



THEORIES ABOUT A POTENTIAL CORRELATION BETWEEN PUBIC HAIR REMOVAL AND PREDISPOSITION TO ABORTION

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ABSTRACT

Background: Pubic hair removal (PHR) modifies the body by depilation and epilation procedures but is not included in abortion etiology.

Review Results: The potential predisposition to abortion following PHR is based on the following theories:

Inflammatory neurogenic theory

An inflammation following PHR induces the release of proinflammatory cytokines and the activation of sensory neurons. Miscarriage may result from overactivation of inflammation or may be associated with the sensory pathways of itch.

Neuroendocrine theory

The hypothalamic-pituitary-gonadal axis has neuronal, hormonal and stress regulation. Hair follicles (HFs) function as a peripheral equivalent to the hypothalamic-pituitary-somatotropic axis, the hypothalamic-pituitary-adrenal axis and exhibit elements of the hypothalamic-pituitary-thyroid axis. HFs are an extrapituitary source of prolactin regulated by cytokines with potential abortion effects. Corticotropin-releasing hormone receptors can be found in the endometrium, the central nervous system and skin. The corticotropin-releasing hormone may regulate myometrial contractility during pregnancy.

Theory of arachidonic acid metabolism activation

Arachidonic acid (AA) and prostaglandins are released because of skin injury and erythema, complications occurring following PHR. Abnormal gene expression associated with AA metabolism has been identified in implantation failure. AA metabolism activation towards prostaglandin F_{2α}, stimulating uterine contractions, has been associated with abortion pathogenesis.

Conclusion: PHR has not been reported as a modifiable risk factor of abortion. The present review suggests a potential correlation between PHR and predisposition to abortion, although the evidence confirming or refuting this remains insufficient.

Keywords: abortion, pubic hair removal, risk factors, arachidonic acid, prostaglandins, hair follicle, hypothalamic-pituitary gonadal axis,

BACKGROUND

Pubic hair removal (PHR) is a body modification practice without medical indications, which is not mentioned in abortion etiology [1]. The multifactorial etiology of miscarriage includes anatomical, genetic, autoimmune, immunological and infective factors. Other causes include endocrine disorders such as hyperprolactinemia, thyroid dysfunction, progesterone deficit with excess of Th1 cytokines [2], infectious processes resulting in cytokine-mediated cervico-vaginal inflammation [3]. A prolonged or early exposure to certain Th1 cytokines, IL-1 β and others may cause miscarriage [4]. Inflammatory and immunological dysregulation likely affects the trophoblast invasion [5]. The modifiable risk factors of abortion include alcohol, caffeine, smoking, cocaine [5], environmental contaminants [5, 6], stress, which affects uterine blood flow, inflammation, cortisol and immune balance [7]; excessive labour activity [4] and night labour [8]. Other factors include nutrition deficits, trauma, passive smoking and exposure to heavy metals [9].

Predictive biomarkers of increased abortion risk are also known, such as low levels of human chorionic gonadotropin and plasma kisspeptin, indicative of reduced maternal immune tolerance or aberrant decidualization and implantation, included in the abortion pathogenesis [10].

Pubic hair protects the genital area from pathogens [1]. Comfort and benefit during coitus are reasons for practicing PHR [11]. Depilation methods (shaving, trimming) remove the hair from the skin surface, whereas epilation methods (waxing, laser epilation, electromechanical epilation) remove the shaft of the hair follicle (HF) [12, 13]. Inadequate genital hygiene – lack of it or an excessive one – alters genital homeostasis and is associated with a risk of vulvovaginal infections. Because of residue accumulation resulting in infections, PHR is occasionally recommended, as well as trimming instead of total hair removal, although there is no consensus on that matter [14].

The most frequently used depilation method is shaving [1, 11, 14, 15, 16], and the most common epilation method is waxing [1]. The adverse effects of shav-

ing, such as skin irritation, inflammation, thickening and impairment of epidermal integrity, pseudofolliculitis and hyperpigmented papules, are associated with the release of proinflammatory cytokines from the epidermis, activation of sensory nerves and vasodilation [12]. Other adverse reactions after shaving include pruritus, cuts, ingrown hair [1], folliculitis, dermatitis [11]. Adverse reactions after waxing include pain, edema, irritation, ingrown hair, bleeding [1], folliculitis [13].

