



The Grammar of Science: The Challenge of Competing Outcomes

Jaranit Kaewkungwal*

Mahidol University, Thailand

*Corresponding author email: jaranitk@biophics.org

Received: 11 Nov 2025; Revised: 23 Nov 2025; Accepted: 28 Dec 2025

<https://doi.org/10.59096/osir.v18i4.279021>

In many studies, multiple possible outcomes can occur. Sometimes, another event happens before the outcome of interest, potentially preventing it from occurring. This situation is known as a “competing risk.” This paper discusses how to handle competing risks, which are common in epidemiological research, focusing on time-to-event analyses where individuals are followed from study entry until the event of interest, a competing event, or censoring.

Getting Started with Time-to-event Analysis

Time-to-event data describe how long it takes for a specific event to occur. Time-to-event analysis, or survival analysis, estimates the survival function, $S(t)$ —the probability that an individual has not yet experienced the event at a given time. Depending on the study goal, analyses may compare survival between groups or assess how factors such as age, sex, socioeconomic status, or treatment affect the time to event.¹

In time-to-event analysis, individuals are followed over time, and their hazard of experiencing the outcome, $h(t)$, may change at each time point. The hazard represents the instantaneous risk of the event occurring at a specific time, given that the individual has not yet experienced it, reflecting how risk evolves

over time rather than the total probability of the event. Those who experience the event are called “failures”, while those who do not are considered “censored”. Censoring occurs when an individual (1) does not experience the event by the study’s end, (2) is lost to follow-up or withdraws, or (3) dies or experiences a different event during follow-up.¹ Because of censoring, survival analysis requires specialized methods beyond standard regression.

The Kaplan–Meier (KM) method estimates the proportion of individuals who remain event-free over time.^{2,3} It assumes noninformative censoring, meaning that censored individuals would have had the same probability of experiencing the event as those who remained under observation. By including individuals with incomplete follow-up, KM provides a flexible and widely used tool for time-to-event analysis.¹

Figure 1 illustrates 10 individuals followed over 14 months, showing who experienced the event of interest and who were censored. Those reaching the endpoint (death from the disease) are considered failures, whereas individuals who did not reach the endpoint were censored. Figure 2 presents the hazard function $h(t)$ along with the KM curves for the survival function $S(t)$ and the failure function $F(t)$ (i.e., $1-S(t)$) for the same dataset.

