Table S1: Crosstalk of RAS and Rho in cellular transformation.

Crosstalk of Ras & RhoA promotes/favors transformation (Yes/No/Undetermined)	Ras isoform	Remarks (key findings)	References
Yes	HRAS	RAS increases RhoA activity, through the RAF/MAPK pathway, by downregulating p190GAP. Active RhoA reduces p21 Cip expression.	(16)
Yes	Undetermined	Sustained RAS signaling interfere with the balance of Rac and Rho activity, leading to EMT. RAS downregulates Rac activity which in turn increase RhoA activity.	(19)
Yes	HRAS	RhoA activities are required for the downregulation of p21 Cip1, which enable HRAS transformation.	(18)
Yes	HRAS	Activated RhoA cooperate with activated RAF to induce transformation; dominant-negative RhoA 19N can reverts HRAS transformation. (Interestingly, the coexpression of RhoA 19N with HRAS V12 can restore stress fiber formation in transformed cells; RhoA19N is known to cause stress fiber disruption).	(21)
Yes	HRAS	Active RhoA-GTP are selected for by sustained RAF/MAPK signaling, which is required for proliferation of RAS transformed cells. RAS transformation causes RhoA to be uncoupled from stress fiber regulation.	(17)
Yes	HRAS	Constitutively activated RhoA and HRAS synergistically induce transformation. Dominant-negative RhoA T19N expression reduce RAS transformation.	(60)

Yes	HRAS	Oncogenic HRAS signaling in cell deprived of p53 synergistically enhances RhoA activity, through the p190GAP inactivation.	(15)
Yes	HRAS	Oncogenic HRAS transformation via a RAF independent pathway phenocopy RhoA 63L transformed NIH3T3. This transformation can be inhibited by dominant-negative RhoA T19N mutant.	(61)
Yes	HRAS	TGF-β-ALK5 activation of RhoA is essential for the HRAS mediated transformation.	(59)
Yes	HRAS	RhoA signaling is required to downregulate p27 which facilitated RAS transformation.	(62)
No	HRAS	HRAS transformed fibroblast cells lack stress fiber and cell adhesion. Inactivation of the RhoA-Rock pathway may contribute to RAS transformation.	(25)
No	KRAS	Oncogenic KRAS downregulates RhoA activity, resulting in reduced actin filament and stress fiber.	(23)
No	KRAS	RhoA expression is downregulated in ki-ras transformed NIH3T3; Transformed NIH3T3 displayed loss of stress fibers; Enforced Tropomysoin expression reverts transformed phenotype through Rho upregulation.	(65)
No	HRAS	RAS transformation downregulates Rho activities through Gankyrin. Increased Gankyrin in RAS transformed cells increases RhoA and RhoGDI interaction, leading to inactivation of RhoA.	(64)