Pubic hair removal is associated with minor complications. The most common ones are epidermal abrasion and ingrown hair. Vulvovaginal irritation and infection, as well as sexually transmitted infections, have also been reported [15]. Occurrence of condylomata acuminata has been reported after shaving and waxing [1]. It has not been found whether the sexually transmitted pathogens (chlamydia, gonorrhoea, herpes, papilloma viruses, *Treponema pallidum*) observed more frequently following total PHR result from the PHR procedure itself, micro-trauma or sexual behaviour [1].

Pubic hair grooming malpractică may result in laceration and sexually transmitted infections such as gonorrhoea and chlamydia. The most common genital complications are itching, rash, folliculitis, allergy, and pain [16]. Skin micro-trauma following PHR facilitates pathogen invasion [16]. Pulling out traumatizes HFs, which results in risk of infection, because follicular microbiota cannot be eradicated even by antiseptics with long contact time, and bacterial complications cannot be completely avoided [1].

Mechanical epilation causes perifollicular transient neurogenic skin inflammation, itching, thermal sensitivity, pain, delayed hair regrowth, thinning, lightening of the hair shaft [12].

The increased degranulation activity of perifollicular mast cells, induced by the micro-trauma following mechanical epilation, is long-lasting and associated with substance P release by the sensory neurons. Proinflammatory cytokines such as IL-1 β and tumor necrosis factor α (TNF α), which are released in skin trauma, increase the intracutaneous nerve growth factor (NGF). The increased NGF following epilation may contribute to itch and transient pain or hyperalgesia induced by sensory hyperinnervation. Surrogate markers of neurogenic inflammation, itch, thermoregulation and pain after epilation include NGF, substance P receptor, certain channels of the transient receptor potential (TRP) [12].

The TRP channels are sensory molecules in the skin participating in the pathways of itch, pain and inflammation. The endogenous itch mediators, IL-13 and IL-31, inflammatory mediators, arachidonic acid (AA) and prostaglandins activate certain TRP channels. The TRP channels are expressed on sensory neurons around the HFs as well. The activation of certain TRP channels by physical stimuli results in mast cell degranulation with an inflammatory cascade participating in the initiation of pain and itch. TRP channels are activated by AA as well and play a role in causing pain and itch [17]. By means of the sensory neurons and its effect on the release of inflam-

matory mediators, NGF takes part in nociceptive signaling in generating and maintaining chronic pain in injury [18].

Adverse reactions following laser epilation include pain, impaired sensation, erythema, blistering, crusting [1], petechiae, purpura, ecchymosis [19], burns, hyperpigmentation, hypopigmentation, irritation, folliculitis, contact dermatitis, leukotrichia [20], edema, pruritus or urticaria [21]. Laser epilation destroys the hair follicles by selective photothermolysis [13, 20]. The penetration depth is in the range 4 mm – 2 cm, and reaching the fetus is very unlikely [22]. In three reports, pregnant women were recommended not to resort to laser hair removal due to lack of safety data [23], and to use waxing, shaving or chemical depilation instead [24, 25]. A systematic review supports maternal-fetal safety in cutaneous laser treatment during pregnancy. Although elective laser treatment is not recommended, there is no solid evidence that it induces miscarriage [22].

From the point of view of evolution, the skin is an older organ than the central nervous system (CNS) and human HFs function as a peripheral equivalent to the hypothalamic-pituitary-somatotropic (HPS) axis. They represent a non-classical site for production of growth hormone (GH) [26] that directly acts on the growth hormone receptor (GHR) expressed in the HFs and indirectly on the insulin-like growth factor 1 (IGF-1). Excessive GH and IGF-1 levels are associated with hypertrichosis and hirsutism [27]. GH treatment modulating the IGF-1 levels improves the results of in vitro fertilization at low IGF-1 [28].

HFs are a peripheral equivalent to the hypothalamic-pituitary-adrenal (HPA) axis [26, 27], expressing corticotropin-releasing hormone (CRH) and adrenocorticotrophic hormone (ACTH), and the intrafollicular CRH [27] is regulated by the HF-synthesized cortisol [27, 29].

During pregnancy, the function of the HPA axis is increased. ACTH and cortisol levels are elevated, probably due to placental CRH and ACTH synthesis, maternal pituitary desensibilization to cortisol or increased pituitary response to CRH. Maternal hypercortisolism is considered to occur in pregnancy either because pregnancy is a condition refractory to this hypercortisolism or because the increased progesterone neutralizes it. Hypercortisolism is supposed to be maintained by extrapituitary ACTH and CRH sources, including placental ACTH - an autonomous continuous source, and CRH. Maternal hypercortisolism in early pregnancy protects the fetus by deactivating glucocorticoids. Increased ACTH at the end of pregnancy suggests the presence of ACTH not controlled by feedback. Premature activation of placental CRH activates the HPA axis and can induce delivery. CRH receptors (CRH-Rs) can be found in the placenta, endometrium, CNS and skin. During pregnancy, CRH may regulate myometrial contractility by acting directly, as well as through ACTH, and facilitates decidualization and implantation.

