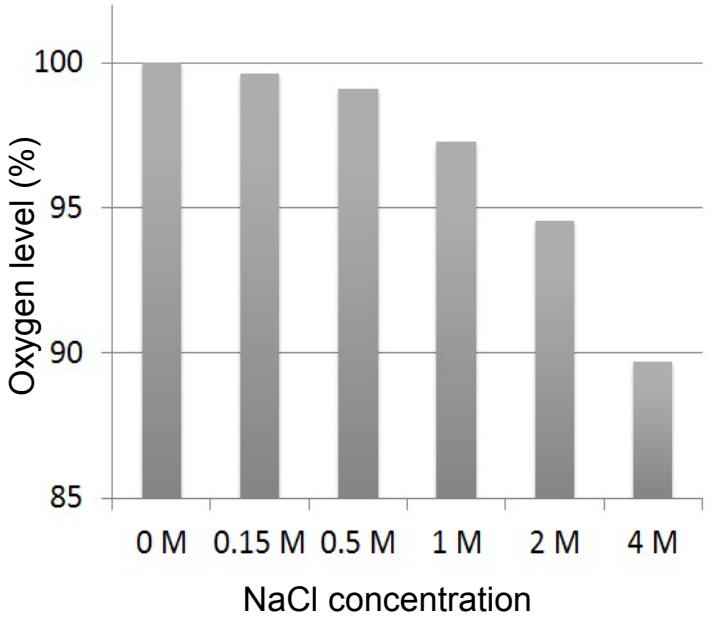
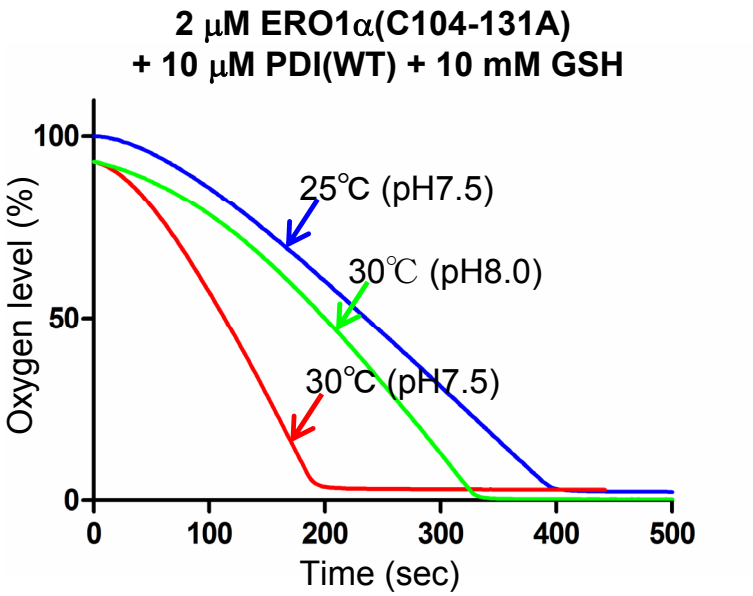
Supplemental Figure 1

A



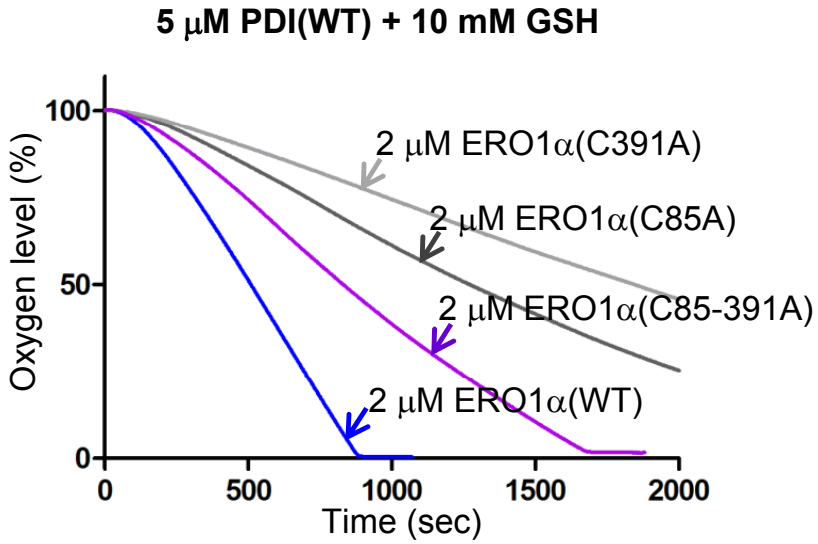
B

0℃	20℃	40℃	60℃	
0.049	0.031	0.023	0.019	(cm^3/cm^3)



