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ZINC CONTENT IN BERRIES - THE IMPORTANCE FOR HUMAN HEALTH

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Berries have become inevitable part of the diet of conscious men. Even though berries are not the richest source of zinc, there are agricultural practices that increase zinc content in surface soils. Being markedly involved in the human physiology and biochemistry, zinc is also a common component of plant metabolic pathways. A zinc defficiency occurs in plants mostly in calcareous and alkaline soils, affecting young leaves first. The toxicity and intolerence of zinc, which occur in the soils with prolonged use of fertilizers containing zinc among other elements, are greater problems than zinc deficiency. Zinc toxicity is often associated with the deficiency of magnesium, iron or manganese. In humans, zinc deficiency leads to *acrodermatitis enteropathica*, growth retardation, hypogonadism, depressed mental function, impaired cognitive functions and immune disorders, affecting males and females in the developing world. Serum zinc was correlated with the severity of depression and low serum zinc levels were found in depressed patients, suggesting that serum zinc could be the marker of depression. Diverse neurodegenerative processes, as Alzheimer's disease, may change the cellular zinc level, raising it to the level where zinc contributes to the progression of the disease. Zinc plays a pivotal role in few signal transduction and gene expression pathways, including that of cytokine genes. Zinc also holds a key position for multiple functions of cell metabolism, retinal development and specific retinal functions. Zinc in therapeutic dosages was effective in decreasing the incidence of infections. Zinc lozenges reduced the duration and severity of the common cold. *Acta Medica Medianae 2016;55(4):73-81.*

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Zinc Content in Berries - The Importance for Human Health

Berries have become an inevitable part of everyday meals of all diet-conscious men. These fruits are rich in phytonutrients like polyphenols (flavonoids, anthocyanins), vitamins (vitamin C, B, A and K) and minerals (Ca, Mg, Zn), which give them their unique and pleasant taste. They are at the same time low in fats, which makes them a popular and enjoyable food or snack. The juices obtained from berries have a specific flavor and aroma that makes them true energy boosters. All these features make the berries ideal part of the modern man's diet. Dietary guidelines around the world recommend increased intake of fruits and vegetables as an exceptional source of phytochemicals for the prevention of chronic diseases. In addition, different epidemiological studies showed that the consumption of anthocyanins, the predominant class of flavonoids in berries, reduces the risk of chronic noncommunicable deseases (cardiovascular diseases, diabetes, cancer and arthritis) due to specific antioxidative and anti-inflammatory effects they demonstrate in vitro and in vivo (1, 2) (Figure 1).

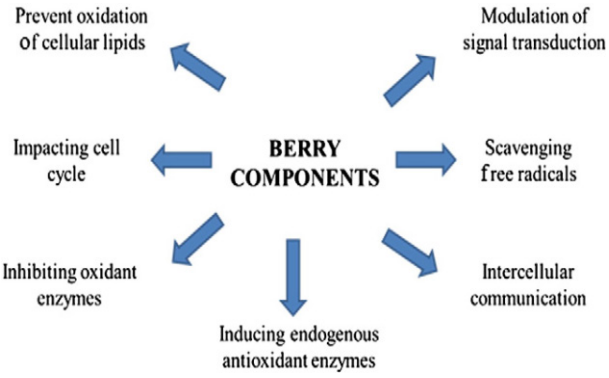

Figure 1. The health benefits of berries (Stapleton et al., 2008)

Berries are at the top of the most important agricultural export items for several years in Serbia, second only to grains. The data from 2012 showed the export of 100.000 tons (mainly of frozen fruit) and given the condition of our agriculture, they are expected to retain that position. The raspberries are the berries primarily cultivated and exported, followed by strawberries and blackberries, while the importance of other species (chokeberry, black currant and bilberry) of this group of fruit is recognized and their production is on the rise (3).

Nowadays, food products have to satisfy numerous quality criteria before commercialization, especially in industrialized countries, where high quality food products have well-defined demanded characteristics. Among these quality criteria, the authenticity and traceability of wild berries and their product are very important. The characterization of berries elements has become the field of interest because their concentration can influence the quality of fruit and eventually the health of consumers. Chemical composition and nutritional value of berries is influenced not only by origin, but by the soil, climate, irrigation, agricultural practice and storage as well (4).

