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Estimating Survival and Cause-specific Mortality from Continuous Time Observations

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Summary

Ecologists must understand the causes and demographic consequences of mortality to properly conserve and manage populations. Survival data collected in continuous time can yield robust survival estimates with high precision, but currently these methods are underused in ecology. Continuous time survival data can be derived from studies ranging from direct observation of stationary nests to radio telemetry of free-ranging animals; consistently, these designs involve intensive monitoring and high probability of detection of individual subjects. Continuous time survival rates are calculated using estimators that define time intervals either by constant risk periods (e.g. Mayfield, Heisey–Fuller) or mortality events (e.g. Kaplan–Meier, Nelson–Aalen); these estimators differ in their assumptions and suitability for specific study designs and datasets. Univariate survival rate comparison using simple nonparametric tests is ill-suited for ecological data because of common irregularities like few mortalities, staggered entry of subjects, and right censoring of survival timelines. Semi-parametric Cox proportional hazard (CPH) models offer robust insight into relative hazard while allowing researchers the flexibility to address study design complexities including multiple predictors, random effects, and time-dependent variables. Fully parametric survival models rarely provide improvement over a semi-parametric approach and require that underlying survival distributions are known, which is uncommon in ecology. An additional advantage of a continuous time study design is that precise timing of the mortality event is determined, potentially also allowing researchers to identify cause of death. Cause of death information is the basis for competing risks analysis, which extends the CPH approach to multiple mortality agents. Although infrequently used in ecology, competing risks analysis can be especially useful in conservation and management by revealing the relative importance of different risk types and whether they are additive or compensatory to other mortality sources. Knowledge gained from competing risks analysis can be especially valuable for appropriately targeting mitigation efforts. Newer approaches in survival analysis, including mixed-effects modeling and Bayesian methods, hold promise for refining inference from continuous time datasets. In sum, research in ecology will benefit from expanded collection and analysis of continuous time survival data, with new tracking technologies like camera-based monitoring and satellite-based radio telemetry being especially noteworthy for supporting novel analysis and insight. Ultimately, better integration of continuous time survival and competing risks analyses will contribute importantly to future advances in population ecology and conservation biology.

6.1 Introduction

Population ecologists have a longstanding interest in understanding the causes and consequences of mortality in organisms. Understanding when and where an animal or plant dies, what is the cause of death, and whether predisposing factors led to the mortality event, is of paramount relevance to research ranging from demographic analysis to conservation biology (Grosbois et al. 2008; Nussey et al. 2008). At its core, an individual's survival rate (or mortality rate, where survival = 1 – mortality) is central to estimates of fitness and thus is a necessary consideration when investigating evolutionary processes (Metcalf and Pavard 2007). Population-level survival and mortality estimates are fundamental to understanding population status and viability, community interactions, and ecosystem resilience (McCallum 2000; Morris and Doak 2004). Yet, population ecologists face substantive logistical challenges when studying survival and mortality in natural settings. Free-living organisms often occur at low density and have cryptic lifestyles, or else exhibit elusive or vagile behavior or prolonged periods of stasis or dormancy. Not only do such constraints make it difficult to implement a robust survival

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monitoring schedule, but they limit the ability to effectively survey mortality risk continuously through time. When survival monitoring is infrequent or has a low probability of detection, status and fate of individuals cannot be inferred directly, which challenges our ability to obtain unbiased survival estimates. In fact, the reliability and rigor of survival research is a general concern in population ecology (McCallum 2000; Williams et al. 2002; Murray 2006), leading to efforts to improve monitoring and estimation through both careful design and implementation of observational studies, and adoption of analytical procedures that provide robust inference while accounting for limitations in ecological datasets.

Contemporary methods in survival analysis repeatedly track uniquely marked individuals until they reach an endpoint like death or loss from the survey (Figure 6.1). Owing to the challenges of monitoring individual free-living organisms and documenting their fate, survival research in ecology can involve indirect and infrequent detection, leading to incomplete confirmation of the organism's status and discrete survey events that are separated by time gaps that can last days, months, or even years. In fact, ecologists commonly survey animals via live-capture, opportunistic field observation, or from noninvasive genetic methods, and then use *capture-mark-recapture* (CMR) statistics to estimate probability of survival (Chapter 7). Survival estimates based on CMR methods are usually approximations (i.e. "apparent survival," sensu White and Burnham 1999) because the sampling protocol is discrete, and mortality, emigration, and other fates usually are not known. Alternative models are being developed to more precisely estimate survival from *discrete time* data (Barbour et al. 2013; Schaub and Royle 2013; Chapter 7), but there is no substitute for monitoring individuals continuously through time and documenting their fate directly. Yet, this protocol requires specialized monitoring and statistical procedures based on *known-fate methods* (sensu White and Burnham 1999). *Continuous time* survival monitoring is logistically more demanding and not possible or

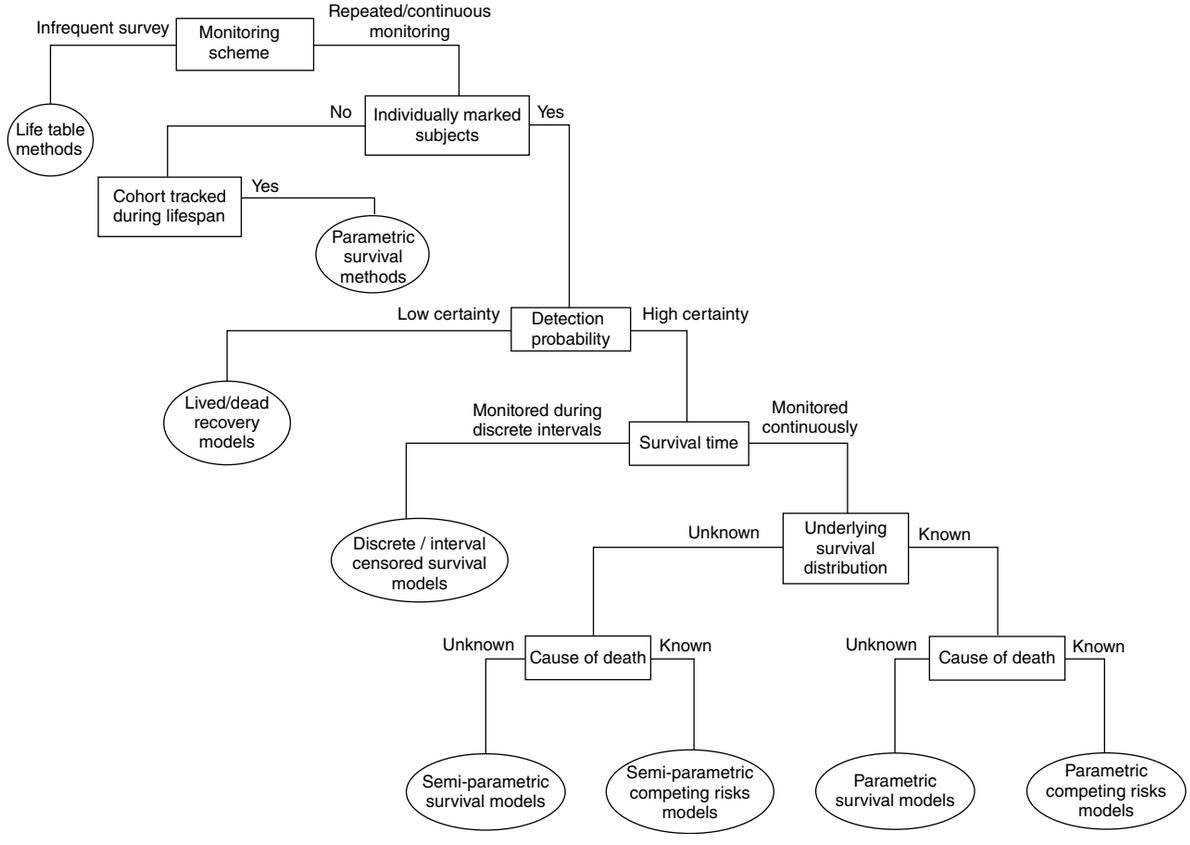

Figure 6.1 Conceptual diagram for selecting a continuous time survival analysis approach. Individuals must be distinguished and monitored repeatedly with high detection probability. Monitoring frequency should be sufficiently intense to develop a continuous survival timeline. Whether the underlying survival distribution and cause of death are known will determine whether fully parametric and competing risks methods can be used. *Source:* Adapted from Murray and Patterson (2006).

appropriate for all field studies, but when available, data from such studies can offer less bias and increased precision compared to their discrete time counterparts. Notably, discrete time and continuous time analytical methods yield similar survival estimates when monitoring and detection frequency converge, even though both approaches remain philosophically and computationally distinct (Efron 1988; Williams et al. 2002).

6.1.1 Assumption of No Handling, Marking or Monitoring Effects

Organisms can be monitored in a continuous or semi-continuous manner using a variety of approaches, and a fundamental assumption in survival research is that researcher activities and marking methods do not affect subjects. Stationary subjects like plants or nest sites can be individually tagged and assessed manually by observers or through deployment of field cameras; many vertebrates and some invertebrates can be tagged and monitored using very high frequency (VHF) transmitters that are detected remotely using radio telemetry; more recently, larger species are monitored using Global Positioning System (GPS) or satellite-based transmitters (Bridge et al. 2011; Mansfield et al. 2012; Kissling et al. 2014). These activities and devices allow researchers to potentially track the status of individuals continuously through time, and thereby facilitate precise death time estimation compared to more passive monitoring approaches that serve in discrete time survival research. Yet, procedures like repeated visitation of nest sites, or capture and handling of animals for radio transmitter deployment, or even the effects of radio transmitters themselves on animal behavior, condition, and fitness, are potentially impactful to subjects. In fact, it is reasonable to suggest that monitoring protocols and devices used in continuous time data collection tend to be more invasive, with more frequent monitoring and more obtrusive tags, than methods used for discrete time survival research. For example, we know that if predators follow human scent when searching for food, nest-site visitation by researchers may increase predation risk (Major 1990; Kurucz et al. 2015; but see Ibáñez-Álamo et al. 2012). We also know that stress associated with animal capture and handling can have marked impacts on post-capture survival probability (Gilbert et al. 2014; Chitwood et al. 2017; DelGiudice et al. 2018). Further, radio transmitters are sometimes deployed on individuals that are either too small or too sensitive to carry the tags (Paquette et al. 1997; Saraux et al. 2011). In fact, some research activities or tag types may have subtle effects that are not easily discerned or quantified using standard monitoring approaches (Hamel et al. 2004; Brooks et al. 2008; Ludynia et al. 2012; Vandenabeele et al. 2012). Although assessing the pros and cons of different monitoring and tagging methods is beyond the scope of our chapter, we remind that the potential effect of proposed monitoring activities and devices on subjects should be understood prior to initiating a survival study. Resources are available for evaluating the potential effects of handling and marking (Murray and Fuller 2000; Barron et al. 2010), and guidelines are available to help design field procedures having minimal researcher impacts (Hawkins 2004; Casper 2009). Thus, at the outset of a continuous time survival study researchers should consider carefully whether research activities are likely to affect subjects, how field procedures and tags can be adjusted to minimize potential negative effects, and if necessary, how such effects can be detected, quantified, and addressed via data processing or analytical adjustments.

6.1.2 Cause of Death Assessment

Continuous time detection of individuals provides additional benefits by allowing researchers to determine cause of death through contemporaneous recovery of carcasses and assessment of the death site. Plants and animals normally succumb to a variety of mortality agents, and knowing what agents are implicated is important for providing a range of options in survival analysis (Figure 6.1). *Cause of death* information is especially relevant when researchers aim to focus mitigation efforts to forestall population decline or promote recovery. Yet, there are obvious logistical challenges in determining cause of death in the field, including that carcasses are difficult to recover and post-mortem exams often provide equivocal results. Even survival studies prioritizing cause of death determination can fail to confirm the source of mortality for a sample of subjects, or else confound proximate causes of death or the factors directly causing mortality (e.g. predation, starvation) with the ultimate causes (e.g. sublethal infection, malnutrition). For example, even though predation is well-known as the primary proximate cause of death in snowshoe hares (*Lepus americanus*), an experimental manipulation of sublethal parasites in hares showed that ultimately, parasitism (Murray et al. 1997) and declining body condition (Murray 2002) contribute importantly to hare predation. Likewise, sublethal parasitism increases predation risk in Red Grouse (*Lagopus scotica*; Hudson et al. 1992) whereas poor body condition predisposes coral reef fish (*Pomacentrus amboinensis*) to predation (Hoey and McCormick 2004). Thus, additional information about predisposing factors is important for comprehensive cause of death assessment. Similarly, whether specific risk factors incur additive or compensatory mortality on populations remains a longstanding, albeit understudied, interest in ecology (Burnham and Anderson 1984; Boyce et al. 1999; Murray et al. 2010),

and an understanding of proximate and ultimate causes of death is an important component of such an assessment. Accordingly, continuous time survival research can contribute to disentangling the roles of different causes of death and their demographic implications.

In this chapter, we review continuous time survival analysis in field-based ecology. Continuous time survival methods (i.e. "time-to-failure" or "time-to-event" models) are appropriate in research situations where survival monitoring is frequent with a high detection probability. Herein, we highlight important considerations and limitations when designing continuous time survival research (Winterstein et al. 2001; Williams et al. 2002; Murray 2006), and focus on the application of multiple-variable regression-based approaches which are especially well-designed for the challenges associated with observational field research. Our review extends to methods that account for cause-specific hazards and competing risks. *Competing risks analysis* is well-established in disciplines like epidemiology and industrial design (Crowder 2001; Pintillie 2006; Austin et al. 2016), but until recently was rarely used in ecology (Heisey and Patterson 2006). Owing to this oversight, and because collectively these methods are rarely covered in ecology undergraduate curricula and related statistical texts, review of this topic is important for students and researchers in population ecology.

6.1.3 Historical Origins of Survival Estimation

Human demographic analysis served as basis for contemporary methods in survival analysis, and an early demographer, John Graunt (1620–1674), accessed public records of births and deaths in Renaissance London to conduct "political arithmetick" in an effort to reveal demographic trends. Graunt pooled death records into 6–10 year (age) intervals to show changes in probability of survival and age-specific mortality (Figure 6.2). These records reveal a steady decline in survival probability as individuals age, such that by age 60 only 10% of the initial cohort remained alive and by age 80 only 1% had survived. This pattern translates to high mortality in age classes spanning 0–60 years and a lower risk in later ages, likely reflecting disproportionate loss of particularly frail individuals in the younger age classes.

In principle, we can evaluate these survival and mortality rates using either a cumulative function representing probability of survival considering all previous age classes (Figure 6.2a), or as age-specific mortality rates that reflect probability of death during a specific time interval (Figure 6.2b). Note that Figures 6.2a and b could have been expressed as the mortality function and the age-specific survival rate, respectively, because of the aforementioned "1 minus" property between survival and mortality.

Today, Graunt's analysis serves as an important foundation in statistical demography (Wainer and Velleman 2001; Egerton 2005); a similar example in ecology comes from Adolph Murie's study of Dall sheep (*Ovis dalli*) skulls collected over several years near Mt. McKinley, Alaska (Murie 1944). Murie estimated age of death from tooth wear and developed *life tables* to calculate mortality and lifespan in a manner similar to Graunt's approach with human public records (Box 6.1). Murie showed that Dall sheep have qualitatively similar survival patterns to those seen in humans, except that the decline in survival probability in the middle age classes is more restrained (Figure 6.2c). Age-specific variation results in lower age-specific mortality among middle-aged sheep compared to young and old individuals (Figure 6.2d).

