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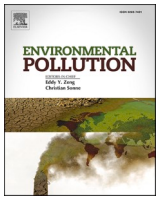

Goslings, but not adults, show physiological alterations associated with mercury contamination in wild Arctic barnacle geese (*Branta leucopsis*)

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A B S T R A C T

The Arctic is increasingly exposed to anthropogenic contaminants, yet their physiological effects on wildlife remain poorly understood. Mercury (Hg) is of particular concern because it accumulates in polar ecosystems and biomagnifies along food webs. We investigated associations between blood Hg concentrations and multiple physiological markers in wild barnacle geese (*Branta leucopsis*) breeding in Svalbard. Using blood samples from both adults and their goslings, we quantified Hg concentrations, oxidative damage (lipid peroxidation measured as 4-hydroxynonenal, 4-HNE), metabolomic profiles, and telomere length. Hg concentrations were higher in adults than in goslings. In goslings, but not adults, higher Hg levels were associated with lower 4-HNE, reduced concentrations of several metabolites, and a positive tendency in telomere length, suggesting age-specific physiological responses. These patterns in young may reflect compensatory antioxidant responses at low exposure levels, a coordinated metabolic constraint during rapid growth, and a possible growth-related processes influencing telomere dynamics. These findings suggest that even low Hg environmental exposure may be associated with alterations in physiological pathways during early life stages of wild birds.

1. Introduction

The Arctic, commonly perceived as a pristine and static environment, is increasingly affected by climatic changes and chemical contamination. High air temperatures are reshaping the Arctic landscape six to seven times faster compared to the rest of the world (Bradley et al., 2025). This is driven by multiple interacting physical mechanisms (Previdi et al., 2021), such as the loss of reflective sea ice, which in turn decreases the Earth's albedo and consequently increases the solar energy absorption by the ocean surface, amplifying regional warming and accelerating the sea-ice decline in a positive feedback loop (Dai et al., 2019). Additional mechanisms include heat transport by ocean currents (Beer et al., 2020), and a reduced Planck response (Pithan and Mauritsen, 2014). While climate change dominates discussions of Arctic transformation, chemical contamination represents a parallel and interacting pressure, the biological consequences of which are less well understood. Environmental contamination in the Arctic arises from two main pathways: local sources and long-range transport. Local sources include fossil fuel combustion, industrial activities such as mining and oil extraction, waste incineration, and emissions from maritime traffic, with consequent enhancements of pollutants as nitrogen oxides (NOx), sulphur dioxide (SO_2) and particulate matter (Law et al., 2017). Long-range transport allows pollutants emitted in distant regions to reach the Arctic via atmospheric circulation and ocean currents, resulting in the deposition and accumulation of persistent contaminants far from their original source (Li et al., 2024). These processes, combined with low temperatures, limited microbial activity, snow and ice cover (Imoobe et al., 2024) make Arctic ecosystems a global sink for contaminants (Varotsos and Krapivin, 2018), where pollutants are transported in and accumulate due to limited removal processes. Persistent substances can be taken up by organisms and, through bioaccumulation and biomagnification, are likely to reach elevated concentrations in species at high trophic levels.

A recognised and pervasive contaminant in the Arctic is mercury (Hg). Hg is released locally through industrial activities such as mining (Marquès et al., 2017), and can be transported from lower latitudes to

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the poles via atmosphere and ocean currents. Once in the Arctic, chemical and photochemical processes associated with sea ice, catalyse the oxidation of atmospheric gaseous Hg^0 into its reactive forms (Hg^{2+} and Hg^{+1}), which are then deposited in the environment (Ariya et al., 2004). These inorganic Hg forms can be converted by microbial processes in soils and aquatic systems into methylmercury (MeHg), the most toxic and bioavailable form of Hg (Clarkson and Magos, 2006). Wildlife are exposed to Hg primarily through the diet (Rice et al., 2014). Once absorbed in the gastrointestinal tract, Hg enters red blood cells, where it binds to erythrocyte haemoglobin (Hong et al., 2012). Due to its electrophilic nature, Hg can reacts with nucleophilic groups, such as thiols and selenols in proteins, altering their redox state, leading to oxidative stress and ultimately impairing the function of biomolecules (Farina and Aschner, 2019).

Field and laboratory studies have demonstrated that Hg exposure in animals can reduce reproductive output (Mills et al., 2020; Paris et al., 2018; Zabala et al., 2023), compromise immune (Han et al., 2023; Hawley et al., 2009; Ibañez et al., 2024; Lewis et al., 2013) and endocrine function (Nugegoda et al., 2025; Whitney and Cristol, 2018), as well as alter behaviour (Brittain et al., 2023; Grunst et al., 2026; Scheiber et al., 2018; Swaddle et al., 2017). Experimental studies in Arctic barnacle goslings *(Branta leucopsis)* have shown that goslings exposed to environmental background Hg concentrations exhibited altered behavioural stress responses compared to unexposed conspecific (Scheiber et al., 2018). Moreover, Hg concentrations were associated with altered immune responses and increased expression of pro-inflammatory genes (Han et al., 2023). In the same species, Hg has also been linked to neurochemical changes, including altered dopamine D2 receptor levels in the brain (van den Brink et al., 2018). Despite extensive evidence for phenotypic effects, the physiological mechanisms through which Hg influences wild populations of Arctic barnacle geese remain poorly understood, particularly regarding the cellular mechanisms involved.