The analysis of variance shows that the differences in metal content between berries are statistically significant ($p < 0.05$) for iron, manganese, potassium, phosphate, magnesium and sodium and not for zinc. Regarding metal content, the variables responsible for the main difference between *Rubus* and *Ribes* genus, are phosphate, potassium and magnesium, while the results for zinc and calcium suggest that the contents of these metals are not linked to genetic origin. The author obtained this data for the fruit cultivated in the same field and under the same conditions, and concluded that the principal factor that determines the content of metals and phenolics in the berries, is genome. These data implied that berries could be differentiated on the basis of their metal, anthocyanins and polyphenols content (4).

Zinc in plants

Numerous studies have confirmed the enormous biological significance of zinc in and for the human body. Although it is a rare element in the Earth's crust (52-80 mg/kg) (5), it is a well known fact that agriculture increases zinc content in surface soils, as well as nonferrous metal industry and fertilizers. Zinc can also be found in water, where it originates from mine and industrial drainage, urban runoff, and from the erosion of soil particles containing Zn (US EPA 1980). The zinc content in plants differs considerably „reflecting the impact of different factors of various ecosystems and of genotypes"(6).

The availability of Zn depends on its physicochemical form. The free ionic form of Zn is totally available to a plant, while as a chelate, precipitate or part of live or dead soil biomass, it is partly or completely unavailable to a plant (7). Also, there are many factors that determine the availability of zinc, such as the soil pH and structure, activity of microorganisms, and water content. Among them, soil pH (which can vary from 4.0 to 9.0) exhibits an impressive control over the free ion concentration. An increase of one pH unit, in 5.5 -7 range, causes a 30±45-fold decrease in aqueous Zn^{2+}-ion concentration (7).

According to Kabata-Pendias (5), the highest concentrations of zinc are found in cereal grains (18-33 mg/kg mean value worldwide) being the lowest in rice and the highest in oats. The problem that occurs with zinc in grains is that it is bound tightly due to high phytate and dietary fibers contents, and its bioavailability is limited.

Being an important factor in human physiology and biochemistry, zinc is also a common component of plant metabolic pathways. The involvement of zinc in plant metabolism is essential since it is an active compound of dehydrogenases, proteinases, peptidases and phosphohyrolases. Zinc also stabilizes cellular components and influences cellular membrane stability (5).

Zinc defficiency (below 20 mg/kg) can occur in plants mostly in calcareous and alkaline soils, under acid and semi-acid conditions. The deficiency affects young leaves first. Zinc-defficient plants are undeveloped and short too due to inadequate growth hormone supply (5). Zn-deficiencies also appear in highly weathered tropical soils that cover about 20 % of the world's land surface in the tropics and subtropics. Almost half of the agricultural soils from India, one-third of the agricultural soils of China, 14 Mha in Turkey and 8 Mha in Western Australia are considered Zn-deficient for plants (7).

However, an even bigger problem then Zn-deficiency are the toxicity and intolerence of zinc in plants which occurs in soils with prolonged use of fertilizers that contains zinc, as well as with inputs from other pollution sources which increase zinc content in soils. Zn-toxicity is often associated with a joint deficiency of Mg, Fe or Mn due to a competitive uptake between the polyvalent cations (8). These states are species- and genotype-dependent and rely on the plant growth stage. Eisler (9) reported that sensitive plants wither when the soil Zn concentration exceeds 100 mg/kg and photosynthesis is stopped when the content is over 178 mg Zn/kg. Some species have shown the ability to accumulate larger amount of zinc (*Thlaspi spp*. can contain more than 10.000 mg/kg) and are used for phytoremediation of soil (10).

Since papers related to berries mainly allude to phytochemicals as pivotal for positive effects on human body, we would like to make reference to the content of zinc in berry fruit, as well as comparing zinc content to recommended daily intake and review zinc roles.

Considering that Recommended Dietary Allowance (RDA) (National Research Council, 2001) for zinc has been set at 11 milligrams per day for men and 8 milligrams per day for women,

Table 1. Zinc content in berries from the U.S. Department of Agriculture Nutrient Database (11)

Berry	mg Zn/100g
Blackberry, raw	0.53
Bluberry, raw	0.16
Cherry, sour, red, raw	0.10
Cranberry, raw	0.10
Currant, black, raw	0.27
Currant, red and white, raw	0.23
Gooseberry,raw	0.12
Raspberry, raw	0.52
Strawberry, raw	0.14

it is obvious that zinc findings are relatively low in berries (the zinc values in fresh berries are given in Table 1.(11)). This problem can be solved either by increasing the concentrations of the nutrient in plants or by increasing its bioavailability in food, or both. The bioavailability of micronutrients can be improved by either increasing the quantity of substances in plant foods that enhance the absorption and utilization of micronutrients or by decreasing the quantity of dietary antinutrients that inhibit micronutrient absorption. However, plant breeding and genetic engineering techniques have the greatest potential to increase Fe and Zn content in plants (12). Since these minerals, as well as the compounds favoring or inhibiting their uptake in humans (phytic acid, phenolic compounds, ascorbic acid), have specific roles in plants, their uptake by the plant from the soil as well as their concentrations in different plant organs is strictly regulated (13). This means that alteration of their concentrations would change the entire physiology of the plant (7).

