Antioxidant Activity of Blood Plasma in Individuals with Neoplasms

A. V. Marusin, V. B. Salyukov, and E. Yu. Bragina

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In 3 groups of patients with neoplasms plasma antioxidant activity was measured by the yield of TBA-reactive products generated in lecithin—Fe^{2+} model system. Patients with laryngeal, tongue, oropharyngeal, oral, and laryngopharyngeal tumors showed lower plasma antioxidant activity compared to patients with breast, lung, and bronchial cancer ($p<0.005$). In women with breast cancer plasma antioxidant activity increased with age ($p<0.05$). Possible mechanisms underlying these fluctuations are discussed. It is assumed that plasma antioxidant activity is relatively stable and modulates body resistance to tumor process.

Key Words: *antioxidant activity; blood plasma;* vari*ability; neoplasm*

The role of reactive oxygen species in carcinogenesis and aging is well established. In light of this, the antioxidant system can be regarded as a limiting factor determining adaptive capacities of the organism, life span, and, possibly, the risk of tumor formation [8,12]. *In vivo* studies showed that tumor cells possess high antioxidant activity (AOA) and can immediately release prostaglandins upon contact with macrophages, neutrophils, and natural killers. This phenotype provides two mechanisms of effective local defense against immune effectors, which ten- and hundred-fold decreases rejection of tumor cell [1]. However, published data on the content and activity of antioxidant enzymes, low molecular antioxidants, and the state of AOA during tumor growth are contradictory [2,12]. Possible changes in AOA during carcinogenesis, their dependence on tumor localization, and the correlation between AOA variability and the risk of cancer remain unclear.

Here we measured plasma AOA in patients with tumors of different localization and variability of this parameter depending on patient's sex and age.

Institute of Medical Genetics, Tomsk Research Center, Siberian Division of the Russian Academy of Medical Sciences. ***Address for correspondence:*** marussin@img.tsu.ru. Marusin A.V.

MATERIALS AND METHODS

Forty-five men and 39 women (age 30-84 years, mean 55.2 years) patients of outpatient department of the Institute of Oncology were included in the study. The patients were divided into 3 groups: breast cancer (BC, n=36, women); lung and bronchial cancer (LBC, n=19, 16 men and 3 women); laryngeal, tongue, oropharyngeal, oral, and laryngopharyngeal cancer (OPC; n=29, men).

Plasma AOA was evaluated by inhibition of peroxidation in a model system containing oxidation substrate, inductor of oxidation, and plasma sample [3].

The method was elaborated in the Laboratory of Molecular and Cell Radiobiology (Institute of Medical Radiology, Obninsk) and based on spectrophotometric measurement of malonic dialdehyde (MDA) produced during Fe^{2+}-induced peroxidation of lecithin liposomes in the presence of plasma samples [4].

Variability of plasma AOA was characterized by lognormal distribution and the data were subjected to logarithmic transformation (after multiplying by 100 to exclude negative values) and analyzed by parametric tests.

The results were processed using Statistica 5.0 software.

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RESULTS

In OPC group, plasma AOA was lower than in BC and LBC groups (Fig. 1; $p<0.005$). The revealed difference cannot be explained by patient's age or gender. OPC patients did not differ by age from LBC patients (59.28±10.60 and 61.11±8.46 years, $p>0.05$), while AOA in women with BC did not differ from that in LBC patients, but BC patients were younger than OPC and LBC patients (48.81±10.10 years).

We previously showed that AOA did not depend on age, at least in the interval from 18 to 65 years [4,5]. No differences in AOA between practically healthy men and women were found. These results agree with the concept that total AOA is relatively stable and does not vary with age [11]. The content and activity of some antioxidant enzymes and low-molecular-weight antioxidants considerably decreased only in 75-80-year-old individuals [6,9].

