Mean arterial pressure (MAP), the average blood pressure (BP) over a cardiac cycle, is an important measurement of the overall circulating pressure load. Adequate MAP (at least 60 mm Hg) is needed to sustain sufficient perfusion pressure for tissue perfusion so as to reduce hypoperfusion and organ failure, furthermore, prevent death. MAP is highly associated with systolic BP (SBP) and diastolic BP (DBP). Previous studies have confirmed MAP as an important independent predictor of metabolic syndrome, cardiovascular disease (CVD) and cardiovascular death. However, unlike the well-established strong association between MAP and cardiovascular mortality, the evidence on the predictive utility of MAP in the risk of all-cause death remains controversial. MAP was found to be comparable to SBP in predicting the risk of CVD mortality and all-cause mortality across all age and gender groups in the Chicago Heart Association cohorts, while every 10 mm Hg increase in MAP was reported to be independently associated with a 14% increase in the risk of all-cause mortality in the elderly with isolated systolic hypertension in the USA. However, in Middle Eastern populations without baseline CVD and antihypertensive medications, MAP showed insignificant relation with all-cause mortality among the elderly, although it was strongly associated with an increased risk of CVD mortality and all-cause mortality in those aged <60 years. This evidence based on the middle-aged or elderly populations is inadequate to define the role of MAP in predicting all-cause death among the young, who are generally ‘healthy’.

Sun et al evaluated the associations between MAP and all-cause mortality in adults aged between 18 and 40 years, filling a gap in our understanding of the relationship between MAP and all-cause death in the general population. The authors analysed data from the US National Health and Nutrition Examination Survey (1999–2006) and concluded that there was no significant association between MAP and all-cause mortality in a young adult population. Unlike previous studies, this ‘healthy’ population has no history of CVD, low rates of smoking and obesity, and relatively normal SBP levels. Therefore, this study with relatively short follow-up may lack the power to address all-cause mortality. The findings do not come as a surprise.

Moreover, as MAP was estimated indirectly rather than directly measured, the calculation methods may affect the results. In this paper, the authors used the standard calculation for MAP (DBP plus one-third of pulse pressure (PP)) regardless of heart rate (HR), which could introduce errors and be less accurate than the new formula, namely, MAP = DBP + [(0.33 + (HR × 0.0012)) × (PP)]. This limitation also makes the use of MAP as a risk predictor inferior to SBP in clinical practice. Current guidelines for hypertension diagnosis and management neither define an optimal MAP as a target nor use MAP in risk calculations. SBP remains the main parameter representing the risk of CVD posed by high BP. Using MAP in combination with SBP does not improve prognostication. In this regard, whether MAP can be used as an independent predictor of all-cause mortality remains to be answered by more research, especially in the young.

MAP increases with age as a result of arterial stiffening. The findings of Sun et al, on the other hand, reflect the importance of lifestyle management in BP lowering from an early age.

CVDs such as sepsis in the ‘healthy’ young adult participants, which limited the assessment of the association between MAP and death. The regulation of MAP is through a complex interaction between the cardiovascular, renal and autonomic nervous systems. Although the impact of MAP on all-cause mortality in young adults has not been demonstrated, it still seems prudent to maintain MAP within the normal range (70–100 mm Hg) at all ages. Nevertheless, this study is valuable in revealing the association between MAP and all-cause death in the young, thus prompting more studies on MAP and more studies focusing on early BP control.

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