Decreasing the quantity of dietary factors that inhibit trace element bioavailability may be another approach to increase the nutritive value of crops as sources of micronutrients. Some of the antinutritives are phytic acid, lecitins, and tannins. The action of these inhibitors in foods can be reduced with proper food processing procedures or by plant breeding programs. However, additional research is needed to evaluate the effects of decreased phytin on seed viability and agricultural production before seed and crops with low phytic acid concentration are used on a wide scale (14). Moreover, phytate, tannins and other dietary components have anticancer activity and could perhaps explain why plant-based foods have been related to decreased risk of many chronic diseases (15).

Zinc deficiency

The essentiality of zinc was established in 1963. Before that, it was believed that zinc deficiency in humans had never occurred because the food analysis showed sufficient amounts of zinc in human diets (16). However, in early 1970s Barnes and Moynahan (17) reported that a fatal genetic disorder – *acrodermatitis enteropathica* – was caused by zinc deficiency presenting with the patient's incapability to absorb dietary zinc. Zinc supplementation cured this disorder. After a while, the Food and Nutrition Board of the National Academy of Sciences established the recommended dietary allowances for zinc for humans. Since then, the agricultural and pharmaceutical industries were required by law to label the zinc contents of their products.

Even though it was believed that zinc deficiency was rare in humans, the clinical pictures similar to those reported for zinc-deficient dwarfs from the Middle East were common in many developing countries, where primarily cereal proteins were consumed by the population. Few reports have also shown that zinc deficiency affected both males and females. It has been documented that a nutritional deficiency of zinc affects today more than 2 billion people in the developing world. The clinical manifestations related to zinc deficiency include growth retardation, hypogonadism, impaired cognitive functions and immune disorders (16).

The zinc deficiency affects not only the developed countries, but also the developing world. Newborns, children, pregnant women and old people are the populations at highest risk. This deficiency tends to appear when there is a low zinc intake, an increased loss of zinc from the body, or with increased Zn requirements of the body. The early sign of zinc deficiency is the loss of taste. Zinc deficiency has been linked with anorexia, dermatitis, poor wound healing, hypogonadism with impaired reproductive capacity, impaired immune function, and depressed mental function (18). Zinc deficiency in mothers has been associated with increased incidence of congenital malformation in infants (19).

Zinc homeostasis and bioavailability

The human body contains zinc in all tissues and fluids. The largest pools of zinc are the muscles and bones, liver and skin as well, and just 5% occurs extracellularly (12). A small amount of readily available zinc is stored in the liver, kidney and pancreas (20). Beside its function as an active centre of more than 1000 different enzymes that occur in all six groups of the IUBMB (International Union of Biochemistry and Molecular Biology) enzyme classification (6), Zn maintains the structure and function of membranes, either stabilizing thiol groups and inhibiting oxidative damage by binding to membranes at sites that otherwise might be occupied by metals with a redox potential, or by scavenging free radicals by bonding with metallothionein (12).

The body does not have depots of zinc. About 1% of the total body zinc needs to be restored daily by food intake (21) and longed periods of zinc reduction cannot be compensated. A systemic zinc deficiency can be the result of: 1. decreased dietary intake, 2. decreased absorption, 3. increased elimination, 4. mutations in the ZIP4-transporter encoding gene the (i.e. acrodermatitis enteropathica), important in intestinal uptake, 5. tissue and cellular redistribution or use of certain medications (penicillamine, diuretics, antimetabo-

lites, valproate and iron salts) (22, 23). Serum/ plasma zinc levels, which normally range 70–250 mg/dl, reflect the intake and respond quickly to any alterations of zinc levels. Other biomarkers of the zinc status can be altered too, such as the activity of zinc-dependent enzymes alkaline phosphatase and 24 h urinary zinc excretion (24).