Hg interacts with sulfhydryl groups of glutathione, an important antioxidant (Clarkson, 1997), but is also able to bind to cysteine residues in proteins and to cellular membranes (Notariale et al., 2022). This alterations potentially lead to compromised redox balance and altered cellular metabolism, with consequences on DNA sequences as telomeres. Telomeres are the end regions of linear chromosomes, which play a fundamental role in maintaining chromosome stability by protecting them from degradation and end-to-end fusions (Muraki et al., 2012). Telomere length gradually shortens with each cell division, due to the inability of DNA polymerases to fully replicate the ends of the lagging strand (Wynford-Thomas and Kipling, 1997), however, oxidative stress can exacerbate this shortening (von Zglinicki, 2002). Telomeres are particularly susceptible to oxidative damage (Barnes et al., 2019), as ROS tend to attack their rich guanine bases (Wang et al., 2010) generating nucleotide modification as the oxidation in the 8-oxo-7,8-dihydroguanine (8-oxo-dG) form (Singh et al., 2019). Such damage can interfere with DNA replication, causing further loss of telomeric repeats, and accelerate telomere shortening beyond that caused by the end replication problem alone (Barnes et al., 2019). However, although telomere shortening is generally considered the predominant age-related pattern, telomere lengthening has also been documented in several species, including house sparrows (*Passer domesticus*) (Pepke et al., 2023), Magellanic penguins (*Spheniscus magellanicus*) (Cerchiara et al., 2017), and Seychelles warblers (*Acrocephalus sechellensis*) (Brown et al., 2022). Several possible mechanisms may contribute to this phenomenon, such as telomerase activity which restore telomeric sequences (Rubtsova et al., 2012), increased turnover of hematopoietic cells (Ma et al., 2023), or other processes that remain poorly understood. The coexistence of telomere shortening and lengthening indicates that telomere length may be better viewed as a dynamic trait that reflects an individual's current physiological condition and the trade-offs associated with resource allocation and somatic maintenance. Telomere length dynamics therefore, could serve as a marker of cellular and organismal health, reflecting both the natural aging process and the cumulative impact of environmental stressors on physiology (Fasching, 2018). Although telomere length derives from cellular processes, its variation could also have consequences for the individual, reflecting overall physiological condition and predicting outcomes such as survival and mortality risk. A meta-analysis across wild vertebrate populations demonstrated that individuals with shorter telomeres face higher mortality risk, indicating that telomere length could not only reflect cellular health but can also be associated to survival and fitness outcomes (Wilbourn et al., 2018).

Here, we explored whether Hg contamination is associated with oxidative damage, metabolic variation, and telomere length in wild barnacle geese (*Branta leucopsis*) in Svalbard. Adults and goslings were captured, and a small blood volume was collected from the brachial vein. In red blood cells, we quantified Hg concentrations, 4-hydroxynonenal (a product of lipid peroxidation) as a biomarker of oxidative stress (Kim et al., 2015), profiled target metabolites, and assessed telomere length. We tested the following hypotheses: i) individuals with higher Hg concentrations in red blood cells would exhibit elevated 4-HNE levels, consistent with increased oxidative stress; ii) higher Hg concentrations would be associated with altered metabolic profiles, reflecting disruption of redox balance and impact on cell physiology; and iii) higher Hg concentrations would be associated with shorter telomeres, consistent with accelerated telomere erosion mediated by redox disruption, iv) the effects differ between adults and goslings. Together, these hypotheses aim to test whether Hg exposure in Arctic wildlife is associated with measurable changes in key biological pathways in an Arctic bird species, indicating that environmentally relevant concentrations of Hg may induce physiological effects.

2. Material and methods

2.1. Samples collection

The study was conducted in the area of Ny-Ålesund (78°55′N, 11°56′E), in Svalbard (Spitsbergen, Norway). The area is used by geese for nesting and post-breeding family aggregation. The samples were collected after the breeding season, when geese form family groups comprising of adults and goslings. Sampling was carried out over two days, on 31 July and 9 August 2024. Geese were captured using standard walk-in traps, weighed and measured (tarsus, head, body mass), and marked with uniquely numbered leg rings. Blood samples (around 200 μl) were collected from the brachial vein using a 25-gauge needle and a heparinized capillary tube. After handling, all individuals were safely released at the site of capture. Whole blood samples were centrifuged (microcentrifuge VWR® MiniStar silverline) for 10 min at 2000 g to separate plasma from erythrocytes, and samples were stored at −20 °C. For previously ringed adult birds, ring records were used to assign the minimal age of each animal, while for all goslings the age was set at 0 years. A total of 83 geese were sampled for this study from the tundra habitat, comprising 34 goslings and 49 adults. Sex distribution was similar across groups (goslings: 18 males, 16 females; adults: 23 males, 26 females), while adults spanned a wide age range, from 1 to 20 years, with most individuals between 1 and 3 years of age.

2.2. Mercury analysis

Total Hg (THg) was measured using a Direct Hg Analyzer (DMA-80, MLS Mikrowellen-Labor-Systeme GmbH, Germany), quantified as the absolute amount (ng) and subsequently expressed as concentration normalized by sample mass (μg of THg/kg of red blood cells). Around 0.02 g of red blood cells per sample (wet weight) were inserted into a nickel sample boat and weighed. In the DMA-80, samples were thermally decomposed in an oxygen stream, and Hg was quantified after gold amalgamation and thermal desorption using atomic absorption spectrometry at 253.7 nm.Analytical blanks (26 in total) were included

in each measurement batch, at the start and end of the run, and every 5–7 samples. The measurement included a total of 4 batches, defined as a continuous analytical run performed within a single day, including calibration, a sequence of samples, internal quality control samples, and procedural blanks. Accuracy was verified using certified reference materials (DORM-3; fish protein from the National Research Council of Canada, with certified Hg concentration), yielding mean recoveries (±SD) of 99.67 ± 1.64%. The certified reference material was analysed once per analytical batch, resulting in a total of four CRM measurements across the measurement. The laboratory's QA/QC procedures also included regular participation in the QUASIMEME international performance study program, consistently achieving satisfactory results. To further assess measurement reliability, a subset of samples (37 out of 92, corresponding to 40% of the samples) was analysed in a double-blind fashion. These samples were selected because sufficient biological material was available, while samples with limited remaining material were analysed only once to preserve material for additional analyses in future. Repeatability of the Hg levels obtained from these replicate measurements was high (R = 0.80, 95% CI = 0.67–0.89; permutation test P = 0.004). Median values of relative standard deviations (RSD) were 8.7% (IQR: 5.2–14.6) in adults and 14.0% (IQR: 11.3–24.5) in goslings. Values of Hg concentration are reported as μg/Kg of red blood cells.

2.3. LC-MS analysis

For each individual, 50 μL of 100% acetonitrile (Thermo Scientific Chemicals; Cat. No. 042266) was added to approximately 0.015 gr of red blood cells, which were then lysed by sonication for 60 s at 25 KHz in ice water. Afterwards, samples were centrifuged at 8.000 x g for 10 min, and the supernatant collected for measurement of both 4-hydroxynonenal (4-HNE) and metabolites. The LC-MS analysis was conducted on a Shimadzu LC-MS-8045 (Shimadzu, Kyoto, Japan).

