International Journal of Advanced Research in Biological Sciences
ISSN: 2348-8069
www.ijarbs.com
DOI: 10.22192/ijarbs Coden: IJARQG(USA) Volume 5, Issue 10 - 2018

Research Article

DOI: http://dx.doi.org/10.22192/ijarbs.2018.05.10.016

An update on micro RNA in sickle cell disease

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Abstract

It is shown that sickle cell disease or sickle cell anaemia is a life -long blood anomaly expressed by erythrocytes that assume an abnormal, rigid, sickle shape. Sickle cell anaemia is caused by a point mutation in the β - globin chain of haemoglobin, causing the amino acid glutamic acid to be replaced with the hydrophobic amino acid valine at the sixth position. MicroRNA-144 modulates oxidative stress tolerance and is associated with anaemia severity in sickle cells disease, miRNA-144 directly regulate nuclear factor erythroid -2- related factor 2, a central regulator of cellular response oxidative stress and modulates the oxidative response in K562 cell line and primary progenitor cells. The paper was written to update the world on the microRNA and sickle cell disease.

Keywords: Update, micro RNA, Sickle Cell Disease

Introduction

It is shown that sickle cell disease or sickle cell anaemia is a life -long blood anomaly expressed by erythrocytes that assume an abnormal, rigid, sickle shape. The sickling occurs because of a mutation in the haemoglobin gene (Obeagu *et al.*,2015).
Sickle cell anaemia is caused by a point mutation in the β - globin chain of haemoglobin, causing the amino acid glutamic acid to be replaced with the hydrophobic amino acid valine at the sixth position. The β - globin gene is found on the short arm of chromosome 11. Under low - oxygen conditions, the absence of a polar amino acid at position of six of the β - globin chain promotes the non - covalent polymerization of haemoglobin, which distorts red blood cells into a sickle shape and decreases their elasticity. The loss of red blood cell elasticity is central of the pathophysiology of sickle cell disease (Obeagu *et al.*, 2015).

Micro RNA and sickle cell disease

Given the potential limitations of sensitivity and size bias for the traditional means of isolating and characterizing erythrocyte RNA, it remained possible that erythrocytes contain RNA species not previously identified. There are several lines of evidence that human mature erythrocytes, although lacking in ribosomal and large-sized RNAs, contain diverse and abundant microRNAs.

Several microRNA (miR 320, let 7s, miR 181, miR 141,) were over represented in the HbAA erythrocytes. While microRNA (miR 299, miR 144, miR 140, miR 451) were over represented in HbSS erythrocytes (Swem *et al.* 2018). HbSS erythrocytes have a high level of miR 451 which was found to be translocated unto plasmodium falciparum, this observation led to the possibility that micro RNA composition may contribute to the malaria resistance notes for sickle

erythrocytes. MiR-320 played an important role for the down-regulation of its target gene, CD71 during reticulocyte terminal differentiation. Further investigation revealed that poor expression of miR-320 in HbSS cells was associated with their defective down regulation of CD71 during terminal differentiation. Microrna-144 is a family of microRNA precursors found in mammals, including humans. The 22 nucleonic mature Micro-RNA sequent is excised from precursor hair pin by the enzyme Dicer (Yu *et al.*, 2002). In human, micro-144 has been characterised as a common microRNA signature (Wang *et al.*, 2014) of a user of different tumours. GATA4 is thought to activate transcription of the micro-144 precursor (Zhang *et al.*, 2010). Microrna-144 function in a cluster with microRNA-451, this locus regulated the expression of a number of genes whose products are involved in erythropoiesis (Rasmussen *et al*., 2010). One of the identified targets of microRNA-144 is insulin receptor.

MicroRNA-144 modulates oxidative stress tolerance and is associated with anaemia severity in sickle cells disease, miRNA-144 directly regulate nuclear factor erythroid -2- related factor 2, a central regulator of cellular response oxidative stress and modulates the oxidative response in K562 cell line and primary progenitor cells. Also increase in miRNA-144 is associated with reduced NRF_2 levels in HbSS reticulocytes and decrease glutathione regeneration and attenuated antioxidant capacity in HbSS erythrocytes, thereby providing a possible mechanism for the reduced oxidative stress tolerance and increased anaemia severity seen in HbSS erythrocytes. this finding suggest that erythroid microRNAs can serve as genetic modifiers of HbS-related anaemia and can provide novel insights into the clinical heterogeneity and pathobiology of sickle cell disease (Sangokoya *et al.*, 2010).

Conclusion

Also increase in miRNA-144 is associated with reduced NRF_2 levels in HbSS reticulocytes and decrease glutathione regeneration and attenuated antioxidant capacity in HbSS erythrocytes, thereby providing a possible mechanism for the reduced oxidative stress tolerance and increased anaemia severity seen in HbSS erythrocytes.

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Quick Response Code	Subject: Medical Sciences
DOI:10.22192/ijarbs.2018.05.10.016	

How to cite this article:
Emmanuel Ifeanyi Obeagu. (2018). An update on micro RNA in sickle cell disease. Int. J. Adv. Res. Biol. Sci. 5(10): 157-158.
DOI: http://dx.doi.org/10.22192/ijarbs.2018.05.10.016