JETIR.ORG ISSN: 2349-5162 | ESTD Year : 2014 | Monthly Issue
JOURNAL OF EMERGING TECHNOLOGIES AND INNOVATIVE RESEARCH (JETIR)
An International Scholarly Open Access, Peer-reviewed, Refereed Journal

Antifertility Effects on Medicinal Plants

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Abstract -

Everyone can refer to plants that are helpful in treating a range of ailments that arenot responsive to allopathic treatment as "medical plants," using this widely used term. Theseinclude the rising human fertility rate and birth control, both of which have become significant problems in recent years. Current information on therapeutic plants with anti- fertility properties that has been demonstrated by science is provided by this review. This study includes information about the parts used, family, chemical elements found in plants, and botanical name. Traditional medical and surgical procedures continue to be popular and trusted even with the rapid advancement and broadening of these fields. Several studies have indicated that

traditional herbal remedies have anti-fertility effects. This review aims tohighlight findings from studies on.

The purpose of this review was to give a thorough overview of the medicinal plants that different tribes and ethnic groups use around the world to treat female infertility. We conducted a thorough review of the literature by examining scholarly articles and traditional textbooks, as well as by consulting reputable international scientific databases. Using terms like "antifertility," "anti-implantation," "antiovulation," and "antispermatogenic" and activity of plants, we searched CENTRAL, Embase, and PubMed. Antifertility agents are plants, including their parts and extracts, that have historically been used to promote antifertility. In this paper, various medicinal plants have been reviewed for thorough studies such as Andrographis paniculate, Michelia champaca L, Azadirachta indica, Apium graveolens, Withania somnifera, Hydropiper polygonum, Cannabis sativa. It seems that many of these therapeutic plants function by means of an antizygotic mechanism. This review unequivocally shows that it is time to go beyond experimental research in order to find new possible chemical constituents from medicinal plants; the mechanisms underlying plant extracts and their active constituents should be further studied. With more investigation and study, this review establishes a strong basis for future research on the effectiveness of plants that women use as traditional antifertility remedies today and may prove to be effective in thefuture.

Keywords - Fertility, Infertility, antispermatogenic, antiovulation, Estrogen, Progesterone, Pregnancy.

Introduction -

Oral contraceptives, commonly known as antifertility medicines, are medications that regulate fertility.[1] These medications have an impact on and are involved in female ovulation and the menstrual cycle. Birth control tablets contain a combination of estrogen and progesterone. When an antifertility drug stops ovulation, implantation, fertilization, zygote destruction, or abortion in females, it is considered active. It inhibits testosterone, stops spermatogenesis in men, and has an impact on organ gonadotrophin or sperm mortality. Many emerging nations are currently controlling their population sizes.[2] In albino male andfemale rabbits, oxyphenbutazone, indomethacin, and acetyl salicylic acid exhibit antifertility effects by inhibiting prostaglandin production. In male rabbits, oxyphenbutazone and indomethacin have an impact on the reproductive process. Many emerging nations currently regulate the size of their populations. In albino male and female rabbits, oxyphenbutazone, indomethacin, and acetyl salicylic acid decrease prostaglandin production and exhibit antifertility effects. In male rabbits, the reproductive process is impacted by oxyphenbutazone and indomethacin.[3] Plant-based medications are gaining traction over synthetic ones because of their low toxicity and historical use in traditional medical systems like Ayurveda. Family planning has been encouraged through a number of contraceptive methods. However, because to the severe side effects of synthetic steroidal contraceptives, interest in native herbsfor potential contraceptive benefits has recently grown. Therefore, it is necessary to look for acceptable native plant items that could be utilized in place of tablets.[4]

Brahmi (Bacopa monnieri), a well-known herb with a positive impact on cognition, isanother classic example.

Although the phytochemical and analytical parameters were not disclosed, testing the antifertility capability of an aqueous extract of Brahmi in male mice demonstrated reversible male infertility. Additionally, the study found that the percentage of testis seminiferous tubules with damaged tissue was 96.27% after 28 days of Brahmi administration compared to 54.13% after 56 days of treatment. This finding has generated controversy.[5] Numerous herbs and plant extracts are utilized to cure and alleviate a variety of physical and mental diseases, in part because population increase is a major contributor to poverty and pollution in developing nations.[6] Numerous possible infertility treatments havebeen studied over time, including immunological, hormonal, and pharmaceutical approaches.[7]

Mechanism of action of Antifertility Plants -

Medicinal plants have been shown to have antifertility effects through a variety of mechanisms of action. One of these mechanisms is their influence on sex hormones, which is particularly useful for treating breast pain, menopausal symptoms, enlarged prostates, regularizing menstrual cycles, and suppressing fertility [8]

