I read with concern the recent commentary by Spitz et al. (1) in which they advocate training current and future cancer epidemiologists in the complexities of the “-omics” (i.e., genomics, proteomics, metabolomics, and epigenomics), with the corollary of advanced data management and analysis training to handle the reams of “big data” generated by these technologies. Essentially, in their view, cancer epidemiologists should be trained in computer science and molecular biology so that they will contribute meaningfully to moving epidemiology away from being considered a “soft” science. Implicit in their commentary is the foundational belief that molecular approaches to investigating the causes of cancer offer the most promise for preventing cancer incidence and mortality.

Arguably, the current paradigm for cancer research rests within the somatic mutation theory, which characterizes cancer as a cell-based, genetic, molecular disease (2). The promise of the relentless, well-funded search of genetic evidence was that it would first provide a cogent explanation of the disease and then the means to prevent cancer incidence and mortality. Instead, judging by candid admissions made by leading cancer researchers reflecting on the so-called war on cancer (3, 4), those promises remain mostly unfulfilled.

If the goal of epidemiology is to study the causes and distributions of diseases in human populations so as to identify ways to prevent and control disease (5), then cancer epidemiology has had several notable successes, including the decrease in the number of cases and deaths from smoking-related cancers (6) and the precipitous drop in breast cancer incidence that was likely an unintended consequence of hormone treatments (7). The body of research driven by the somatic mutation theory is largely unrelated to these few measurable successes; no big “wins” in our field have been scored from decades of focusing on genetic mutations. Unfortunately, many cancer rates are increasing for unknown reasons (8), and for most cancers, there are still no robust prevention strategies. Treatments based on somatic mutation theory have, so far, been fleeting and temporary (3).

It is perhaps time for cancer epidemiologists to explore alternative theories of cancer causation. The tissue organization field theory (9), which posits that cancer is a tissue-based rather than a cell-based disease, has great potential for reframing the way we think about cancer causation and the means to treat and even prevent cancers. Sonnenschein and Soto (10) compellingly refute the classic hallmarks of cancer and propose switching from the reductionist philosophy shadowing the somatic mutation theory to a more plausible and productive organicist approach. Exploring the potential of this theory in human populations will require cancer epidemiologists to think differently about which exposures to investigate, what new kinds of collaborators to recruit, and how epidemiologic methods might be used in innovative ways. The decision of whether to train future (or retrain current) cancer epidemiologists as molecular geneticists and/or big data analysts should rest on personal preferences, because the record thus far shows that such efforts may not result in those big wins in the war on cancer that are our ultimate goal as cancer epidemiologists.

Also, while we’re rethinking the whole paradigm, can we please stop talking about this effort in military terms? Perhaps gardening is the more accurate, realistic metaphor—find out what improves the “soil” of tissues and organs, favors their healthy growth and maintenance, and discourages the “weeds.”

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REFERENCES


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