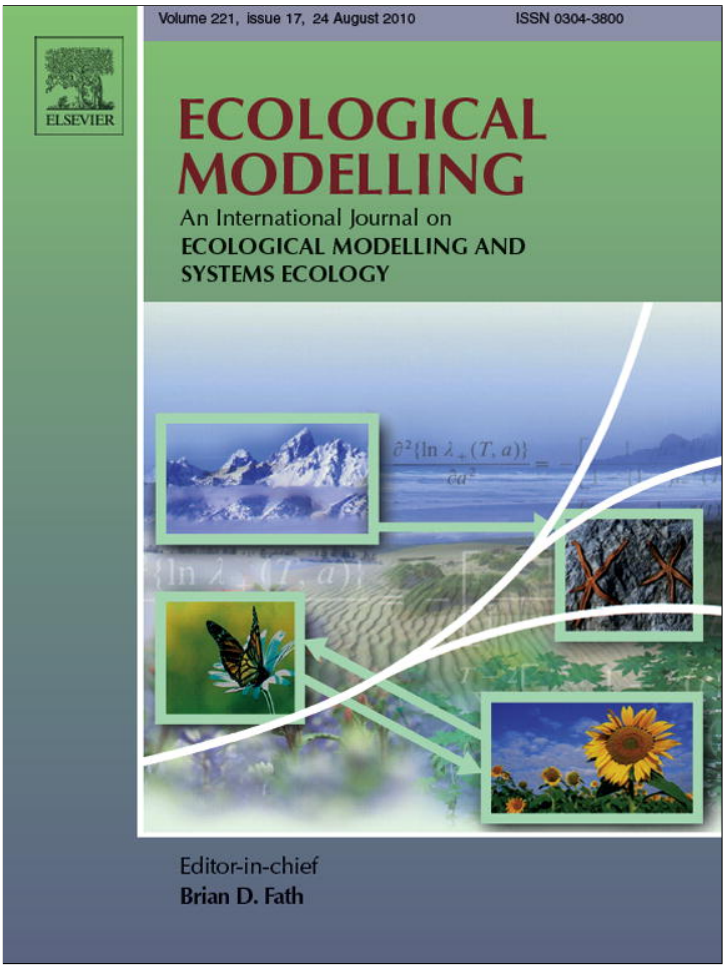

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Modelling the effects of UV radiation on the survival of Antarctic krill (*Euphausia superba* Dana) in the face of limited data

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ABSTRACT

Antarctic krill, *Euphausia superba*, is a keystone species of the Antarctic ecosystem. A fishery for krill may compete with land-based predators (penguins and seals), particularly during the breeding season. The Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR) is moving towards management in small scale units. The management models specify predation and fishing mortality as space and time dependent but do not yet include non-predation natural mortality. Krill are known to be highly susceptible to ultraviolet radiation (UV) but there are limited empirical data. We develop a model for krill mortality caused by UV and parameterize and assess it by comparison with experimental data. The analysis allows us to identify key parameters that should be measured in future experiments and also leads to suggestions about modification of experimental procedure. We illustrate the method for krill found in the Livingston Island area and show that (a) it is possible to estimate the component of natural mortality due to UV-induced damage and (b) that cohorts born in 1979, 1984, or 1997 have different survival in the first 5 years of life, associated with differential UV exposure. In particular, those born in 1997 may have experienced as much as 10% lower survival than those born in 1979. The method developed here allows a potentially important source of krill mortality to be incorporated into the management models and suggests key experiments and field work in the future.

1. Introduction

The Antarctic krill, *Euphausia superba* (henceforth krill), is a species of primary importance in the Southern Ocean, being the major prey of many vertebrate species (Smetaceck and Nicol, 2005). A fishery for krill has been a major fishery in the region since the late 1970s (Croxall and Nicol, 2004). There is recent concern that catches will increase in part due to new harvesting and processing technologies and in part to a likely increase in demand for krill-based products, such as aquaculture feed or krill oil for human consumption (Nicol et al., 2000). Furthermore, the Southern Ocean is a site of rapid potential climate change and there is concern that such change will affect krill (e.g. Wiedenmann et al., 2008, 2009).

The Southern Ocean krill fishery is managed by the Commission for the Conservation of Antarctic Marine Living Resources (CCAMLR), which has adopted an ecosystem-based, precautionary approach towards the krill fishery (Reid et al., 2005). A key principle of the Convention for the Conservation of Antarctic Marine Living Resources is that the effects of a krill fishery on predators should be reversible on a reasonable time scale. Annual catch limits are currently well below the estimated total krill biomass. For example, catch limits in the south Atlantic, southeast Indian Ocean, and southwest Indian Ocean sectors are 4.0 million tonnes, 440,000 tonnes, and 450,000 tonnes respectively while biomass estimates are 44.3 million tones, 4.83 million tones, and 3.9 million tonnes, respectively (Croxall and Nicol, 2004; more details can be found at the CCAMLR site http://www.ccamlr.org/pu/e/e_pubs/cm/drt.htm). The intention of such catch limits is to limit the potential for ecosystem-wide depletion of the krill.

