Corrigenda

The oncogenic *RAS2^{val19}* mutation locks respiration, independently of PKA, in a mode prone to generate ROS

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We have repeated all the respiration experiments in the above paper with lower concentrations of TET and CCCP (20–100 μ M TET and 1–5 μ M CCCP) since the concentrations originally used may potentially cause non-specific effects. Using the lower range of TET and CCCP concentrations, we find that the respiratory state value (RSV) differences are still significant (10.65 in the *RAS2*^{val19} cells against 69.61 in wild-type cells). Thus, the conclusions of the paper remain unchanged. Nonetheless, the distinction should be made that the *RAS2*^{val19} mutant shows a respiration closer to state 4 than the wild type but is not completely non-phosphorylating. The corrected RSV table (Table I) is as follows:

Table I. Basal respiration rates and RSVs of different strains

Strain	Respiratory characteristics	
	Basal rate (µM O ₂ /min)	RSV
wt	13.36 ± 2.02	69.61 ± 3.9
RAS2 ^{val19}	4.49 ± 1.03	10.65 ± 4.45
wt + Yep13	14.61 ± 1.40	34.81 ± 2.7
$RAS2^{val19} + Yep13$	7.85 ± 0.57	9.35 ± 5.6
wt + Pde2p	23.27 ± 3.66	42.47 ± 2.8
$RAS2^{val19} + Pde2p$	13.76 ± 3.99	6.16 ± 10
wt + Yepd	17.80 ± 1.59	51.49 ± 4.76
RAS2val19 + Yepd	3.16 ± 1.26	15 ± 2.89
wt + UCP1	18.15 ± 6.62	22.59 ± 1.49
RAS2val19 + UCP1	14.61 ± 1.40	53.67 ± 9.56
bcy1-13	3.09 ± 1.23	44.23 ± 2.1