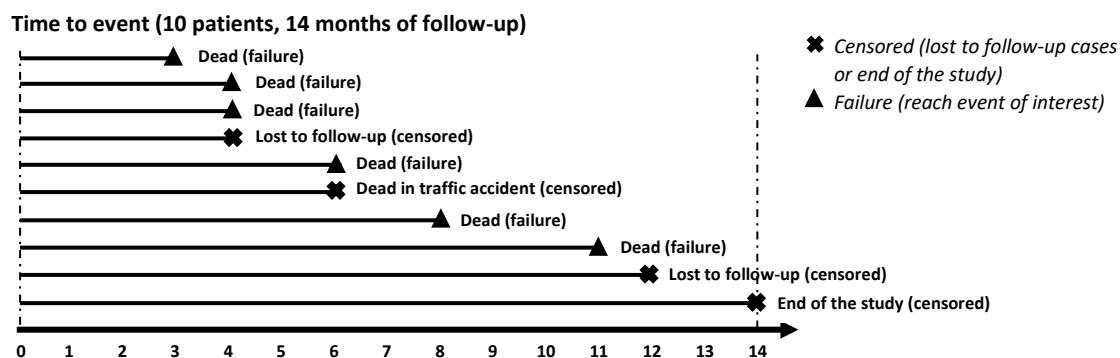


Figure 1. Event occurrence and censoring during 14-month follow-up

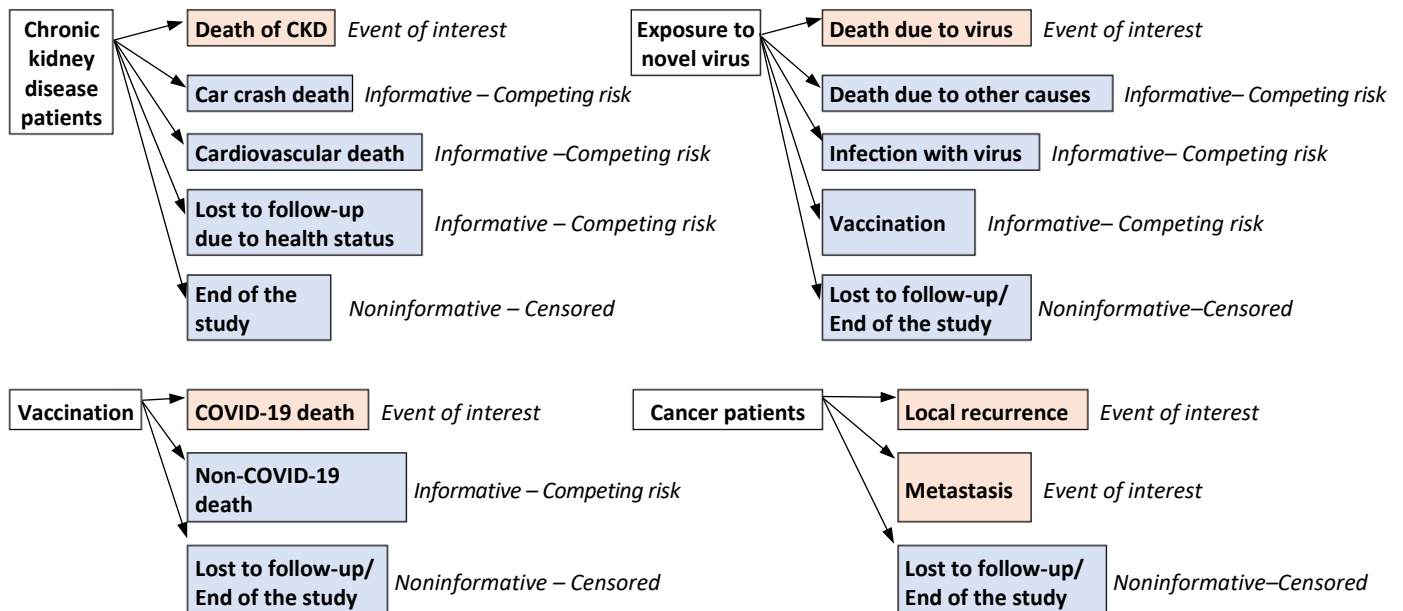


Figure 4. Examples of different competing risks events

Ignoring other possible events can give misleading results.^{7,8} When there is a competing risk situation, the KM method will treat competing events as censored, assuming that competing events are independent—like standard censoring—which is often unrealistic.^{1,4}

Competing Risks: Analysis & Models

Competing risk regression is usually based on two types of hazard: the cause-specific hazard (CSH) and the subdistribution hazard (SDH). In the presence of competing risks, CSH and SDH produce different hazard functions for the same event. While both account for competing risks, they use different risk sets, which leads to different interpretations of covariate effects.⁹

Cause-specific Hazard

The cause-specific hazard (CSH) measures the risk of a specific event occurring at a particular time,

considering only individuals who have not yet experienced any event.^{6,9} The group of individuals who are still at risk at a given time is called the risk set. Once someone experiences a competing event, they are removed from the risk set and no longer contribute to the calculation of the hazard for future times. Figure 5 illustrates a scenario in which 20 patients are followed over time, and each can experience Event 1, Event 2, or no event (censored). From Months 0 to 3, all 20 are in the risk set. At month 3, one person experiences Event 1 and none experiences Event 2, giving a CSH of $1/20=0.05$ for Event 1 and $0/20=0.00$ for Event 2. At month 4, 19 people remain in the risk set because one individual has already had Event 1. That month, two people experience Event 1 and one experiences Event 2, resulting in a CSH of $2/19\approx0.11$ for Event 1 and $1/19\approx0.05$ for Event 2. This process continues over time, showing the instantaneous risk of each event among those still in the risk set.