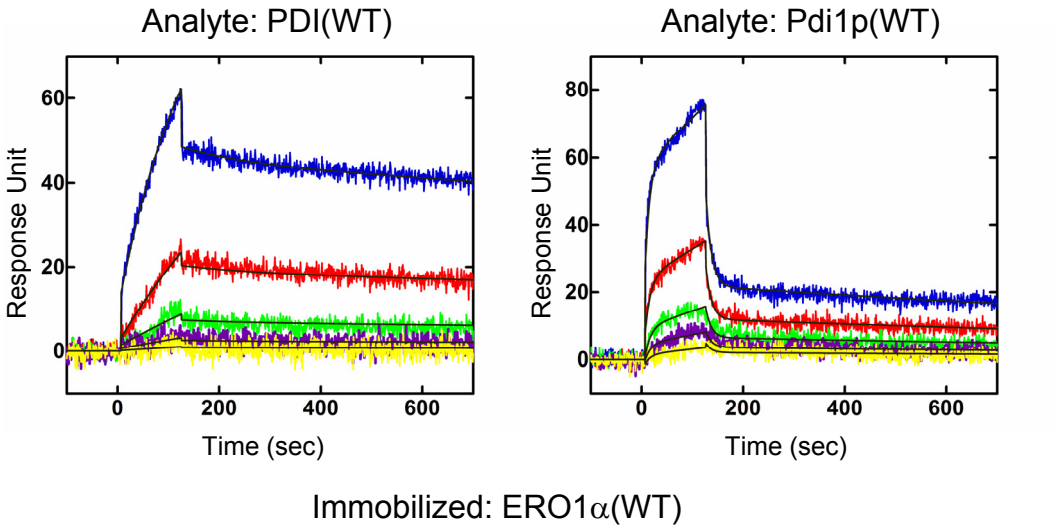
Supplemental Fig. 1 NaCl concentration or temperature dependence of the amount of dissolved oxygen. *A*. The amount of dissolved oxygen in air-saturated assay buffer (50 mM HEPES [pH 7.5] and 2 mM EDTA, 25℃) at each NaCl concentration detected by a Clark-type oxygen electrode. Dissolved oxygen level at 0 M was regarded as 100%. *B*. Upper column shows the amount of dissolved oxygen in the water at each temperature (2011 MARUZEN Co. Ltd., Publishing Division). The lower graph shows the oxygen consumption rates under each condition, as depicted in each curve, based on the absolute amount of dissolved oxygen. The initial amount of dissolved oxygen at 25℃ was taken to be 100% and the initial amount at 30℃ was estimated to be ~93% from the data of the upper column

Supplemental Figure 2



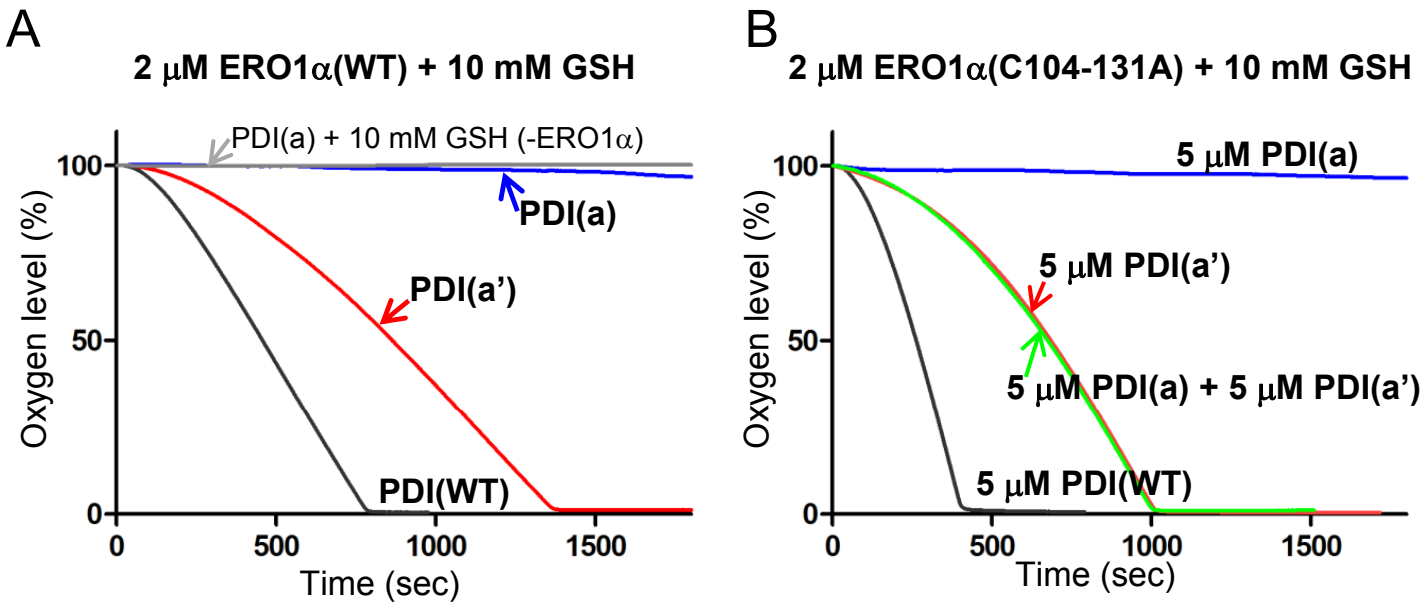
Supplemental Fig. 2 Oxygen consumption of 5 μM human PDI in the presence of 10 mM GSH by wild-type ERO1α or its mutants, including C85A, C391A, or C85A and C391A.

Supplemental Figure 3



Supplemental Fig. 3 Affinity measurements between human PDI and ERO1α(WT) or yeast Pdi1p and ERO1α(WT) by SPR spectroscopy. ERO1α was immobilized on a biosensor chip, and PDI or Pdi1p were injected as analytes. Blue, red, green, purple, and yellow curves correspond to 36, 12, 4, 1.33, 0.44 μM analyte, respectively.

Supplemental Figure 4



Supplemental Fig. 4 Oxygen consumption of 5 μM human wild-type PDI or its active site mutants (PDI(a) and PDI(a')) in the presence of 10 mM GSH by wild-type ERO1α *(A)* or the constitutively active mutant *(B)* without mutation of C166. These data indicated that the C166A mutant does not change the specificity of ERO1α for PDI, whereby ERO1α preferentially oxidizes PDI(a').