

# Telomere length and cerebrovascular diseases

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The telomere length (TL) and telomerase activity may claim the role of genetic markers of biological age of blood vessels. Some authors believe that leukocyte TLs are associated with oxidative stress and inflammation even in healthy people, which suggests that systemic oxidative stress and inflammation associated with cardiovascular risk may accelerate telomere shortening, thereby mitigating the effects of the hereditary component of TL. In addition, given the systemic effects of oxidative stress, inflammation, and similar indicators of shortening of TL in somatic tissues, the rate of the shortening of TL in the blood cells reflects this in the vascular tissue. This theory implies that the shortening rate of TL can serve as the clinical biomarker of cardiovascular mortality. While many potential pathophysiological mechanisms have been proposed, there is not yet a consensus as to which are most important in causing cerebrovascular morbidity/mortality. **The aim** of our study is to determine the relationship between telomere length and telomerase activity with indicators of oxidative stress in patients with cerebral atherosclerosis (CA) and diabetes mellitus.

**Methods.** The clinical and instrumental study involved 161 patients with CA.

**Results.** The patients were divided twice into the 2 groups depending on the relative TL and telomerase activity.

It has been found that the patients with the CA with long telomeres have statistically significantly higher level of catalase comparing with the group of short telomeres. Also, in the group with high telomerase activity, the catalase (CAT) and superoxide dismutase (SOD) levels are statistically significantly lower than in the group with low telomerase activity. It should be noted that the patients with long and short telomeres are comparable in age, and the age of the patients with low telomerase activity was statistically significantly greater.

**Conclusions.** In the patients with the CA the association of some markers of oxidative stress (CAT, SOD, glutathione (GSH) with telomere length and telomerase activity was detected, regardless of the presence of concomitant diabetes mellitus. The most stable direct correlation in this category of the patients was found between GSH and telomerase activity ( $r = 0.48$ ), that may indicate the key role of GSH in the rate of telomere shortening and the development of atherosclerosis.

	Long telomeres (n = 56)	Short telomeres (n= 30)	p
Catalase (CAT)	599.68 (526.662 – 637.987)	532.645 (324.36 – 554.195)	0.023
Glutathione (G)	3.52±0.2	3.58±0.19	0.481
Superoxide dismutase (SOD)	8.51 (8.03 – 9.33)	8.03 (7.87 – 8.51)	0.301
Thiobarbituroreactive Substances (TBARs)	17.61 (16.4 – 18.827)	16.585 (15.94 – 19.06)	0.664
Glycation End Products (AGE)	31.1±7.6	32.4±7.5	0.683
Age	65.8±10.9	61.7±9.2	0.079
Female	69,6%	70,0%	0.99
Male	30,4%	30,0%	0.99
Patients with ischemic stroke	51,8%	33,3%	0.11
Patients with diabetes mellitus	28,6%	33,3%	0.81

	High telomerase activity (n = 56)	Low telomerase activity (n = 30)	p
Catalase (CAT)	549.405 (369.85 – 590.1)	604.47 (544.615 – 699.038)	0.026
Glutathione (G)	3.59±0.19	3.46±0.18	0.134
Superoxide dismutase (SOD)	8.03 (7.75 – 8.37)	9.23 (8.55 – 9.79)	0.002
Thiobarbituroreactive Substances (TBARs)	16.97 (16.025 – 18.775)	17.81 (16.355 – 19.165)	0.727
Glycation End Products (AGE)	32±6.8	31.1±9.1	0.772
Age	61 (57 – 70)	66 (61 – 79)	0.021
Female	67,9%	73,3%	0.63
Male	32,1%	26,7%	0.63
Patients with ischemic stroke	44,6%	46,7%	0.99
Patients with diabetes mellitus	30,4%	30,0%	0.99