COMMENT ON CHENG ET AL.

Trends and Disparities in Cardiovascular Mortality Among U.S. Adults With and Without Self-Reported Diabetes, 1988–2015. Diabetes Care 2018;41:2306–2315

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We read with great interest the recently published article by Cheng et al. (1). The authors note that there was a dramatic increase in prevalence (age-standardized) between the 1980s and 2010 (3% and 9%, respectively) in the population with diabetes compared with the population without, along with a decrease in the excess risk of cardiovascular disease (CVD). The authors attribute this excess risk reduction to an improvement in primary and secondary diabetes care.

However, it must be remembered that the diabetes diagnostic criteria have changed over the years. In 1997, the fasting blood glucose threshold for diagnosis went from 140 to 126 mg/dL, precisely in view of the increased risk of CVD in patients with blood glucose levels between 126 and 140 mg/dL (2). This change, combined with more frequent screening in the population at an ever-younger age (3) and greater disease awareness in the population (4), means that self-reported diabetes no longer describes the same disease described in the 1980s.

This phenomenon implies not only that some individuals with diabetes in the 1980s were included in the healthy population (which mathematically has a minimal impact on the CVD incidence comparison between the population with and without diabetes, as the authors rightly note in their discussion of the study's limitations), but mainly that the population with diabetes in the 1980s suffered from much more severe disease (the relationship between blood glucose level and cardiovascular risk showed a dose-response effect). Diabetes was also usually diagnosed later, and therefore patients were at higher risk of complications, as demonstrated by the phenomenon of "legacy" (metabolic memory) (5), than was the case with diabetes diagnosed in the 2000s.

The change in definition and the increased ability to diagnose this condition at least partially justify the dramatic prevalence increase, which is probably also due to a real increase in diabetes and its causes, such as obesity. These changes also justify the reduction of excess risk through a dilution effect of the denominator of the population with diabetes, which progressively includes people with lower blood glucose levels and a shorter duration of prediagnosis disease and therefore less risk. Nevertheless, the change in diagnostic criteria and the increase in diagnosis of a subgroup of the population with diabetes with fewer complications do not impact the reduction of the cardiovascular risk gap between women and men in this population, compared with the gap existing in the general population. The main findings of Cheng et al. are thus valid and worth investigating further to understand underlying mechanisms.

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