However, in many areas, the fishery operates in the main feeding grounds of krill-dependent predators, such as seals and penguins (Croxall and Nicol, 2004), during the breeding season. Krill biomass on the scale relevant to predators fluctuates widely from year to year (Mackintosh, 1972, 1973; Murphy et al., 1998); the population sizes and breeding success of many krill predators are linked to local abundance of krill (Croxall et al., 1988; Reid and Croxall, 2001). Because of the potential for competition between the fishery and the krill predators at a local scale, the CCAMLR is moving towards

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management in Small Scale Management Units (SSMUs; Constable and Nicol, 2002; Hewitt et al., 2004).

A better understanding of the potential conflicts between the fishery and krill predators requires identifying and understanding the factors driving the variability in krill biomass. Among the questions related to management in small scale units are: (a) how should the overall catch limit for an area be subdivided among the SSMUs? (b) How should the development of the fishery be limited until this decision is made? (c) Which of predator demand, krill biomass, or demand minus biomass are most important in effective management of krill? One of the models used by the CCAMLR to answer these questions is the Krill Predator Fishery Model (KPFM, Watters et al., 2006, 2008, 2009; Hill et al., 2007a,b).

In the KPFM, the number of krill in a given SSMU at the start of season $t+1$ is determined by the number present at the start of season t, mortality, migration, and recruitment. In principle, mortality can have three components: (i) predation, (ii) fishing, and (iii) natural, non-predation mortality. In the KPFM, the third source of mortality is currently set equal to 0, so that the focus is on the suite of predators (baleen whales, seals, penguins, and fish) and the intensity of fishing in time and space. However, there always exist non-predation sources of mortality and in every ecological system, it is a challenge to compute the intensity of such mortality. Recruitment of krill in the KPFM in a SSMU is an asymptotic function of adult abundance in a previous time period, with a lag determined by the age at which krill recruit to the adult stock, and is influenced by time-dependent environmental conditions. Currently in the KPFM such environmental factors are treated as a time- and spatially varying anomaly (but spatially and seasonally specific) in the function relating recruitment and adult stock.

Various studies have explored the environmental influence on krill biomass, including how sea ice may affect reproductive success (e.g. Siegel and Loeb, 1995; Hewitt et al., 2003; Wiedenmann et al., 2009) or how variation in temperature experienced by krill may impact growth, which in turn will influence biomass (Wiedenmann et al., 2008). However, the impact that increases in ultraviolet (UV) radiation, a known mortality agent of krill, may have on krill biomass has largely been ignored. UV radiation is known to generate reactive oxygen species (Halliwell and Gutteridge, 2007) and such free radicals are implicated in all theories of aging and senescence. A number of authors (Mangel, 2008; Dowling and Simmons, 2009; Monaghan et al., 2009) have proposed that oxidative stress mediates and constrains the life histories of organisms and thus should be considered when constructing demographic and population models.

The DNA of Antarctic krill has the highest concentrations of A-T base pairs of any metazoan organism (Jarman et al., 1999) and UVB (280–320 nm) radiation particularly damages DNA at adjacent thymine residues (see, e.g. Browman, 2003). Thus Jarman et al. (1999) suggest that krill may be particularly susceptible to damage from UVB (cf. Lopez-Martinez et al., 2008). Krill also have comparatively quick and efficient DNA repair mechanisms (Malloy et al., 1997) and all seven mycosporine-like amino acids (MAAs) that are believed to have photoprotective properties (Newman et al., 2000; Karentz and Bosch, 2001), suggesting that defense against and repair of UV-related damage is part of krill evolutionary history. The depletion of ozone above the Antarctic (the 'Antarctic ozone hole'; Karentz, 1991; Rowland, 2006) increased the already high levels of levels UV radiation that organisms experience in the Antarctic and lead to suggestions of catastrophic effects of increased UV, which have not been observed (Karentz, 1991). However, the effect of changing UV radiation due to the ozone hole (Solomon et al., 2007, p. 553) on the biomass of krill available for predators is unknown.

