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Review Article | International Journal of Hematology and Blood Disorders | Open Access

# Antioxidants In The Management of Sickle Cell Anaemia

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**Received: August 23, 2018; Accepted: September 17, 2018; Published: September 18, 2018**

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## Abstract

The burden of anaemia associated with sicke cell disease is major haemoglobinopathies causing a lot of challenge globally. It is still highly prevalent in Nigeria not minding all the efforts to reduce the menace. It presents with so many challenges and complications to the patients. A lot of efforts are going on to enhance the lives of the patients. Sickle cell anaemia patients have hype metabolic rates with elevated production of reactive oxygen free radicals (OFRs) which are destructive to cells especially if there is low total antioxidant status as seen among sickle cell anaemia patients. It is important to administer enough foods and fruits that are rich in antioxidants to improve the well-being of the patients. This paper was written to enlighten the world and those involved with the care of sickle cell patients on the need to manage them with antioxidants and the resultant positive effect that will ensue on the patients.

**Keywords:** Antioxidants; Management; Sickle Cell Anaemia; Crisis

## Introduction

Sickle cell anaemia is a genetic condition which is marked by sickling of red blood cell (RBCs) under deoxygenation [1]. It is highly prevalent among African descents especially in areas malaria infection is endemic because of selective advantage of sickle cell trait [2]. Sickle cell patients are still seen in Nigeria upon all the efforts for genetic counseling before marriage made by the health workers. Sickle cell anaemia is a point mutation in β- globin chain. There is indication that there is increased oxidation in sickle cell subjects in relation to healthy individuals with increased release of reactive radicals which are highly damaging to the cells in the system of the patient leading to crisis in them with resultant shortened life span if not handled properly. There is reduction in total antioxidants status of the subjects with exacerbation of pains in them [3, 4].

A certain level of oxidation happens in majority of human diseases and is suspected to make a significant contribution to the process leading to the disease formation [1]. Little of oxygen free radicals (OFRs) are usually produced in aerobic organisms. Reduced OFRs are shown to be important in most of cellular processes, but retenton of OFRs may destroy biological macromolecules, resulting to cytotoxicity, hypersentivity, mutagenicity and /or carcinogenicity [5].

## Role of Antioxidants

The antioxidant defense against free radicals attack may be responsible for the susceptibility to disease pathogenesis as some studies have shown reduced antioxidant levels in some patients [6, 7].

Free radicals are not completely decreased chemical species that have a single unpaired electron in an outer orbit. Energy created by these unstable elements is given through reactions with adjacent molecules, with the capacity to destroy lipids, proteins and nucleic acids [8]. Autocatalytic reactions stimulated by free radicals change susceptible molecule to free radicals to produce the chain destruction [9, 10]. Free radical attack has been associated to numerous disease situations [1].

Some antioxidants like Vitamin E, Vitamin A and ascorbic acid and reduced glutathione in the cytosol [6] inhibit the initiation of free radical destruction. Also, some enzymatic systems lead to the inactivation of free radical reactions. These break down hydrogen peroxide and superoxide anion. The enzymes are often positioned near the sites of production of the oxidants. Catalase present in peroxisomes decompose hydrogen peroxide, $H_2O_2$ ($2H_2O_2 \rightarrow O_2 + 2H_2O$). Superoxide dismutase's seen in many cell types change superoxide's to inactivate molecules, $H_2O_2$ ($2O_2^- + 2H \rightarrow H_2O_2 + O_2$) [11]. This group is shown to involve both the manganese-superoxide dismutase which is seen in the cytosol. Glutathione also gives defense against cellular damage by detoxifying oxygen free radicals.

Where there is insufficient antioxidant function, cells undergo serial biochemical and morphologic alterations as they are continuously injured and ultimately causing apoptosis. In a lot of disease situations, the outcome of promoted free radical synthesis is related to the net balance between free radical formation and termination.

Also to serving as an oxygen radical scavenger γ- tocopherol scavenges reactive nitric oxide species and hinders prostaglandin E2 have been shown to mediate inflammation [12]. Other amine antioxidants function as lipid peroxide and scavenge ROS [9, 10]. Individuals confirmed with Sickle cell disease have a greater requirement for the antioxidant (Glutathione) to facilitate metabolic processes, than healthy individuals.

The total antioxidant status (TAS) is reported reduced in sickle cell anaemia due to increased release of oxygen free radicals (OFRs) generated in the body by the metabolic processes. Sickle cell patients are faced with many complications that could lead to increase release of OFRs to overwhelm the antioxidant status. Adequate administration of antioxidants will be of great help to improve the well-being of the patients. The patients should eat food and fruits rich in antioxidants to build their immunity and naturally fight many opportunistic infections that enhance release of OFRs that could lead to apoptosis of cells with attendant shortened life.

## Conclusion

It has been shown that antioxidants administration especially in food and fruits will be highly beneficial to the care of sickle cell disease subjects. This will help to reduce crisis, inflammation and shortened life of the patients. The patients are encouraged to take more fluids and nutritive foods to enhance their well-being and productivity in life. There is hope for those living with sickle cell anaemia. Research in antioxidants is becoming more dynamic and promising and will be utilized to management diseases. More researches should be done in antioxidant utilization in sickle cell anaemia to enhance the life of those affected. This will reduce economic and human loss as a result of this menace that is genetically formed by point mutation in β- globin chain.

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