Note that the parallels between the Graunt and Murie datasets extend beyond the basic shape of the survival functions. The life table approach used by both researchers constitutes a rather blunt survival analysis and differs from modern methods by being retrospective and reconstructive by using information from individuals who have already died rather than those whose risk was actively monitored (Figure 6.1). Use of age at death data is a *cross-sectional* assessment of a given population rather than a *longitudinal* study that follows survival of a cohort of individuals through real time. In fact, the human and sheep studies hinge on assumptions related to temporal consistency in both mortality risk and mortality reporting, and that the population age distribution and size are virtually stationary (Anderson et al. 1981); these assumptions are probably unrealistic and untestable for either dataset. More broadly, life table methods are especially problematic for long-term study of organisms living in highly variable environments where survival monitoring is opportunistic and probability of detection is low. For example, if emigrating sheep are subject to higher mortality risk but leave no evidence of their fate during field collections, they would be under-represented and the resulting survival estimate would be biased. Similarly, if smaller or more fragile lamb skeletons are less likely to be detected, juvenile survival may be overestimated (Gilbert et al. 2014). Fundamentally, life tables fail to track how individual mortality risk varies through time. In contrast, a well-designed continuous time survival study actively tracks individuals as they experience individualized and variable risk exposure. The specific approaches and limitations of life table methods in ecology, including as a means of survival analysis, are discussed elsewhere (Hastings 1997; McCallum 2000; Neal 2004). The remainder of our chapter focuses specifically on survival monitoring of individual subjects in continuous time.

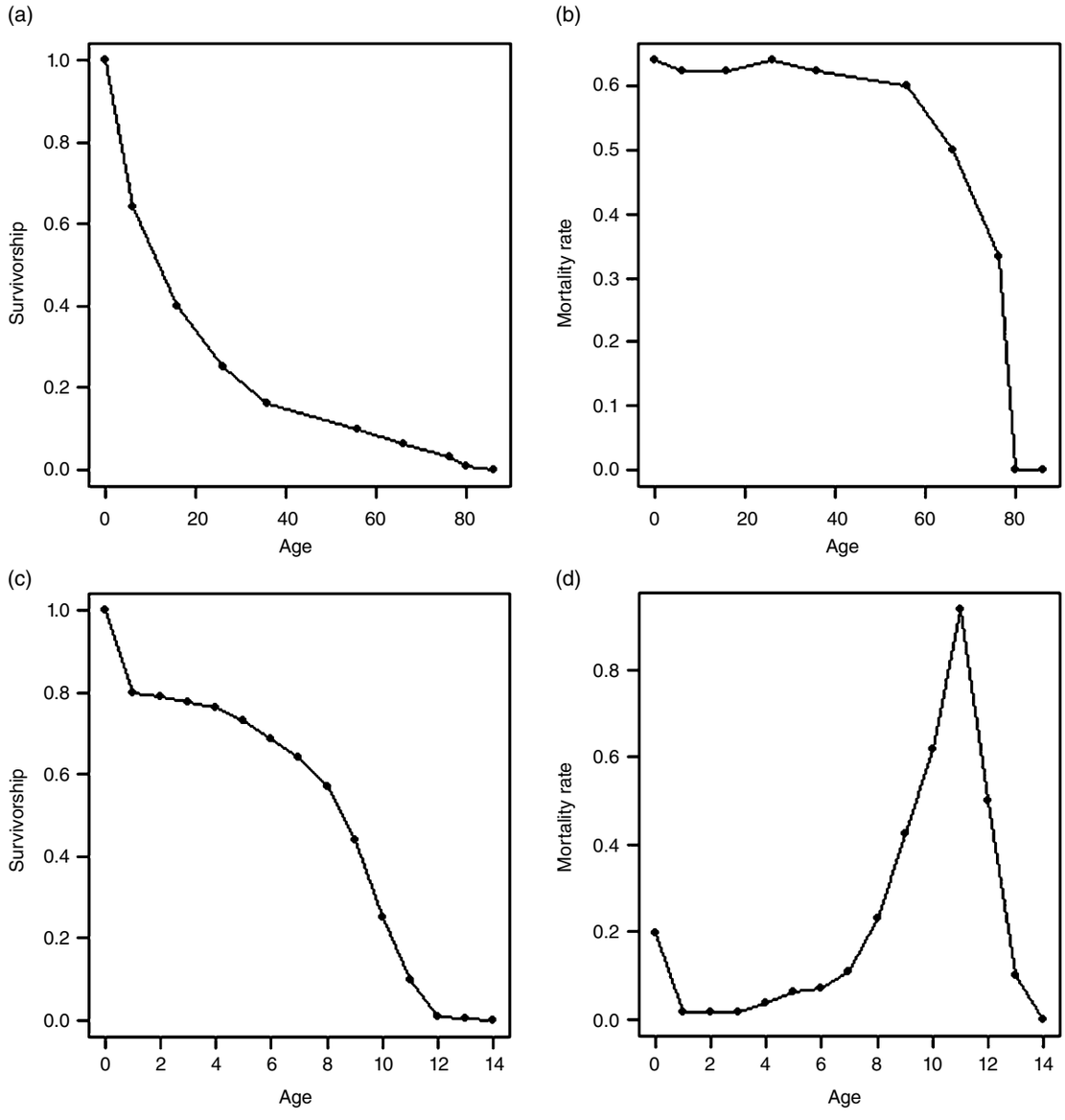

Figure 6.2 Cumulative survivorship (a) and (c) and age-specific mortality rate (b) and (d) in humans (approximately 8 year block) and Dall Sheep. Datasets for humans and sheep were from Graunt (1662) and Murie (1944), respectively.

6.2 Survival and Hazard Functions in Theory

To properly track risk through continuous time, we must establish representative survival timelines for individual subjects. Quantitative survival estimation measures the *cumulative* survival function $S(t)$ and the hazard function $h(t)$ of individuals. Assume that T is a non-negative random variable denoting time-to-failure (i.e. death), so that the survivor function represents the *cumulative probability of survival* of an individual at least until a specified time, t;

$$S(t) = \Pr(T \geq t). \tag{6.1}$$

The survivor function reflects the probability that there is no event prior to t, which is identified as the start of the monitoring period. Therefore, the survival rate equals 1 when $t = 0$ and decreases to zero as t approaches infinity (Figure 6.3a). Indeed, across a sufficiently long study duration all subjects will eventually succumb to mortality,

Box 6.1 Life Table Analysis

Murie (1944) collected skulls from Dall sheep found dead near Mt. McKinley in Alaska. Skulls were aged based on tooth wear, allowing estimation of the number of animals dying in each age class (x). We can then use a life table approach to reconstruct the demography of the population. From an initial sample of 608 sheep skulls, Murie calculated the number left alive (n_x) when expressing the starting population as 1000 individuals (i.e. conversion factor: 1000/608 = 1.645). Number of sheep dying in age class x is: $d_x = n_x - n_{x+1}$. Proportion of the total population surviving (l_x) is: $l_x = n_x/n_0$ and the mortality rate (q_x) is: $q_x = d_x/n_x$. The average number of individuals alive in each age class (L_x) is: $L_x = (n_x + n_{x-1})/2$. We can determine age-specific life expectancy (e_x) by calculating: $e_x = T_x/n_x$, where $T_x = \sum_x^{\infty} L_x$. The life table for the Dall sheep population is provided below, and proportion surviving and mortality rate relative to age are graphed in Figure 6.2c and d, respectively.

Age class (x)	Number alive (n_x)	Number dying (d_x)	Proportion surviving (l_x)	Mortality rate (q_x)	Avg. no. alive in age class (L_x)	T_x	Life expectancy (e_x)
0–1	1000	199	1.000	0.199	900.5	7053	7.0
1–2	801	12	0.801	0.015	795	6152.5	7.7
2–3	789	13	0.789	0.016	776.5	5357.5	6.8
3–4	776	12	0.776	0.015	770	4581	5.9
4–5	764	30	0.764	0.039	749	3811	5.0
5–6	734	46	0.734	0.063	711	3062	4.2
6–7	688	48	0.688	0.070	664	2351	3.4
7–8	640	69	0.640	0.108	605.5	1687	2.6
8–9	571	132	0.571	0.231	505	1081.5	1.9
9–10	439	187	0.439	0.426	345.5	576.5	1.3
10–11	252	156	0.252	0.619	174	231	0.9
11–12	96	90	0.096	0.937	51	57	0.6
12–13	6	3	0.006	0.500	4.5	6	1.0
13–14	3	3	0.003	1.000	1.5	1.5	0.5

although in a time-to-failure context, those who are lost from the study and thus succumb to an unknown fate only contribute to $S(t)$ while they are successfully detected. Variable T can be considered as either continuous or discrete, with the former tallying death over distinct, fixed-length time intervals and the latter tracking mortality across largely uninterrupted timelines. For example, if researchers monitor turtle nest survival by relocating nests on foot, daily visitation is probably necessary to achieve the uninterrupted timeline for a continuous time analysis; less frequent detection will result in discrete time intervals with uncertain timing of death events, potentially warranting a discrete time analysis.

As time progresses, the survival function based on cumulative probability of survival inevitably declines, as did survival functions for humans (Figure 6.2a) and Dall sheep (Figure 6.2c). The shape of the survival function reflects how mortality risk changes with time, with steeper declines indicating increased risk as the individual ages. Thus, we can consider the cumulative distribution function of the survival timeline, $F(t)$, which reflects the probability of dying by T and is the complement of the survivor function:

$$F(t) = 1 - S(t) = \Pr(T \le t). \tag{6.2}$$

The trajectory of the cumulative distribution function under a range of scenarios is described in Figure 6.3b. It may be helpful to consider the cumulative distribution function as equivalent to a cumulative mortality function.

The *hazard function* $h(t)$ is the fundamental unit in the statistical analysis of survival and represents the instantaneous failure rate, or in other words, the instantaneous risk of death conditional upon the subject's survival to the beginning of the time interval (Cleves et al. 2010). Expressed in terms of probabilities, the hazard function is:

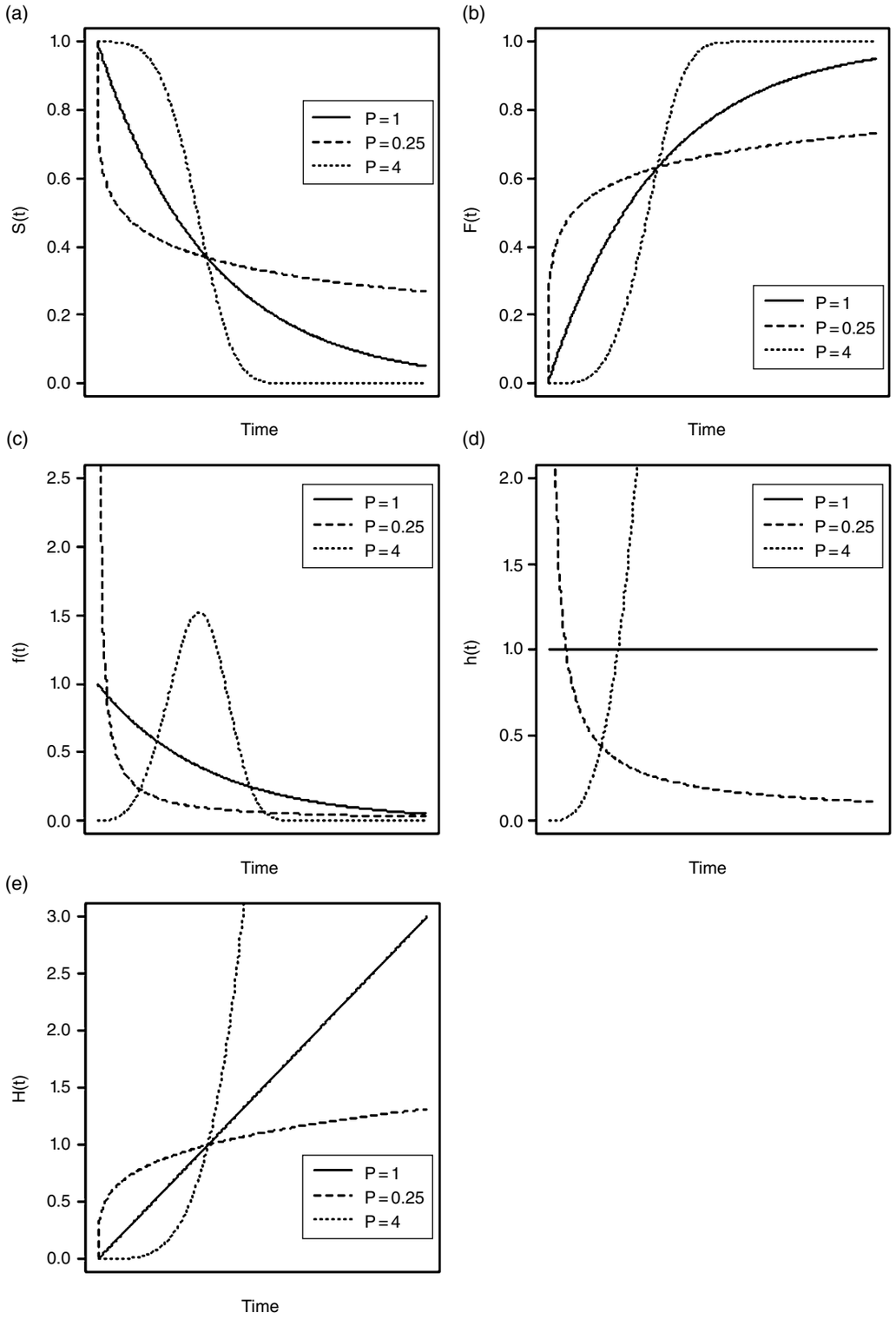

Figure 6.3 Theoretical functions relating cumulative survival (a), cumulative distribution (b), probability density (c), hazard (d), and cumulative hazard (e) in hypothetical scenarios. Parameter *P* refers to the dimensionless shape parameter defined by a Weibull distribution.

$$h(t) = \lim_{\Delta t \to 0} \frac{\Pr(t \le T < t\Delta t \mid T > 1)}{\Delta t}. \quad (6.3)$$

Equation (6.3) illustrates that the probability of survival past T is a function of the risk as time approaches zero (Figure 6.3c), meaning that the hazard rate represents the risk of death over an infinitesimally small time unit. Practically speaking, to achieve $\Delta t \to 0$, survival status should be monitored over short time intervals with a high probability of detection, although to some degree these criteria can be relaxed if hazard is very low. For instance, in the case of turtle nest monitoring it may be possible to relax daily monitoring if mortalities are especially infrequent or if the timing of death can be estimated with little error. Ultimately, continuous time survival analysis requires that subjects are monitored intensively so that timelines are largely uninterrupted, and timing of mortality is precisely known.

