Backlash greets ‘bad luck’ cancer study and coverage
How subtleties got lost in the telling

By Jennifer Couzin-Frankel

On New Year’s Day, some striking news broke in Science: a piece by this reporter called “The bad luck of cancer” and the research study it was based on, published simultaneously in the journal. As Science’s story and many other news accounts of the paper explained, the study authors concluded that simple “bad luck”—their description of random mutations accumulating in healthy stem cells—could explain about two-thirds of cancers. That would exceed the risk of environmental and genetic factors combined.

Readers wasted little time in skewering the authors, mathematician Cristian Tomasetti and cancer geneticist Bert Vogelstein of Johns Hopkins University in Baltimore, Maryland. “Seems some cancer researchers are simply running out of intelligent questions or arguments,” read one of the 210 online comments on Science’s news piece. Reporters, including this one, fared worse. “Please, journalists, get a clue before you write about science,” pleaded an irate columnist in The Guardian by an evolutionary biologist and a statistician. Critics charged that media stories misinterpreted the study, seeking a streamlined message that downplayed the fact that the study didn’t include all cancers. In fact, it omitted two of the most common, prostate and breast, because data needed to include them were lacking.

Still, was the “two-thirds” figure actually referring to a fraction of cancer cases? Tomasetti had explained to Science that “if you go to the American Cancer Society website and you check what are the causes of cancer, you will find a list of either inherited or environmental things. We are saying two-thirds is neither of them.” He also confirmed the news story’s language describing the study before it was published.

In a follow-up interview after the uproar, Tomasetti clarified that the study argued that bad luck explained two-thirds of the variation in cancer rates in different tissues—a subtly different claim. Some tissues are overtaken by cancer more readily than others, and mutations in stem cells explain two-thirds of that variability, Tomasetti and Vogelstein concluded.

Despite the confusion among reporters, Tomasetti did not feel they had been careless—quite the contrary. “Overall, the reporters who interacted with us made a very honest and sincere effort to be as accurate as possible,” he says. And, he believes, he did his best to convey his findings to nonexperts. “If given enough time, or space, I can explain the subtleties of any given scientific result to anyone really,” but there were only so many hours he could spend speaking with reporters on deadline. The material is complicated even for statistical gurus, he believes. He has been busy preparing a technical report with additional details, and Johns Hopkins also sent a follow-up explainer to journalists and posted it online.

“It’s too easy to blame the media,” says David Spiegelhalter, a biostatistician at the University of Cambridge in the United Kingdom who blogs at Understanding Uncertainty. In this case, he felt, “the gist of the coverage is very reasonable—most cases of cancer are due to chance.”

Many scientists felt the paper’s authors were also guilty of oversimplifying. The paper included a striking figure splitting cancers into green and blue categories. The green were cancers “mainly due” to random mutations—suggesting, the authors wrote, that they were less likely to bow to prevention. However, that category included esophageal cancer and melanoma, both thought to have strong environmental drivers—heavy alcohol consumption for esophageal cancer, for example, and sun exposure for melanoma. Melanoma sat just inside the border of green country—but that was enough to incense some readers.

“They’ve ignored some of the fundamental lifestyle factors,” said Graham Colditz, a cancer prevention specialist at Washington University in St. Louis. Vogelstein says his paper doesn’t dispute that the environment contributes to melanoma. “This is a mathematical theory,” he says, and it doesn’t explain every facet of every cancer it includes.

Anne McTiernan, a physician and epidemiologist at the Fred Hutchinson Cancer Research Center in Seattle, Washington, believes the authors had “good intentions,” but she criticizes their assumption that a correlation between stem cell divisions and cancer risk means that one causes the other, something their data didn’t prove. Tomasetti agrees, but he notes that “all the biology we have on this topic supports causality.”

A key unanswered question is whether the furor will dampen cancer prevention efforts. “The message shouldn’t be, ‘Oh, it’s all chance, there’s nothing we can do about it,’” says Timothy Rebbeck, a cancer prevention specialist at the University of Pennsylvania. “There should be something we can do about it,” because risk varies so much among individuals. If anything, he says, the study points to the value of prevention. It shows huge risk gaps between cancers driven by the environment or genetics—such as lung cancer in smokers—and cancers at the same site without a clear cause.

Spiegelhalter isn’t surprised that coverage of the paper had its flaws. “It’s one of those things that’s so superficially simple,” he says, “and yet the superficial simplicity is not correct.”