However, published data on sex- and age-related changes in AOA are contradictory [6,9,11,13,14]. Some authors reported age-dependent changes in the integral parameters of the antioxidant system with age attesting to impaired oxidation resistance [13]. Sex-related differences in plasma capacity for scavenging free radicals generated in a model system were found [11].

Reduced plasma AOA in OPC patients compared to other groups can be explained by different contribution of exogenous factors determining localization of neoplasms. We hypothesize that individuals with low AOA under the effect of adverse environment more often develop OPC, while the development of BC and LBC in subjects with high AOA depends on endogenous factors, in particular, on decreased activity of DNA repair systems, which is an independent etiological factor of carcinogenesis [10].

The present study revealed an age-dependent increase in AOA in women with BC (Fig. 2; $r=0.42$, $p=0.02$). The revealed correlation attests to age-related differentiation of BC development in individuals with high AOA, rather than to age-related AOA variability. In other words, the disease develops later in individuals with high plasma AOA.

Thus, the integral AOA evaluated by inhibition of Fe^{2+}-induced peroxidation of lecithin liposomes (generation of TBA-reactive substances) in the presence of plasma samples does not change with age and may play a role in organism's resistance to tumor development.

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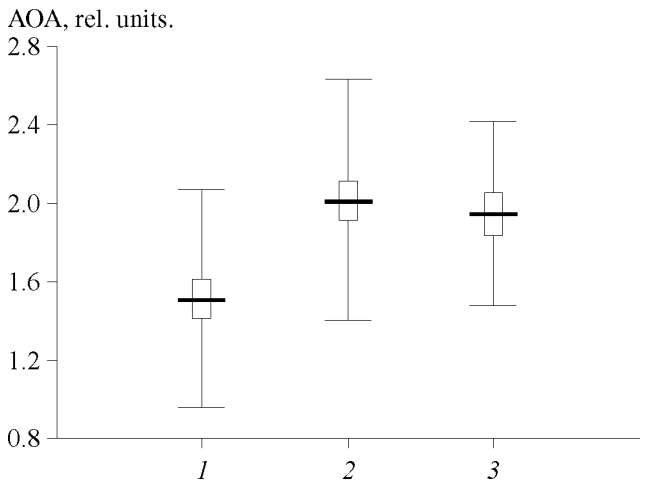

Fig. 1. Plasma antioxidant activity (AOA) in patients with laryngeal, tongue, oropharyngeal, oral, and laryngopharyngeal cancer (*1*, *n*=29), breast cancer (*2*, *n*=36), lung and bronchial cancer (*3*, *n*=19).

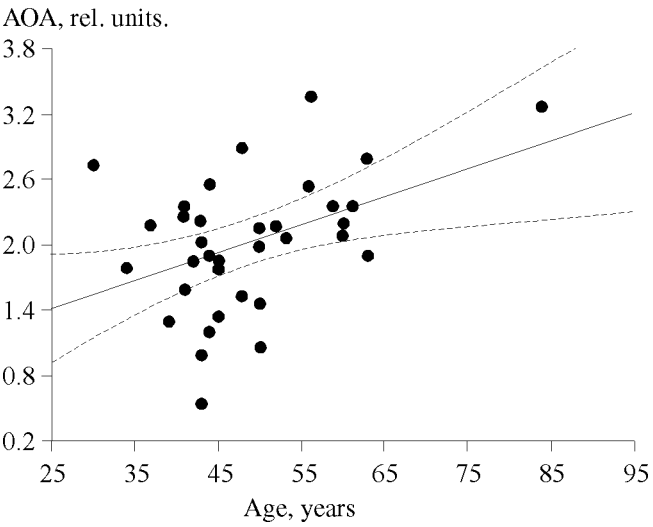

Fig. 2. Plasma antioxidant activity in patients with breast cancer of different age. Regression equation: $y=(0.0255\pm0.094)x+(0.7822\pm0.4702)$; $p=0.011$. Dotted line — 95% confidence interval for regression line.

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