An important difference between the survival and hazard function is that while survival rates are bounded from zero to one and the survival function experiences a constant decline when plotted against time, hazard rates can range from zero (no risk) to infinity (certainty of death) and can increase, decrease, or follow irregular patterns depending on how risk changes with time. If mortality risk increases dramatically with age, the survival function declines rapidly while its corresponding hazard function increases; conversely, if risk decreases with age, the survival function declines gradually and the hazard declines as well (Figure 6.3c). The hazard function is linked to its survival function through the corresponding *probability density function*, $f(t)$, where:

$$h(t) = \frac{f(t)}{S(t)}. \quad (6.4)$$

Note that the notation used implies that the capitalized term (i.e. $F(t)$) represents the cumulative sum, whereas the lowercase (i.e. $f(t)$) is its first derivative [Figure 6.3]. To estimate the hazard function from the survival function, we note that the first derivative survival function, $S'(t)$, and probability density function, are linked [$f(t) = -S'(t)$]. Accordingly, we recognize that hazard and survival functions are related such that if hazard is constant through time, $h(t) = \lambda$, then $S(t) = e^{-\lambda t}$. We can then derive the survival function directly from the hazard function by:

$$S(t) = \exp\left[-\int_0^t h(u)du\right], \quad (6.5)$$

which also allows us to back-calculate to obtain the hazard function from the survival function:

$$h(t) = -\left[\frac{{}^{dS}(t)/_{dt}}{S(t)}\right]. \quad (6.6)$$

These algebraic exercises demonstrate that the survival, hazard, and related functions are all directly linked. To further illustrate this point, we can invoke an additional function, the *cumulative hazard function*, $H(t)$, which measures the accumulation of hazard at a given point in time and can be represented as:

$$H(t) = \int_0^t h(u)\,du. \quad (6.7)$$

$H(t)$ reflects the sum of hazards experienced by a subject and thus is a measure of risk accumulation. In other words, the derivative (slope) of the cumulative hazard function provides a time-specific estimate of hazard, and logically the accrual of this hazard can increase or remain stationary but never decrease with time (Figure 6.3e).

From the above calculations, we note that the cumulative hazard function, $H(t)$, is related to our other functions of interest, namely the hazard (6.7a), cumulative survival (6.7b), cumulative distribution (6.7c), and probability density functions (6.7d):

$$h(t) = \frac{d}{dt}H(t) \quad (6.8a)$$

$$S(t) = \exp\{-H(t)\} \quad (6.8b)$$

$$F(t) = 1 - \exp\{-H(t)\} \quad (6.8c)$$

$$f(t) = h(t)\exp\{-H(t)\}. \quad (6.8d)$$

At this juncture, we can consider more explicitly the linked expression of these various functions. As it turns out, the hazard function can follow a variety of functional forms, the most versatile being the *Weibull function*. The Weibull invokes a dimensionless shape parameter (p) that specifies how hazard varies through time. We can express the Weibull hazard function as:

$$h(t) = pt^{p-1} \quad (6.9a)$$

and the four corresponding related functions as:

$$S(t) = \exp(-t^p) \quad (6.9b)$$

$$F(t) = 1 - \exp(-t^p) \quad (6.9c)$$

$$f(t) = pt^{p-1}\exp(-t^p) \quad (6.9d)$$

$$H(t) = t^p \quad (6.9e)$$

Figure 6.3 illustrates several of these functions across a sample of p values.

6.3 Developing Continuous Time Survival Datasets

Continuous time survival monitoring gives rise to data that are comprised of consecutive non-negative observations from the same individual. As such, survival data do not conform to standard assumptions of independence

and normality, and therefore require specialized dataset structure and analytical techniques. Further, continuous time survival analysis was originally developed for human medicine and industrial design, where subjects are tracked with relative ease, continuity, and certainty of detection compared to ecological studies. Concerns regarding the rigor of survival datasets and analyses in ecology highlight a need for careful data collection, dataset structuring, and data exploration, to avoid weak inference and biased results (Winterstein et al. 2001; Williams et al. 2002; Murray 2006).

6.3.1 Dataset Structure

We consider an example with a continuous time survival dataset for five individuals in a hypothetical population (Table 6.1). A survival dataset normally should reference when observations begin and end for each individual, and in standard survival analysis subject fate is recorded as a binary variable (death: "1"; non-death: "0"). Individuals failing to die during the study are *right-censored* and also coded as "0" (see ID no. 2 and 5, Table 6.1). As discussed below, censoring includes all non-mortality outcomes such as intentional withdrawal from the study, loss of contact, emigration, and study termination prior to a subject's death (Collett 2003; Murray 2006). *Left-censoring* refers to staggered entry and the timing that a new subject is recruited to the study.

Continuous time studies aim to eliminate interruptions in subject timelines, and Murray (2006) provides recommendations for establishing continuous time survival monitoring schedules. However, even with intensive monitoring longer gaps may arise when subjects are not surveyed or detected for extended periods. For

Table 6.1 Hypothetical continuous time survival dataset.

Id	Timein	Timeout	Days	Fate	Birthday	Mass	Sex	Ageclass	Year	COD1	COD2
Basic											
1	21jan12	22july13	548	1	15jan11	1250	1	1	2		
2	21jan11	27may12	492	0	2jan08	1100	0	3	1		
3	10feb12	12oct12	245	1	20jan10	1340	1	2	2		
4	21jan12	07mar13	411	1	8jan11	1190	1	1	2		
5	15may12	01dec12	200	0	10jan11	1350	0	1	2		
5	12feb13	11jun13	119	1	10jan11	1350	1	1	2		
Time-dependent											
1	21jan12	22july13	548	1	15jan11	1250	1	1	2		
2	21jan11	27may12	492	0	2jan08	1100	0	3	1		
3	10feb12	12oct12	245	1	20jan10	1340	1	2	2		
4	21jan12	07mar13	411	1	8jan11	1190	1	1	2		
5	15may12	01dec12	200	0	10jan11	1350	0	1	2		
5	12feb13	11jun13	119	1	10jan11	1350	1	2	3		
Competing risks											
1	21jan12	22july13	548	1	15jan11	1250	1	1	2	1	0
2	21jan11	27may12	492	0	2jan08	1100	0	3	1	0	0
3	10feb12	12oct12	245	1	20jan10	1340	1	2	2	1	0
4	21jan12	07mar13	411	1	8jan11	1190	1	1	2	0	1
5	15may12	01dec12	200	0	10jan11	1350	0	1	2	0	0
5	12feb13	11jun13	119	1	10jan11	1350	1	2	3	0	1

The dataset consists of five subjects (Id), each was recruited (Timein) and exited (Timeout) the study on a known date. The total number of days monitored is recorded for each individual (Days) as is the fate (1 = death, 0 = censor). Birthdate was known for each individual. When each subject was recruited to the study, its body mass (Mass: continuous variable), gender (Sex: binary variable), age category (Ageclass: categorical) and year of capture (Year) were recorded. In the Basic table, each row represents a continuous timeline and Subject no. 5 is distinguished by having two entries, reflecting a gap when the individual was not monitored. The Time-dependent dataset is extended to convert Ageclass and Year into time-dependent covariates that are adjusted at the beginning of each calendar year. The Competing risks dataset further extends the dataset to differentiate individuals according to their cause of death (COD1, COD2). Note that this data structure can be altered depending on the software package used and whether the study is designed to monitor a cohort with fixed recruitment timing (i.e. no staggered entry) or if all subjects are monitored to the time of death.

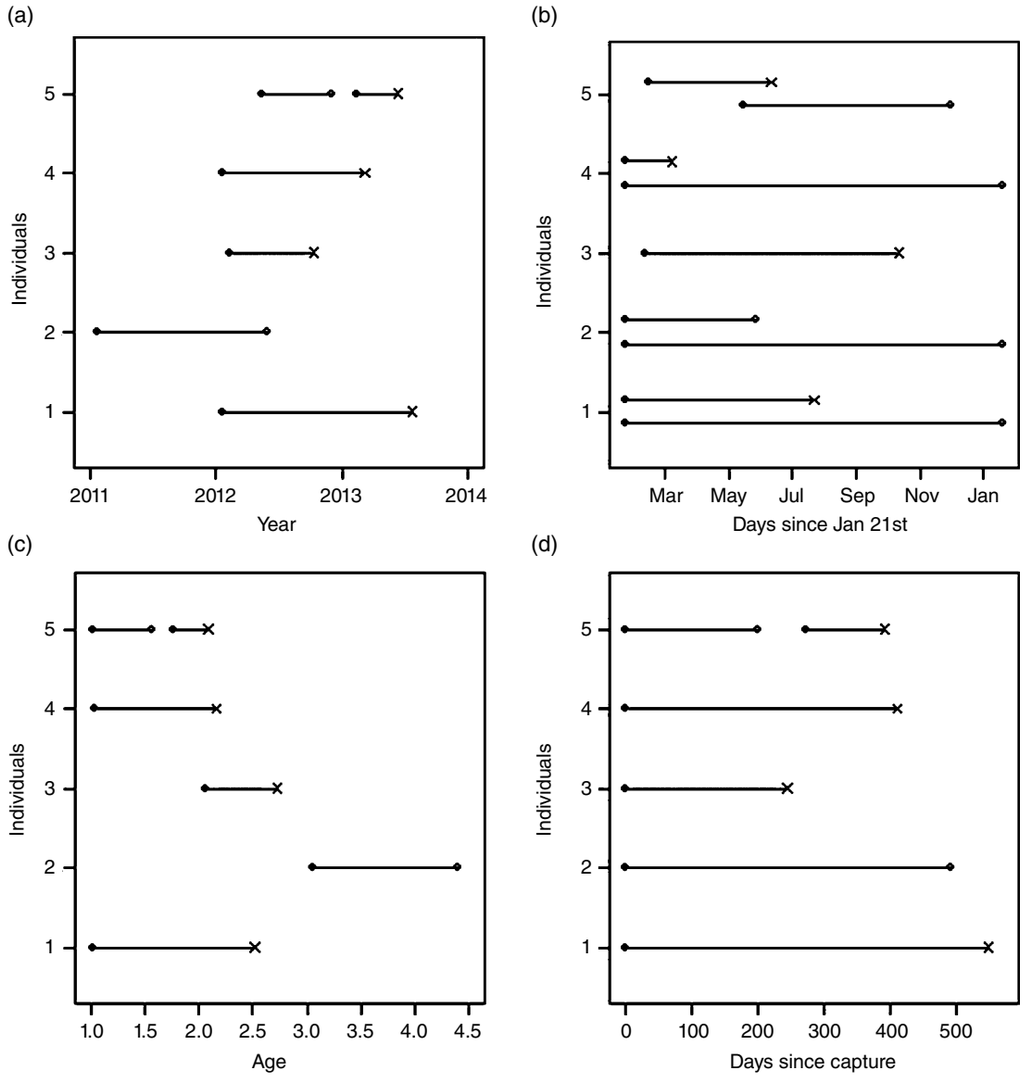

Figure 6.4 Timelines and fates of hypothetical individuals in a continuous time survival analysis. Each panel reflects a different time origin and structure for the same data. The five subjects correspond to those described in the basic dataset from Table 6.1, and are displayed according to study-related (a), recurrent (b), age (c), and time since capture (d) study designs.

example, in nest survival studies, periods of inclement weather may preclude daily visitation by researchers, whereas in radio telemetry research animals may temporarily leave the study area or else transmitters can fail and only be replaced at a later date. Normally, such gaps in an individual's timeline should be censored, which results in subject timelines being comprised of distinct uninterrupted segments punctuated by gaps with no data (see id no. 5, Table 6.1; Figure 6.4a). Yet, such gaps remain a point of contention as some authors suggest that missing observations can be imputed using standardized procedures, although the appropriate structure of such imputation models remains in doubt given that the dependent variable includes both event (i.e., death) and time (Van Buuren et al. 1999; White and Royston 2009). A critical point here is that, as a general rule, gaps in survival timelines should never be interpolated manually because this will bias the survival estimate in favour of subjects who re-enter the dataset and against those who fail to do so (Winterstein et al. 2001).

In theory, continuous time survival studies should detect mortality events more or less when they occur, leaving little uncertainty in the estimated death time. However, low detection success often adds uncertainty to the estimated timing of death, as may be the case when a subject is found dead after a gap in detection. How one

corrects for the gap between the last live detection and the mortality event can be problematic because the common approach is to assume that mortality occurred at the *midpoint* of the monitoring gap (Winterstein et al. 2001; Murray 2006). In theory, this approach will underestimate variance and inject bias in the survival estimate (Lindsey and Ryan 1998; but see DeCesare et al. 2017). Likewise, assuming that several mortality events with uncertain dates occur at the midpoint of a monitoring gap creates *tied death times*, which is mathematically not possible in a continuous time context (Murray 2006). Some remedies for tied death times, including death time randomization or application of survival timeline likelihood functions, are implemented as defaults in some survival analysis software (Kalbfleisch and Prentice 2002; Collett 2003). Ultimately, while these adjustments may help address uncertainty in mortality timing, as a general rule, datasets that have extensive gaps in survival timelines and high uncertainty in death times may be best suited for approaches based on discrete time (Chapter 7) or interval censoring (Singer and Willett 1991).

6.3.2 Right-censoring

Right-censoring occurs when an individual's ultimate disposition is not known (see Id no. 2 and 5, Table 6.1). As a general guideline, right-censoring should be kept to a minimum and also be random or "noninformative" (Collett 2003). However, censoring rates can be a problem in ecological research because of the challenges associated with intensively tracking a representative sample of subjects under unpredictable field conditions to precisely estimate timing of the death event (Murray 2006). For example, field studies of long-lived organisms are almost always completed before all subjects have died, meaning that datasets inevitably include right-censored individuals. Likewise, if nests that are located in dense vegetation are less likely to be relocated at all times during the study, or if smaller individuals are prone to lose radio transmitters and thus succumb to an unknown fate, censoring will be biased. Field adjustments can be implemented to address censoring issues, including broadening relocation efforts to improve detection success or extending the study duration until most individuals have died. Sometimes, sources of censoring can be identified and reclassified using ancillary information obtained when the subject was last detected (Hays et al. 2007). Diagnostic tests for *informative censoring* involve assessing the significance of covariates potentially associated with censoring status, or plotting survival time of known deaths versus censored individuals (Oakes 2001; Collett 2003).

As an example of the problems that can arise with censoring, Smith et al. (2010) studied survival of recolonizing wolves (*Canis lupus*) for 22 years in the northwestern United States, where packs ranged over broad areas and were heavily persecuted by humans. A longstanding concern is that wolf survival estimates could be inflated if emigrating animals have a higher mortality risk or if radio-collared individuals are killed illegally and their transmitters are intentionally destroyed, leaving no evidence of their fate. To minimize overall censoring rates and the potential for informative censoring, Smith et al. (2010) intensified searches for missing animals when radio contact was lost and deployed new transmitters whenever animals were recaptured. Prior to their survival analysis, the authors confirmed that survival time was similar between censored and noncensored groups and that other potential confounding factors like proximity to human activity or to the edge of the monitoring area did not influence censoring patterns. Because censoring rates were comparable overall and only slightly higher for animals residing in remote locations, Smith et al. (2010) concluded that informative censoring was likely negligibly associated with emigration from the study area rather than from human persecution. In contrast, Liberg et al. (2011) found that high censoring rates among radio-collared wolves in Sweden were attributable to *cryptic poaching*, which took place when animals were killed intentionally by humans who then disposed of the transmitter and associated evidence of mortality. Consequently, an analysis that reclassified previously censored wolves as dead revealed an unsustainable mortality rate for the Swedish wolf population. This sensitivity analysis highlights both the potential role of informative censoring on bias in survival estimates, as well as the importance of intensive monitoring and data exploration for deciphering censoring patterns in survival datasets.

Once detected, informative censoring may be addressed either by establishing confidence intervals (CI) for survival times of censored animals or by conducting the survival analysis separately across censoring categories. It is also possible to develop survival models where censoring status is parameterized explicitly, although this latter option may involve making untestable assumptions about survival times and censoring patterns (Collett 2003). As a general observation, survival studies in ecology should pay much closer attention to censoring patterns, including reporting overall censoring rates, testing for potential censoring bias, and adopting appropriate corrective measures (Murray 2006).