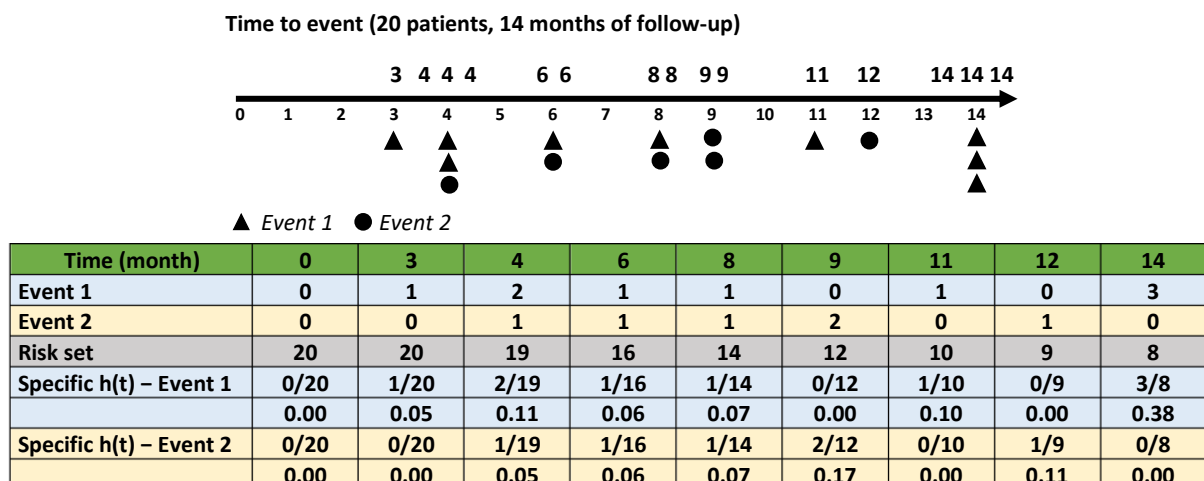


Figure 5. Cause-specific hazard (CSH) schematic

Subdistribution Hazard

The subdistribution hazard (SDH) is a method used to estimate the probability of different events when competing risks are present. Unlike the CSH model, which removes individuals from the risk set once they experience a competing event, the SDH keeps these individuals in the risk set along with those who are still event-free. This approach allows direct estimation of the cumulative incidence of each event while still using a Cox regression framework.^{6,10} The SDH function shows the risk of a specific event at a given time, including both people who haven't had any events yet and those who have experienced competing events.⁹ As

shown in Figure 6, suppose 20 patients being followed over time, where they can experience Event 1, Event 2, or be censored. From months 0 to 3, all 20 are in the risk set. At month 3, one person experiences Event 1 and none experiences Event 2, so the $SDH = 1/20 = 0.05$ for Event 1 and $0/20 = 0.00$ for Event 2. At month 4, the risk set for Event 1 is now 19 (one person already had Event 1), while the risk set for Event 2 remains 20, since no one has had Event 2. That month, two people have Event 1 and one has Event 2, giving $SDH = 2/19 \approx 0.11$ for Event 1 and $1/20 = 0.05$ for Event 2. This continues over time, showing how the SDH accounts for competing events while estimating the probability of each specific event.

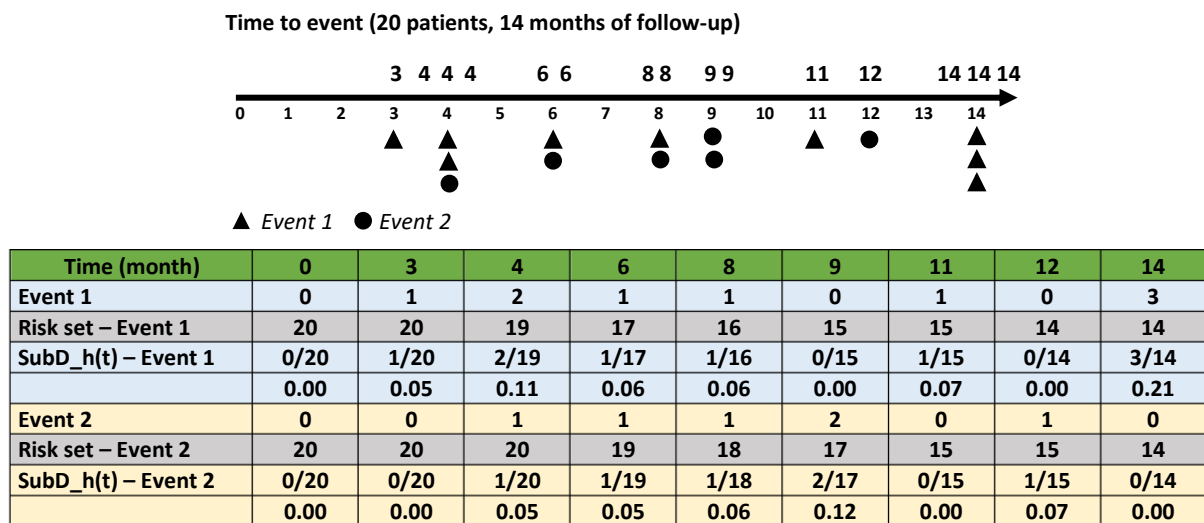


Figure 6. Subdistribution hazard (SDH) schematic

Cumulative Incidence Function

The Cumulative incidence function (CIF) is used instead of standard methods like KM, which can overestimate the risk when competing events are present.¹¹ The CIF shows the probability that a specific event occurs by a certain time while taking competing events into account.^{6,12,13} When there is only one type of event, the CIF is the same as 1–KM. With multiple competing events, the CIF is calculated from cause-specific hazards but does not assume that the events are independent, providing a more accurate estimate of event probabilities.^{6,14}

