شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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Zagazig University
Benha Faculty of Medicine
Dermatology and Andrology Department.

CATALASE AND GLUTATHIONE PEROXIDASE IN PATIENTS WITH VITILIGO

٤١٧٤ ص

Thesis
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Abbreviations

AOC	Antioxidant capacity.
bFGF	Basic fibroblast growth factor
6 BH_4	(6R) – L- erythro 5,6,4,8 tetrahydrobiopetrin.
CAT	Catalase enzyme.
DHI	5 – 6 Dihydroxy indole.
DHICA	Dihydroxy indole carboxylic acid
DOPA	Dihydroxy phenyl alanine
GPX	Glutathione peroxidase.
GR	Glutathione reductase.
GSH	Reduced glutathione.
GSSG	Oxidized glutathione.
ICAM-1	Intercellular adhesion molecule-1
MCH	Melanin concentrating hormone.
MCHR 1	Melanine concentrating hormone receptor 1
5 MOP	5 Methoxypsoralen.
8 MOP	8- Methoxypsoralen.
Mn SOD	Manganese superoxide dismutase
MSH	Melanocyte stimulating hormone.
PPARS	Peroxisomal Proliferator activator receptors.
ROS	Reactive oxygen species.
SOD	Superoxide dismutase.
IL	Interleukin.
TNF α	Tumour necrosis factor alpha
TRP- 1	Tyrosinase related protein 1
TRP- 2	Tyrosinase related protein 2
UVA	Ultra violet A
UVB	Ultra violet B

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Introduction

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Vitiligo is a common pigmentary disorder that affects individuals of all ethnic origins equally, with a prevalence close to 0.5- 5% of the world population **(Boisseau- Garasaud et al., 2000).**

The aetiology of vitiligo is still unknown. Beside the most popular autoimmune theory, several groups had shown the involvement of oxidative stress in the pathophysiology of the disease **(Maresca et al., 1997).**

The accumulation of hydrogen peroxide and low catalase levels had been demonstrated in the epidermis of vitiligo patients **(Schallreuter et al., 1999).**

The observations of **Tobin et al., (2000)** had shown that melanocytes are still present in the epidermis of patients with long duration vitiligo. Melanocyte cultures were successfully established from depigmented epidermal suction blister tissue of all 12 randomly selected patients and these cells produced melanin. Furthermore the vacuolation of melanocytes of patients with active disease was reversible upon exogenous addition of bovine catalase to the culture medium.

In addition, the presence of clustered and single premelanosomes in basal and suprabasal keratinocytes of lesional and normal epidermis, as well as the retention of single

melanocyte in lesional epidermis, was demonstrated by light and electron microscopy. Upon topical application of a narrow band UVB- activated pseudocatalase, vacuolation, granulation and dilatation of endoplasmic reticulum completely recovered but the ectopic premelanosome shedding remained. Taken together, these observations indicated that melanocytes are never completely absent in the depigmented epidermis and that these melanocytes can recover their functionality in vivo and in vitro upon removal of hydrogen peroxide **(Tobin et al., 2000).**

Few disturbances of antioxidants had been described in the blood of vitiligo patients, apart from an elevation of selenium, an important factor for glutathione peroxidase activity **(Beazly et al., 1999).**

Yildirim et al., (2003) studied, the role of oxidative stress in the pathogenesis of generalized vitiligo. Superoxide dismutase, glutathione peroxidase and glutathione levels in erythrocytes and serum malondialdehyde and nitric oxide were investigated in 24 patients with generalized vitiligo and 20 healthy controls. Results indicated that significantly increased levels of erythrocyte superoxide dismutase, serum malondialdehyde, and nitric oxide were associated with a marked reduction in erythrocyte glutathione peroxidase and glutathione activites in patients with generalized vitiligo. These observations suggested that the presence of an imbalance of