6.3.3 Delayed Entry and Other Time Considerations

Survival estimates can be influenced by the structure of the *time scale* used in analysis (Fieberg and DelGiudice 2009), and continuous time survival datasets should be

coded with time scales that are biologically sensible and that reflect study design considerations (Figure 6.4). This point is especially relevant to ecological studies, where mortality risk can be sensitive to the effects of age, seasonality, or calendar time. For example, if tree survival is repeatedly influenced by seasonal drought (Mueller et al. 2005), recurrent time (i.e. an annual cycle) may be the appropriate time scale for survival estimation; if mammals are subject to especially high mortality immediately postpartum (Gilbert et al. 2014), age-based tracking may be warranted (i.e. tracking starts at birth). In our basic dataset for a hypothetical organism (Table 6.1), calendar time was selected as the time scale because of the long duration of monitoring and lack of age-related or seasonal mortality pulses. Thus, researchers should consider the range of time scales that are possible (Figure 6.4), and select the best option for their study circumstances. Likewise, selecting the appropriate time unit for analysis (i.e. days, months, years) is important because it can influence the interpretation and utility of survival estimates and related model coefficients.

In many field studies, individuals are recruited through an extended period sometimes spanning months or years, leading to *delayed entry* (i.e. left-censoring or staggered entry) (Table 6.1; Figure 6.4a). Variation in timing of entry poses a number of challenges including that early mortalities can bias survival estimates if the initial sample size is small and some subjects die before all are recruited (Murray 2006; Fieberg and DelGiudice 2011). Sometimes, the survival dataset can be left-truncated to exclude early recruits, as was done by Smith et al. (2010) for a handful of wolves that were monitored during the 1980s. Here, the dataset was restricted to subjects monitored after 1994, which was justified because a small minority of animals were monitored during the early years of the study, survival estimates were comparable before versus after this date, and temporal variables did not distinguish early versus later recruits in an exploratory survival analysis (Smith et al. 2010). Occasionally, handling or marking protocols themselves may influence survival, and measuring changes in subject condition or behavior immediately post-capture can reveal the potential benefits of left truncating individual survival timelines prior to their full recovery (Dechen Quinn et al. 2012).

Left truncation also inevitably arises when survival monitoring is limited to select time periods. For neonatal ungulates, recruitment to a survival study typically happens within days after birth, when juveniles can be effectively captured and equipped with radio transmitters (Murray 2006). However, neonatal ungulates commonly experience high predation risk within hours after birth and this mortality pulse is usually missed when animals are recruited using traditional protocols and timelines (Gilbert et al. 2014). Thus, survival rates for juvenile ungulates are regularly overestimated, and this bias is specifically due to left truncation of subject timelines. Thus, researchers should conduct appropriate data exploration and report potential survival estimate bias from left truncation (Fieberg and DelGiudice 2011).

6.3.4 Sampling Heterogeneity

Across a cohort of subjects, individuals with *higher frailty* will die sooner, leaving those with lower risk to contribute disproportionately to mortality rate estimation. Likewise, if an initial group of subjects is recruited and tracked, over time the cohort of survivors will be entirely comprised of older individuals that may have higher or lower hazards. In either scenario, lack of replacement leads to progressive sampling heterogeneity, which biases the survival estimate and limits its generality (Zens and Peart 2003; Prichard et al. 2012). It follows that such problems will be aggravated by high mortality rates, prolonged study duration, or high initial heterogeneity among subjects. Thus, proper study design and subject recruitment are crucial for limiting progressive *sampling heterogeneity*, and important corrective measures include delayed recruitment of representative subjects to replace those that have died (Murray 2006). To detect whether a survival dataset is affected by sampling heterogeneity, baseline hazards can be plotted and checked for temporal variation (Vaupel and Yashin 1985). From an analytical perspective, sampling heterogeneity can be evaluated using study entry time as a covariate in survival models (Collett 2003; Murray 2006; Prichard et al. 2012), although this approach may cause unwanted correlation between the predictor and response variables.

Sometimes researchers intentionally recruit different types of subjects into a survival study, thereby making sampling heterogeneity an implicit aspect of study design. Smith et al. (2010) radio-collared wolves that were representative of the larger population as well those that were recently involved in livestock depredation. Baseline mortality risks were markedly higher for wolves that killed livestock, so Smith et al. (2010) necessarily treated each group separately in analyses and used only the representative sample in deriving survival estimates for the broader wolf population. Thus, researchers should be aware of how different sampling regimes can affect sampling heterogeneity and attendant survival estimates, and thereby assess the composition of the sampling population at different points during the study. When diagnostic tests reveal sampling heterogeneity and the potential for biased survival estimates, necessary adjustments may involve restricting the analysis to subjects that best reflect the population of interest.

6.3.5 Time-dependent Covariates

As individuals go through life, susceptibility to risks will vary with time; risks tend to increase or decrease according to intrinsic factors like age, body condition, or reproductive status, and to extrinsic factors like temperature, predator numbers, or season. Whenever possible, survival datasets should capture such variability by including time-dependent (or time-varying) covariates that account for temporal changes in risk owing to variability in predictor variables (Kleinbaum and Klein 2011). For example, if we want to document the effect of environmental conditions on survival of an endangered cactus at the northern edge of its geographical range, we should include daily temperature measurements in the survival dataset so we can relate daily temperature variation to plant survival.

Time-dependent or "time-varying" covariates are designed to reflect timing of the change in risk factors. Specifically, the dataset is constructed to reflect potential temporal change in the variable's impact on risk status of the individual. In Table 6.1, under the Basic classification, the five subjects are classified with a fixed value for the Age class and Year variables, meaning that these values represent conditions at the time that subjects were first recruited into the study. These values do not consider the time-varying nature of age or year on subject mortality risk. However, because subject no. 5 was monitored for an extended period during which it transitioned to an older age category and year of study, appropriate adjustments to these variables are needed to reflect temporal variability in risk. Under the Time-dependent classification (Table 6.1), Id no. 5 is recognized as being in a higher age category and subsequent year during its second monitoring period. Here, the Age class and Year variables were adjusted to represent the subject's current status with respect to those variables rather than conditions at the time of recruitment to the study, as was the case for the Basic classification.

Time-dependent variables can be incorporated into survival datasets by splitting subjects into different observation periods. As a rule of thumb, extrinsic variables are easier to present in a time-dependent context because they may not require intensive monitoring of subjects and related data on location, behavior, or condition. In the cactus example, coding for extreme weather conditions would be a simple matter of relating the recorded daily temperature, or daily minimum temperature, with the outcome of our survival monitoring for that time interval. The frequency of updating time-dependent variables, and the degree that subject timelines will need to be split to accommodate time-dependent variables, will depend on both the objectives of the study and the availability of ancillary information to include in the dataset. Intensive (i.e. daily) timeline splitting is required to document the effect of a specific temperature threshold on cactus survival, but it may be less crucial if we are more concerned with the cumulative effect of low temperature or with a factor that varies little over time. Another consideration is that time-dependent covariates can be coded either in real-time or following time delays to reflect lags between exposure to risk factors and expression of related changes in risk. For female wolves, reproductive status might be classified as a time-delayed time-dependent variable, because physiological effects of pregnancy and lactation on mortality risk may be most important especially in the months following parturition (Smith et al. 2010). Note that there are a variety of considerations and coding options that can guide the structure of time-dependent variables in a survival dataset (Kleinbaum and Klein 2011), and this aspect of dataset design requires careful evaluation of both data availability and study objectives.

6.4 Survival and Hazard Functions in Practice

6.4.1 Mayfield and Heisey–Fuller Survival Estimation

To understand how survival and hazard rates vary through time or relative to different factors, we first review the more common continuous time survival estimators. The Heisey–Fuller, Mayfield, and related estimators track survival over distinct time intervals where risk remains constant within an interval. The Kaplan–Meier and Nelson–Aalen estimators let mortality events rather than changes in risk determine the duration of time intervals. The *Heisey–Fuller estimator* (HF) was developed specifically for radio telemetry research where a group of individuals are monitored intensively for survival status and fate, whereas the Mayfield and related estimators originate from field studies involving continuous time monitoring of bird nest failure (Mayfield 1975; Williams et al. 2002). For illustrative purposes, we focus on the HF estimator, but the Mayfield estimator and related methods share a similar foundation and provide largely consistent results (Trent and Rongstad 1974; Heisey and Fuller 1985; Williams et al. 2002). We compute HF survival by:

$$\hat{S} = 1 - \frac{d}{r}, \tag{6.10}$$

where d is the number of deaths and r is the cumulative duration of monitoring. In nest survival or radio telemetry studies where individuals are monitored on a daily basis, parameter r is the number of individual days of exposure, and the term d/r is the *crude mortality rate*,

where "crude" means that all causes of death are included. Confidence limits for the HF and related estimators can be calculated using methods outlined in Johnson (1979), Bart and Robson (1982), Heisey and Fuller (1985), and Powell (2007). Because in HF survival estimation risk is constant within a time interval, d/r should remain consistent from beginning to end of the interval. Accordingly, the HF is expanded to an interval spanning t days by: $S(t) = S^t$, and to i intervals by:

$$\hat{S} = \prod_{i=1}^{I} S_i. \tag{6.11}$$

For example, Wirsing et al. (2012) monitored mortality rates of nests of painted turtles (*Chrysemys picta*) and snapping turtles (*Chelydra serpentina*) by daily checks at nest sites for the duration of the nesting period (June–September). Most nest losses were due to predation by raccoons (*Procyon lotor*). They monitored 94 painted turtle and 198 snapping turtle nests, documented 54 and 166 nest mortality events, and accumulated 3561 and 3121 observation days, respectively. Using the HF, the daily survival rate was markedly higher for painted turtles [painted: 1 – (54/3561) = 0.98484; snapping: 1 – (166/3121) = 0.94681)]. When extrapolated to a roughly four-month (110-day) nesting period (which assumes a constant mortality risk through the four-month period), nest survival is almost 19% for painted turtles and roughly zero for snapping turtles (painted: $0.98484^{110} = 0.18631$; snapping: $0.94681^{110} = 0.00245$). As an aside, when working with daily survival rates it is helpful to retain multiple digits after the decimal because rounding error can alter rates when extrapolating to longer time intervals. For painted turtle nests, $0.98484^{110} = 0.186$; $0.9848^{110} = 0.185$; and $0.984^{110} = 0.189$.

The HF estimator is attractive because survival estimates are easily calculated from summary survival data on number of deaths and number of days of exposure. The model is robust to censoring, provided that censoring is random, and overall this estimator is especially useful for populations exposed to variable risk, where risk remains constant over shorter time periods that can be pooled into distinct time intervals. However, whether risk remains constant within an interval is critical to model performance, even though researchers rarely verify this assumption (Stanley 2004; Murray 2006). In fact, we show later that HF estimates for turtle nest survival are biased because risk was not constant through the nesting period. Further, the HF model can be biased if survival is monitored across irregular and infrequent intervals because of the imprecision in estimating timing of a mortality event; in such cases application of a likelihood-based alternative is advised (Hensler and Nichols 1981). Last, another important issue with the HF estimator is that while the rates themselves are easy to compute by hand or with a spreadsheet, the CI are less straightforward and to date researchers have done so mainly using a DOS-based program (`MICROMORT`, Heisey and Fuller 1985). To our knowledge, there is no accessible MS Windows-based replacement for calculating HF CI, meaning that on a long enough timeline, the HF estimator may become obsolete and its effective survival may drop to zero.

6.4.2 Kaplan–Meier Estimator

The *Kaplan–Meier estimator* (KM) is the most popular survival estimator and is widely encountered in both medical and ecological literature. KM estimation considers the timing of death events as the determinant of interval endpoints, so if there are k unique death times, the corresponding survival rate is the product of survival during each interval, i:

$$\hat{S} = \prod_{i=1}^{k} \left[1 - \frac{d_i}{v_i}\right], \tag{6.12}$$

where d_i is the number of deaths and v_i is the number of subjects at risk during i. KM variance is calculated as:

$$\operatorname{var}\left(\hat{S}\right) = S^2 \left[\prod_{i=1}^{n} \left(\frac{d_i}{v_i(v_i - d_i)}\right)\right]. \tag{6.13}$$

A distinct feature of the KM estimator is that time interval endpoints are defined by actual mortality events and the focus of the estimator is on the individual subject rather than on the time interval; this is philosophically consistent with contemporary individual-based approaches to survival analysis, making the KM estimator a logical complement to standard analysis. The relationship between constant survival and interval endpoints follows a step-function, with Figure 6.5a showing high initial mortality during the early nesting period and lesser mortality later in the season, for both turtle species. Seasonal changes in mortality risk also emphasize why our earlier use of the HF estimator likely was not appropriate for determining turtle nest survival rates. The KM probability of cumulative survival for the 110-day nesting period is 0.357 (95% CI = 0.243, 0.524) for painted turtles and 0.165 (0.120, 0.226) for snapping turtles; these estimates differ substantially from the earlier estimates calculated using HF estimation (painted turtle: 0.186; snapping turtle: 0.002) and doubtless reveal bias in the original HF estimates (Table 6.2). However, the HF estimates could be improved to some extent by first tallying mortality risk within shorter time intervals when risk is more constant.

We can also determine nest mortality risk (hazard) by first estimating the cumulative hazard from the survival function ($H(t)$, see Eq. 6.8b) and then differentiating

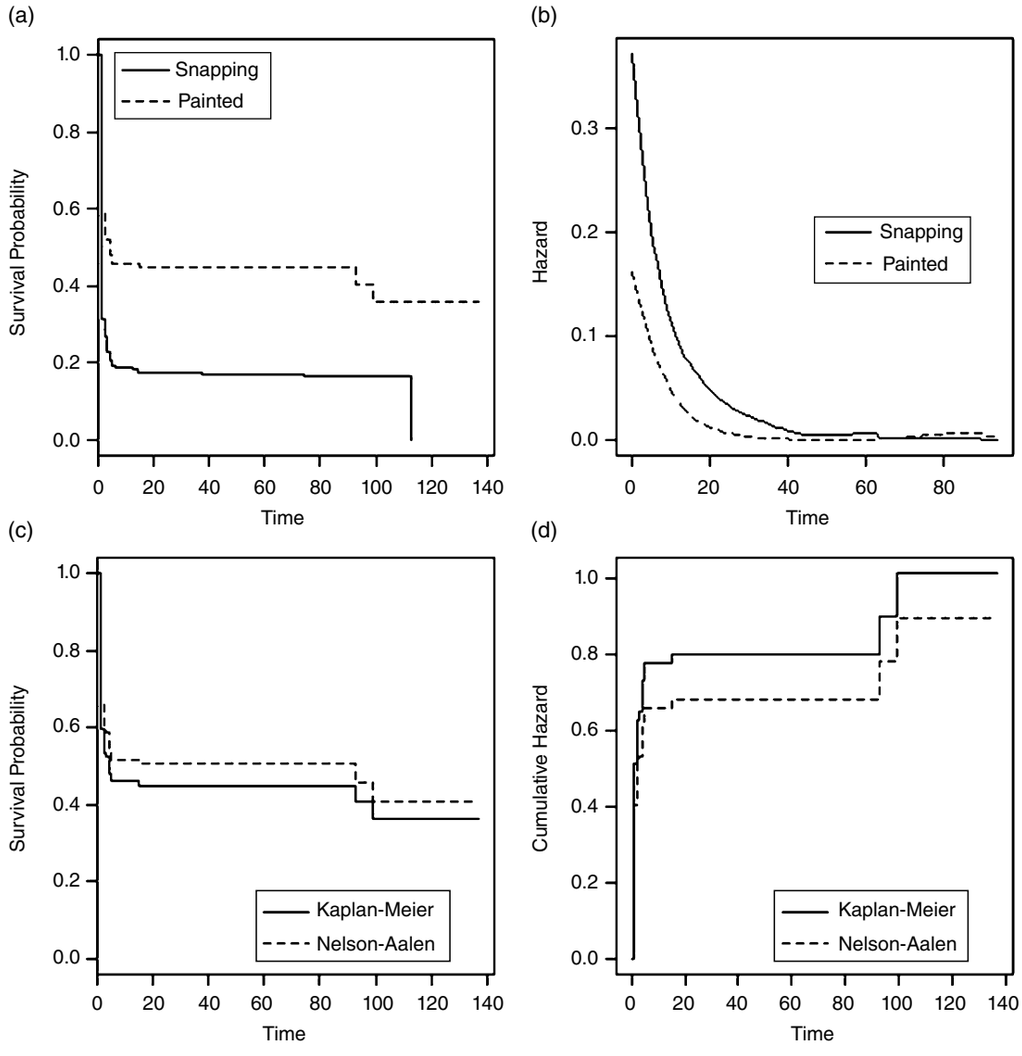

Figure 6.5 Kaplan–Meier survival functions (a), and hazard functions (b) for snapping turtle and painted turtle nests relative to time since clutch initiation. For snapping turtles only, panels (c) and (d) compare Kaplan–Meier and Nelson-Aalen survival (c), and Kaplan–Meier and Nelson–Aalen failure (d) rates. 95% CI are omitted for clarity. *Source:* The dataset was modified from Wirsing et al. (2012).