These observations raise the questions that are the focus of this paper. First, is it possible to compute the non-predation natural mortality of krill induced by UV radiation? Second, did krill born at different times over the last 30 years, and thus exposed to potentially different UV profiles, have different rates of non-predation natural mortality? To answer these questions, we develop a method that allows one to relate the intensity of UV radiation to the non-predation natural mortality of krill, parameterize it with limited empirical data (thus showing the gaps in those data) and then compute the relative survival of 3 cohorts of krill whose lives span non-overlapping intervals. In the course of achieving these goals, we develop a computational algorithm for including a previously untreated source of mortality in the KPFM. In addition, we show how one can proceed to make predictions about ecosystem change and effects on organisms in the situation of having limited data.

2. Materials and methods

In an ideal situation, an enormous amount of laboratory and field data would be available to support the development of any model, particularly for purposes of parameter estimation and confrontation between the models and data (Hilborn and Mangel, 1997). Unfortunately that is not the case for krill and UV and there are only two empirical studies in this situation. Newman et al. (1999) conducted a laboratory study and Ban et al. (2007) a field study of the effects of UV on krill survival. Each study has difficulties of interpretation because of the empirical set-up (explained below). In a situation such as this, one could simply give up and assert that until much more empirical work were conducted there is no point in attempting prediction or modelling. However, problems are not solved by ignoring them (Feller, 1971). A key role of theory and modelling in ecology is to help guide empirical work (Mangel, 2006) and clearly theory and modelling are most useful before all the data are collected. We subscribe to this philosophy and will show how the models can guide future empirical studies of UV and krill.

2.1. A model for UV-induced damage in two different experiments

Newman et al. (1999) conducted an experiment in which five non-larval krill were kept in the dark, or exposed to low UVB, or high UVB for 8 h in each of 6 days. They measured the number of individuals that died on each day (Fig. 1). UVB causes damage to DNA, protein and lipid (Hessen, 2002) and such damage at the molecular and cellular level can be expressed as mortality at the organismal level (Mangel and Munch, 2005). Clearly, UV can also have non-fatal effects, but there are no data concerning krill on which to build a model for these effects (see Section 4).

Thus consider the damage $D_k(i,t)$ on day i at time t (measured as fraction of a day, so that $0 \le t \le 1$) during UV treatment k. We assume that damage increases in proportion to the intensity of UV, $U_k(i,t)$ with rate constant γ so that

$$\frac{dD_k}{dt} = \gamma U_k(i,t) \tag{2}$$

In each of the UVB treatments $U_k(i,t)$ is a constant, u_k for $0 \le t \le 1/3$ (day) and 0 for $t > 1/3$ (night). We thus conclude that for $t \le 1/3$

$$D_k(i,t) = D_k(i,0) + \gamma u_k t \tag{3}$$

and for $t > 1/3$

$$D_k(i,t) = D_k(i,0) + \frac{\gamma u_k}{3} \tag{4}$$

We combine Eqs. (3) and (4) with the linking condition from 1 day to the next that $D_k(i,0) = D_k(i-1,1)$ and the dynamics

$$D_k(i,t) = D_k(i-1,1) + \gamma u_k t \tag{5}$$

We let $S_k(i,t)$ denote the fraction of individuals alive at time t within day i in treatment k, given that they were alive at the start

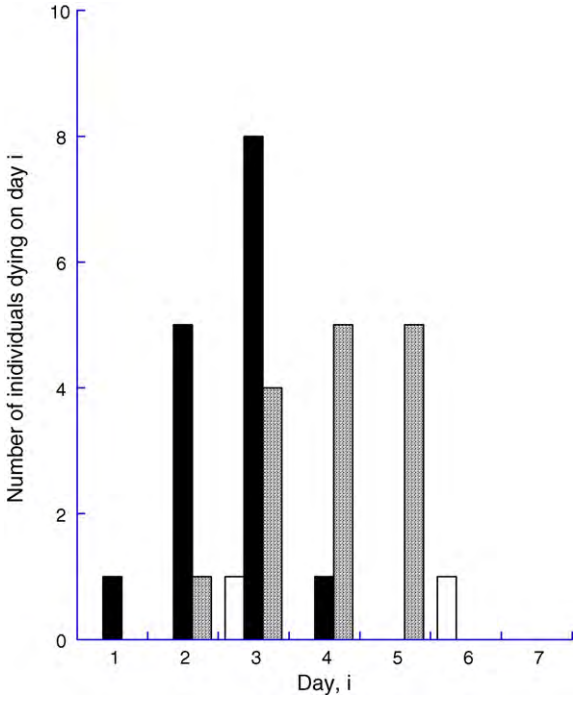

Fig. 1. Newman et al. (1999) conducted three replicated trials in which five non-larval krill were kept in the dark, or exposed to low UVB, or high UV- for 8 h each day and measured the number of individuals who died on each day of the experiment. The summarized data, combining replicated trials, show the number of deaths each day of the experiment according to treatment (clear – dark; stipple – low UV-B; black – high UV-B).

