

Table 4. Reduction Potential and Biological Status of Cells

Cell line	Treatment <sup>a</sup>	E <sub>hc</sub> /mV for GSSG/2GSH (pH)				Ref.
		Proliferating	Confluent	Differentiating	Apoptotic	
HL-60	1 $\mu$ M staurosporine	-239 $\pm$ 6 <sup>b</sup>			-167 $\pm$ 9 <sup>b</sup>	[102]
HL-60	Overexpressing Bcl-2 + 1 $\mu$ M staurosporine	-230 <sup>b</sup> to -205 <sup>b</sup>			no apoptosis at E $\leq$ -205 <sup>b</sup>	[102]
Normal fibroblast	Untreated	-222 (7.0) <sup>c</sup> -247 (7.4)	-188 (7.0) <sup>c,d</sup> -213 (7.4)			[103]
Fibrosarcoma	Untreated	-213 (7.0) <sup>c</sup> -238 (7.4)	-213 (7.0) <sup>c,e</sup> -238 (7.4)			[103]
HT29	5 mM sodium butyrate	-258 (7.39) <sup>f</sup>		-201 (7.40) <sup>f</sup>		[30]
HT29	25 $\mu$ M benzyl-isothiocyanate	-244 (7.30) <sup>f</sup>		-160 (7.45) <sup>f</sup>		[30]
Murine hybridoma <sup>g</sup>		-235 <sup>b</sup>			-170 <sup>b</sup>	[30,176]
CRL-1606 murine hybridoma <sup>g</sup>	Untreated	-232 (7.0) <sup>c</sup> -257 (7.4)				[53]
Jurkat	Untreated	-240 <sup>b</sup>				[177]
WAL-2A human lymphocyte	Untreated	-237 <sup>b</sup>				[177]
WAL-2A human lymphocyte	$\rho^0$ (no mitDNA)	-233 <sup>b</sup>				[177]

<sup>a</sup> Changing cells from proliferation to another biological state.

<sup>b</sup> The data were adjusted to the measured cellular pH, but the pH was not reported.

<sup>c</sup> This reported E<sub>hc</sub> assumed pH = 7.0. The E<sub>hc</sub> below is adjusted to pH 7.4 with Eqn. 14.

<sup>d</sup> These cells were contact-inhibited.

<sup>e</sup> These cells were not contact-inhibited, thus, they continue to proliferate.

<sup>f</sup> This pH was determined experimentally.

<sup>g</sup> These cells are a fusion product of a myeloma and a B lymphocyte.

- [30] Kirlin, W. G.; Cai, J.; Thompson, S. A.; Diaz, D.; Kavanagh, T. J.; Jones, D. P. Glutathione redox potential in response to differentiation and enzyme inducers. *Free Radic. Biol. Med.* 27:1208–1218; 1999.
- [53] Hwang, C.; Sinskey, A. J.; Lodish, H. F. Oxidized redox state of glutathione in the endoplasmic reticulum. *Science* 257:1496–1502; 1992.
- [102] Cai, J.; Jones, D. P. Superoxide in apoptosis: mitochondrial generation triggered by cytochrome c loss. *J. Biol. Chem.* 273:11401–11404; 1998.
- [103] Hutter, D. E.; Till, B. G.; Greene, J. J. Redox state changes in density-dependent regulation of proliferation. *Exp. Cell Res.* 232:435–438; 1997.
- [176] Jones, D. P.; Maellaro, E.; Slater, A. F. G.; Orrenius, S. Effects of N-acetyl-L-cysteine on T-cell apoptosis are not mediated by increased cellular glutathione. *Immunol. Lett.* 45:205–209; 1995.
- [177] Cai, J. Y.; Wallace, D. C.; Zhivotovsky, B.; Jones, D. P. Separation of cytochrome c-dependent caspase activation from thiol-disulfide redox change in cells lacking mitochondrial DNA. *Free Radic. Biol. Med.* 29:334–342; 2000.