Several studies have reported an association between air pollution and endothelial dysfunction, especially in individuals having diabetes. However, very few studies have examined the impact of air temperature on endothelial function. The objective of this analysis was to investigate short-term effects of temperature and ozone on endothelial function in individuals having diabetes. Moreover, we investigated interactive effects between air temperature and air pollution on markers of endothelial function. Between November 2004 and December 2005 flow-mediated dilatation (FMD), nitroglycerin-mediated dilatation (NTGMD) and several blood markers representing endothelial function were measured using brachial artery ultrasound on four consecutive days in 22 individuals with type-2 diabetes mellitus in Chapel Hill, North Carolina (USA). Daily measurements of meteorological parameters, ozone and particulate matter with an aerodynamic diameter ≤2.5 μm (PM<sub>2.5</sub>) were obtained from fixed monitoring sites. We used additive mixed-models adjusting for time trend, day of the week, relative humidity and barometric pressure to assess temperature and ozone associations with endothelial function. A 1 °C decrease in the 24-h temperature average was associated with a decrease in mean FMD on the same day (−2.2% [95%-confidence interval: [−4.7; 0.3%]]) and with a delay of one and four days. A temperature decrement also led to an immediate (−1.7% [−3.3; −0.04]) decrease in NTGMD. Moreover, we observed an immediate (−14.6% [−26.3; −2.9%]) and a one day delayed (−13.5% [−27.0; 0.04%]) decrease in FMD in association with a 0.01 ppm increase in the maximum 8-h moving average of ozone. Temperature effects on FMD strengthened when PM<sub>2.5</sub> and ozone concentrations were high. The associations were similar during winter and summer. We detected an association between temperature decreases and ozone increases on endothelial dysfunction in individuals having diabetes. We conclude that endothelial dysfunction might be a possible mechanism explaining cardiovascular events in association with environmental stimuli.