To quantify 4-hydroxynonenal (4-HNE), a C18 type column was used (Phenomenex 1.7 μm 2.1 × 50mm). The mobile phases were A, Millipore H_2O + 0.1% FA (v/v), and B, acetonitrile + 0.1% Formic Acid (v/v). The analysis started at a flow of 0.3 mL/min as 95% A for 1 min, then dropped gradually to 0% A over 4 min, kept there for another 30 s and went back to 95% A in 30 s, for 4 min. Total run time was 10 min. The instrument was operated in positive ionization mode in the multiple reaction monitoring (MRM). 4-HNE was monitored at the [M+H]+ of mother to daughter products of 157.00 -> 55.15 (CE = −23 eV), 157.00 -> 69.15 (CE = −15 eV) and 157.00 -> 139.30 (CE = −13 eV). The presence of 4-HNE in samples was quantified using a standard calibration curve of pure 4-HNE (CAS: 75899-68-2, Merck) at the following concentrations: 0.0001, 0.001, 0.01, 0.1, 0.25, 0.5, 0.75, 1, 2.5 and 5 μM. The injection volume used was 1 μL. Chromatographic peaks corresponding to 4-HNE were identified using all fragments within the same retention time as that of pure standards. Peak areas were used to interpolate concentrations based on the standard curve and expressed as μg of 4-HNE per gram of erythrocytes. Peak area showed a high repeatability across replicates (R = 0.995, 95% CI = 0.983–0.998; permutation test P = 0.001). Values of 4-HNE concentration are reported as μM of 4-HNE per gram of red blood cells.

Targeted amino acid calibration standards were prepared using an Amino Acids Mix Solution (Merck, 79248). Primary metabolites were analysed by LC/MS by using the method package "LC-MS/MS Method Package for Primary Metabolites Ver. 2.0 (Shimadzu Corporation, 2019)". In brief, 1 μL of sample is introduced into a Shimadzu LCMS 8045 using MilliQ Water with 0.1% Formic Acid and Acetonitrile with 0.1% Formic Acid as mobile phases. A Discovery HS F5-3 (3 μm × 150 mm x 2.1 mm, Supelco, 567503-U) column was used as the analytical column. The stock solution contained 17 amino acids, each at 2.5 mM in 0.1 M HCl and cystine at 1.25 mM. Serial dilutions were prepared to obtain eight calibration levels ranging from 0.005 to 10 μM. Targeted metabolomic analysis was performed following the manufacturer's LC/MS/MS Method Package, were the amino acids are quantified using external calibration curves. Linear regression showed high correlation coefficients (R^2) between 0.987 and 0.999 across analytical replicates. Amino acids (aspartic acid, serine, alanine, cysteine, glycine, histidine, threonine, glutamic acid, proline, lysine, arginine, valine, methionine, tyrosine, isoleucine, leucine, phenylalanine) were quantified using calibration curves and expressed as μM per gram of red blood cells. Organic acids (malic, pyruvic, lactic, uric, and citric acid) and asparagine could not be absolutely quantified due to the lack of certified standards and are therefore reported as peak areas normalized to red blood cell mass, providing a semiquantitative estimate of their abundance.

2.4. Telomere length quantification

Genomic DNA was isolated from 5 μL of red blood cells using the Blood DNA Preparation Column Kit (Jena Bioscience GmbH), according to the manufacturer's instructions. The DNA concentration and purity were assessed with a NanoDrop-One spectrophotometer (Thermo-Scientific). Telomere length was measured by quantitative real-time amplification method (qPCR) (Cawthon, 2009), using GAPDH as the reference single-copy gene (Atema et al., 2013). As species-specific GAPDH primers were not available for barnacle geese, primers were designed using Primer3 (v2.4.0) via the Primer3 web interface (release 4.1.0). Due to the lack of a fully annotated genome for *Branta leucopsis*, primer design was based on a homologous genomic sequence from the phylogenetically closely related species *Anser cygnoides* (isolate HZ-2024a). The GAPDH primers were 5′- TGAAAATTGTCAGGTAAGGA GAAGG-3′ (forward) and 5′-AGTACAGATGCTTTTCTTCACACAT-3′ (reverse). Telomeres were amplified using the primers TEL1 (5′-CGGTTTGTTTGGGTTTGGGTTTGGGTTTGGGTTTGGGTT-3′) and TEL2 (5′-GGCTTGCCTTACCCTTACCCTTACCCTTACCCTTACCCT-3′) (Criscuolo et al., 2009). The qPCR was performed using a Bio-Rad CFX Opus 384 (Bio-Rad) using 5 ng of DNA with 1 μL of each primer (forward and reverse) at a concentration of 10 μM, in 10 μL of QuantiFast mix with SYBR green, with a final reaction volume of 20 μL. Each sample was run in triplicate, and for each individual, GAPDH and telomere reactions were processed in the same plate. The qPCR program consisted of an initial denaturation at 95 °C for 5 min, followed by 40 cycles of 95 °C for 10 s, 60 °C for 15 s, and 72 °C for 60 s, and a subsequent melt curve from 72 °C to 95 °C in 1 °C increments, with pre-melt conditioning (90 s at the first step and 5 s thereafter). Means of Ct values were retained for statistical analysis if the difference between duplicates was ≤0.3 Ct (Rampazzo et al., 2010). Runs efficiencies were between 95 and 110%, with R^2 between 0.97 and 0.99. Telomere length was expressed as the telomere-to-single-copy gene ratio (T/S), calculated as 2^(−ΔCt), where ΔCt is the difference between Ct value of telomere reaction minus the Ct value of the GAPDH reaction. This metric reflects the relative amount of telomeric DNA compared with the stable GAPDH amplicon. T/S values were further normalized against an internal control sample to correct for intra- and inter-assay variation (Criscuolo et al., 2009).