Hyperzincemia and elevated 24 h urinary zinc excretion can be the consequence of high zinc intake, occupational exposure (in welding), high intestinal absorption, or diminished intestinal excretion. This disorder involves dysfunction of the immune system, fever, headaches, cramps, nausea, vomiting, diarrhea, loss of appetite and demyelination (25, 26).

The absorption of zinc has been thoroughly studied. This process includes intestinal mucosal cells uptake from the intestinal lumen, transfer of the nutrient across the mucosal cells, and transport from the intestinal cells to other tissues and organs. A mineral nutrient must be consumed in a convenient form that can be taken up by the mucosal cell directly or in a form that can be converted in the intestine to the absorbable form (12).

The Zn homeostasis is maintained by the regulation of alimentary absorption of Zn and by controlling the secretion of endogenous Zn into the intestinal tract. The absorption of Zn occurs in the small intestine in humans, although the maximum absorption occurred in the duodenum in one study (27) and the jejunum in another (28). The duodenum was the main site of Zn absorption in two studies conducted on rats, concluding that Zn was secreted into the lumen of the small intestine along its entire length. Zinc can enter the intestine with salivary, gastric, pancreatic and biliary secretions. Also, the pancreas can release an exocrine ligand that binds dietary Zn and intensify Zn absorption in the jejunum during subacute Zn deficiency (29).

Phytate has been considered to be an antinutrient due to its ability to bind minerals. It has been suggested that phytate reduced Zn bioavailability due to Zn coprecipitation as a Zn-Ca-phytate complex. An inhibitory effect of phytate on Zn absorption has been studied and it was found that Zn absorption in humans is not diminished by Ca or nonphytate P at concentrations that are generally present in human diets (30-32). However, a high Ca intake by either milk, CaH_2PO_4 or $CaCO_3$ depressed net Zn absorption in humans (33). Food processing (cooking, fermentation, germination, malting and soaking, treatment with phytase), increase Zn bioavailability in foods by reducing the quantity of dietary phytate (34, 35).

Moreover, some dietary components or ingredients have been studied and their effect on Zn bioavailability in humans and experimental animals has been examined. The results were not always consistent. Some promoters of the bioavailability of Fe or Zn, or both Fe and Zn, are various organic acids (ascorbic, citric, malic and fumaric acids) and several amino acids (methionine, cysteine, histidine) (12).

Zinc and depression

Zinc connection with depression has been widely reported. This metal is found in many glutamatergic nerve terminals in its ionic (Zn^{2+}) form (36). It has been reported that several diverse treatments (acute or chronic treatment with zinc, co-administration of zinc and antidepressants at subeffective doses) produces antidepressant-like effect in experimental animals predictive of antidepressant activities (37, 38). The opposite – depressive effect was also confirmed where rats and mice were fed with zinc-deficient diets exhibited depressive-like behavior, which was reversed after the use of antidepressants (39).

When humans are concerned, it has been found that serum zinc is correlated with the severity of depression (40). Low serum zinc levels were found in patients with depression (41). It was found that 25 mg of zinc for 12 weeks, improved the effectiveness of antidepressant treatment in patients who were previously resistant to conventional pharmacotherapy (42). It was also found that depressed patients had serum zinc levels lower than control subjects, and after an antidepressant treatment, these values increased, suggesting that serum zinc may be a marker for depression (43). Women with a lower intake or without zinc supplementation had more symptoms of depression than women with higher intakes or those who took zinc supplements (44).

Swardfager (45) noticed that zinc plays a role in depression by modulating oxidative stress, inflammation, neuroplasticity and neurogenesis. It was observed that chronic fatigue syndrome patients, who usually exhibit depressive symptoms, have lower zinc serum levels, which lead to increased oxidative stress, impairment of the immune system and increased inflammatory signs. These changes may contribute to the symptoms of depression (46).

The role of zinc in Alzheimer's disease

Alzheimer's disease (AD) is the most common form of senile dementia, characterized by a progressive deterioration of cognitive abilities (47). In addition to a significant loss of cholinergic neurons, AD is characterized by the accumulation of protein aggregates, the main constituent of which is amyloid-β protein. Amyloid-β originates from APP protein by the cleavage of one of three proteinase enzymes called α, β, and γ secretases (48, 49).

Manosso has reviewed the current studies that associate zinc with the pathogenesis of AD, focusing on the interactions of zinc with proteins that are commonly and significantly altered in this disease. It was proposed that diverse neurodegenerative processes may perturb the cellular zinc le-

vel, raising it to the level where zinc contributes to the disease progression. This phenomenon may affect the plaque development and modify zinc content in the tissue through immuno-inflammatory and/or regenerative responses (36).