Urocortins belong to the CRH family. The pituitary gland is the site with the highest immunoreactive urocortin in humans. In ovine pregnancy, cortisol stimu-

lates pituitary urocortin, which suggests activation of the HPA axis. Human placenta, chorion and amnion express urocortin 1. Endometrial urocortin 1 takes part in decidualization and implantation, whereas the placental one stimulates the release of prostaglandin and placental ACTH and is likely to participate in controlling the delivery process. Urocortin 1 and 2 have been identified in the myometrium. Urocortin 2 interacts with the myometrial corticotropin releasing hormone receptors 2 (CRH-R2s) and stimulates myometrial contractility [30].

The skin and HF are an extrapituitary source of prolactin (PRL) – a pluripotent hormone associated with HF reproduction and growth, which elongates the hair shaft in humans by autocrine/paracrine regulation. In the cutaneous context, PRL exhibits cytokine-like properties and is a mediator along the brain-skin axis; the prolactin receptor belongs to the family of cytokine receptors [29]. Prolactin in HF is regulated by substance P and the proinflammatory cytokine interferon-gamma (IFN- γ) [27]. PRL expression in HF is increased by IFN- γ and decreased by the epidermal substance P and TNF- α in HF [29].

HF are an extrapituitary source of thyrotropin-releasing hormone (TRH) of the hypothalamic-pituitary-thyroid axis. TRH and estradiol regulate prolactin in HF [27]. TRH stimulates prolactin release [31]. The neuroendocrine regulation of kisspeptin neurons dominates and controls the gonadotropin releasing hormone (GnRH) in the HPG axis. [32]. It is the kisspeptin hypothalamic neurons and the kisspeptin produced by them, and not GnRH, that express progesterone receptors (PR) and estrogen receptor α (ER α), and kisspeptin may be found in the limbic cerebral areas and the placenta [32, 33, 34].

The HPG axis coordinates reproduction and fertility and has neuronal, hormonal and stress regulation and interacts with and can be influenced by the HPA axis – CRH, ACTH, glucocorticoids, cortisol and kisspeptin modulate GnRH. The progesterone synthesized in the brain and allopregnanolone, its neuroactive metabolite, increase during stress and are associated with neuro-modulatory and antigonadotropic properties induced mainly by metabolization and not through PR signaling. Stress-induced suppression of the reproductive kisspeptin-GnRH axis has been suggested in female mice [33, 35].

Lesional epidermis, having itchy urticarial papules and pregnancy plaques, is positive for PR, whereas non-lesional epidermis does not express ER and PR [36]. Exposure to certain endocrine disrupting chemicals (EDCs) in rodents affects the HPG and HPA axes [34] and may result in thyroid dysfunction in humans as well [34, 37]. These chemicals act through non-steroid receptors [34] and hormonal receptors, as a result of which inflammation [34] and epigenetic modifications may occur [34, 37]. The main source of exposure is dermal absorption [37]. The hypothalamus controls homeostasis as a result of the neuronal potential for sensory and hormonal input integration [34]. Exposure to EDCs influences GnRH and kisspeptin neurons by activating ERs, androgen receptors or the aryl hydrocarbon receptor during pregnancy.

Kisspeptin neurons react to metabolic and physiological stress factors, suppressing reproduction. Because of lower estradiol, EDCs are associated with a higher incidence of miscarriage [38]. Recurrent spontaneous abortion (RSA) is closely related to the overexpression of arachidonic acid metabolism. Aberrant gene expression of cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2), thromboxane A2 receptor (TBXA2R) and prostaglandin F2 α (PGF2 α) in the uterus of the rat contributes to uterine contraction and RSA. AA is released during miscarriage and delivery, which increases PGF2 α , prostaglandin E2 (PGE2) and thromboxane A2 (TXA2) and may be associated with the process of abortion and delivery in humans. Prostaglandins stimulate uterine contractions, while PGF2 α , an AA metabolite, is important in abortion. Maintaining an inflammatory condition during pregnancy provides maternal tolerance to the fetus, overactivation of inflammation may result in spontaneous abortion. COX-1 participates in platelet aggregation control. COX-2 takes part in inflammation, pathophysiological processes and catalyzes AA conversion into prostaglandin in the human body. Increasing COX-2 may result in vascular inflammation, coagulation reaction, and miscarriage in early pregnancy. Activation of TXA2, an AA metabolite, aggregant, procoagulant, vasoconstrictor, as well as of TBXA2R, may be a factor for recurrent abortion due to thrombosis causing ischemia, hypoxia and embryo death. [39]. Genes associated with AA metabolism are abnormally expressed in recurrent implantation failure [40]. AA promotes hair growth [41]. Increased concentrations of AA, PGE2, PGF2 α have been observed in erythema [42].

