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The expression of microRNA-451 in human endometriotic ...

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4325675>

Human subjects and tissue acquisition. As no difference in **MIF mRNA**, protein or **miR-451 expression** was noted among stages of the menstrual cycle, stages of **endometriosis** or influenced by medications, data were collapsed and analyzed as **ectopic** versus **eutopic** tissue for **MIF mRNA** and protein as well as **miR-451 expression**.

Cited by: 21

Author: Amanda Graham, Tommaso Falcone, Wa...

Publish Year: 2015

Pathogenesis and Pathophysiology of Endometriosis

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3836682>

Endometrial cell survival. The eutopic endometrium from women with **endometriosis** shares certain alterations with **ectopic lesions** that are not observed in the endometrium from healthy women. Up-regulation of the **anti-apoptotic gene** BCL-2 has been shown in both eutopic and ectopic endometrium from affected women (31).

Cited by: 627

Author: Richard O. Burney, Linda C. Giudice

Publish Year: 2012

Endometriosis: Exploring the Roles of MicroRNA-451 and MIF

<https://consultqd.clevelandclinic.org/endometriosis-roles-micorna-451-macrophage...> ▾

Nov 23, 2015 · The **expression** of microRNA-451 in human endometriotic lesions is inversely related to that of macrophage migration inhibitory factor (MIF) and regulates MIF **expression** and **modulation of epithelial cell survival**. Hum Reprod 2015;30(3):642-652.

Author: Amanda, Amanda Todorovich

Altered expression of microRNA-451 in eutopic ...

<https://academic.oup.com/humrep/article/30/12/2881/2380152> ▾

Sep 14, 2015 · **Expression** of each of the three targets was up-regulated in **eutopic** and **ectopic endometrium of baboons with endometriosis compared** with controls (Fig. 1B, C and D).

Cited by: 13

Author: N.R. Joshi, R.W. Su, G.V.R. Chandramou...

Publish Year: 2015

Author: Joshi, N.R.

Augmented cell survival in eutopic endometrium from ...

<https://rbej.biomedcentral.com/articles/10.1186/1477-7827-3-45> ▾

Sep 08, 2005 · The aim of this study was to evaluate if the balance between cell proliferation and apoptosis is changed in eutopic endometrium from women with endometriosis throughout the menstrual cycle by studying **bax** (pro-apoptotic), **c-myc** (regulator of cell cycle) and **TGF-beta1** (involved in cell

INTRODUCTION

Endometriosis (EMs) is a chronic and recurrent, but benign, disease in women of reproductive age, with a morbidity of approximately 10%. It is characterized ¹by the presence of functional endometrial glands and stroma outside the uterine cavity^[1,2]. Typical symptoms of EMs include cyclic pelvic pain, dysmenorrhea, dyspareunia, and infertility. Previous studies have reported that EMs patients have a high risk of developing gynecological tumors and autoimmune disorders^[3,4]. Thus, EMs can cause severe psychological and physiological harm to those affected by it and imposes a substantial social burden^[5,6].

Despite its high prevalence and incapacitating symptoms, the etiology of EMs is not clear. Evidence suggests that it is a multifactorial disease. Retrograde menstruation, immune system disorders, and genetic and environmental factors have been proposed as susceptibility factors for EMs^[7-9]. The susceptibility factor of retrograde menstruation proposed by Sampson is the most widely accepted^[10]. However, ⁴almost all women of reproductive age exhibit some degree of retrograde menstruation, and only 10% to 15% suffer from EMs^[11,12]. Recently, more evidence has emerged to support the theory that genetic changes in the eutopic endometrium may be the key molecular events in the pathogenesis of EMs^[13].

MicroRNAs (miRNAs) are short noncoding RNA molecules that regulate genetic expression post-transcriptionally and are implicated in several ³biological

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miRNA-451 expression in eutopic endometrium contributes to the pathogenesis c



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Altered expression of microRNA-451 in eutopic ...

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4643526>

MiR-451 expression was decreased in eutopic and ectopic endometrium of both baboon and women with endometriosis Our data suggest that **miR-451** is significantly ($P < 0.001$) down-regulated after the induction of **endometriosis** in EUE collected at 3 and 15 months and in endometriotic lesions collected at 15 months (Fig. 1 A).

Cited by: 14

Author: N.R. Joshi, R.W. Su, G.V.R. Chandramo...

Publish Year: 2015

The expression of microRNA-451 in human endometriotic ...

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4325675>

Although expressed by endometriotic lesion tissue, the **expression** and function of **miR-451** as related to **endometriosis** remains controversial. **miR-451** was initially reported (Pan et al., 2007) as one of the most differentially expressed **miRNAs** between normal **eutopic endometrium** and both **eutopic** and **ectopic endometrial** tissue from women with ...

Cited by: 23

Author: Amanda Graham, Tommaso Falcone, Wa...

Publish Year: 2015

Altered expression of microRNA-451 in eutopic ...

<https://reference.medscape.com/medline/abstract/26370665> ▾

The altered **expression** of **miR-451** was validated in the **eutopic** and **ectopic endometrium** of additional baboons between 3 and 15 months following disease induction. Timed **endometrial** biopsies from women with and without **endometriosis** were also used to validate the **expression** of **miR-451**.

Altered expression of microRNA-451 in eutopic ...

https://www.researchgate.net/publication/282036380_Altered_expression_of_microRNA-451...

Sep 22, 2015 · The altered expression of **miR-451** was validated in the **eutopic** and **ectopic endometrium** of additional **baboons** between 3 and 15 months following disease induction.

Endometriosis: Exploring the Roles of MicroRNA-451 and MIF

<https://consultqd.clevelandclinic.org/endometriosis-roles-microrna-451-macrophage...> ▾

Nov 23, 2015 Thus, we propose that in the **pathogenesis** of **endometriosis**, elevated **miR-451** may function to regulate **MIF** expression, which in turn contributes to the pathogenesis of the disease.