The synaptic vesicles in the brain contain 15% of zinc. Smaller amount of zinc may be loosely bound to proteins and amino acids within the cytoplasm. These are labile, or chelatable forms of zinc, while the rest is tightly bound. The hippocampus contains the highest concentrations of chelatable zinc, where vesicular zinc is localized in neurons using glutamate as a neurotransmitter (50). Even though glutamate is the major neurotransmitter in the hippocampus and cortex, not all neurons that use glutamate contain chelatable zinc. A link of excitatory amino acid with AD has been suggested (51). The hypothesis suggested that in AD, there are initial increases in intracellular chelatable zinc which is then released, precipitating β-amiloid proteins and compromising neuronal viability. The resulting immunological and inflammatory responses are partially responsible for the modifications of tissue levels of chelatable zinc. Abnormally high and low intracellular chelatable zinc could be involved in neuronal death by different mechanisms (52).

Increases of chelatable zinc occur as a secondary response to oxidative stress. Chelatable zinc increases in the neurons undergoing apoptosis (53) and other cells induced to die via apoptosis (54). Moreover, a loss of cholinergic neurons may cause increased hippocampal content of zinc in AD (55). Increases in chelatable zinc may initiate neurodegenerative processes by direct activation of transcription factors, protein kinases and DNA synthesis and by inhibition of enzymes critical for cell survival. Zinc inhibits brain sodium-potassium adenosine triphosphatase (Na-K-ATPase) and mitochondrial energy production at micromolar concentrations. Inhibition of Na-K-ATPase and complexes I and II of the mitochondrial respiratory chain causes neuronal death (52).

Zinc and immunity

Low serum zinc level is a sensitive marker of immune activation and inflammation (56) and elevated production of pro-inflammatory cytokines, as interleukin-6 may be one of the causes of zinc deficiency (57). Zinc plays a pivotal role in few signal transduction and gene expression pathways, including that of cytokine genes (58). Prasad showed that the activity of serum thymulin, a thymus-specific hormone involved in T-cell functions, was decreased in mildly zinc-deficient subjects (59).

The positive role of zinc with ascorbic acid, L-carnitine and methylmethionine sulfonium chloride (vitamin U) has been discovered. It was reported that these complexes exhibited an insulinomimetic activity in diet-induced metabolic syndrome in rats. The World Health Organisation (WHO) has issued a warning about pandemic outbreak of this syndrome, the dominant underlying risk factors of which are abdominal obesity and insulin resistance. It is a well known fact that adipose tissue expresses and secretes a panel of adipocytokines (plasminogen activator inhibitor-1,2, TNF-α), which contribute to the development of vascular diseases, hyperlipidemia, hyperglycemia and insulin resistance. Thus the reduction of abdominal adipose tissue is the first line of treatment or prevention for metabolic syndrome (60). Insulinomimetic activity of Zn(II) ion complexed with maltol, amino acids, picolic acid and their derivatives has been also confirmed by Joshikawa and others (61). These results indicate that metabolic syndrome could be prevented through the reduction of visceral adipose tissue content and/or improvement of blood fluidity brought about by a Zn(II) complex with vitamin U and vitamin C (60).

Ophthalmic role of zinc

Zinc is present in all retinal cells, being particularly concentrated in photoreceptors. Zinc, as an essential part of zinc-finger DNA binding proteins, influences the expression of numerous zinc-binding proteins (62) and is engaged in the transcriptional regulation of binding elements (63). The ability of zinc to inhibit oxidation is concentration dependent, since zinc in high concentrations is toxic to the retina (64). Significantly, retinal zinc concentrations are affected in age-related macular degeneration (AMD) (65).

Zinc has a key role in multiple functions of general cell metabolism (mitochondrial function, gene expression, antioxidant defense, DNA repair mechanisms, cell proliferation, cell differentiation, apoptosis), retinal development and specific retinal functions (phototransduction, rhodopsin recovery, neurotransmission). Zinc also participates in the conversion of light stimulation into an electrical signal (phototransduction); intracellular signals within the photoreceptors (rhodopsin deactivation and/or regeneration); and/or communication between photoreceptors and other retinal neurons and glial (Muller) cells. Reisomerization of 11-cis-retinal from all-trans retinol is mediated by a metalloenzyme, retinol dehydrogenase, which requires catalytic Zn^{2+} at the active site of the enzyme (62).