Table 6.2 Survival and failure rate estimates (±95% CI) during 110-day intervals for painted turtle and snapping turtle nests exposed to predation risk.

	Painted turtle	**Snapping turtle**
Heisey–Fuller survival	0.186 (0.119, 0.292)	0.002 (0.001, 0.006)
Kaplan–Meier survival	0.357 (0.244, 0.524)	0.165 (0.120, 0.226)
Nelson–Aalen survival	0.410 (0.288, 0.583)	0.274 (0.213, 0.354)
Kaplan Meier failure	1.030 (0.646, 1.411)	1.802 (1.487, 2.120)
Nelson–Aalen failure	0.892 (0.54, 1.245)	1.295 (1.038, 1.546)

Confidence intervals (CI) for the Heisey–Fuller estimator are obtained through Taylor series approximation (Heisey and Fuller 1985). Models were based on a modified dataset with a subset of variables and results differ from published estimates (Wirsing et al. 2012).

($h(t)$, see Eq. 6.8a). Practically speaking, this procedure is usually accomplished by using a nonparametric smoothing spline that is set to an appropriate *bandwidth* (Mueller and Wang 1994; Hess et al. 1999; Tsai et al. 1999). Bandwidth selection is largely arbitrary but the resulting graphed function should provide sufficient variation to reveal the most biologically meaningful patterns in hazards. For both turtle species, hazards decline dramatically with nest age, although there may be a slight increase in risk after 60 days (Figure 6.5b).

One drawback of the KM approach is that a new parameter is estimated with each death event, leading to lack of parsimony in studies having many deaths. Another issue is that in theory tied death times are not possible and require special treatment (Pollock et al. 1989). Since the KM estimator was initially developed for clinical research, the basic estimator is designed to deal with studies where subject recruitment occurs at the outset, but when staggered entry is present the *generalized Kaplan–Meier* model (GKM) makes adjustments for the conditional probability of late recruits (Pollock et al. 1989). For instance, the GKM would have been the appropriate estimator for survival analysis of wolves (Smith et al. 2010) or turtle nests (Wirsing et al. 2012), had calendar time rather than survival time been the temporal unit of interest. Yet, it is important to highlight that notwithstanding its advantages for late recruits, the GKM estimator is sensitive to small sample sizes, especially when mortalities occur early in the study before the full complement of subjects is under observation (Woodroofe 1985). Likewise, both the KM and GKM estimators give rise to absurd values in long-term studies when the entire initial group of subjects dies before subject recruitment is complete (Murray 2006). Thus, researchers should be judicious as to whether to include delayed entry of subjects in a survival analysis if KM or GKM survival estimation is a priority. Instead, left truncation may benefit datasets that have a small sample of early recruits. Currently, most statistical software packages do not account for staggered entry or GKM, so the default in almost all applications is the standard KM estimator.

In general, KM estimation may be superior over HF if risk varies widely with time (Figure 6.5a). In contrast, HF estimation may be more appropriate if mortality events occur in pulses or are particularly common or rare. Both HF and KM estimators are distinguished by how an interval endpoint is defined, and the methods are convergent when intervals are similarly identified (Pollock et al. 1989).

6.4.3 Nelson–Aalen Estimator

Owing to the limitations associated with both HF and KM estimation, there is a counting process analogue, the *Nelson–Aalen estimator* (NA), that can provide improved estimates, especially when sample sizes are small. The NA is often computed in the context of cumulative hazards. Recall that $H(t) = -\ln\{S(t)\}$; the cumulative hazards NA estimator is, through i time intervals:

$$\hat{H}(t) = \sum_{(j \mid t_i \le t)} \frac{d_i}{v_i}, \tag{6.14}$$

where v_i is the number at risk at t_i and d_i is the number of deaths at t_i (Cleves et al. 2010). When dealing with small sample sizes, KM survival estimates and NA cumulative hazards tend to be superior to their counterparts, and as a general rule NA tends to overestimate survival whereas KM overestimates cumulative hazards (Table 6.2). Differences between the estimators become more pronounced as sample size diminishes (Figure 6.5c, d), but both estimators are convergent when sample sizes are large. Also, in contrast to the KM estimator, the NA estimator is robust to gaps when no subjects are at risk. Nevertheless, the NA estimator seems to be underused in ecological studies, as most researchers seem to accept the default KM, perhaps without fully appreciating its potential limitations. At a minimum, simulations with variable sample size and variation could be used to establish a general rule of thumb for when KM versus NA estimation should be used in survival estimation.

6.5 Statistical Analysis of Survival

6.5.1 Simple Hypothesis Tests

Sometimes, it may be convenient to initiate a survival analysis by comparing survival functions between populations or treatments without explicitly considering a suite of predictor variables. Such tests are structured around the null hypothesis of no differences between groups, and can provide preliminary insights and summary statistics related to the primary factors imposing risk. HF estimates can be compared using confidence limit overlap, using the z-statistic for two groups (Hensler and Nichols 1981; Bart and Robson 1982) or contingency tables for larger groups (Heisey and Fuller 1985; Sauer and Williams 1989). Survival data obtained from the KM estimator can be compared using a variety of nonparametric likelihood or rank tests (Box 6.2). Survival studies in ecology sometimes restrict their analysis to these simple tests, despite that they were originally designed for evaluating straightforward hypotheses from clinical trials having balanced designs, randomization, consistent distribution of mortality events, and limited delayed entry or censoring (Murray 2006). These conditions are unlikely to be upheld in a majority of ecological datasets, making these tests inappropriate for the multitude and complexity of factors affecting mortality risk under field conditions.

6.5.2 Cox Proportional Hazards

Risk factors in observational field studies are more appropriately analyzed using multivariate regression approaches. The *Cox proportional hazards model* (CPH) is the most widely used survival analysis method in the medical sciences and is firmly established in ecological research (Williams et al. 2002; Murray 2006). The model is based on the partial likelihood of the hazard, $h_i(t)$, where the ith individual is subject to a vector of covariates potentially influencing risk; $x_I = (x_{i1}, x_{i2},\ldots, x_{ip})$. The corresponding CPH model is:

$$h_i(t) = h_0(t)\exp\left(\beta_1 x_{i1} + \beta_2 x_{i2} + \ldots + \beta_p x_{ip}\right), \quad (6.15)$$

where $h_0(t)$ is the *baseline hazard* (constant-only model) for an individual with covariate vector $x_I = (0,0\ldots0)$, and β is an unknown parameter (Murray 2006). The model is considered *semi-parametric* because the underlying shape of the hazard function remains unspecified, such that $h_i(t)$ and $h_j(t)$ differ only in that their ratio $[h_i(t)/h_j(t)]$ is proportional through time and differs by the exponential term related to the covariates. It follows that the absence of a specified hazard function makes the CPH model particularly versatile for the challenges of ecological survival research, where the functional form of baseline hazard is rarely known. CPH models generate *hazard ratios* (HR) or *model coefficients* (β) that are related by HR = exp(β), such that HR > 1.0 (or $\beta > 0$) indicates increased risk whereas HR < 1.0 (or $\beta < 0$) indicates reduced risk, relative to the baseline.

Wirsing et al.'s (2012) analysis of turtle nest survival considers a variety of risk factors potentially affecting nest predation. As an extension of our earlier survival estimate calculations (Table 6.2), multivariate CPH models (Table 6.3) reveal that for painted turtles, corridor proximity (Corridor: dummy variable, 1 = yes) is negatively associated with risk whereas date of nest laying (Datelaid: calendar date) is positively associated with risk. In other words, because Corridor has hazard <1.0, and 95% CI that do not overlap 1.0, we can infer that proximity to a corridor reduces predation risk. Datelaid has a hazard ratio > 1.0 (and 95% CI do not overlap 1.0), implying that nests laid later in the nesting season incur higher relative risk. We note that risk decreased by 81.4% (1 − 0.186 × 100) when the nest was within a corridor, and risk increased by 4.3% (|1 − 1.043| × 100) for each day that egg laying was delayed. Additionally, predation risk increased by 14% (|1 − 1.014| × 10 × 100) for every 10 m increase from vegetative cover (Veglaid variable was coded as continuous, in meters), but because the 95% CI for Veglaid overlapped 1.0, we infer that the effect of this variable was not statistically significant. For snapping turtle nests, risk increased by 22% (|1 − 1.022| × 10 × 100) for every 10 m increase in distance from water (Waterdist was coded as continuous, in meters), and with lesser (nonsignificant) influence from distance to vegetation (Vegdist was coded as continuous, in meters) and effect of disturbed habitat (Disturbed was

Table 6.3 Hazard ratios (HR) (±95% CI) from semi-parametric Cox proportional hazards (CPH) models, and hazard rates from parametric exponential and Weibull regression models, for nests of painted turtles and snapping turtles.

	Variable 1	Variable 2	Variable 3
Painted turtle	Corridor	Datelaid	Vegdist
Cox	0.186 (0.064, 0.542)	1.043 (1.014, 1.074)	0.997 (0.978, 1.016)
Exponential	0.129 (0.045, 0.369)	1.132 (1.088, 1.177)	1.013 (0.995, 1.031)
Weibull*	0.129 (0.056, 0.473)	1.062 (1.029, 1.096)	0.999 (0.980, 1.019)
**Weibull shape*	0.406 (0.326, 0.507)		
Snapping turtle	Disturbed	Vegdist	Waterdist
Cox	2.901 (0.832, 10.116)	0.997 (0.986, 1.010)	0.050 (0.023, 0.109)
Exponential	2.349 (0.735, 7.510)	1.000 (0.988, 1.012)	1.030 (1.014, 1.04)
Weibull*	2.988 (0.901, 9.920)	0.997 (0.985, 1.009)	1.025 (1.006, 1.043)
**Weibull shape*	0.509 (0.456, 0.568)		

Models for each species included only significant variables identified by Wirsing et al. (2012). Variables Corridor and Disturbed are dummy variables identifying whether the nest is on a predator travel corridor or in disturbed habitat, respectively (yes = 1), Datelaid is the Julian date when the nest was first laid, Vegdist and Waterdist is the distance to vegetation and open water, respectively. Models were based on a modified dataset with a subset of variables and results differ from published estimates (Wirsing et al. 2012).

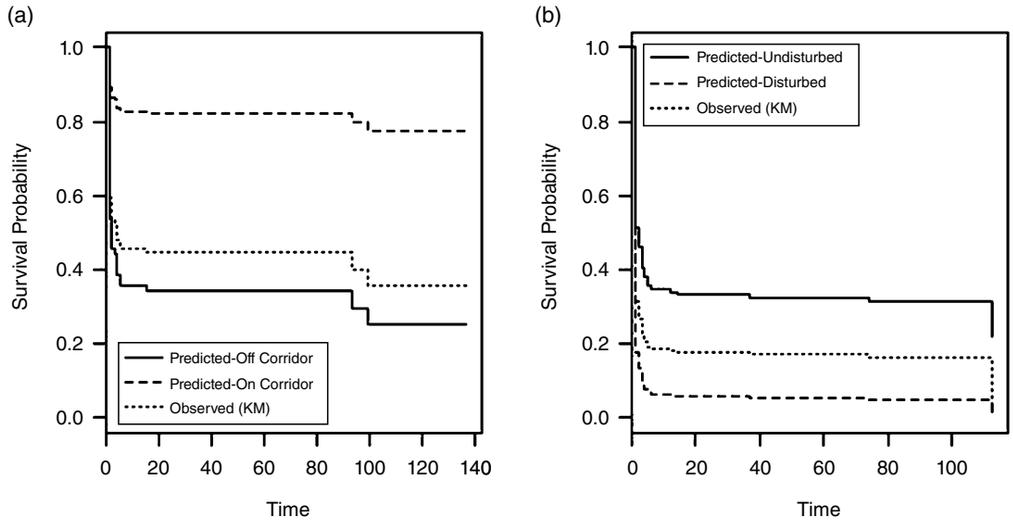

Figure 6.6 Survival probability for nests of snapping turtles (a) and painted turtles (b). Functions include Kaplan–Meier survival rates derived from observed data as well as those predicted by nest site location relative to habitat disturbance (a) and travel corridors (b) from a univariate Cox proportional hazards (CPH) model. *Source:* The dataset was modified from Wirsing et al. (2012).

coded as dummy variable, 1 = yes). In a similar study, Leighton et al. (2011) used CPH to determine sea turtle nest survival using a continuous time monitoring approach and determined that risk increased according to vegetative cover and time during the nesting season.

Once a hazard model is generated, it is possible to describe survival differences between groups by comparing HR (Table 6.3) or computing adjusted survival curves, which are computationally distinct from KM estimates because they factor in the possible effects of covariates on survival. Figure 6.6a and b provide survival curves for painted turtle and snapping turtle nests in light of predictor variables included in hazard models for each species. It is clear that there are notable differences in the estimates provided by the adjusted survival curves versus the KM estimates (Figure 6.5a) or hazards (Figure 6.5b) examined earlier.

CPH analysis requires continuous survival timelines but when ecological datasets are punctuated by *monitoring gaps* (i.e. low or variable detection probability), a counting process analogue of CPH can be used: the *Andersen–Gill model* (AG, Hosmer et al. 2008). We discussed counting processes previously in the context of the NA cumulative hazards estimator. Here, the counting process considers the survival timeline as an accumulation of conditional independent steps, which is a function of the total number of events (deaths) and the cumulative intensity process of the events up to *t*. Functionally, the counting process approach discretizes units of survival such that individual observations in time rather than continuous timelines underlie the analysis; the two approaches converge when there are no gaps in survival timelines (Murray 2006). For example, Johnson et al. (2004) used AG rather than CPH models to evaluate mortality risk among radio-collared grizzly bears (*Ursus arctos*) because the sample included many punctuated timelines and thereby favored a counting process approach. Similarly, Liebezeit and Kendall (2009) modeled the role of industrial activity on nest mortality in a variety of shorebirds and passerine birds; discontinuous nest survival timelines necessitated the application of AG rather than CPH models. Note, however, that researchers do not usually need to be concerned about the specific application of AG models in survival analysis because these are invoked as a default in most software programs when subject timelines are punctuated.

