SUPPLEMENTARY TABLES

Supplementary Table 1. Leave-one-out sensitivity analyses on LPO.

	Pooled SMD [95% CI]	Between-study heterogeneity
Overall	-2.00[-2.91, -1.10]	$p < 0.0001$; I^2=79%
Omitted study		
Carretero M (2009) female	-1.86[-2.85, -0.88]	$p < 0.0001$; I^2=81%
Carretero M (2009) male	-2.21[-3.20, -1.22]	$p = 0.0003$; I^2=78%
Garcia J (2011)	-1.99[-3.08, -0.91]	$p < 0.0001$; I^2=82%
Gutierrez-Cuesta J (2007)	-1.75[-2.63, -0.86]	$p = 0.002$; I^2=74%
Nogues MR (2006) female	-2.08[-3.14, -1.03]	$p < 0.0001$; I^2=82%
Nogues MR (2006) male	-2.29[-3.13, -1.44]	$p = 0.005$; I^2=70%
Okantani Y (2002)	-1.83[-2.81, -0.85]	$p = 0.0003$; I^2=78%

Supplementary Table 2. Leave-one-out sensitivity analyses on carbonylated protein.

	Pooled MD [95% CI]	Between-study heterogeneity
Overall	-5.74[-11.03, -0.44]	$p < 0.00001$; I^2=93%
Omitted study		
Caballero B (2008)	-6.38[-13.25, 0.48]	$p < 0.00001$; I^2=95%
Garcia JJ (2011)	-6.40[-13.29, 0.49]	$p < 0.00001$; I^2=95%
Gutierrez-Cuesta J (2007)	-3.00[-4.59, -1.40]	$p = 0.81$; I^2=0%
Okantani Y (2002)	-6.84[-13.44, -0.24]	$p < 0.00001$; I^2=92%

Supplementary Table 3. Leave-one-out sensitivity analyses on GPx.

	Pooled SMD [95% CI]	Between-study heterogeneity
Overall	3.33[1.89, 4.78]	$p = 0.06$; I^2=65%
Omitted study		
Carretero M (2009) female	3.53[0.91, 6.14]	$p = 0.02$; I^2=81%
Carretero M (2009) male	2.61[1.77, 3.46]	$p = 0.30$; I^2=5%
Okantani Y (2002)	4.04[2.38, 5.71]	$p = 0.20$; I^2=40%

Supplementary Table 4. Leave-one-out sensitivity analyses on GRx.

	Pooled SMD [95% CI]	Between-study heterogeneity
Overall	2.59[0.50, 4.68]	$p < 0.00001$; I^2=90%
Omitted study		
Caballero B (2009)	3.35[0.69, 6.00]	$p < 0.00001$; I^2=91%
Carretero M (2009) female	1.87[-0.13, 3.87]	$p < 0.0001$, I^2=88%
Carretero M (2009) male	1.65[-0.16, 3.45]	$p = 0.0001$, I^2=86%
Nogues MR (2006) female	3.35[0.62, 6.09]	$p < 0.00001$, I^2=90%
Nogues MR (2006) male	2.92[0.01, 5.83]	$p < 0.00001$, I^2=92%

Supplementary Table 5. Leave-one-out sensitivity analyses on GSH/GSSH ratio.

	Pooled MD [95% CI]	Between-study heterogeneity
Overall	1.12[0.77, 1.47]	p = 0.12; I^2=53%
Omitted study		
Carretero M (2009) female	0.99[0.66, 1.33]	p = 0.21; I^2=36%
Carretero M (2009) male	1.25[0.93, 1.58]	p = 0.24; I^2=27%
Garcia JJ (2011)	1.12[0.44, 1.81]	p = 0.04; I^2=76%

Supplementary Table 6. PubMed search strategy (01 August 2019).