The CSH and cause-specific cumulative incidence function (csCIF) measure the event rate among those still at risk, but they assume that different events do not influence each other, which may not always hold true.⁶ In contrast, the SDH and subdistribution cumulative incidence function (sdCIF) account for competing events by keeping individuals who have already experienced competing events in the risk set, along with those who are still event-free.⁶ This approach allows direct calculation of the cumulative

probability of each event over time. The SDH can also show how covariates affect this probability through the subdistribution hazard ratio (SHR). Models such as the Fine and Gray SDH model are especially useful for predicting an individual's absolute risk of an event while accounting for competing risks.^{10,13}

Interpretation and Application of Competing Risks Models

The CIF is calculated by integrating the hazard over time, weighted by overall survival. The hazard ratio (HR) represents the relative change in the instantaneous risk among individuals who are still event-free. As an example adapted from a COVID-19 vaccination study, suppose 10,000 people are followed for one year.¹⁵ Vaccination status is the exposure, COVID-19 death is the event of interest, non-COVID-19 death is treated as a competing event, and those who survive the year are censored. Using a CSH model (Cox), the hazard ratio is $HR_{vax} = 0.40$ (95% CI 0.32–0.51), meaning vaccinated individuals have a 60% lower instantaneous risk of COVID-19 death among those still alive and event-free. Using a SDH model

(Fine & Gray), the subdistribution hazard ratio is $SHR_{vax}=0.65$ (95% CI 0.54–0.78), reflecting a 35% lower cumulative incidence of COVID-19 death over 1 year, accounting for competing deaths. At 12 months, the CIF shows 0.8% for vaccinated and 2.0% for unvaccinated individuals, thus a risk difference between the two groups is 1.2 percentage points.

The choice of hazard function in competing risks analysis depends on the research question. As noted in the literature, the CSH is most useful for etiologic research, which seeks to understand what causes an event and how risk factors directly affect it.^{3,6,11,15,16} In contrast, the SDH is better suited for prognostic research, which focuses on predicting the overall chance of an event over time while accounting for competing events. SDH models also estimate the cumulative incidence, making them more useful for real-world risk prediction.

Returning to the COVID-19 vaccination study example, the CSH model shows how vaccination affects the immediate risk of COVID-19 death among people who are still alive and have not experienced any event, reflecting the biological effect of the vaccine. The SDH model, on the other hand, takes competing events—such as death from other causes—into account and shows how vaccination affects the overall cumulative risk of COVID-19 death, which is more useful for predicting outcomes in the population. We can say that SDH model estimates effects in relation to the cumulative incidence, making their results closely tied to—and therefore strongly influenced by—the frequency of competing events in the population. In contrast, the CSH model evaluates effects independently of how often competing events occur, underscoring the greater robustness and stability of CSH estimates when competing-risk incidence varies.^{6,13}

When reporting CSH and SDH models, always include the CIF to visualize event probabilities over time. Clearly indicate whether hazard ratios come from a CSH or SDH model, because their values and interpretations differ. Remember, SDH reflects cumulative incidence in the presence of competing events and does not measure the exact causal effect of a covariate.

Guidelines for Researchers

Competing risk models are most useful when follow-up is long enough to observe a sufficient number of events. Because there is no simple test for informative censoring, it is important to ensure that individuals with and without events were handled consistently. These models are typically considered when competing

events are fairly common—affecting $\geq 10\%$ of the population or occurring as often as the primary event.¹

Competing risks analysis considers multiple types of events, giving more accurate predictions for individual outcomes. However, it can be biased if exposures or conditions change over time. In such cases, multistate models are preferable, as they track individuals through multiple states—such as treatment, relapse, and death—capturing complex transitions over time.¹⁶

When you have multiple outcomes, don't settle for just one—pick the method that tells the whole story.

Acknowledgements

An AI tool, ChatGPT (OpenAI, 2025), was used to generate language suggestions during the preparation of this manuscript, and all outputs were reviewed for accuracy and appropriateness.¹⁷ The author reviewed, edited, and take responsibility for the final content.