2.5. Statistical analysis

All statistical analyses were performed in R (version 4.4.2). Significance was taken at $\alpha \leq 0.05$. To summarize variation in metabolite concentrations, Principal Component Analysis(PCA) was performed including the 23 metabolites analysed (amino acids: asparagine, aspartic acid, serine, alanine, cysteine, glycine, histidine, threonine, glutamic acid, proline, lysine, arginine, valine, methionine, tyrosine, isoleucine, leucine, phenylalanine; organic acids: malic acid, pyruvic acid, lactic acid, uric acid, citric acid). The Kaiser-Meyer-Olkin (KMO) index was 0.83, and Bartlett's test confirmed that the correlation matrix differed significantly from an identity matrix ($\chi^2 = 3079$, df = 253, $p < 0.001$), indicating that dimensionality reduction was appropriate. The first principal component explained 51% of total variance and reflected

coordinated variation across amino acids and metabolites, and thus extracted for subsequent analyses as a summary index of the individual metabolic profile. Body condition was calculated by dividing body mass by tarsus length cubed, a common measure of energy reserves and health status in birds, representing animal mass corrected for structural size (Labocha and Hayes, 2012).

To test the possible effect of Hg concentration on variation of 4-HNE, metabolite profile (PC1), and telomere length, three separate linear models were fitted, one for each physiological trait. This included, as independent, variables the interaction between Hg concentration and ontogenetic stage (adults or goslings), the interaction between body condition and ontogenetic stage, sex, and age. As was not possible to estimate individual age for goslings, but all hatched within the same short breeding period, their age was collectively set as zero. In this way, the age term in the models reflects the age only within adults. For adults, age was defined based on the year of first appearance on the island, which provides a reliable proxy of chronological age. The interaction between Hg concentration and ontogenetic stage was included to test whether the effect of Hg differed between adults and goslings. Similarly, the interaction between body condition and ontogenetic stage allowed us to assess whether the relationship between body condition and each physiological biomarker varied across life stages. Model residuals were checked for normality using the Shapiro–Wilk test and for heteroscedasticity using the Breusch–Pagan test. The dependent variables 4-HNE and telomere length were log-transformed to improve normality of the models' residuals. For the metabolite model a robust linear model was fitted using the *lmrob* function from the *robustbase* package (Maechler et al., 2021) to account for deviations from normality and heteroscedasticity in the residuals. The first principal component (PC1) as dependent variable was not log-transformed to improve normality of the model residuals because principal component is centered and scaled by construction and can take negative values, making logarithmic transformation inappropriate in this case.

The first component (PC1) showed consistently positive and relatively homogeneous loadings across metabolites, indicating a general metabolic concentration gradient. The highest contributions came from several amino acids (histidine, leucine, isoleucine, proline, tyrosine, phenylalanine, methionine, arginine, alanine) and from two organic acids (lactic and pyruvic acid). To complement the PCA and identify which metabolites varied with Hg concentration in each life stage, we ran separate univariate linear models for each metabolite in adults and goslings. Metabolite concentrations were scaled to account for differences in variance structure between adults and goslings. For adults, each metabolite was modelled as a function of Hg concentration, body condition, sex, and age; for goslings, as a function of Hg concentration, body condition and sex. For each model, the regression coefficient and p-value for the effect of Hg concentration was extracted, and the p-values corrected for multiple testing using the Benjamini–Hochberg false discovery rate (FDR). Pyruvic acid could not be modelled in goslings because LC-MS peak intensities were too low or undetectable in this age class, resulting in missing values for all gosling samples.

3. Results

3.1. Adults show higher Hg concentrations than goslings

Hg concentrations ranged from 0.72 to 14.90 μg/kg (mean = 2.36 μg/kg, median = 1.98 μg/kg, SD = 1.92 μg/kg), with a moderately right-skewed distribution characterized by predominantly low to intermediate values and with a few high Hg concentrations in adults. Adults had higher Hg concentrations than goslings (Welch two-sample *t*-test: t = 3.46, df = 65.03, p = 0.001; 2.82 vs 1.60 μg/kg). No differences in Hg concentrations were detected between males and females in adults (Welch two-sample *t*-test: t = −0.37, df = 42.59, p = 0.716; males: 2.70 μg/kg, females: 2.93 μg/kg; 95% CI: −1.51 to 1.04 μg/kg) or goslings (t = 0.49, df = 27.39, p = 0.628; males: 1.67 μg/kg, females: 1.52 μg/kg; 95% CI: −0.45 to 0.73 μg/kg).

3.2. 4-HNE was associated with Hg levels in goslings but not in adults

In adults (the reference group in the model), Hg concentration did not show a significant association with 4-HNE. Goslings had a lower baseline 4-HNE levels than adults, and body condition was not associated with 4-HNE levels in both adults and goslings, nor were there any differences between males and females. Age also did not show a significant effect on Hg concentrations in adults. The interaction between Hg concentration and ontogenetic stage indicated that the relationship between Hg and 4-HNE differed between adults and goslings, with an overall slope of approximately – 0.85 for goslings. The interaction between body condition and ontogenetic stage provided no indication that the relationship between body condition and lipoperoxidation differed between adults and goslings. The model accounted for 49% of the variation in log-transformed 4-HNE (adjusted $R^2 = 44\%$), and showed a strong overall fit ($F_{7,71} = 9.83$, $p = < 0.001$). The model outcome is reported in Table 1.

3.3. Metabolite concentrations were associated with Hg levels in goslings, but not adult geese

The metabolomic profile further supported an ontogenetic divergence in physiological responses to Hg exposure. Goslings showed lower values along the main metabolic gradient (PC1 values), reflecting reduced concentrations of amino acids and organic acids compared to adults. In adults, PC1 was not associated with Hg concentration. In contrast, the interaction between Hg and ontogenetic stage showed that goslings exhibited a more negative association between Hg and metabolic gradient, pointing to a shift in their metabolic profile with increasing Hg levels. Body condition was negatively associated with ammino and organic acids concentrations in adults, and this relationship did not differ between age classes. The robust model explained 71% of the variation in the metabolic gradient (adjusted $R^2 = 0.68$), with results reported in Table 2.