REVIEW RESULTS

The correlation between pubic hair removal as a modifiable risk factor and predisposition to abortion has not been studied to date; this review suggests that there may be such a correlation on the basis of the following theories:

1. Inflammatory neurogenic theory

An inflammation following shaving releases proinflammatory cytokines and activates sensory nerves. Mechanical epilation causes inflammation and micro-trauma, resulting in liberation of substance P by the sensory nerves [12]. IL-13 of the sensory pathways of itch takes part in the etiology of spontaneous abortion [4, 17]. Maintaining an inflammatory condition during pregnancy provides maternal tolerance to the fetus. Overactivation of inflammation may result in miscarriage [39].

2. Neuroendocrine theory

The HPG axis coordinates reproduction and fertility and has neuronal, hormonal and stress regulation [33]. From the point of view of evolution, the skin is the oldest endocrine organ [2, 6, 26]. HF function as a peripheral equivalent to the hypothalamic-pituitary-somatotropic axis [26], the hypothalamic-pituitary-adrenal axis with CRH expression and exhibit elements of the hypothalamic-pituitary-thyroid axis. HF are an extrapituitary source of prolactin regulated by substance P and the proinflammatory cytokine IFN- γ [27]. PRL expression in

HF is increased by IFN- γ and decreased by the epidermal substance P and TNF- α in HF [29]. Hyperprolactinemia, thyroid dysfunction [2], prolonged exposure to certain proinflammatory cytokines of the Th1 type, where IFN- γ and TNF α belong, exposure to IL-1 β and others may cause spontaneous abortion [4, 43]. CRH-Rs are found in the endometrium, the central nervous system and skin. CRH may regulate myometrial contractility during pregnancy [30].

3. Theory of activation of arachidonic acid metabolism

Arachidonic acid (AA) promotes hair growth [41]. Increased concentrations of AA, PGE₂, PGF₂ α are concomitant with erythema [42]. Arachidonic acid, prostaglandins [12], proinflammatory cytokines like IL-1 β [4] are released following skin injury [12] but have been reported in the etiology and pathogenesis of spontaneous abortions [4, 39]. Trauma is associated with abortion [9]. Abnormal gene expression associated with AA metabolism has been identified in implantation failure [40]. Activation of AA metabolism towards PGF₂ α , stimulating uterine contractions, has been associated with abortion pathogenesis [39].

CONCLUSION:

PHR has not been reported to date as a modifiable risk factor of abortion. The present review suggests a potential correlation between PHR and predisposition to abortion, although the evidence confirming or refuting this remains insufficient.

Abbreviations

AA - arachidonic acid
 ACTH - adrenocorticotropin hormone
 CNS - central nervous system
 COX-1 - cyclooxygenase-1
 COX-2 - cyclooxygenase-2
 CRH - corticotropin-releasing hormone
 CRH-Rs - corticotropin-releasing hormone receptors
 EDCs - endocrine disrupting chemicals
 ER α - estrogen receptor α
 GH - growth hormone
 GHR - growth hormone receptor
 GnRH - gonadotropin-releasing hormone
 HF - hair follicles
 HPA axis - hypothalamic-pituitary-adrenal axis
 HPG axis - hypothalamic-pituitary-gonadal axis
 HPS axis - hypothalamic-pituitary-somatotropic axis
 IFN- γ - interferon-gamma
 IGF-1 - insulin-like growth factor-1
 NGF - nerve growth factor
 PGE₂ - prostaglandin E₂
 PGF₂ α - prostaglandin F₂ α
 PHR - pubic hair removal
 PR - progesterone receptors
 PRL - prolactin
 RSA - recurrent spontaneous abortion
 TBXA₂R - thromboxane A₂ receptor
 TNF α - tumor necrosis factor α
 TRH - thyrotropin-releasing hormone
 TRP channel - transient receptor potential channel
 TXA₂ - thromboxane A₂

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