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Iron and Folate Supplementation versus Lead Altitudes in Pregnant Anemic Women

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Editorial

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Editorial

Human exposure to lead occurs through various sources like lead based paints, battery recycling, lead containing pipes or lead-based solder in water supply systems, industrial processes such as lead smelting and coal combustion, grids and bearings, etc.. Lead is concerned to a broad range of physiological, biochemical and behavioral malfunctions. There are a number of reports and literature, which reflect initiates oxidative damage to heart, kidney, reproductive organs, brain and erythrocytes [1]. But, there are very few reports revealing association between iron intake and environmental lead exposure during pregnancy. Therefore, the present 'Editorial' is focused at current research pertaining to the effect of iron and folate supplementation on biochemical modulation of definite lead toxicity markers linked with pregnancy.

Lead has been well documented to cause anemia through different pathways by decreasing the red blood cell survival and/or by inhibiting haem synthesis. Data obtained from a study reveal the levels of Hb, Fe, Ferritin to decrease in pregnant anemic women, recovering significantly in all supplemented groups [2]. Furthermore, TfR levels increased significantly in all anemic groups and reversed after treatment. Authors earlier reported that daily oral iron supplementation improved body iron in iron deficient women [3]. The consistent decreases in sTfR in the supplemented groups were probably due to an increase in iron supply, or to a decrease in iron requirement while the iron stores are being replenished. It is more likely that Pb is inadvertently uptaken through pathways intended for Fe [4]. Studies recommend that the divalent metal transporter 1 (DMT1) is a transporter for both Fe and Pb in the small intestine and body Pb levels have been regulated in harmony with Fe status. DMT1 has been reported to elevated in iron deficiency and lesser in iron overload [5]. The biological mechanisms come into sight in certain studies suggesting the protective effects of recommended oral Fe supplementation against Pb toxicity [2,3,6]. A considerable reduction of delta aminolevulinic acid dehydratase (D-ALAD) following a significant increase of zinc protoporphyrin (ZPP) and aminolevulinic acid in anemic women has been well documented, but reversed after treatment [2]. However, in the abundance of hemoglobin (Hb), even in serious case of lead intoxication, increased ZPP is relatively harmless probably constituting less than 1% of the total Hb production [7].

The activity of antioxidant enzymes, namely, CAT, SOD and GSH decreased significantly in pre-treated anemic women, while their activities recovered significantly after oral iron supplementation. The decreased activities of SOD may be due to targeting the sulfhydryl groups, concomitant with replacement of zinc ions by lead serving as important co-factors for these antioxidant enzymes inactivating them [8]. Besides, lead inactivates glutathione (meant for protection of cells against free radicals) by binding to sulfhydryl groups. Consequently, synthesis of GSH from cysteine via the γ-glutamyl cycle occurs, which is usually not effective in replenishing the supply of GSH [9].

A recent relevant study has successfully noticed significantly augmented level of LPO and PC, in iron deficient anemic women and even after treated subjects [2]. There are reports on significant acceleration of RBC's lipid peroxidation in IDA, reflecting the lipids in RBCs likely to be susceptible to peroxidation in the pathophysiology of IDA. Lead causes hemoglobin oxidation, directly causing RBC hemolysis most

probably due to inhibition of ALAD, resulting in an increased concentration of substrate ALA in both blood and urine. These elevated ALA levels generate hydrogen peroxide and superoxide radical concomitant with interaction of oxyhemoglobin, resulting in the generation of hydroxyl radicals [10]. Progression of all the above mentioned mechanisms enables cell's extreme vulnerability to oxidative stress [11], thus leading to cell death [12].

It is well known that lipid peroxidation is a free-radical-mediated phenomenon and that the lipids in RBCs are susceptible to peroxidation in the pathophysiology of iron deficiency anemia. There are reports that the iron doses used for correcting iron deficiency anemia may further elevate the lipid peroxidation products, mainly due to increased bioavailability of elemental free iron in gastrointestinal mucosal cells of the subjects [13]. Moreover, reports increased peroxidative damage of RBC membrane proteins, as measured by the protein carbonyl content, in the iron supplemented groups. This may be due to Fe mediated generation of ROS that probably enhances peroxidative damage of both the proteins and lipids [12,14].

The levels of vitamin C and E have been documented to decrease considerably in iron deficient anemic women and further decreasing after treatment. This may be consequent of its property of quenching ROS along with metal chelation enabling it a prospective detoxifying agent for lead. The similar may be factual for vitamin E as its serum levels were also noticed to decline in pre-treated as well as post-treated women. Vitamin E is a vital lipid-soluble and chain-breaking antioxidant, dynamically involved in the inhibition of propagation of free radicals generation during oral iron treatment in anemic patients especially under the course of pregnancy [15].

Conclusion and Future Perspectives

Conclusively, iron and folic acid supplementation considerably revert the lead levels in supplemented groups. Perturbation of prooxidants and antioxidants in pregnant anemic women directs definite oxidative stress, probably due to Pb intoxication. Besides, Iron deficiency anemia is connected with elevated blood lead levels, increasing lead absorption followed by an additional independent negative impact on fetal development. Thus, it is vital that bioavailability of lead via whichever means should be controlled and precise steps can be considered to decrease the prevalence of anemia during pregnancy. Regulatory and health agencies should also consider this as a priority and make more generous efforts en route for resolving this public health concern.

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