To identify which metabolites contributed to the differences between goslings and adults in their association between Hg and metabolome, the PCA was complemented with a set of univariate linear models run separately for ontogenetic class. For each metabolite, the association between its concentration and Hg levels was tested, while controlling for body condition, sex, and age (age included only in adults). This approach allowed the quantification of metabolite-specific responses to Hg exposure and to compare effect sizes between life stages. The resulting pattern revealed a divergence between adults and golsings. In goslings, several amino acids showed negative associations with Hg concentration after FDR correction, indicating a possible coordinated reductions in metabolite concentration with increasing Hg. In contrast, only few metabolite showed a positive association with Hg in adults. The direction and magnitude of metabolite-specific slopes are summarized in the heatmap in Fig. 1, and full model outputs for each metabolite are provided in Table 3.

Table 1
Results from linear model explaining variation in log-transformed 4-HNE concentrations in adult geese and goslings. Estimates (β), 95% confidence intervals (CI), and p-values are reported for each predictor. All numeric predictors were scaled prior to analysis. Significant effects are reported in bold.

Predictors	Estimates	CI	p
Intercept	0.09	−0.37 – 0.55	0.684
Hg concentration	0.09	−0.14 – 0.32	0.421
Size = gosling	**−2.80**	**−4.26 – -1.34**	**<0.001**
Body condition	−0.30	−0.72 – 0.12	0.153
Sex = female	−0.14	−0.55 – 0.27	0.509
Age in adults	−0.18	−0.42 – 0.07	0.150
Hg concentration × size = gosling	**−0.94**	**−1.79 – -0.09**	**0.031**
Size = gosling × body condition	−0.28	−1.64 – 1.09	0.687

Table 2
Results from robust linear model explaining variation in metabolic profile (PC1) in adult geese and goslings. Estimates (β), 95% confidence intervals (CI), and p-values are reported for each predictor. All numeric predictors were scaled prior to analysis. Significant effects are reported in bold.

Predictors	Estimates	CI	p
Intercept	2.91	1.35 – 4.46	<0.001
Hg concentration	0.04	−0.47 – 0.54	0.882
Size = gosling	**−6.22**	**−7.89 – -4.55**	**<0.001**
Body condition	**−1.82**	**−2.98 – -0.67**	**0.002**
Sex = female	−0.18	−0.82 – 0.46	0.575
Age in adults	0.27	−0.60 – 1.14	0.542
Hg concentration × size = gosling	−1.30	−1.99 to −0.62	**<0.001**
Size = gosling × body condition	1.15	−0.29 – 2.59	0.116

3.4. No associations between Hg and telomere length was detected in either goslings or adults

Telomere length showed no clear association with Hg concentration in adult geese. Body condition, sex, and age were also not related to telomere variation. However, the interaction between Hg and ontogenetic stage indicated a tendency for goslings to a positive relationship between Hg and telomere compared to adults. Overall, the model explained around 11% of the variation in telomere length (adjusted R^2 = 1%) but showed weak overall fit ($F_{7,66} = 1.13$, $p = 0.356$). The model outcome is reported in Table 4.

An overview of the descriptive statistics for all measured variables, including the mean, standard deviation, median, and range, is provided in Table S1 of the supplementary materials. A graphical overview of the relationships between Hg concentrations and physiological traits is reported in Fig. 2.

4. Discussion

The Arctic, long considered a pristine and unpolluted environment, is increasingly affected by both local and long-range anthropogenic contaminants (Muir et al., 2025). Evaluating the effects of these contaminants on local wildlife is essential, as many species have evolved under low exposure to anthropogenic chemicals, and may consequently have limited physiological capacity to cope with novel or increasing contaminants burdens. This study aimed to test whether Hg levels in Arctic geese are associated with oxidative damage, metabolic variation and telomere length. We found age-dependent associations between Hg exposure and physiological variables. In adult geese, 4-HNE and telomere length were not affected by blood Hg concentrations, while metabolite profiles were primarily influenced by body condition. In goslings, higher Hg concentrations were associated with lower 4-HNE, reduced metabolite profile, but not with telomere length.

Hg concentrations in our study ranged between 0.72 and 14.90 μg/kg of red blood cells, and were lower than those previously reported in literature for other Arctic birds in Svalbard. A broader synthesis of Hg contamination in Arctic sea and shorebirds (Chastel et al., 2022) report that blood concentrations (dry weight) in northern fulmars (*Fulmarus glacialis*) ranged for instance between 70 and 410 μg/kg, from 110 to 660 μg/kg in glaucous gulls (*Larus hyperboreus*), and from 360 to 2830 μg/kg in black-legged kittiwakes (*Rissa tridactyla*). In alcids, concentrations were generally lower, with ranges from 10 to 230 μg/kg in Brünnich's guillemots (*Uria lomvia*), 100 to 260 μg/kg in common murres (*Uria aalge*), 40 to 150 μg/kg in little auks (*Alle alle*), and 60 to 240 μg/kg in Atlantic puffins (*Fratercula arctica*). Common eiders (*Somateria mollissima*) exhibited a range between 50 and 340 μg/kg. In a previous study on Barnacle goose in Svalbard (Han et al., 2023), hepatic Hg concentrations ranged from 9 to 13 μg/kg (dry weight). Although comparisons among tissues should be interpreted with caution, the concentrations measured in the present study are broadly consistent with previous estimates for the same species, and indicate relatively low Hg exposure compared with many Arctic seabirds. These relatively low concentrations are likely consistent with the trophic ecology of barnacle geese, which are predominantly herbivorous and therefore occupy a lower trophic position compared with piscivorous or benthic-feeding seabirds, where biomagnification of Hg leads to higher exposure levels.

Hg is known to perturb the cellular redox balance by binding to thiol (-SH) groups in proteins (Ajsuvakova et al., 2020), depleting crucial antioxidants like the glutathione (Farina and Aschner, 2019), and disrupting other enzymes involved in the antioxidant system (e.g. thioredoxin reductase), leading to oxidative stress (Rupa et al., 2023). A useful biomarker of oxidative stress is 4-HNE (Kim et al., 2015), a product of lipid peroxidation (Schaur et al., 2015). In addition, 4-HNE may also have a role in signaling, acting as a second messenger of oxidative stress, triggering antioxidant defense pathways that limit both its own formation and strengthen cellular protection against oxidative damage (Csala et al., 2015). In adult geese, Hg concentration was not associated with 4-HNE levels, suggesting that Hg exposure may not be a major driver of lipid peroxidation at the concentrations observed in this study. In goslings, however, Hg concentration was negatively associated with 4-HNE levels. This pattern could reflect differences in oxidative

