCA results for body condition index

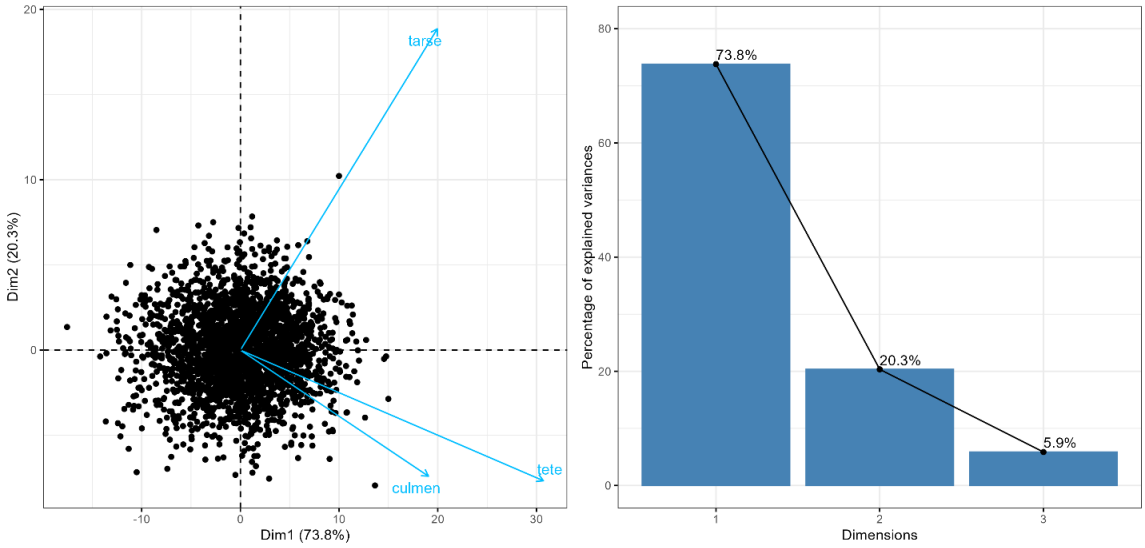

Figure S4.2: Graphical representation of the principal component analysis (PCA) of culmen, head and tarsus length in greater snow geese.

Annexe S4.3: Complete model selection for CMR survival models

Table S4.2: Complete results of model selection for survival in the first and second year after capture of greater snow geese marked in spring 2009 in relation to relative telomere length (RTL), body condition (COND), body size (SIZE) and experimental treatment (food addition; TRT, first year only). Survival in subsequent years (years 3 to 13) was considered constant (A). Model selection for the encounter probability was run beforehand and is presented in (B), where Time (T) was tested against constant encounter. K: number of parameters, ΔQAICc: difference in QAICc between the current and the top-ranked mode (N = 559).

1st year survival	2nd year survival	Recapture	Recovery	K	Deviance	ΔQAICc
intercept	intercept	T	Constant	14	2850.26	0.00
Constant	T	T	Constant	16	2845.53	0.28
Constant	Size	T	Constant	17	2843.01	0.31
TRT	Constant	T	Constant	17	2843.99	1.10
COND	Constant	T	Constant	16	2847.88	1.73
RTL	Constant	T	Constant	16	2847.44	1.83
SIZE	Constant	T	Constant	16	2847.58	1.95
Constant	Cond	T	Constant	17	2845.20	2.08
Constant	RTL	T	Constant	17	2845.39	2.24
TRT*RTL	Constant	T	Constant	20	2838.04	2.50
TRT+RTL	Constant	T	Constant	18	2843.71	2.94
TRT+COND	Constant	T	Constant	18	2843.84	3.05
TRT+SIZE	Constant	T	Constant	18	2843.89	3.10
Constant	$RTL+RTL^2$	T	Constant	18	2844.26	3.40
TRT*SIZE	Constant	T	Constant	20	2839.77	3.91
TRT*COND	Constant	T	Constant	20	2842.20	5.88
A) Model selection for recapture and recoveries						
TRT+SIZE+COND		T	Constant	26	2835.02	0.00
TRT+SIZE+COND		Constant	Constant	15	2865.81	2.07
TRT+SIZE+COND		T	T	37	2820.18	11.43
TRT+SIZE+COND		Constant	T	26	2850.30	12.43