Search	Query	Items found
#1	("melatonin"[MeSH Terms] OR "melatonin"[All Fields] OR "n acetyl 5 methoxytryptamine"[All Fields])	25030
#2	((((("brain"[MeSH Terms] OR "brain"[All Fields]) AND ("aging"[MeSH Terms] OR "aging"[All Fields] OR "agings"[All Fields] OR "ageing"[All Fields] OR "ageings"[All Fields])) OR ("sensation"[MeSH Terms] OR "sensation"[All Fields] OR "senses"[All Fields]) OR ("geriatrics"[MeSH Terms] OR "geriatrics"[All Fields] OR "geriatric"[All Fields] OR "gerontol"[All Fields])) AND "cellular senescence"[MeSH Terms]) OR "cellular senescence"[All Fields]	18,940
#3	(("rodentia"[MeSH Terms] OR "rodentia"[All Fields] OR "rodent"[All Fields] OR ("rats"[MeSH Terms] OR "rats"[All Fields] OR "rat"[All Fields]) OR ("mice"[MeSH Terms] OR "mice"[All Fields]) OR ("mice"[MeSH Terms] OR "mice"[All Fields] OR "mouse"[All Fields]) OR ("rats"[MeSH Terms] OR "rats"[All Fields] OR "rattus"[All Fields]) OR "mus"[All Fields]) AND "SAM"[All Fields]) OR "samp"[All Fields] OR "SAMP8"[All Fields] OR "SAMP10"[All Fields] OR (("aging"[MeSH Terms] OR "aging"[All Fields] OR "senescence"[All Fields]) AND ("mice"[MeSH Terms] OR "mice"[All Fields]))	53,179
#4	#2 OR #3	67,390
#5	#1 AND #4	303

Supplementary Table 7. Explanations for the full-text article exclusions.

SL/NO	Title	Reasons
1	Gutierrez-Cuesta J, Tajes M, Jimenez A, Camins A, Pallas M. [Effects of melatonin in the brain of the senescence-accelerated mice-prone 8 (SAMP8) model]. Rev Neurol. 2011; 52: 618–22.	Review
2	Asai M, Ikeda M, Akiyama M, Oshima I, Shibata S. Administration of melatonin in drinking water promotes the phase advance of light-dark cycle in senescence-accelerated mice, SAMR1 but not SAMP8. Brain Res. 2000; 876: 220–4.	Unrelated outcome
3	Lardone PJ, Alvarez-García Ó, Carrillo-Vico A, Vega-Naredo I, Caballero B, Guerrero JM, Coto-Montes A. Inverse correlation between endogenous melatonin levels and oxidative damage in some tissues of SAM P8 mice. Journal of Pineal Research. 2006; 40: 153–7.	Wrong study organ
4	Rosenfeld SV, Togo EF, Mikheev VS, Popovich IG, Khavinson VK, Anisimov VN. Effect of epithalon on the incidence of chromosome aberrations in senescence-accelerated mice. Bull Exp Biol Med. 2002; 133: 274–6.	Wrong study design
5	Shibata S, Asai M, Oshima I, Ikeda M, Yoshioka T. Melatonin normalizes the re-entrainment of senescence accelerated mice (SAM) to a new light-dark cycle. Adv Exp Med Biol. 1999; 460: 261–70.	Unavailable
6	Parisotto EB, Vidal V, García-Cerro S, Lantigua S, Wilhelm Filho D, Sanchez-Barceló EJ, Martínez-Cué C, Rueda N. Chronic Melatonin Administration Reduced Oxidative Damage and Cellular Senescence in the Hippocampus of a Mouse Model of Down Syndrome. Neurochem Res. 2016; 41: 2904–13.	Wrong animal model
7	Morioka N, Okatani Y, Wakatsuki A. Melatonin protects against age-related DNA damage in the brains of female senescence-accelerated mice. J Pineal Res. 1999; 27: 202–9.	Wrong animal model
8	Cristòfol R, Porquet D, Corpas R, Coto-Montes A, Serret J, Camins A, Pallàs M, Sanfeliu C. Neurons from senescence-accelerated SAMP8 mice are protected against frailty by the sirtuin 1 promoting agents melatonin and resveratrol. J Pineal Res. 2012; 52: 271–81.	*Ex-vivo*