of the day. We assume that mortality rate has a damage independent component m_0 and a damage-dependent component m_d. Then survival is given by

$$\frac{1}{S_k}\frac{dS_k}{dt} = -[m_0 + m_d D_k(t)] \tag{6}$$

so that

$$-\log(S_k(i,1)) = \int_0^1 [m_0 + m_d D_k(i,t)]\,dt \tag{7}$$

We integrate from $0 \le \tau \le 1/3$ and from $1/3 \le \tau \le 1$ to obtain

$$-\log[S_k(i,1)] = m_0 + m_d D_k(i,0) + \frac{5}{18} m_d \gamma u_k \tag{8}$$

In summary, the model of damage and survival can be summarized as:

A. Set $D_k(0,0) = 0$ (because there is no reinforcement of damage, initial damage can be absorbed into m_0).
B. For $i > 0$ iterate according to $D_k(i,0) = D_k(i-1,0) + 1/3\gamma u_k$.
C. For each i, compute $S_k(i,1) = \exp(-m_0 - m_d D_k(i,0) - 5/18 m_d \gamma u_k)$.

We determine m_0, m_d and γ from the number of individuals who died on days 1–6 in the dark, low UVB, and high UVB treatments and the number of survivors of the dark treatment (Fig. 1).

We let

$$p_k(i) = \Pr(\text{an individual dies on day } i \text{ in treatment } k, \text{ given it is alive at the start of day } i) \tag{9}$$

Clearly $p_k(1) = 1 - S_k(1,1)$. For $i > 1$

$$p_k(i) = \left[\prod_{i'=1}^{i'=i-1} S_k(i',1)\right](1 - S_k(i,1)) \tag{10}$$

since to die on day i an individual must survive from day 1 to $i-1$ (the product) and then not survive on day i. The probability of being alive at the end of the experiment is

$$p_R = \left[\prod_{i=1}^{7} S_k(i,1)\right] \tag{11}$$

We let $L(m_0, m_d, \gamma)$ denote the likelihood of the parameters given the data: the number of individuals A_{ijk} alive on day i of replicate j in treatment k, from which we find the number of individuals N_{ijk} who die on day i of replicate j in treatment k. Here $k = 1$ corresponds to the dark; $k = 2$, 3 to the Low UVB ($0.378\,\mathrm{W\,m^{-2}}$) and high UVB ($0.918\,\mathrm{W\,m^{-2}}$) treatments of Newman et al. (1999), respectively. We let R_{jk} denote the individuals surviving the entire experiment in replicate j of treatment k, so that $R_{11} = 4$, $R_{21} = 5$, and $R_{31} = 4$ and all others are zero.

On any day in any replicate of any treatment, the number of individuals who die will be a binomial random variable with parameters A_{ijk} and $p_k(i)$. The contribution to the likelihood from this ijk-tuple is

$$\binom{A_{ijk}}{N_{ijk}} p_k(i)^{N_{ijk}} (1 - p_k(i))^{A_{ijk} - N_{ijk}} \tag{12}$$

The contribution of the survivors of replicate j in treatment k to the likelihood is $p_R^{R_{jk}}$.

In consequence, the likelihood is

$$L(m_0, m_d, \gamma) = \left[\prod_{k=1}^{3}\prod_{j=1}^{3}\prod_{i=1}^{7} \binom{A_{ijk}}{N_{ijk}} p_k(i)^{N_{ijk}} (1 - p_k(i))^{A_{ijk} - N_{ijk}}\right] \times \left[\prod_{k=1}^{3}\prod_{j=1}^{3} p_R^{R_{jk}}\right] \tag{13}$$

Discarding terms that do not depend upon the parameters, the log-likelihood $l(m_0, m_d, \gamma)$ is

$$l(m_0, m_d, \gamma) = \sum_{k=1}^{3}\sum_{j=1}^{3}\sum_{i=1}^{7} N_{ijk} \log(p_k(i)) + (A_{ijk} - N_{ijk}) \log(1 - p_k(i)) + \sum_{k=1}^{3}\sum_{j=1}^{3} R_{jk} \log(p_R) \tag{14}$$

To implement Eqs. (10)–(14), we obtained the original, rather than summarized, data (the $\{N_{ijk}\}$) from Dr. S. Newman, via Dr. S. Nicol (Australian Antarctic Division).

We found the Maximum Likelihood Estimates (MLEs) of each parameter (m_0^*, m_d^*, γ^*), through systematic search of the parameter space (Hilborn and Mangel, 1997). We conducted a Chi-square goodness of fit test of the model by computing the predicted number of individuals dying on each day in the low UVB and high UVB treatments to confirm that we cannot reject the best-fitting model.