6.5.3 Proportionality of Hazards

The CPH approach is based on an assumption of *proportional hazards* across the model, meaning that there should be a linear relationship in how hazards vary according to predictor variables. The assumption can be verified using a variety of graphical approaches, and hazard proportionality between painted and snapping turtle nests can be assessed using linearity in plots of log(survival) vs. log(time) or -log(-log(survival)) vs. log (time) (Kleinbaum and Klein 2011). Our graphical assessment of the assumption using either method (Figure 6.7a and b) reveals largely parallel lines, and therefore proportional hazards. Note that here we could have used natural logs rather than $\log_{10}$ in our tests, and different software

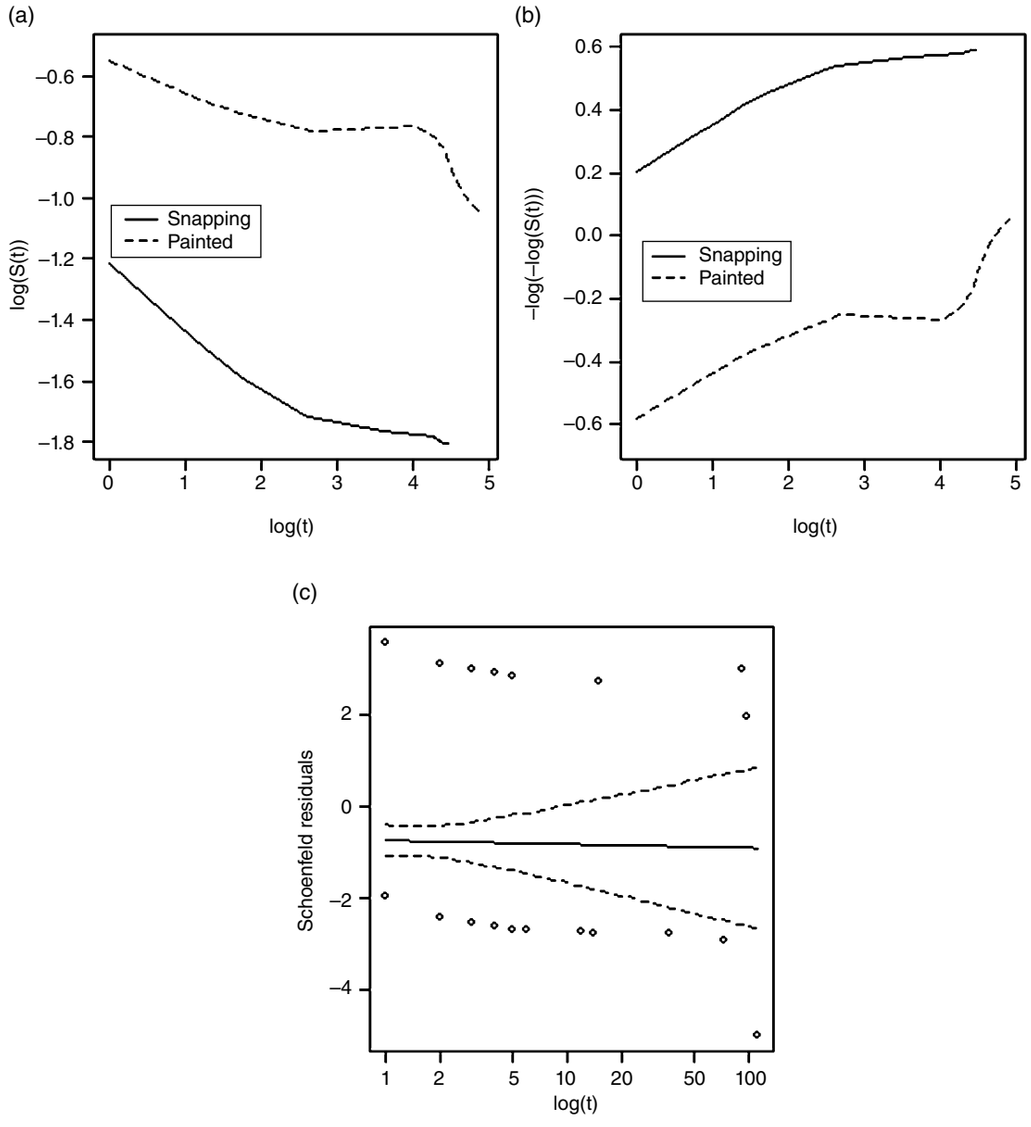

Figure 6.7 Graphical tests of the assumption of hazard proportionality from a Cox proportional hazards (CPH) model comparing nest survival of snapping and painted turtles. The most common tests are provided, including log(S(t)) (a), −log(−log(S(t)) (b), and Scaled Schoenfeld residuals (c). Dashed lines in panel c refer to 95% CI. *Source:* The dataset was modified from Wirsing et al. (2012).

programs use different transformations as defaults. Alternatively, we could plot the relationship between KM survival functions versus those from a CPH model with the predictor in question, and gauge parallelism between observed and expected functions (Kleinbaum and Klein 2011). Naturally, when predictor variables are continuous they should be reclassified into categories for proper graphical evaluation of the proportional hazards assumption.

Hazard proportionality can also be checked via analysis of residuals or model re-estimation (Therneau et al. 1990; Gail 1991). The primary tool for model checking in CPH analyses is the *Schoenfeld residuals*, which is based on the contribution of each covariate to the fit of the model specifically at each failure time. If residuals are distributed randomly, then there is no inherent structure and the proportional hazards assumption is met. For example, Schoenfeld's residuals from a simple univariate model comparing predation across turtle species are distributed randomly, with no inherent trend (Figure 6.7c), and with no statistically significant difference ($X^2 = 0.006$, $p = 0.94$). Thus, hazard proportionality is implied for

Box 6.2 Testing for Equality of Survival Curves

Simple tests are available for testing the hypothesis of no differences between two or more survival functions. These tests have limited utility in ecology but may be used when necessary, such as to test for differences between predictors that lack hazard proportionality. Let $t_1 < t_2 < \ldots < t_k$ represent the ranked failure times, with d_j being the number of deaths at t_j and n_j the number of subjects at risk prior to t_j; d_{ij} and n_{ij} are the same units for group i, $i = 1,\ldots, r$. The equality of survivor functions leads to the test:

$$H_0 = \lambda_1(t) = \lambda_2(t) = \ldots = \lambda_r(t), \tag{6.16}$$

where $\lambda(t)$ corresponds to the hazard function at t versus the alternate hypothesis that one or more $\lambda_i(t)$ is different for some t_j. If the null hypothesis is true, the expected number of failures in group i at t_j is $e_{ij} = n_{ij}d_j/n_j$ and the corresponding test statistic is:

$$\mathbf{u}' = \sum_{j=1}^{k} W(t_j)\left(d_{1j} - e_{1j}, \ldots, d_{rj} - e_{rj}\right). \tag{6.17}$$

Note that $W(t_j)$ is a weighting function that is zero when n_{ij} is zero. There are several different weighting functions that give rise to different test statistics, depending on data structure and limitations (StataCorp 2007).

Test Weight at each death (t_j)	Comments
Logrank 1	Best when hazards are proportional
Wilcoxon n_i	Best when hazards are not proportional and death and censoring patterns are similar
Tarone-Ware $\sqrt{n_i}$	Best when hazards are not proportional and test is less susceptible to death and censoring patterns
Peto-Peto-Prentice $\hat{S}(t_i)$	Best when hazards are not proportional and test is less susceptible to death and censoring patterns
Fleming-Harrington $\hat{S}(t_{i-1})^p\left\{1-\hat{S}(t_{i-1})\right\}^q$	Test is flexible, if $p > q$, more weight to early deaths, if $p < q$ more weight to late deaths, if $p = q = 0$, test reduces to logrank test

the model under consideration. In cases where complex multivariate hazard models are considered, it is appropriate to check the fit of Schoenfeld residuals both for individual predictors as well as for the global model. Cleves et al. (2010) outline a variety of additional tests that can be used to further gauge hazard proportionality in CPH models.

If hazards are not proportional, a number of options are available, including developing separate models across the problematic variable and using nonparametric methods to compare survival rates. Better yet, a stratified CPH model can be fit, where different baseline hazards are assumed across the problematic variable. Specifically, stratification allows the form of the underlying hazard to vary across levels of the stratification variable, and thus enables model fitting without estimating the effect of the problematic covariate. However, predictors that are stratifiable usually must be binary or categorical, and the stratification process precludes direct assessment of the role of the predictor on risk, except through nonparametric tests (Box 6.2).

There are two other alternatives to stratification when faced with models failing to adhere to hazard proportionality. First, the time axis can be adjusted to accommodate shorter time periods that actually conform to the proportionality assumption. Second, it may be possible to use time-dependent variables to explicitly model nonproportionality or else develop models based on accelerated failure or additive hazards (Hosmer et al. 2008; Kleinbaum and Klein 2011). However, such methods are rarely invoked in ecological research, perhaps because the assumption of hazard proportionality itself is so rarely tested or because the complexity of such techniques tends to exceed the quality of many survival datasets (Murray 2006).

6.5.4 Extended CPH

In ecology, subject mortality risk is regularly influenced by *dynamic factors* such as time-dependent variation in weather, age, condition, or behavior. As described in Section 6.3.4, time-dependent covariates open a wealth of possibilities for evaluating the role of time-sensitive predictors on risk. Here we elaborate more formally on the extended CPH model, which includes one or more time-dependent variables. Recall that time-dependent variables can be either intrinsic or extrinsic and they are adjusted during the subject's timeline, as it progresses through distinct stages for the variable in question (see Id no. 5, Ageclass and Year variables, Time-dependent section, Table 6.1). We consider the expanded CPH model as:

$$h_i(t) = h_0(t)\exp\left[\left(\beta_1 x_{i1} + \beta_2 x_{i2} + \ldots + \beta_{p1} x_{ip1}\right) + \left(\alpha_1 x_{j1}(t) + \alpha_2 x_{j2}(t) + \ldots + \alpha_{p2} x_{jp2}(t)\right)\right]. \tag{6.18}$$

where x_i and x_j represent time-independent and time-dependent covariates, respectively, and β's and α's

represent coefficients associated with time-independent and time-dependent variables, respectively. The extended CPH model assumes that the effect of a time-dependent variable on hazard at t is restricted to the value of the variable at t, and therefore does not consider its value either before or after the specified time (Kleinbaum and Klein 2011). Further, because the proportional hazards assumption is no longer satisfied for all variables under the extended CPH model, computation of the hazard ratio for the model now involves two sets of predictors, time-independent and time-dependent, and the hazard ratio becomes a function of time.

Extended CPH models are both flexible and robust to the complexities of field research, and thus should receive greater attention when risk is dynamic. We invoked time dependency in our earlier example of turtle nest survival by re-estimating rainfall and local turtle nest density on a daily basis (Wirsing et al. 2012). Similarly, Liebezeit and Kendall (2009) studied nest survival in birds and included temporal variability in risk by adjusting predictors through short time intervals. Smith et al. (2010) tallied wolf survival across three-month intervals and adjusted age, social status, dispersal status, and habitat-related predictors for each interval. In each case, time-dependent variables were assumed to be constant within time intervals and variable across intervals.

Note that the extended CPH model mimics the approach used for AG modeling, except that the CPH model structure is modified directly, whereas the AG model uses a counting process approach to accommodate time-dependent variables. In the aforementioned study of grizzly bear survival, dynamic variables representing age, road density, and habitat type were used in an AG modeling context to assess time-dependent mortality risk determinants (Johnson et al. 2004). To conclude this section, we submit that nominal additional investment in study design, data collection, or data structure and analysis may allow researchers to provide an important time-dependent context to studies that otherwise could yield weak or biased survival inference. A helpful first step would be to use simulations to clarify the optimal coding and time interval structure to best capture the dynamic effects of time-dependent variables on survival estimation.

6.5.5 Further Extensions

The CPH model has additional features that allow further refinements and improvements. Contemporary survival analysis hinges on the assumption that subjects are independent, but in reality individuals often share higher or lower frailty based on intrinsic or extrinsic similarities. Siblings may have related mortality risk depending on their genetic makeup, whereas group-living subjects may be exposed to particular *mortality agents* that affect the entire unit. In such cases, it is appropriate to generate robust standard errors that reflect shared mortality risk, a procedure that is analogous to adding a random variable to a regression model to explain correlation between clustered data (Kleinbaum and Klein 2011). Because the hazard in a *shared frailty model* is computed according to the assigned clusters, the analytical context reflects lack of independence among subjects, leading to increased precision in the estimated error. For example, in bird nest survival studies a frailty component can account for differences in predation risk between clusters of nests on the same plot (Liebezeit and Kendall 2009), whereas in wolf studies similar adjustments can improve the precision of estimated hazards for members of a common pack who share comparable exposure to natural and anthropogenic risks (Smith et al. 2010). It is also possible to use mixed-effects CPH models to explicitly estimate random coefficients to further refine comparisons between groups of subjects (Freitas et al. 2008), and such additions may be useful when dealing with unobserved heterogeneity that has strong effects on risk. However, to date such refinements and extensions of the CPH model have not gained strong traction in the ecological literature, possibly owing to sample size or study design limitations that constrain performance of more complex models.

6.5.6 Parametric Models

We consider that CPH models are well-suited for answering most survival questions in ecology, but in rare cases it may be appropriate to use fully *parametric models* to address uncertainty about the shape of the hazard function or to produce age-specific survival functions (Allison 1995; Collett 2003). For instance, Griffin et al. (2011) used parametric models to assess the role of climate and predators on the survival of neonatal elk (*Cervus elaphus*). Animals were radio-collared shortly after birth and researchers could infer birth date and age of individuals. Similarly, nest survival studies often infer the age of the nest based on the timing of its first discovery or nest and egg characteristics (Liebezeit and Kendall 2009; Wirsing et al. 2012). Accordingly, researchers can estimate the *baseline hazard* for the group of subjects according to age, and thereby use a parametric approach when estimating hazards. Yet, for most ecological studies the extent that a parametric approach will improve on inference that could be derived from a semi-parametric CPH analysis is not evident (Korschgen and Kenow 1996; Olson et al. 2014). For many field studies, researchers do not know either the underlying shape of the hazard function or the age distribution of subjects, meaning that parametric models are

often simply not a suitable option. Thus, even when parametric models can be applied to ecological datasets they may not provide marked improvements in terms of hazard estimates, parameter precision, or model fit (Murray 2006).

In the case of predation on turtle nests (Wirsing et al. 2012), the date of nest deposition was known and therefore hazard functions could be estimated parametrically. To fit a parametric model it is necessary to select an underlying statistical distribution; the simplest of these is the *exponential distribution*, which assumes constant survival, followed by the *Weibull distribution*, which allows survival to monotonically increase or decrease (see Eq. 6.3). In the case of turtle nest predation (Wirsing et al. 2012), our previous CPH models fit to each species revealed a series of HR that are qualitatively different from the hazards for the same parameter estimated using the exponential model (Table 6.3). The difference was not surprising because we know from Figure 6.5 that hazard varies markedly during the nesting season and thus that the exponential distribution is an inappropriate fit. In fact, when we fit the more flexible Weibull model to the data, the hazard estimates for most predictors tend to be closer to the HR from CPH. Further, we note that the estimate for parameter p, the dimensionless shape unit defining the trend in the survival function, is below the value of one for either turtle species (Table 6.3). A shape unit <1.0 indicates an overall decline in hazard with time; we had qualitatively identified this trend earlier, after fitting hazard functions described in Figure 6.6b. To conclude, although fully parametric models may be warranted in specific instances when survival functions are known or if they serve as a basis for hypothesis testing, in reality there are few ecological applications where parametric approaches provide either improved parameter estimation or novel insights into the drivers of mortality risk.

