As exciting as the latest scientific research can seem, it can be hard to discern its true relevance. This context and significance often gets lost in translation when in transit from lab bench to journal article to the public domain. From there, the information takes on a life of its own like a bad game of telephone. Today, just calling something a “study” and a large one at that is enough to elicit knee-jerk tacit acceptance of assumed credibility and validity. Such will likely be the case with a newly published *BMJ* piece on maternal prenatal caffeine intake and being overweight in childhood.

But, putting research into context requires a lot more unpacking – the boring reality is sometimes it is worthwhile, sometimes it isn’t and, often, it falls into that nebulous in between category. And, when it comes to prospective observational epidemiological studies like this one, the subject matter may change but the use of routinely flawed methodology can create rather than discover associations mathematically made to fit into a desired framework.

Even these authors acknowledge

> “observational studies can never establish causality”

Why can these study designs be so problematic?

Let’s take this recent publication whose stated objective was
“to study the association between maternal caffeine intake during pregnancy and the child’s weight gain and overweight risk up to 8 years.”

Here are some issues in it, to name a few: data was self-reported for intake of 255 dietary items at 22 weeks gestation-- “which can induce misreporting, it is a limitation” -- and in parental-reporting measurements; growth models estimated missing body size data; factors that influence infant findings (e.g. maternal and paternal height/weight parameters, presence or absence of gestational diabetes) were “considered but not included in the final model;” how and what confounders were adjusted remain quite vague (e.g. was the infant formula or breastfed, what was the feeding schedule, what perinatal, family history and other environmental factors were at play - these are among those not addressed).

Even the authors suggest [2]

“our findings might be explained by residual confounding of non-accounted factors related to an overall unhealthy lifestyle and high caffeine consumption; though exclusion of smokers and very high caffeine consumers did not modify results.”

Consideration of a host of confounders that influence growth in infancy and childhood including but not limited to diet, exercise, underlying medical conditions and genetics were not explored.

So, what’s the big deal?

In general, the goal is to advance or course correct medical understanding and innovation. Deficiencies in data collection and interpretation shift the focus away from the findings and diminish their value, while impacting human behavior. This tends to serve getting published more than it does progress.

This particular work supports existing recommendations to restrict maternal caffeine intake during pregnancy; and, reaffirming that certainly has merit given the importance of protecting you and your baby from avoidable harm (see current policy here [3], here [4] and here [5]). Making the leap that there is significant correlation with being overweight in childhood, without accounting for a number of meaningful confounders, is a tougher sell based on this study design.

That’s why it is crucial not to ignore - or de-prioritize - other substantial influences that contribute to pediatric obesity, a multi-factorial problem.

To appreciate the frequency and extent such limited studies are taking place and how they can devalue legitimate science, review my articles Cannabis and Strokes: Linked Or Not? [6] and Hey Media, Even Harvard Can Get It Wrong [7]. With the scientific community in a constant battle for resources, very real tangible funds can get reallocated away from more higher risk, higher reward research.