Ban et al. (2007) conducted an experiment similar to those of Newman et al. (1999). However, they used calyptopsis larvae rather than juveniles and did not report overall UVB intensity, so that a direct comparison is problematic since larval krill are likely to be differentially sensitive to UV than juvenile krill. Still, the comparison is worthwhile because of the independence of the two sets of

experiments, so we used two values for UV intensity reported by Newman et al. (1999) to predict survival in the experiments of Ban et al. (2007).

2.2. Survival consequences of UV-induced mortality

The MLE parameters cannot be used directly in a population model because in the experiments krill are starved and diel vertical migration is impossible (see Section 4 for more about how our models can influence future empirical work on this subject). Hence mortality rates are substantially greater in the experiments than in nature. In this section, we explain the requisite modifications of the survival model and then use it to derive predictions about the consequences of UV-induced mortality.

We follow Siegel (2005) and Wiedenmann et al. (2008) to characterize vertical migration of krill: age −1 and older krill in summer (January–March) occupy surface waters (0–50 m) during both day and night. In the spring (October–December) and in the fall (April–June), krill occupy waters between 100 and 150 m during the day, and move up to between 50 and 100 m at night. During the winter (July–September), krill spend the day between 250 and 350 m, and migrate up to the region between 100 and 150 m at night. We assume that krill occupy surface waters (0–50 m) completely during their first year of life.

As described above, the values of damage independent mortality m_0 and damage-dependent mortality m_d determined in the analysis of the experiments of Newman et al. (1999) are overestimates. In the laboratory, with adequate food and ambient light conditions, absent of predation, the probability that a krill survives a single year is 0.9 (S. Kawaguchi, personal communication), *i.e.* a daily mortality rate ≈ 0.000274. This can be considered an estimate for m_0 the non-damage mortality rate, and thus treated as the rate of non-predation natural mortality under the best possible circumstances.

To account for a lower damage-dependent mortality rate than that computed from the experiments, we multiply m_d^* by $\eta_m < 1$. Thus, for a krill with accumulated damage d, the non-predation daily mortality rate is $m_0 + \eta_m m_d^* d$. Since η_m cannot be estimated from existing data (suggesting crucial experiments, see Section 4) we treat it as a parameter and condition results on this parameter.

We let $S(t,y)$ denote the probability that a krill survives to age t in year y, so that $S(0,y) = 1$. If $D(t|y)$ is the damage that has accumulated due to exposure to UV radiation by day t of life in year y

$$S(t+1,y) = \exp(-m_0 - \eta_m m_d^* D(t|y))S(t|y) \quad (15)$$

with the understanding that when $t = 365$, $y \to y+1$ and t is reset to 1. To model the accumulation of damage, we use the analog of Eq. (5), but accounting for fractions of the day $f(t)$ of daylight and that krill do not spend all of their time at the surface. We denote by $k_e(j_d)$ the extinction coefficient for UV when a krill is at depth $\delta(j_d)$ on day j_d. Thus, the protection afforded to it by water is $\sigma(t) = \exp(k_e\delta(j_d))$ and Eq. (5) becomes

$$D(t+1|y) = D(t|y) + \frac{\gamma^* U(t,y) f(t)}{\sigma(t)} \quad (16)$$

where $U(t,y)$ is the intensity of damaging UV radiation on day t in year y. The procedure described in Eqs. (15) and (16) allows us to (i) compute the non-predation natural mortality of krill induced by UV radiation and (ii) compare the differential survival of cohorts of krill born at different times and thus experiencing potentially different UV profiles.

2.3. An illustrative calculation at Livingston Island

As an illustration, we focus on 62°S (Livingstone Island, the sampling site of Kawaguchi et al., 2007) and assume that krill do not leave the area (one of the assumptions about movement in the KPFM; the other being passive drift). We use the model of Forsythe et al. (1995) to compute day length as a function of Julian day. We assume that UV exposure parallels visible exposure (see Section 4 for alternatives).

Although the work of Jarman et al. (1999) and Newman et al. (1999, 2000) gives indication that krill are susceptible to UV, the precise nature of that susceptibility is unknown. The standard method for measuring the susceptibility of a species to UV is through the Action Spectrum (AS) or Biological Weighting Function (BWF), which convolves the irradiance an organism experiences with its biological effect (Cullen and Neale, 1997). However, the BWF for *E. superba* is unknown. The BWF varies widely across organisms (Browman et al., 2000a,b; Maragoni et al., 2006) and the information is conflicting about what to choose for the BWF of krill. Kouwenberg et al. (1999) conclude that the BWF for cod (*Gadus morhua*) eggs is similar to that for naked DNA. Browman (2003) conclude that a Calanoid copepod is more sensitive to UV damage than cod. Tartarotti et al. (2000) conclude that the BWF for the copepod *Boeckella gracilipes* more closely resembles that for UV-induced erythema, rather than that for naked DNA and Williamson et al. (2001) show that the BWF for *Calanus* eggs and *Daphnia* are similar to each other, but quite different from cod eggs.