6.6 Cause-specific Survival Analysis

6.6.1 The Case for Cause-specific Mortality Data

We began our chapter with a description of John Graunt's analysis of human mortality patterns in Renaissance London. Perhaps Graunt's most lasting contribution was the cataloging of causes of death befalling London residents during a time when sundry mortality agents prevailed across Europe. Graunt's original records report as many as 80 mortality agents, including the rather colorful "king's evil" (tubercular infection of lymph nodes), "hanged and made-away themselves," and "lunatique" (Graunt 1662). Yet, in retrospect one must question the accuracy and variety of Renaissance death records given the limited clinical and diagnostic tools available at that time. In contrast, contemporary ecologists are more restrained in their attribution of causes of death, with rarely more than four to six different proximate causes tending to be reported in a given study (Suzuki et al. 2003; Collins and Kays 2011; Tidemann and Nelson 2011).

Proper identification of cause of death is crucial when determining the relative importance of different risk factors for targeted mitigation. For example, if a population is declining due to predation, targeted management actions require an understanding of the prevalence of predation as a cause of death and which segments of the prey population are most vulnerable (e.g. juveniles, malnourished, or naïve individuals). Yet, there may be multiple predator species killing prey, but perhaps not all individuals are equally susceptible to risk from the same predators. Sympatric wolves and cougars (*Felis concolor*) kill different age and sex cohorts in natural populations of elk (*C. elaphus*, Husseman et al. 2003), as do lynx (*Lynx lynx*) and red fox (*Vulpes vulpes*) preying on roe deer (*Capreolus capreolus*, Melis et al. 2013). Note that it is possible that mortality agents target individuals that have higher frailty due to other factors, as is expected when predation is directed at sick, lame, or otherwise compromised individuals. Snowshoe hares and red squirrels (*Tamiasciurus hudsonicus*) are killed by a variety of predators. Body condition affects vulnerability of hares to risk of predation by specific predators, whereas for red squirrels, body condition does not influence predation risk (Wirsing et al. 2002). It follows that in such cases, predation is the proximate cause of death but malnutrition or other factors can be important ultimate causes. When both *proximate* and *ultimate* causes of death are involved in mortality, their respective role in population dynamics can become especially complex and difficult to disentangle.

The onus is on researchers to adopt field protocols that increase the likelihood of confirming proximate and ultimate causes of death. Frequent detection of subjects allows mortality events to be confirmed and diagnosed shortly after their occurrence, when evidence on the carcass itself and at the death site can be especially informative. With the advent of satellite-based telemetry, it is now possible to determine cause of death for migratory animals that range over expansive spatial scales (Hays 2014; Klaassen et al. 2014). But even intensive field research inevitably includes deaths that are attributed to unknown causes. The uncertainty reflects real-life challenges in conclusively determining cause of death from evidence at the death site, as well as difficulties associated with necropsy of incomplete or decomposed carcasses. Follow-up investigation and ancillary investigation may help discern the cause of death in instances where

primary evidence is inconclusive. For example, Hays et al. (2003) inferred high rates of fishing-related mortality in leatherback turtles (*Dermochelys coriacea*) by focusing on unnatural changes in the location or movements of satellite-based radio transmitters. Alternatively, various diagnostic, genetic, and related tests are now available to complement traditional approaches in identifying cause of death, and thereby help reduce the proportion of mis-assigned or unknown mortality events (Onorato et al. 2006; Wengert et al. 2012; Mumma et al. 2014). In a detailed study of mortality of fishers (*Pekania pennanti*), researchers used gross necropsy, histology, toxicology, and molecular methods to distinguish among five proximate causes of death, several of which required multiple approaches for conclusive assessment (Wengert et al. 2013; Gabriel et al. 2015). A number of resources are available for diagnosis of cause of death for wildlife species (Friend et al. 1999; Mineau and Tucker 2002; Wengert et al. 2012; Alt and Eckert 2017).

It may also be possible to adopt field protocols to assess the potential for misdiagnosis of cause of death: in a study of radio-collared snowshoe hares, Murray et al. (1997) showed that deployment of hare carcasses in the field did not result in scavenging by predators, implying that predation rates in radio-collared hares were unlikely to be overestimated due to mistaking scavenging for predation. In contrast, deployment of carcasses of Ruffed Grouse (*Bonasa umbellus*) revealed relatively high rates of scavenging and carcass displacement (Bumann and Stauffer 2002). Regardless, cause of death assessment in field research should include prompt carcass retrieval, necropsy, and assessment of the death site, in conjunction with assessment of the timing of death. Ultimately, studies seeking to develop a robust assessment of cause-specific mortality rates and their demographic importance are compelled to redouble efforts to confirm the proximate and ultimate cause of death for study individuals.

6.6.2 Cause-specific Hazards and Mortality Rates

The quantitative analysis of cause of death allows us to assign specific risks to different mortality agents. To that end, we revisit our earlier expressions of survival and hazard and consider analogues for multiple risk types. Recalling that hazard is the instantaneous risk of death, then if death can occur from $p = 1,\ldots, q$ causes and T is the time to death from any cause, the *cause-specific hazard* for cause p at time t is:

$$h_p(t) = \lim_{\Delta t \to 0} \frac{\Pr(t \le T < t + \Delta t, \textit{death from } p \mid T \ge t)}{\Delta t} \tag{6.19}$$

for T equal to the time of death from any cause (Cleves et al. 2010). Accordingly, we can consider that different causes of death are "competing" to define T, and therefore the total risk of any death type is $h(t) = \sum_j h_j(t)$, with the probability of death from cause p being $p(t)/h(t)$. If subjects die from either risk types p or q, the probability of death (cause-specific hazard) from p is $h_p(t)/\{h_p(t) + h_q(t)\}$ and the cause-specific hazard from q is one minus the above probability.

In reality, risk types p and q may not act independently, for example when particularly vulnerable individuals experience high risk from more than one mortality agent. This is an important point because the one-to-one correspondence between cause-specific hazard and the cumulative incidence of risk in standard survival analysis is lost in the case of cause-specific risk. Rather, we extend the survivor function to include multiple risks; $S(t) = \exp\{-H_p(t) - H_q(t)\}$, where $H_p(t)$ and $H_q(t)$ are the cumulative hazards from each risk type, respectively. The implications of this distinction for competing risks analysis are important, because the *cause-specific cumulative incidence* (i.e. $F_p(t)$) requires multiple risk types for proper calculation. If the term (1 – KM) estimates the failure function for standard survival data across all causes of death, the analogous term is not relevant to cause-specific failure because it assumes independence among risk types (Cleves et al. 2010; Andersen et al. 2012).The *cumulative incidence function* (CIF) is the appropriate unit in cause-specific mortality estimation, where the cumulative failure rate for risk type p ($F_p(t)$) now considers the association between $h_p(t)$ and related covariates. The relationship between $\text{CIF}_p(t)$ and cause-specific hazards can be summarized by: $p(t) = \int_0^t h_p(x)S(x)dx$, where x is a specified time. Practically speaking, it is more appropriate to consider a modified failure function rather than a modified survivor function when dealing with competing risks, and this is why researchers usually use CIF as the unit of interest (Cleves et al. 2010). However, the CIF can be biased low when there is staggered entry or a paucity of early recruits due to left truncation (Woodroofe 1985; Tsai 1988; but see Heisey and Patterson 2006). Alternatively, the HF estimator can be expanded to deal with multiple fates by considering that m_{ip} is the probability that an individual who is alive at the beginning of interval i dies as a result of mortality cause p (Heisey and Fuller 1985). The number of deaths in i from cause p (d_{ip}) is:

$$\hat{m}_{ip} = \frac{d_{ip}}{r_i}. \tag{6.20}$$

Collectively, the probability of death from cause p during interval i is the sum of the probability that the subject survives to a given day and then dies on the same day

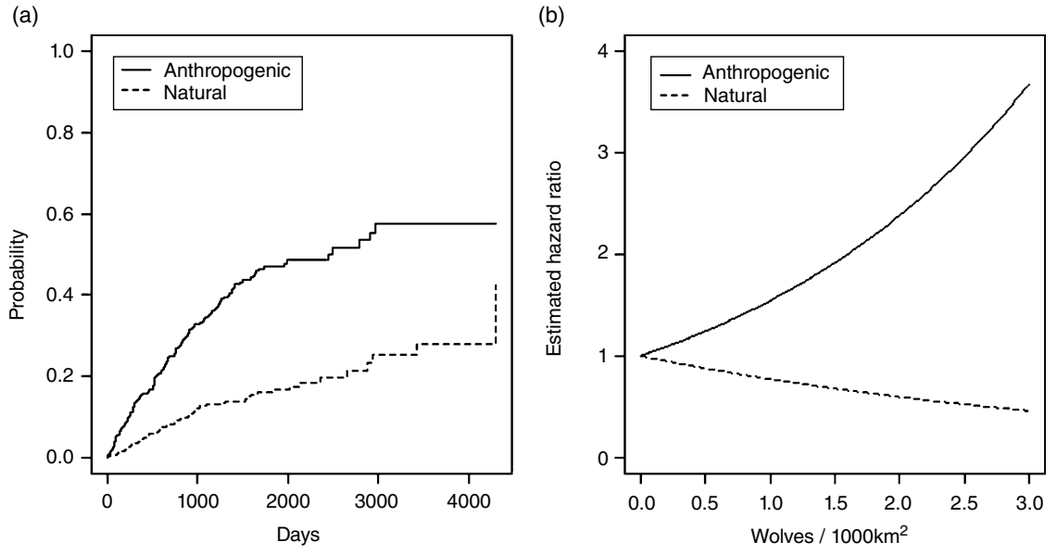

Figure 6.8 Cumulative incidence functions (CIF) (a) and estimated hazards (b) for wolves. Hazards are estimated from a competing risks model including wolf population density. The dataset was modified from Murray et al. (2010).

due to cause p, which can be calculated using the geometric progression:

$$\hat{M}_{ip} = m_{ip} + s_i m_{ip} + {s_i}^2 m_{ip} + \cdots + {s_i}^{(L_i-1)} m_{ip} = \left(\frac{m_{ip}}{1-s_i}\right)\left(1-{s_i}^{L_i}\right). \quad (6.21)$$

It is important to recognize that the first quantity in the second expression above, $\left(\frac{m_{ip}}{1-s_i}\right)$, is the relative risk of death from cause p in interval i, whereas the second quantity, $(1-{s_i}^{L_i})$, is the total probability of death, all sources combined, during i (Heisey and Fuller 1985). More intuitively, if we accept that m represents a specific time unit (usually days), the expression reveals how on day 1 of interval i the subject experiences a mortality rate m_{ip}; on day 2 the mortality rate is a product of risk from m_{ip} and survival during the previous day (s_i), and so forth (Williams et al. 2002).

Our example dataset for a wolf population in western United States (Murray et al. 2010; Smith et al. 2010) included 170 anthropogenic mortalities and 63 natural mortalities, and CIFs were markedly higher for anthropogenic than natural risk (Figure 6.8a). Likewise, if we assume a single interval of constant hazard through the 22-year study, which is unlikely but useful to illustrate the point, 90-day cause-specific mortality rates are 0.029 (0.025, 0.035) for anthropogenic and 0.013 (0.010, 0.016) for natural causes where CIs were derived from Taylor series approximation (Heisey and Fuller 1985). Yet, both the CIF figure (Figure 6.8a) and cause-specific mortality estimates do little to inform about the potential inter-relation between different causes of death in the wolf population itself, and therefore should not be the final destination in our cause-specific mortality analysis.

6.6.3 Competing Risks Analysis

Competing or *proportional risks analysis* constitutes an extension of CPH for the case of cause-specific hazards; alternatively one could use fully parametric models to compare CIFs, although this latter approach is tenuous owing to the recognized limitations of parametric survival analysis in ecology. The most practical way to implement competing risks analysis is to use the *data augmentation* approach (Lunn and McNeil 1995), which takes advantage of the additivity of hazard functions by duplicating the dataset for each risk type and dummy coding within each duplicate to identify the appropriate risk. Accordingly, for each duplicate all mortalities from causes other than those outside the specific risk set are functionally right-censored (see Competing risks in Table 6.1). By stratifying according to risk type, we perform multiple regressions simultaneously, allowing us to deal with more than one risk type within the same analysis and to compare covariates that may relate differentially to each risk type.

There is an important note of caution when deciding on how to functionally conduct competing risks analysis. The number of different mortality agents affecting the subject population and the role of each risk on hazard should influence the number and identity of risk types

included in the analysis. Notwithstanding the need to test hypotheses and predictions that have been established a priori (Chapter 2), exploratory analysis should be used to assess the composition of risk type structure in the dataset. If some causes of death are either uncommon or related through proximate-ultimate linkages, categorical outcomes should be pooled to achieve a more parsimonious risk set. Returning to the wolf mortality study, Murray et al. (2010) used a competing risks framework to compare factors promoting mortality from *natural causes* (disease, strife, senescence) versus *anthropogenic causes* (poaching, legal control, vehicle collision). To achieve this level of detail, animals were radio-collared and monitored regularly, and dead animals were recovered and necropsied shortly after mortality was detected. Some causes of death were relatively uncommon, and the objective of the study was primarily to assess the role of humans on wolf mortality risk. Thus, it was appropriate to consolidate the various causes of death into two main categories of risk: natural vs. anthropogenic causes. A subset of wolves (12%) could not be assigned to either of the two main risk types because they died of undetermined causes, but exploratory analysis of survival times and covariate influence confirmed that this group was comparable to the other two main risk types. Wolves dying of unknown causes probably constituted a representative sample of the population (i.e. included animals dying of anthropogenic and natural causes, in representative proportions) and thus could be included in the analysis as right censors. Note that inclusion of these individuals as right censors serves to illustrate two important reminders: (i) causes of death should be confirmed to the fullest extent possible when anticipating competing risks analysis to reduce the number of unknown mortalities; and (ii) all subjects, regardless of their fate, should be retained in the analysis (pending appropriate checks for bias) to ensure that informative censoring does not compromise parameter estimates.

The role of a variety of predictors was examined to identify determinants of anthropogenic and natural deaths in the wolf population (Table 6.4). Overall, animals that were dispersers (Disperser, 1 = yes) had qualitatively higher risk from both anthropogenic and natural causes of death compared to the nondispersing group, but the HRs were comparable between the two risk types, and in both cases CIs overlapped 1.0. However, risk of death for animals living in Montana (Montana, coded as 1 = yes) increased for anthropogenic causes but decreased for natural causes. Likewise, mortality risk from anthropogenic causes increased by 54.3% for each unit increase in wolf density ($|1 - 1.543| \times 100 = 54.3\%$), compared to a concomitant decline in risk from natural causes with increasing wolf density ($|1 - 0.772| \times 100 = 22.8\%$). The divergence in risk is especially evident when HR associated with each risk type are graphed against wolf density (Figure 6.5b). However, one must take care when comparing HR from competing risks analysis to their corresponding CIFs, owing to the possibility that causes of death are nonindependent (Andersen et al. 2012). Ultimately, because anthropogenic risks were higher, and natural risks were lower, in both Montana and across wolf density, we infer that the two risk types were not independent. In other words, anthropogenic mortality was to some extent compensatory to natural mortality (Figure 6.5b, see below). The complex relationships and contextual subtleties between causes of death would not have been revealed in the absence of a competing risks analysis isolating the effects of each risk type, specifically through inclusion of an interaction term with predictors (Heisey and Patterson 2006). As an outcome of this analysis, conservation efforts designed to manage wolf deaths from anthropogenic causes should focus primarily on the Montana population and relative to increasing wolf numbers. In a similar scenario, Robinson et al. (2015) used a CIF-based approach to compare cause-specific mortality rates of Amur tigers (*Panthera tigris*) to assess the role of a novel cause of death, canine distemper virus, on the tiger population.

