		р38МАРК		JNK-1		ERK-1	
Cytosol	Microsomes	activity	1/2 variation	activity	1/2 variation	activity	1/2 variation
ctl	Untreated	1.00	0.00	1.00	0.00	1.00	0.00
	+ Azc	12.52	0.23	2.18	0.27	2.89	0.32
	+ Tun	7.24	0.26	2.69	0.13	1.10	0.10
-Nck	+ Azc	13.00	0.45	3.78	0.57	1.34	0.19
	+ Tun	9.06	0.19	2.97	0.15	0.10	0.03
-Nck	+ Azc	7.09	0.60	2.01	0.06	3.64	0.33
+ 3SH3-Nck wt	+ Tun	5.91	0.21	1.64	0.04	1.46	0.47
-Nck	+ Azc	13.83	0.81	3.64	0.19	1.84	0.23
+ 3SH3-Nck mut	+ Tun	8.74	0.02	2.99	0.09	0.88	0.22
-Shc	+ Azc	10.43	0.27	2.08	0.11	3.34	0.18
	+ Tun	7.15	2.69	2.48	0.06	1.52	0.56
-Shc	+ Azc	15.56	0.35	1.74	0.42	1.82	0.17
+ SH2-Shc wt	+ Tun	11.89	0.51	1.36	0.03	0.75	0.10
-Shc	+ Azc	10.05	0.28	2.07	0.05	2.90	0.15
+ SH2-Shc mut	+ Tun	8.36	0.20	2.45	0.06	1.32	0.07

Table 1. Impact of immunodepletion of Shc and Nck on ER-stress-mediated activation of SAPK/MAPK pathways. ER microsomes immuno-isolated from non-treated, 10 mM Azc or 10 μg/ml Tun treated cells for 10 min were incubated with control RLC (Ctl), or RLC that had been previously immunodepleted of Nck (-Nck) either in the presence or absence of 10 μg recombinant Nck(3SH3) wt, Nck(3SH3) mutant or Shc(2SH2) wt or Shc(2SH2) mutant. ERK-1, JNK-1 or p38<sup>MAPK</sup> activities were determined as described in Experimental Procedures. Results are presented as fold increase of kinase activity upon Azc (light grey) or Tun (dark grey) treatment over control (n=2, value ± 1/2variation).

Supplementary data Nguyên et al.