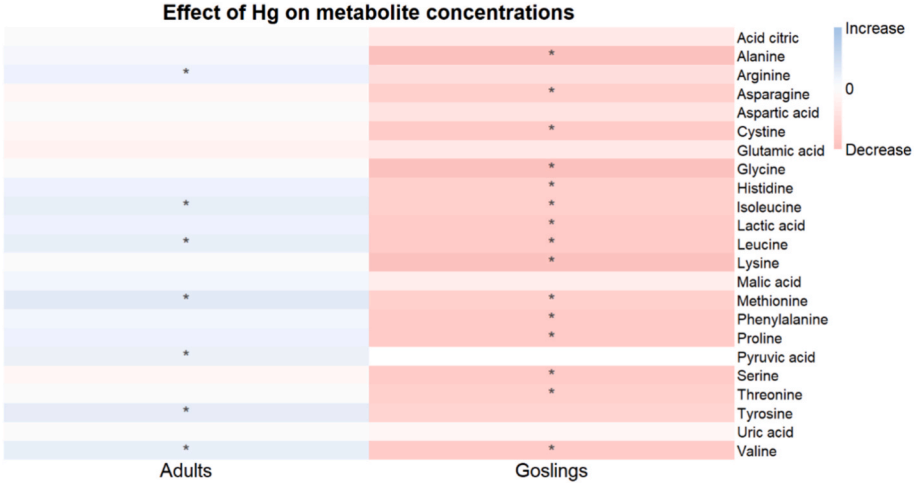

Fig. 1. Heatmap showing standardized regression coefficients (β) for the effect of Hg concentration on each metabolite, obtained from linear models controlling for relevant covariates (adults: body condition, sex, and age; goslings: body condition and sex). Positive values in blue indicate higher metabolite concentrations with increasing Hg, whereas negative values in red indicate decreases. Asterisks indicates metabolites with a significant Hg effect after FDR correction (FDR <0.05). (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

Table 3
Results of univariate linear models testing the association between Hg concentration and each metabolite, analysed separately for adults and goslings. All metabolite concentrations were z-standardized prior to modelling, allowing effect sizes (β) to be directly comparable across compounds. Reported β, 95% confidence intervals (CI), and FDR-corrected p-values refer to the coefficient of Hg concentration only. Pyruvic acid could not be modelled in goslings because LC–MS peak intensities were undetectable in all juvenile samples, as shown by the NA.

	Adults			Goslings		
Metabolite	*β*	*CI*	*p*	*β*	*CI*	*p*
Acid citric	0.01	−0.25 – 0.27	0.926	−0.47	−1.41 – 0.47	0.350
Alanine	0.15	−0.11 – 0.41	0.361	**−1.39**	**−2.16 – -0.62**	**0.010**
Arginine	0.27	0.06 – 0.49	0.044	−0.70	−1.63 – 0.23	0.180
Asparagine	−0.14	−0.38 – 0.11	0.390	**−1.06**	**−1.95 – -0.18**	**0.033**
Aspartic acid	−0.08	−0.33 – 0.17	0.702	−0.62	−1.53 – 0.30	0.222
Cystine	−0.14	−0.38 – 0.11	0.390	**−1.18**	**−2.01 – -0.34**	**0.018**
Glutamic acid	−0.24	−0.50 – 0.01	0.113	−0.53	−1.46 – 0.40	0.294
Glycine	−0.05	−0.31 – 0.22	0.847	**−1.46**	**−2.23 – -0.68**	**0.010**
Histidine	0.27	0.02 – 0.51	0.076	**−1.06**	**−1.93 – -0.20**	**0.029**
Isoleucine	**0.36**	**0.12–0.60**	**0.027**	**−1.11**	**−1.96 – -0.26**	**0.025**
Lactic acid	0.26	0.01 – 0.52	0.082	**−1.21**	**−2.05 – -0.37**	**0.018**
Leucine	**0.33**	**0.09–0.57**	**0.035**	**−1.23**	**−2.06 – -0.39**	**0.018**
Lysine	0.05	−0.19 – 0.30	0.806	**−1.36**	**−2.18 – -0.54**	**0.011**
Malic acid	0.23	−0.01 – 0.47	0.113	−0.44	−1.39 – 0.52	0.374
Methionine	**0.42**	**0.20–0.65**	**0.009**	**−1.04**	**−1.94 – -0.15**	**0.038**
Phenylalanine	0.19	−0.07 – 0.45	0.252	**−1.21**	**−2.05 – -0.36**	**0.018**
Proline	0.29	0.04 – 0.54	0.065	**−1.20**	**−2.04 – -0.37**	**0.018**
Pyruvic acid	**0.30**	**0.06–0.55**	**0.044**	NA	NA	NA
Serine	−0.13	−0.39 – 0.13	0.435	**−1.22**	**−1.95 – -0.48**	**0.011**
Threonine	0.04	−0.22 – 0.30	0.847	**−1.09**	**−1.95 – -0.23**	**0.028**
Tyrosine	**0.39**	**0.16–0.62**	**0.016**	−0.93	−1.84 to −0.03	0.061
Uric acid	−0.03	−0.30–0.23	0.847	−0.14	−1.08 – 0.81	0.770
Valine	**0.34**	**0.11–0.57**	**0.027**	**−1.15**	**−2.02 – -0.29**	**0.024**

Table 4
Results from robust linear model explaining variation in telomere length in adult geese and goslings. Estimates (β), 95% confidence intervals (CI), and p-values are reported for each predictor. All numeric predictors were scaled prior to analysis.