Table 6.4 Cause-specific hazard ratios (HR) (±95% confidence intervals [CI]) for variables included in a multivariate competing risks model for wolves.

Variable	Anthropogenic	Natural
Disperser	1.684 (0.803, 3.531)	1.662 (0.850, 3.249)
Montana	5.421 (2.668, 11.02)	0.482 (0.233, 0.995)
Wolf density	1.543 (1.234, 1.930)	0.772 (0.545, 1.094)

Source: Data adapted from Murray et al. (2010).
The analysis was restricted to wolves that were representative of the population and thus excluded individuals captured following livestock depredation.

6.6.4 Additive Versus Compensatory Mortality

Ecologists have long been preoccupied with understanding whether different risk types affect population dynamics, and in particular whether anthropogenic mortality, usually due to human harvest, is *additive* to other causes of death and thereby constrains population growth (Burnham and Anderson 1984; Boyce et al. 1999; Sandercock et al. 2011). Alternatively, harvest or other sources of mortality may play a *compensatory* role, meaning that their demographic influence is lessened by the fact that some mortality occurs irrespective of whether the cause of interest is implicated. For example, earlier we discussed instances when predators kill prey that are doomed to die from other causes; this type of mortality requires

distinction between the proximate and ultimate cause of death and constitutes compensatory mortality because killed individuals would not otherwise contribute to population growth. At one extreme, compensation can be complete and all mortality replaces potential mortality from other causes; at the other extreme, mortality is fully additive. Studies often reveal *partially compensatory* effects (i.e. partially additive), depending on the magnitude and individual targets of the source of mortality. For example, power-line collisions are only partly compensatory to other sources of mortality in juvenile White Storks (*Ciconia ciconia*, Schaub and Lebreton 2004). This means that overall mortality rates in the stork population are higher due to such mortality. The status of a particular mortality agent in the context of the *additive-compensatory mortality continuum* will determine its influence on the population trajectory (Péron 2013). Interest in additive versus compensatory mortality has dominated longstanding discussions related to harvest management, population recovery, and species conservation (Burnham et al. 1984; Conroy and Krementz 1990; Pöysä et al. 2004; Cooley et al. 2009; Sandercock et al. 2011).

By allowing interaction terms between risk type and predictors, *competing risks models* offer a direct and robust individual-based tool in the assessment of additive versus compensatory mortality (Heisey and Patterson 2006; Murray et al. 2010). However, to date few studies have exploited this approach, preferring instead to focus on population-level changes in different risk types through space or time. Here, the focus is on the change in cause-specific mortality or HR and whether different risk types follow similar or opposite trends, which would indicate additive or compensatory effects, respectively. For example, in a large-scale harvest experiment, Sandercock et al. (2011) used a cause-specific hazards framework to show that the relative importance of hunting mortality in Willow Ptarmigan (*Lagopus lagopus*) often varied inversely with natural mortality, indicating that hunting is partially compensatory. Robinson et al. (2014) used changes in cause-specific mortality following a temporal shift in cougar harvest strategy to infer that mortality due to hunting of adult cats was an additive source of mortality. Griffin et al. (2011) showed that predation by grizzly bears was negatively related to survival of elk calves and therefore constituted additive mortality, whereas predation by other predator species had no discernible demographic significance and thus was compensatory. Bastille-Rouseau et al. (2016) found that predation on caribou calves (*Rangifer tarandus*) by invasive coyotes (*Canis latrans*) in Newfoundland was at least partly additive and therefore potentially contributing to caribou population decline.

Traditionally, the population-level approach to the additive–compensatory debate has involved regressing survival against cause-specific mortality, where rates are tallied for one or more populations, usually on an annual basis. Here, a negative slope implies additive mortality, with a steeper slope implying stronger additivity. For example, using annual cause-specific mortality rates for three separate wolf populations (Murray et al. 2010), we infer by the qualitative difference between slopes that anthropogenic mortality is more strongly additive in Montana and Idaho than in the Greater Yellowstone Area (Figure 6.9). Notably, the range of observed anthropogenic mortality rates is much greater in Montana, providing extra reassurance about the additive effects of anthropogenic mortality on that wolf population. The findings are consistent with other studies using a regression-based approach to infer additive effects of anthropogenic mortality on wolf populations (Creel and Rotella 2010; Sparkman et al. 2011).

When regression-based models are used to infer the demographic importance of a particular mortality agent, regression slopes are compared statistically to determine whether populations differ in the role of additive–compensatory factors (Zar 1999). One caution is that observations such as those in Figure 6.9 are serially autocorrelated when collected sequentially from the same population, leading to lack of independence. More importantly, survival and cause-specific mortality rates themselves are not independent, meaning that this approach may be biased toward showing additivity. To some extent, bias may be mitigated by calculating a corrected standard error of the slope, or perhaps by using a mixed-model design (Otis and White 2004; Schaub and Lebreton 2004). More recently, Servanty et al. (2010) advocated use of a *state-space approach* to properly assess correlations between causes of death, whereas Péron (2013) presented a metric for quantifying additivity based on the variance–covariance structure of the mortality rates themselves. Notwithstanding these developments, we must stress that population-level approaches relating cause-specific mortality to survival fail to take full advantage of continuous time information provided by tracking individual variability through space and time. In fact, Figure 6.8b presents a substantially more convincing case that anthropogenic mortality in wolves is partly compensatory than does Figure 6.9. Ultimately, competing risks analysis and supporting figures allow researchers to more fully dissect mechanisms underlying the additive–compensatory interplay than is possible with a population-level analysis. Thus, when combined with a study design based on a before-after-control-impact approach (Robinson et al. 2014) or replicated experimental treatments (Sandercock et al. 2011), competing risks models

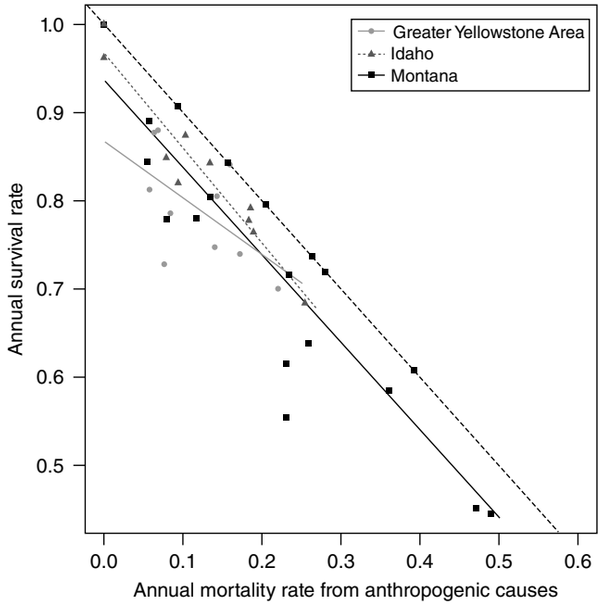

Figure 6.9 Tests for additive mortality in wolves from three populations, using regression of annual survival rate versus annual rate of anthropogenic mortality. Survival and mortality rates were calculated using Heisey–Fuller estimates. The dashed line has a slope = −1.0 which is expected with complete additivity. *Source:* The dataset was modified from Murray et al. (2010).

offer an especially powerful tool for rigorously disentangling additive versus compensatory effects of mortality.

6.7 Software Tools

Most advanced statistical software packages have built-in functions for the estimation of the survival curve with Kaplan–Meier models, and parametric and semi-parametric regression analyses. General computing environments or programming languages such as `MATLAB` and `PYTHON` also have functions or modules available for survival analysis. In `SAS`, the `lifetest` procedure is the principal function for calculating survival curves, instantaneous hazard curves, and associated tests. The `phreg` procedure is the main tool for parametric and semi-parametric regression analysis, with several possible specifications including the use of frailty terms and diagnostic tests. In `Stata`, the function `sts` is used for calculating survival functions and survival curves, whereas the functions `stcox` and `streg` can be used for semi-parametric and parametric regression, respectively. In program `R`, the `survival` package offers many functions related to survival analysis and is the primary reference. The `survival` package includes `survfit` to calculate the survival curve and `surv` to create survival objects that are used for semi-parametric (`coxph`) or parametric analyses (`survreg`). The `eha` package also offers different options for parametric and semi-parametric regression, notably using the functions `phreg` and `coxreg`. A distinction between `survreg` and `phreg` is that the former uses an accelerated-failure time model while the latter used a proportional hazards model that will give results analogous to `coxph` or `coxreg`. Other useful packages in `R` include: `muhaz` for the estimation of hazard functions, `coxme` for the addition of mixed-effects to CPH regression, and `cmprsk` for calculation of CIFs in a competing risk framework.

6.8 Online Exercises

In the exercises associated with our chapter, we use both simulated and real data to illustrate some of the main features covered herein. In Exercise 1, we use a simulated dataset to demonstrate preparations for a survival analysis, with particular attention given to the structure of the time variable. Next, we show how to conduct simple hypothesis tests for different covariates, and how to graph a variety of survival functions. In Exercises 2 and 3, we demonstrate how to develop more complex semi-parametric and parametric models to evaluate mortality risk determinants, using an example based on predation by raccoons on the nests of freshwater turtles. We test model assumptions, and then demonstrate how to identify the best out of a suite of candidate models. In Exercise 4, we demonstrate the steps for conducting a competing risks analysis, and generating corresponding graphical functions to compare hazards across alternative risk types for gray wolves exposed to both anthropogenic and natural mortality.

6.9 Future Directions

Understanding the factors affecting mortality risk and fate of organisms will remain a core interest in population ecology and conservation biology. Ecologists have largely abandoned cross-sectional studies of populations (i.e. life table analysis) in favor of longitudinal studies that track individuals through time as they encounter a variety of risks through life. However, despite a longstanding and well-established basis for investigating continuous time survival in observational research in other fields, several improvements are needed to take full advantage of the available opportunities in ecology. At its core, robust continuous time survival analysis relies on quality data consisting of intensive monitoring and high detection probability of individual subjects, prompt detection of

mortality events, and application of contemporary diagnostic tools to improve cause of death determination. Although many research studies are already appropriately designed to meet many of these needs, for others even modest improvements in study design, data collection, and data treatment and analysis will dramatically improve inference. For example, we highlight the development of new technologies like remotely triggered trail cameras, bio-logging, or satellite-based telemetry that allow researchers to monitor individuals much more efficiently and with far greater detail than in the past (Naef-Danzer et al. 2005; Cagnacci et al. 2010). It is possible to use these devices to assign temporally or spatially explicit mortality risk to individuals (Smith et al. 2010; Loveridge et al. 2017). Doubtless, ongoing refinement and miniaturization of these new technologies means that the age of continuous time monitoring for all sorts of organisms is fast approaching, perhaps even for plants and small animals, which currently are under-represented in the continuous time survival literature.

Semi-parametric modeling approaches should meet the survival analysis needs of the vast majority of population ecologists. However, there remains a tendency to develop survival models without appropriately testing for model assumptions, in particular those related to proportional hazards, lack of progressive sample heterogeneity, and random censoring of subjects. These model assumptions are treated seriously in the human medicine and epidemiological literature (Grambsch and Therneau 1994; Leung et al. 1997; Zens and Peart 2003; Hosmer et al. 2008), and should receive similar attention in ecology. Additional concerns relate to sample-size requirements in survival analysis; observational field studies frequently include too few subjects, too few mortalities, or too many censors to provide robust statistical inference or to allow development of complex survival models (Murray 2006). Necessary steps in model diagnostics, including assessing model parsimony and overfitting, are often overlooked steps that relate directly to sample size and statistical power. Adjustments concerning these issues should include not only improved field protocols and refined statistical analysis, but also proper reporting of censoring rates as well as the outcome of exploratory analyses and model validation. Mixed-effects survival models, where random effects can be assigned and explicitly modeled for groups of individuals not under direct control of the observer, add an important dimension to traditional tools for analyzing survival in free-ranging organisms.

Our review is largely focused on survival analysis methods that are readily implemented using information-theoretic methods, including model selection and multimodel inference approaches (Chapter 2). Recent efforts adapted Bayesian methods to survival analysis (Ibrahim et al. 2005), specifically using Markov chain Monte Carlo and Gibbs sampling algorithms to appropriately weigh models according to their posterior probabilities. Bayesian approaches may be an informative solution to traditional survival analysis (Omerlu et al. 2009; Halstead et al. 2012) and thereby offer respite from otherwise thorny issues in standard survival analysis such as small sample size, missing values, censoring, nonproportionality of hazards, and even unknown causes of death. However, as with all Bayesian approaches, these methods rely on representative priors that can be readily translated from existing survival data; currently this may be a challenging requirement given the paucity of reliable and generalizable survival datasets for many taxa. Yet, development and acquisition of new survival data, combined with improved computational efficiency and user interface, will make Bayesian methods increasingly attractive for survival research in ecology.

One important extension of the suite of continuous time survival methods involves *multiple event analysis*, which is closely aligned to competing risks methods but considers rates of nonlethal reoccurring events. This approach takes advantage of the time-to-event origins of the methods discussed in this chapter, and applies them specifically to evaluate rate determinants for repeatable events. In a simple example, Merrill et al. (2010) used reoccurring events analyses (i.e. CPH and parametric analogues) to illustrate how environmental factors influence rates that wolves kill prey. Because wolves were equipped with satellite-based tracking units, monitoring and detection was quasi-continuous and allowed estimation of the number of prey kills per unit time and relative to habitat and prey density. Reoccurring events analysis holds possibilities for assessing rates of a variety of demographic characteristics such as breeding or dispersal or other events that may happen multiple times in an individual's lifetime. To date, reoccurring events in ecology have rarely been considered in an actual time-to-event context or for estimating demographic rates (Bastille-Rousseau et al. 2011; Whittington et al. 2011; McPhee et al. 2012). The logical extension here is the potential to adopt multistate models to evaluate the interplay between variable subject states and corresponding risks. For example, this approach could allow researchers to investigate how dispersal status increases mortality risk that is separate from natural nondispersal risk, while considering that dispersal status is reversible through time; these methods provide a sophisticated approach for assessing co-occurring and reoccurring risk types (Beyersmann et al. 2012; Devineau et al. 2014; Hightower and Harris 2017). However, we caution that these and other advanced methods are developed primarily for fields where sample sizes and study design tend not to limit modeling capabilities or to raise concerns over model complexity and model mis-specification. Thus, the extent that these methods overextend the capabilities of more modest ecological datasets is open for debate.

Ultimately, more careful application of the methods detailed in this chapter, specifically in the context of intensified fieldwork for developing robust continuous time datasets and the use of contemporary methods focusing on individual variability in risk through space and time, will serve well in raising the bar for ecological survival analysis. When combined with more extensive coverage of these topics in quantitative ecology courses and workshops, these advances should improve our capacity for robust survival analysis and thereby place us in a better position to address future questions and challenges related to population viability, sustainable harvest, or environmental impact mitigation.

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