More over plants with estrogenic property can directly authority pituitary action by peripheral modulation of luteinzing hormone (LH) and follicle stimulating hormone (FSH), dwindle their secretions and blocking ovulation.[9]

Estrogen -

A class of steroid hormones known as estrogens is primarily responsible for regulating the responsiveness of the female sex organs and reproduction. The three main endogenous estrogens are estriol, 7-α-estradiol, and estrone. The most biogenic version of 17-estradiol is the strongest. The ovary is where estrogens are biosynthesised.[10] Phytoestrogens are naturally occurring plant estrogens that can bind to estrogen receptors andshare structural similarities with estrogens. Depending on an individual's circulating endogenous estrogen and the quantity and variety of estrogen receptors in their body, phytoestrogens may have both estrogenic and antiestrogenic effects.[11]

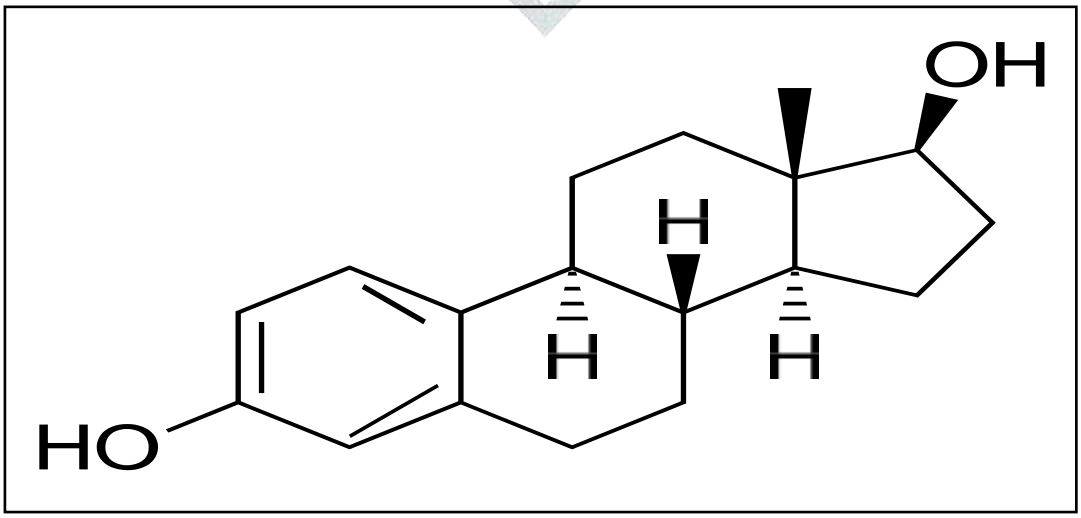

Fig-Structure of Estrogen

Physiology of Estrogen-

The development and maintenance of pregnancy depend on the proliferation and differentiation of uterine epithelial cells, which is triggered by estrogens. Numerous growth factors and progesterone are also crucial for these processes. The development of a model featuring a null ERα (αERKO) has provided us with a tool to ascertain and validate the physiological functions of ERα. Adult αERKO mice are hypoplastic and sterile, with ERα being the major determinant for estrogen activity in the mouse uterus.[12] The secondary sexual traits of females and the changes that occur throughout puberty in girls are mostly caused by the estrogens. Estrogens directly contribute to the uterus, fallopian tubes, and vaginal growth and development. Because estrogen stimulates ductal growth, stromal development, and fat accumulation, it enlarges the breasts.[13]

Meachanism Of Action Estrogen-

Inhibiting the release of gonadotropin-releasing factor by the hypothalamic center can be achieved by estrogen, which in turn stops pituitary gonadotropin secretion and subsequent ovulation. In order for the ova to enter the unprepared uterus and either degenerate or be evacuated, the transit of the ova through the tubules is either hastened or hindered. The delicate progesterone and estrogen balance needed for the blastocyte to implantation in the endometrium is also altered by administered estrogens. In addition,estrogens stimulate the development of hormone receptors, which are essential for the interplay of various hormones. The majority of the effects of estrogens are mediated through intracellular receptor activation. The primary mechanism by which steroids and hormones function is through intracellular receptors.