Thus, to approximate the intensity of damaging radiation, we use erythemal radiation, which is the integral of ground-level irradiance between 280 and 400 nm, weighted with the Action Spectrum proposed by McKinlay and Diffey (1987) (also see Fritz et al., 2008). We downloaded TOMS data (Eck et al., 1995), to find the local noon surface erythemal irradiance (in $\mathrm{mW\,m^{-2}}$) around the 15th day of each month from 1978–1993 and 1996–2005 (no data are available for 1994–1996). We then interpolated between monthly values to obtain a daily intensity of weighted UV.

To focus on potential changes in survival of cohorts of different birth years, we assume a constant extinction coefficient, $k_e = 0.15$ (Newman et al., 2003). Clearly, this would be a poor approximation for a particular field application since the extinction coefficient will depend upon the opacity of the water and thus indirectly on phytoplankton concentrations (e.g. Browman, 2003) and UV exposure will depend upon cloud cover (Sobolev, 2000), all of which are measurable for a particular field application. However, this assumption is completely appropriate for illustration of our method, since it allows focus on the key components in our analysis. The value of extinction coefficient that we choose corresponds to relatively clear water; see Fig. 4 of Browman (2003). We then compute the non-predation survival of krill born in 1979, 1984, and 1997 over the first 1650 days of life. These intervals allow maximum use of the TOMS data while having non-overlapping intervals. We use a range of values of η_m in Eq. (15), our main tool for answering the two motivating questions.

3. Results

3.1. Inference from the Newman et al. experiments and comparison with the experiments of Ban et al.

We found that the likelihood surface is broad and relatively flat around the Maximum Likelihood Estimates (MLEs) $m_0^* = 0.00856$, $m_d^* = 0.352$, and $\gamma^* = 1.88$. The Chi-square test to assess the fit of these maximum likelihood parameters to the data gave test probability $p = 0.64$, so that the model cannot be rejected. Based on the MLE parameters, we predict an expectation of 4.97 deaths in the high UVB treatment and 4.50 deaths in the low UVB treatment.

Ban et al. (2007) did not provide UVB intensity, only intensities at 4 wavelengths. To compare their survival curve with predictions made using the parameters from the experiments of Newman et al. (1999), we used the high UVB intensity that Newman et al.

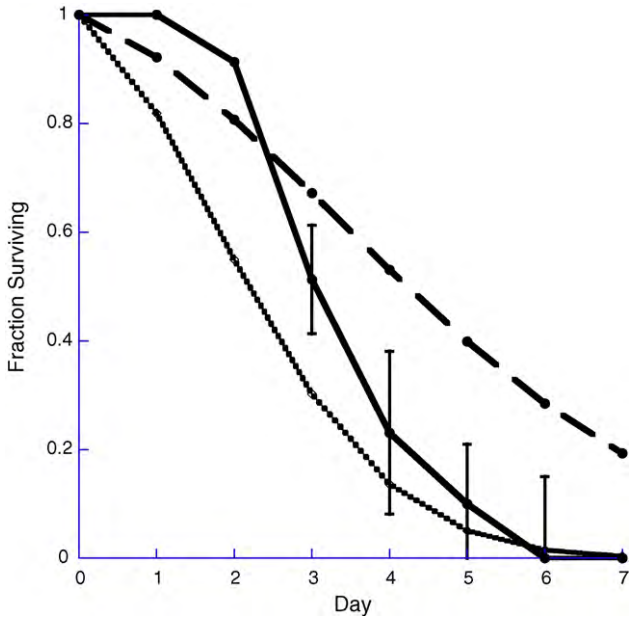

Fig. 2. We have used Eqs. (2)–(6) to estimate the parameters that characterize the relationship between UV damage and survival for the experiments of Newman et al. (1999). Ban et al. (2007) conducted an experiment that can be considered an *in situ* version of the experiments of Newman et al. (1999). However, they did not measure UVB intensity. To compare the survival curve they obtained (circles, with standard deviations shown by error bars) with predictions made using the parameters from the experiments of Newman et al., we used the high UVB intensity that Newman et al. (1999) used (dotted line) and the high UVB reported by Newman et al. (1999) for Palmer Station on the West Antarctic Peninsula (dashed line). The predicted curves include the empirical results. Detailed quantitative comparisons cannot be made for reasons explained in the text.