Predictors	Estimates	CI	p
Intercept	0.14	−0.06 – 0.35	0.169
Hg concentration	0.00	−0.10 – 0.11	0.927
Size = gosling	0.21	−0.50 – 0.92	0.551
Body condition	0.02	−0.17 – 0.22	0.826
Sex = female	0.06	−0.14 – 0.25	0.546
Age in adults	0.07	−0.03 – 0.17	0.147
Hg concentration × size = gosling	0.48	−0.06 – 1.01	0.083
Size = gosling × body condition	0.05	−0.60 – 0.70	0.875

physiology between age classes, or alternatively indicate a non-linear response to Hg contamination, as a compensatory response in terms of increased antioxidants. Such a hypothesis is supported by previous studies in which different animal species (*Mus musculus*, *Pseudosciaena crocea* and *Gavia immer*), exposed to Hg showed an increase in antioxidant enzymes in a dose dependent manner (Hussain et al., 1999; Kenow et al., 2008; Wu et al., 2018). This pattern may also reflect developmental differences in oxidative physiology, whereby rapidly growing individuals, in better condition due to higher genetic quality or a more nutrient-rich diet, uptake more Hg from diet but can also allocate more resources to protection from oxidative damage. Some individuals may therefore accumulate higher Hg concentrations in blood, while potentially also exhibiting greater capacity to counteract oxidative stress via enhanced antioxidant defences. Anyway, in the interpretation of these patterns, it is important to consider that given the low Hg concentrations measured in our study, the observed patterns likely reflect responses to low-level Hg exposure, which may diverge from the classical toxicological effects of Hg.

We used targeted metabolomics of red blood cells for amino acids and organic acids to provide a snapshot of the cellular metabolic state. Amino acids and organic acids are linked to fundamental cellular processes as they reflect protein turnover, energy metabolism, redox balance, and nitrogen handling. Variations in metabolite concentrations can indicate shifts in cellular homeostasis, oxidative stress, and metabolic reprogramming. Metabolite pools have been shown to be sensitive to environmental stressors, including exposure to metal contaminants (Kim et al., 2024). In particular, perturbations in amino acid profiles and organic acid intermediates are considered early and integrative markers of metal-induced cellular stress, as they capture downstream consequences of oxidative damage, impaired energy production, changes in anabolic–catabolic balance and protein turnover (Akash et al., 2023). In adult geese, variation in the metabolites in red blood cells was associated with individuals' body condition, but not with Hg levels. Geese in better condition showed lower metabolites scores, suggesting reduced concentrations of amino acids and organic acids in red blood cells. This pattern may be consistent with the idea that adults in good physiological state may rely less on circulating metabolites, either because they sustain more efficient metabolic regulation or because they experience lower rates of protein and energy turnover. In contrast, goslings did not show a comparable association between body condition and metabolite variation, likely because their metabolic profiles are dominated by the demands of rapid growth. However, in goslings, metabolites concentration in red blood cells, summarized by the first PCA axis, were negatively associated with Hg concentration. Because the main axis explained more than half of the total variance, the observed relationship may suggests that Hg exposure is associated with broad changes in metabolic state rather than alterations in only a few compounds. This interpretation is further supported by single metabolite models analysis, which revealed how a large subset of metabolites displays significant negative associations with Hg. Specifically, the amino acids alanine, asparagine, cysteine, glycine, histidine, isoleucine, lactic acid, leucine, lysine, methionine, phenylamine, proline, serine, threonine and valine decrease significantly with increasing Hg concentrations. This represents a decrease of approximately 60% of the overall measured metabolites, indicating a coordinated metabolic response. This pattern may be consistent with consistent with a systemic limitation of anabolic processes or a reallocation of resources toward maintenance and stress mitigation. In contrast, adults show a much weaker and less consistent pattern, with only few metabolites associated with Hg in a positively way, as arginine, isoleucine, leucine, methionine, pyruvic acid, tyrosine and valine, which represent about the 30% of the metabolites analysed. The absence of a coordinated decline across metabolites, together with positive associations between Hg concentration and few metabolites, may indicate a greater capacity of adults to buffer or compensate for Hg exposure. Taken together, these findings suggests that Hg exposure does not uniformly affect metabolic activity across life stages, but instead

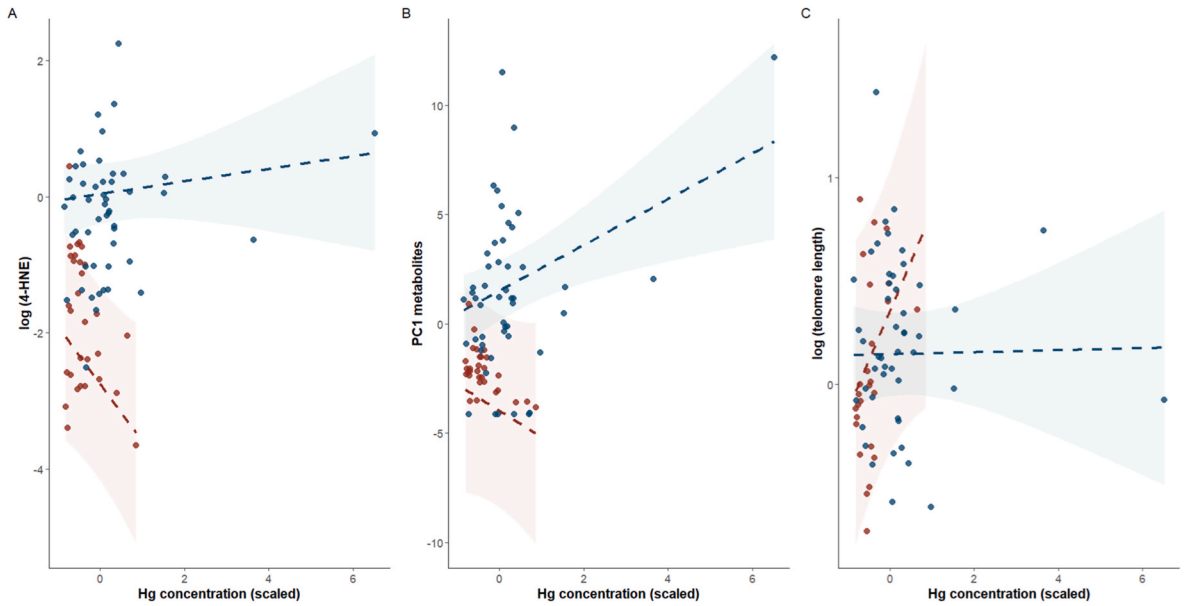

Fig. 2. Relationships between Hg concentration and physiological traits in adult geese (blue) and goslings (red), based on linear models including Hg concentration, ontogenetic stage, body condition, sex, and age. Lines represent model predictions within the observed range of Hg values for each age class, with shaded areas being the 95% confidence intervals. Points represent observed data. (A) Oxidative damage (log-transformed 4-HNE) decreased with Hg concentration in goslings but not in adults. (B) The metabolic gradient (PC1) showed a negative association with Hg in goslings. (C) Telomere length showed no clear association with Hg in adults, whereas goslings showed a tendency toward a positive relationship between telomere and Hg concentration. (For interpretation of the references to colour in this figure legend, the reader is referred to the Web version of this article.)

induces a metabolic constraint which depend on the ontogenetic stage.