Retinal zinc bioavailability responds quickly to any varitions in serum levels. A specific retinal regulatory mechanism of zinc homeostasis reacts in the early stages of a deficiency. These mechanisms include oxidative stress and photoreceptor disruption. Zinc deficiency results in poor dark adaptation (66).

No retinal abnormalities have been described in animal studies or in patients with zinc overload. This means that in vivo homeostatic mechanisms may prevent zinc from accumulating in the healthy retina (62).

Aging has been associated with zinc depletion. Wills (65) found a reduction in total zinc in the neuroretina of men, although not in women.

This author suggested that processes such as inflammation could trigger the AMD abnormality and result in secondary alterations of zinc homeostasis.

It was also found that zinc protects the retina from oxidative stress-induced pericyte apoptosis, neovascularisation and capillary leakage, and it can be considered beneficial in the prevention of diabetic retinopathy (67).

Therapeutic role of zinc

Zinc has been analyzed and approved by the Food and Drug Administration for the treatment and long-term management of Wilson's disease. Zinc prevents accumulation of copper in genetically susceptible individuals and may decrease copper load in patients with Wilson's disease (68). Zinc was effective in decreasing the incidence of infections in therapeutic dosages (75 mg of elemental zinc daily in three separate doses). Zinc-acetate lozenges as a therapeutic agent reduced by 50% the duration and severity of the common cold (69).

Scientists around the world have been dealing with a couple of major challenges for effective zinc supplementation. The first challenge is the requirement of higher frequency of zinc doses. Weekly doses may not be as effective as smaller doses given daily. Most of the zinc in the human body is situated in the muscle and bone tissues, and is not normally released during zinc deprivation. Second, there are uncertainties about the best type of zinc salts to be used, related to their bioavailability and side effects. Further, zinc absorption is inhibited by fiber and phytase present in food. To date, most zinc supplementation studies in humans have used either sulphate, acetate, gluconate, aminoate, histidine, and methionine. The zinc monomethionine supplement has been proven in independent studies to be absorbed significantly better than other forms of zinc, and to resist dietary fiber and phytate (70).

Conclusion

Berries hold an important position among the fruits that can be attributed to their high antioxidant phytochemical contents. Berries are not so rich in zinc, but because of their beneficial chemical composition they are recommended to be widely consumed.

Zinc has got numerous physiological roles in both human and plants organisms, from boosting of the immune system to the spread of antibiotic-resistant bacteria. On the other hand, when its homeostasis is disrupted, zinc shows an opposite effect and functions as an enemy.

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SADRŽAJ CINKA U JAGODASTOM VOĆU – ZNAČAJ ZA ZDRAVLJE

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Jagodasto voće je postalo neizostavan deo ishrane savremenog društva. Iako ova vrsta voća nije najbolji izvor cinka, postoje načini kojim se povećava količina cinka u zemljištu. Cink u velikoj meri učestvuje u fiziološkim i biohemijskim procesima, ali je i osnovna komponenta metaboličkih puteva biljaka. Deficit cinka u biljkama se javlja uglavnom u krečnjačkim i alkalnim zemljištima i prvenstveno pogađa mlade listove. Veći problem od deficita su toksičnost i intolerancija cinka u biljkama koja se javlja usled produžene upotrebe đubriva, koje sadrži cink. Toksičnost cinka je često udružena sa deficitom magnezijuma, gvožđa ili mangana. Kod ljudi, deficit cinka dovodi do acrodermatitis enteropathicas, zastoja u rastu, hipogonadizma, depresije, umanjene kognitivne funkcije i imunih oboljenja, koje pogađaju osobe oba pola u zemljama u razvoju. Serumski cink korelira sa ozbiljnošću depresije, a niski serumski cink je određen kod pacijanata sa depresijom, pa se pretpostavlja da može biti marker za depresiju. Različiti neurodegenerativni procesi, kao što je Alchajmerova bolest, mogu promeniti nivo cinka u ćeliji, gde doprinosi pogoršanju bolesti. Cink igra značajne uloge u različitim prenosima signala i ekspresije gena, uključujući i gene citokina. Cink ima i ključnu poziciju u metabolizmu ćelije, razvoju retine i njenim specifičnim funkcijama. Cink je u terapeutskim dozama efikasan u smanjenju incidencije infekcija. Lozenge sa cinkom su smanjile trajanje i ozbiljnost prehlade. *Acta Medica Medianae 2016;55(4):73-81.*

Ključne reči: *cink, jagodasto voće, ishrana, biljke, bioraspoloživost, deficit*