Global Journal of Reproductive Medicine
ISSN: 2575-8594

Juniper Publishers
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Clinical Image
Volume 5 Issue 5 - August 2018
DOI: 10.19080/GJORM.2018.05.555675

Glob J Reprod Med

Polyol Pathway Shed Light on Non-Obstructive Azoospermia Testicular Sperm Extraction (TESE) Negative ROS Imbalance

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Submission: July 05, 2018; **Published:** August 24, 2018

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Abstract

In our recent study regarding non-obstructive azoospermia by metabolomics technology, we clearly demonstrated that testicular sperm extraction negative patients are in extreme oxidative imbalance. With a reanalysis of our data and the integration of proteomics data, we found the Polyol pathway is up-regulated in non-obstructive azoospermia testicular sperm extraction negative patients. In this regard, we suggest the contribution of this biochemical pathway in the extreme oxidative imbalance of non-obstructive azoospermia testicular sperm extraction negative patients.

Keywords: Azoospermia; Metabolomics technology; Testicular sperm extraction; Sperm production; Non-obstructive azoospermia; Fingerprinting; Non-invasive method; Proteomics; Polyol pathway, Diabetic patient's; Plasma proteome database; Male reproductive system; Extreme oxidative imbalance; Human protein; Glutathione deficiency; Glutathione redox state; Proteomics analysis

Abbreviations: NOA: Non-Obstructive Azoospermia; TESE: Testicular Sperm Extraction; UPP: Up-Regulated Polyol Pathway

Introduction

Non-obstructive azoospermia (NOA) refers to absence of sperm in the semen because of the abnormal sperm production. It is estimated that 6-10% of infertile men are NOA [1]. In our recent study, we used the untargeted metabolomics and metabolic fingerprinting approach for of seminal plasma in order to develop a non-invasive method for detection of spermatogenesis [2,3]. Following a reanalysis of untargeted metabolomics data, we found that sorbose was up-regulated in NOA the testicular sperm extraction (TESE) negative patients (NOA TESE(-)). This data was in line with the proteomics analysis of seminal plasma from NOA patients conducted by Batruch et al. [4]. With the Integrating of the proteomics and metabolomics data, we found that polyol pathway is up-regulated in NOA TESE(-) patients. It is an old fact that glucose and fructose concentrations are increased in azoospermia patients [5].

To the best of our knowledge, no studies have been done regarding the measurement of redox potential in azoospermia or in NOA. However, it is known from diabetic patient's data redox potential (NAD/NADH and NADPH/NADP) in up-regulated polyol pathway (UPP) is in imbalance [6]. It is known that NADPH acts to promote glutathione redox state while its deficiency causes glutathione deficiency [7]. Bhardwaj et al. [8] have shown that azoospermia patients, in fact have glutathione deficiency. Additionally, we have already shown that NOA TESE (-) are in extreme oxidative imbalance by metabolic fingerprinting. It is postulated that NADH oxidase (NOX1) is responsible for the increased level of ROS in UPP [9]. With a search in seminal plasma proteome database, we found NOX1 activator protein (NOXA1) [10]. Furthermore, with a search in the Human Protein Atlas database we also found that NOX1 is expressed in male reproductive system [11]. By integrating these data, we suggest a hypothesis how UPP leads to extreme oxidative imbalance in NOA TESE(-) patients (Figure 1).

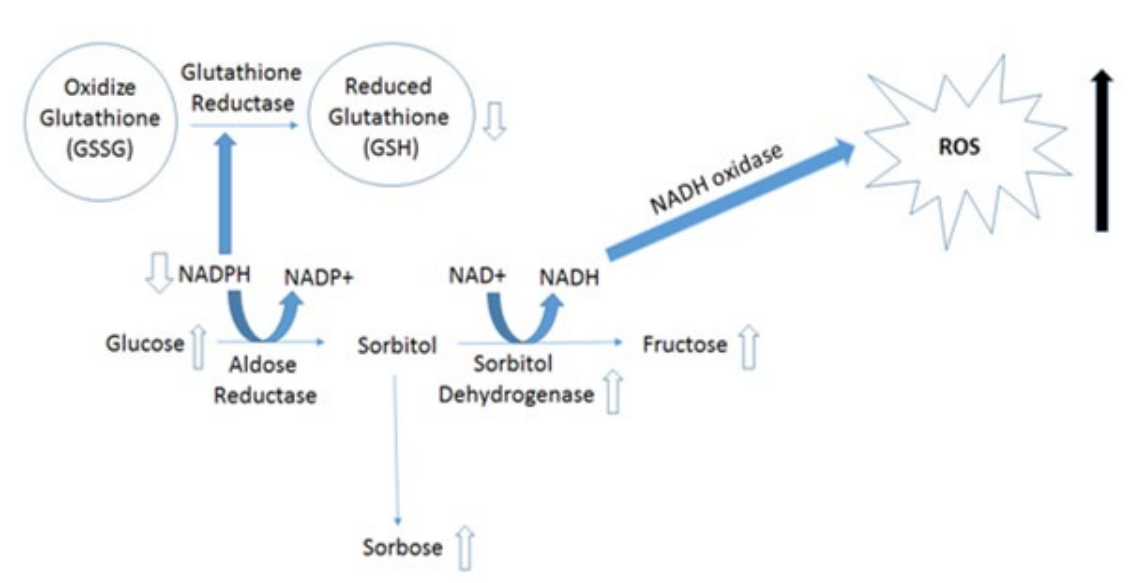

Figure 1: How up-regulated Polyol pathway (UPP) leads to extreme oxidative imbalance in Non-obstructive azoospermia, the testicular sperm extraction negative patients (NOA TESE(-)). NADP+: Nicotinamide adenine dinucleotide phosphate; NADPH: Reduce form of NADP+; NAD+: Nicotinamide adenine dinucleotide; NADH: Reduce form of NAD+; ROS: Reactive oxygen species; :down-regulated; : up-regulated.

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DOI: 10.19080/GJORM.2018.05.555675

How to cite this article: Kambiz G. Polyol Pathway Shed Light on Non-Obstructive Azoospermia Testicular Sperm Extraction (TESE) Negative ROS Imbalance. Glob J Reprod Med. 2018; 5(5): 555675. DOI: 10.19080/GJORM.2018.05.555675.