Suggested Citation

Kaewkungwal J. The grammar of science: The challenge of competing outcomes. *OSIR*. 2025 Dec;18(4):258–63. doi:10.59096/osir.v18i4.279021.

References

1. Maradit Kremers H, Devick KL, Larson DR, Lewallen DG, Berry DJ, Crowson CS. Competing risk analysis: what does it mean and when do we need it in orthopedics research? *J Arthroplasty*. 2021 Oct;36(10):3362–6. doi:10.1016/j.arth.2021.04.015.
2. Basak R, Mistry H, Chen RC. Understanding competing risks. *Int J Radiat Oncol Biol Phys*. 2021 Jul 1;110(3):636–40. doi:10.1016/j.ijrobp.2021.01.008.
3. Dey T, Mukherjee A, Chakraborty S. A practical overview and reporting strategies for statistical analysis of survival studies. *Chest*. 2020 Jul; 158(1S):S39–48. doi:10.1016/j.chest.2020.03.015.
4. Mailman School of Public Health, Columbia University. Competing risk analysis [Internet]. New York: Columbia University; [cited 2025 Nov 1]. <<https://www.publichealth.columbia.edu/research/population-health-methods/competing-risk-analysis>>
5. Gutierrez RG. Competing-risks regression [Internet]. College Station (TX): StataCorp LLC; 2009 Nov 5 [cited 2025 Nov 1]. <https://www.stata.com/meeting/australia09/au09_gutierrez.pdf>

6. Lau B, Cole SR, Gange SJ. Competing risk regression models for epidemiologic data. *Am J Epidemiol.* 2009 Jul 15;170(2):244–56. doi:10.1093/aje/kwp107.
7. Reeve K, Held U. Competing risks survival analysis [Internet]. Zurich: Epidemiology, Biostatistics and Prevention Institute, Biostatistics Department, University of Zurich; 2025 Jun 17 [cited 2025 Nov 1]. 4 p. <https://ebpi.uzh.ch/dam/jcr:e94bba1a-884b-4762-9e5b-65532cc0b250/Competing_Risks_13_06_25.pdf>
8. Scrucca L, Santucci A, Aversa F. Competing risk analysis using R: an easy guide for clinicians. *Bone Marrow Transplant.* 2007 Aug;40(4):381–7. doi:10.1038/sj.bmt.1705727.
9. Jeon Y, Lee WK. Competing risk model in survival analysis. *Cardiovasc Prev Pharmacother.* 2020 Jul;2(3):77–84. doi:10.36011/cpp.2020.2.e11.
10. Breheny P. Competing risks [Internet]. Iowa City: University of Iowa; 2018 Dec 4 [cited 2025 Nov 1]. <<https://myweb.uiowa.edu/pbreheny/7210/f18/notes/12-04.pdf>>
11. Manzoor BS, Adimadhyam S, Walton SM. An introduction to competing risks. *Value & Outcomes Spotlight* [Internet]. 2017 Mar-Apr [cited 2025 Nov 1];3(2):21–2. <<https://www.ispor.org/docs/default-source/publications/value-outcomes-spotlight/march-april-2017>>
12. Royston P, Lambert PC. Flexible parametric survival analysis using Stata: beyond the Cox model. College Station (TX): Stata Press; 2011. 347 p.
13. Fine JP, Gray RJ. A proportional hazards model for the subdistribution of a competing risk. *J Am Stat Assoc.* 1999;94(446):496–509. doi:10.2307/2670170.
14. Dignam JJ, Zhang Q, Kocherginsky M. The use and interpretation of competing risks regression models. *Clin Cancer Res.* 2012 Apr 15;18(8):2301–8. doi:10.1158/1078-0432.CCR-11-2097.
15. Lee SW, Ma D, Davoodian A, Ayutyanont N, Werner B. COVID-19 vaccination decreased COVID-19 hospital length of stay, in-hospital death, and increased home discharge. *Prev Med Rep.* 2023 Apr;32:102152. doi:10.1016/j.pmedr.2023.102152.
16. Sapir-Pichhadze R, Pintilie M, Tinckam KJ, Laupacis A, Logan AG, Beyene J, Kim SJ. Survival analysis in the presence of competing risks: the example of waitlisted kidney transplant candidates. *Am J Transplant.* 2016 Jul;16(7):1958–66. doi:10.1111/ajt.13717.
17. OpenAI. ChatGPT, May 6 version [Internet]. San Francisco (CA): OpenAI; 2025 [cited 2025 Nov 1]. <<https://chat.openai.com>>