(1999) used and the high UVB reported by Newman et al. (1999) for Palmer Station, Antarctica. Our theory brackets the empirical results (Fig. 2); given the disparities between the two experimental approaches (particularly the stage of the organism), we should not expect more precise matching.

3.2. Survival at Livingston Island

In Fig. 3a, we show the survival of the 1979 birth class to day 1650 as a function of η_m, assuming only non-predation natural mortality. When $\eta_m = 0$, krill experience no UV-related mortality. Thus they survive as in the laboratory under ideal conditions. As η_m increases, the non-predation natural mortality due to UV-induced damage reduces survival. In Fig. 3b, we show the survival of the 1984 and 1997 birth classes relative to that of the 1979 birth class as a function of η_m.

When $\eta_m = 0$, the relative survival is 1 since there is no effect of UV on survival. As η_m increases, the relative survival declines and could be as much as 5 or 10% lower for the 1997 birth class. These results cannot be predicted by simply looking at the trajectories of UV intensity (not shown).

4. Discussion

One motivation for the study of Jarman et al. (1999) was the development of the hole in the ozone layer above the Antarctic. Because the possibility of a hole in the ozone layer was not accepted until about 10 years after it was first noticed (Karentz and Bosch, 2001) many of the potential UV-influenced changes could have occurred when no data were collected. Once the hole was recognized, terrible consequences were predicted for the future of the

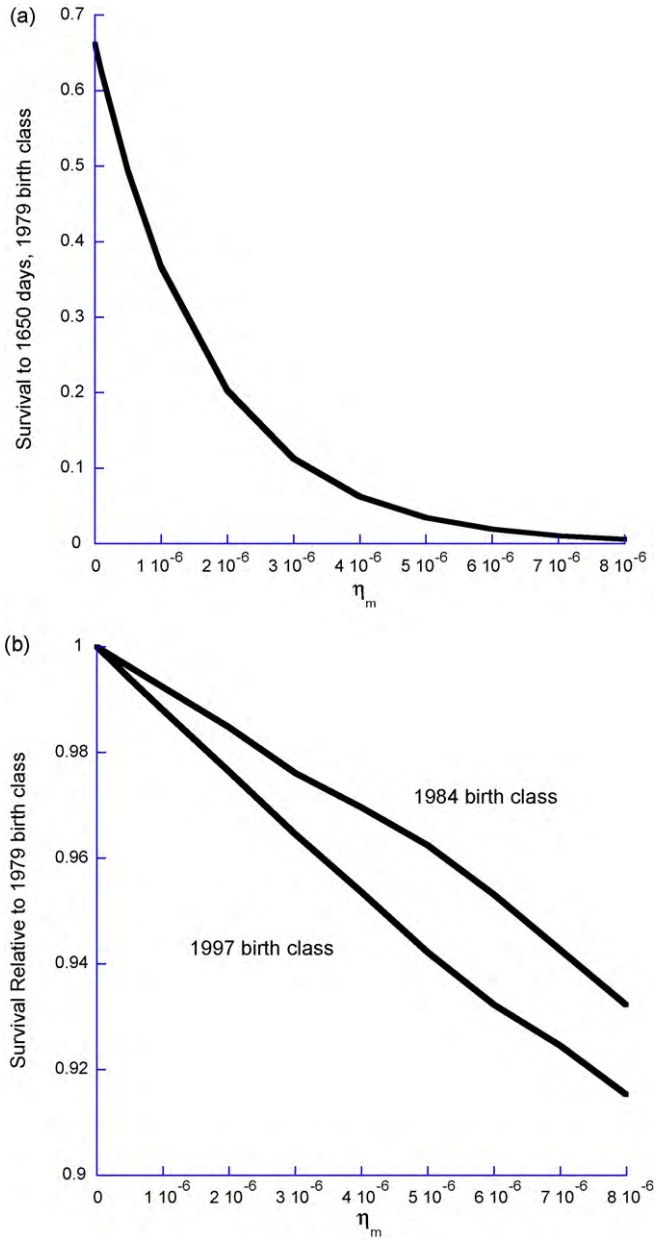

Fig. 3. Answers to the two questions that are the focus of this paper (Is it possible to compute the non-predation natural mortality of krill induced by UV radiation? Are krill born at different times in the trajectory of the development of the ozone hole predicted to have different rates of survival?), for krill around Livingston Island. In panel (a) we show survival, excluding predation, of the 1979 birth class to 1650 days as a function of η_m, the parameter that modulates the survival effect of damage-dependent mortality. When this parameter is 0, there is no effect of damage-dependent mortality and in the absence of predation krill survive in the field as well as in the laboratory under ideal conditions. As η_m increases, damage-dependent mortality is more and more significant and thus non-predation survival declines. In panel (b) we show the relative survival of the 1984 and 1997 birth classes, compared to the 1979 birth class, also as a function of η_m. When $\eta_m = 0$, relative survival is 1 because there is no effect of UV-induced damage on survival. As η_m increases, the contribution of UV-induced damage to non-predation mortality increases and as seen in the figure survival of the 1997 birth class to 4.5 years could be 5–10% lower than that of the 1979 birth class.

