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A commentary on "Linking pediatric obesity to youth-onset type 2 diabetes: integrated longitudinal and cross-sectional evidence from population-based studies worldwide" – Parallel trends are not causal evidence: methodological caution regarding the pediatric obesity–youth-onset type 2 diabetes link

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To the editor,

Soliman et al.[1] synthesized 25 years of population-based data on pediatric obesity and youth-onset type 2 diabetes mellitus (T2DM) and concluded that the evidence confirms "a robust, temporally consistent, and biologically plausible causal relationship" between the 2 epidemics. The clinical message—that primary prevention of childhood obesity is the most powerful available strategy—is one with which we fully agree, and the assembly of registry data from 6 continents is a valuable service to readers. We are concerned, however, that the strength of the causal language exceeds what the assembled design can support, and we note several features of the synthesis that warrant clarification.

First, the analysis is ecological. Temporal parallelism between 2 population-level trajectories, however consistent across countries, cannot establish that one drives the other; many exposures relevant to metabolic risk—ultraprocessed food availability, screen time, sedentary behavior, and diagnostic intensity—have risen along the same 25-year axis. The authors state that temporal parallelism was evaluated through comparative analysis, but no lag, cross-correlation, or time-series modeling is reported, so the claim that obesity temporally precedes T2DM incidence rests on visual inspection of Fig. 1 rather than on a formal test. Given that the review's own data are ordinal and vary in start year by registry, we would suggest that "consistent with" is a more defensible formulation than "confirms."

Second, the internal consistency of the parallelism argument is weaker than presented. Israel showed by far the steepest relative increase (+441%; 0.63→3.41 per 100,000/yr), yet its obesity trend is characterized only qualitatively as rising; a 441% relative change arising from a baseline incidence below 1 per 100,000 is highly sensitive to improvements in case ascertainment and registry completeness over the same interval.[2] Conversely, Japan is described as having plateaued in obesity while its T2DM data derive from a school urine glucose screening program,[3] a detection method not comparable to registry-based ascertainment elsewhere. The 2 extremes of the dataset are therefore the 2 least able to sustain the central claim, and neither is discussed in that light.

Third, the assertion in the section on bariatric surgery that adiposity is "both necessary and sufficient for youth-onset T2DM in the vast majority of cases" is difficult to reconcile with the review's own figures. Approximately 25% of affected youth are not obese at diagnosis globally,[4] rising to roughly 32% in Japan, and the authors themselves emphasize that around 12% of apparent T2DM presentations may harbor monogenic diabetes—a proportion for which no supporting citation is provided. The Teen-Teen Longitudinal Assessment of Bariatric Surgery (Teen-LABS) remission figure of 95% at 3 years, quoted in the abstract, derives from 19 of 20 evaluable participants[5]; presenting this denominator alongside the percentage would help readers calibrate its precision.

Finally, we note 2 citation errors that we would ask the authors to correct. The statement that comparable upward trends were documented in England and Wales is supported by reference 8, which reports type 1 diabetes

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incidence in Finnish children,[6] and the statement for New Zealand cites reference 11 in the original article, an Australian clinical care guideline for type 1 diabetes.[7] In addition, primary data for 5 of the 8 registries in Table 1 (England and Wales, Israel, Australia, New Zealand, and Japan) are attributed to a single source, the narrative review by Perng et al.[8] Newcastle-Ottawa Scale scores of 7/9 to 8/9 assigned on the basis of a secondary synthesis rather than the primary reports may overstate the appraised quality of those entries, and this warrants explicit acknowledgment in a review that adopts a systematic framework.[9]

None of these observations undermines the review's central public health argument, and we share the authors' view that pediatric obesity is the dominant modifiable driver of youth-onset T2DM. Our concern is one of calibration: in a field where surveillance quality, ascertainment methods, and ethnicity-specific body mass index thresholds vary substantially between registries, causal language should be reserved for designs capable of supporting it, and quality appraisal should be traceable to the primary sources. Framing the evidence as strongly suggestive rather than confirmatory would, in our view, better serve both the argument and the readers who will act on it.

See the article "Linking pediatric obesity to youth-onset type 2 diabetes: integrated longitudinal and cross-sectional evidence from population-based studies worldwide" via https://doi.org/10.3345/cep.2026.01228.

See the authors' reply to a commentary on "Linking pediatric obesity to youth-onset type 2 diabetes: integrated longitudinal and cross-sectional evidence from population-based studies worldwide" via https://doi.org/10.3345/cep.2026.02831.

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