We hypothesized that a higher Hg burden would be associated with shorter telomeres through oxidative mechanisms. However, no clear association was detected in adults and goslings, even though the interaction between Hg and ontogenetic class showed a positive trend, although not statistically significant. Several hypotheses could account for the lack of an observed relationship between Hg concentrations and telomere lengths in red blood cells. First, Hg concentrations in this study population may have been too low to induce detectable telomere erosion, even though they were sufficient to induce oxidative damage and alter metabolic profiles in goslings. Therefore, the absence of a statistically significant relationship may likely indicate that a biologically meaningful association between Hg exposure and telomere length in red blood cells was not detectable within the range of Hg concentrations observed in our study. Second, individuals may have maintained telomere length through regulatory or compensatory mechanisms, thereby masking direct negative effects of Hg on telomere attrition. Telomere length does not only reflect biological age or cumulative damage, but also the capacity of an organism to buffer physiological insults and maintain function (Boonekamp et al., 2013). Moreover, both short telomere length (Bauch et al., 2022) and longer telomeres (Davis et al., 2025) have previously been linked to Hg exposure in birds. Consequently, a single measurement of absolute telomere length may not accurately capture the physiological burden of environmental stressors, as it is difficult to separate environmentally induced effects from the variation in inherited telomere length. Telomere dynamics, rather than single measurements, could better reflect cumulative physiological stress (Boonekamp et al., 2014), and has been shown to predict survival and life-history outcomes in natural populations (Bize et al., 2009). While logistical constraints inherent to working on free-living Arctic wildlife precluded repeated sampling of the same individuals, our findings highlight the importance of longitudinal approaches for distinguishing changes in telomere length associated with environmental stressors from variation in telomere length established early in life and influenced by genetic factors.

The physiological mechanisms discussed may have ecological implications. Goslings represent a critical life stage during which acquisition of physiological reserves can influence subsequent survival and recruitment. The observed associations between Hg concentrations and both oxidative status and metabolic profiles suggest that exposure to environmentally relevant Hg concentrations may alter specific physiological processes during development. Although we did not measure traits directly related to fitness, changes in metabolism during early life could potentially influence growth trajectories or the ability to cope with additional environmental stressors. Such sublethal effects may interact with other environmental pressures and contribute to variation in individual performance and population processes. This may be particularly relevant in barnacle geese, which will leave their Arctic breeding grounds in late August to reach the wintering areas in northwestern Europe. The successful completion of the migration depends on the accumulation of sufficient energetic reserves during development. While these potential ecological consequences remain speculative, they highlight the importance of considering how physiological changes associated with contaminants in environment during early life may scale up to influence later life-history stages. We acknowledge that the observational design of this study precludes direct causal inference between Hg concentrations and the observed physiological responses. The interpretation of the results might therefore be influenced by inter-individual biological variability and the consequently limited signal-to-noise ratio, particularly regarding the association between Hg exposure and telomere length. Another possible limitation lies in the low concentrations of mercury in the blood of the geese. While these concentrations were associated with measurable physiological responses in goslings, the resulting patterns may differ from those typically reported under higher exposure regimes, making their biological interpretation less straightforward. Nevertheless, our findings indicate that even background Hg exposure in the Arctic is associated with measurable physiological responses in a free-living bird species during early life stages.

5. Conclusion

In this study we have show how in a remote Arctic ecosystem, low-level environmental Hg concentrations are associated with

physiological variation in a free-living bird species, the barnacle geese (*Branta leucopsis*). Our results indicate possible ontogenetic differences in physiological responses to Hg. Adults exhibited higher Hg concentrations than goslings, however, Hg was not associated with 4-HNE level, cellular metabolism, or telomere length variation. In contrast, although goslings had lower blood Hg concentrations, Hg covaried with alterations in 4-HNE levels and metabolic profiles, while no association with telomere length was detected. By integrating biomarkers reflecting oxidative damage, cellular metabolism, and somatic maintenance, we captured multiple dimensions of physiological state from a single blood sample per individual. Taken together, these results suggest that early life stages may be more physiologically sensitive to Hg exposure than adults. Although Hg concentrations were relatively low and unlikely to induce toxic effects, they were nevertheless associated with variation in oxidative status and metabolism in goslings. More broadly, our findings indicate that even in ecosystems perceived as pristine, subtle physiological perturbations in wild animals can arise from chronic exposure to low contaminant levels, potentially providing early warning signals of environmental change.

CRediT authorship contribution statement

Matteo Schiavinato: Writing – original draft, Visualization, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Sandra Bouwhuis:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Methodology, Investigation, Formal analysis, Data curation. **Wouter Bakker:** Resources, Methodology, Investigation, Data curation. **Elena Johnson-Gittins:** Methodology, Investigation, Data curation. **Sebas Wesseling:** Resources, Methodology, Investigation, Data curation. **Peter Schupp:** Resources, Methodology, Investigation, Data curation. **David Spurgeon:** Writing – review & editing, Validation, Supervision, Investigation, Funding acquisition. **Maarten JJE. Loonen:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation. **Nico van den Brink:** Writing – review & editing, Validation, Supervision, Resources, Project administration, Investigation, Funding acquisition.

Ethics

The sampling was carried out in accordance with EU Directive 2010/63/EU for animal experiments. The study was performed under the ethical approval permit FOTS 30851, issued by the Norwegian Food Safety Authority (Mattilsynet).

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Matteo Schiavinato reports financial support was provided by Koninklijke Nederlandse Akademie van Wetenschappen (KNAW). Maarten J J E Loonen reports financial support was provided by Netherlands Polar Programme of NWO. Matteo Schiavinato, David Spurgeon, Nico van den Brink reports financial support was provided by European Union Horizon (2020), Marie Sklodowska Curie Actions. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envpol.2026.128803.

Data availability

Dataset and R code have been deposited as supplementary materials.

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