Antarctic ecosystem. These seem not to have borne out and it is now recognized that the changes were not as drastic as first predicted but may be more complex (Karentz and Bosch, 2001, p. 12). Our study is an example of such complexity.

The intensity of UV radiation experienced by organisms may fluctuate greatly over short periods of time at the same loca-

tion for a wide variety of reasons (Sobolev, 2000), so that our work needs to be understood in the context of a broad conceptual framework and resulting pattern rather than a specific numerical prediction. Browman et al. (2000a,b) make a similar point: that cloud cover, albedo, water quality and vertical distribution of the mixed layer will have important effects on the level of UVB exposure. Furthermore, organisms have a suite of responses to UVB that includes behavior, increasing melanic pigmentation, use of antioxidant defenses, and repair processes that use UVA and Photosynthetically Active Radiation (Cywinska et al., 2000). Organisms have evolved a wide range of UV-screening compounds (Cockell and Knowland, 1999), since all else being equal it is better to avoid damage in the first place than to have to repair it. Many of these compounds are MAAs, readily found in ice-algae that krill consume (Newman et al., 2000).

Our modelling results are consistent with the field study of Naganobu et al. (1999) on the correlation of krill density and recruitment with the strength of westerly winds, sea ice, and ozone depletion. They found significant correlations between krill density in the Antarctic Peninsula region and parameters characterizing ozone depletion for the period 1977–1997: negative correlations between krill density and the area of the ozone hole, the extent of the ozone hole in the Antarctic peninsula area, and the whole depleted ozone mass; strong positive correlations between krill density and the lowest total ozone and monthly total ozone at the Faraday/Verndasky station. They also found a negative, but statistically not significant, correlation between total ozone in October at the Syowa Station in the Indian Ocean and krill density. However, they found no correlation between ozone depletion and recruitment of krill. Naganobu et al. (1999) consider four hypotheses: (1) increased UVB caused primary production to decline and this caused an increase in the mortality rate of larval krill; (2) UVB has an adverse direct effect on krill; (3) ozone depletion lead to changes in the atmosphere with a negative effect on krill abundance and (4) the correlations are spurious. They consider that the overwhelming weight of the evidence supports hypothesis (2) which is consistent with our results, and our methods now provide the tool for predictive studies.

Our results are also consistent with the study of Browman (2003) on a Calanoid copepod and cod eggs, in which they concluded that an increase in DNA damage due to UV exposure would result in an additional annual mortality of about 10%. However, they measured the BWF, which has not yet been done for krill. Thus, one of the recommendations of our work is the measurement of the BWF for Antarctic krill. This can be done using the methods described by Cullen and Neale (1997) or Kuhn et al. (2000), with careful attention paid to the ratio of UVB to UVA and PAR since they are essential for effective repair (Cywinska et al., 2000; Bancroft et al., 2007).

In principle, the dynamics of damage could depend upon repair, production of new damage, and positive reinforcement of damage (Mangel and Munch, 2005). However, our explorations of a model with all three factors showed that it provided no better fit to the data than a model in which we ignore both reinforcement and repair of damage. Thus, the existing experimental data do not justify a more complicated model with reinforcement of damage or repair of damage. We suggest that future experiments avoid starving the krill, to allow a better estimate of m_0, m_d and η_m. This would also allow the exploration of non-lethal effects of UVB exposure (e.g. reduced growth rate as krill shunt resources from growth to repair, Mangel, 2003; Bancroft et al., 2007). We used fixed, stereotypical vertical migration profiles, rather than allowing them to be determined in a facultative manner; this choice was also determined by the limitations of data. However, it is likely that vertical migration plays an important role in reducing exposure of krill to damaging UV radiation.

We also recommend collecting information on UV radiation in each SSMU with concurrent measurements of krill density and extinction rates in the water column. This will couple our model with *in situ* measurements (e.g. see Eilertsen et al., 2007) eggs and larvae of cod. The output can then feed directly into the KPFM by providing a method for computing the value of non-predation natural mortality.

There can be no doubt marine (and freshwater) organisms such as krill face short term risks from predation and long-term risks from accumulation of UV-induced damage. Hansson and Hylander (2009) have recently given the first empirical example of such a trade-off, in a study involving *Daphnia*, but this has not yet been done for krill. Much work, empirical and modelling, remains to be done for this keystone species.

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