Following their entry into the cell, estrogens combine with particular cytoplasmic binding proteins (receptors) to create a complex. Through a conformational shift, the estrogen-receptor complex is activated. After entering the nucleus, this active complex releases the free estrogen. After that, the estrogen molecule attaches itself to the nuclear chromatin's acceptor sites. The drug's binding to the chromatin material causes changes in therate of synthesis of related enzymes and other proteins as well as an increase or reduction in the production rate of specific types of RNAs.[14]

This means that the biological response is eventually projected by these changes. A examination of the literature suggests that medicinal plants and plant products have an anti- fertility effect. The quest for plants that are both fertility-inducing and anti-fertility has begun in the twenty-first century. It is believed that using herbal medications can be done so withoutcausing any negative effects and that they are safer and less expensive than synthetic medications.[15]

A thorough evaluation of the research on Indian medicinal herbs' ability to prevent conception has been done by Choudhury and Haq, Kamboj and Dhawan, R.G. Mali. It is determined to gather data on these medicinal plants that have anti-fertility properties in this regard. Thus, some effort is made, and the following is a small list of plants that have an anti-fertility effect.[16]

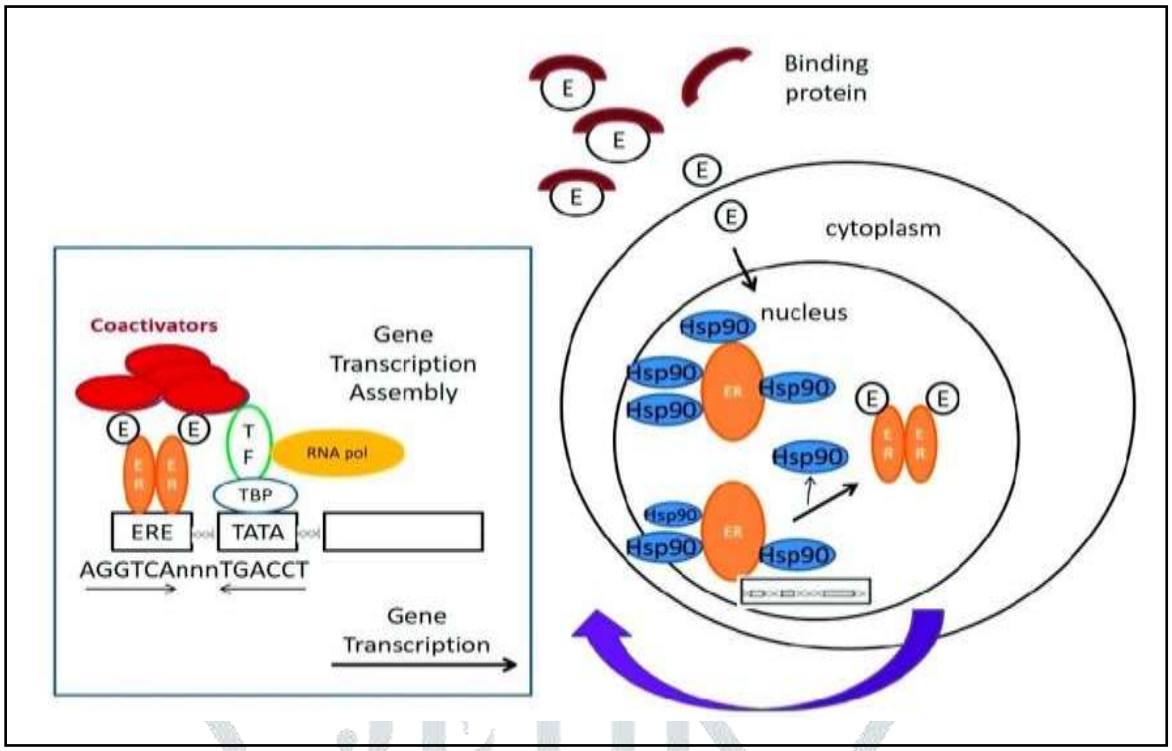

Fig- Mechanism of action of estrogen

Progesterone -

At the start of the 20th century, research into progesterone's physiological effects began.[17] Pregnant animals have been shown to experience abortion when the ovariancorpus luteum is destroyed. An injection of corpus luteum extract could stop this in animals. In 1934, progesterone was eventually extracted from corpus luteum extract by a number of different research groups operating independently. Initially, the drug was referred to asluteosterone in Europe and progestin in the USA. As evidence that this hormone is crucial forpregnancy (pro gestationem [Latin]: promoting gestation), scientists decided to name it progesterone in 1935. [18,19] There are both local and systemic effects of progesterone. Progesterone has a variety of systemic effects, including improving visual memory, promoting the proliferation and differentiation of osteoblasts, stimulating the catabolic metabolism, relaxing smooth muscle cells, increasing calcium and phosphorus excretion, raising basal body temperature, improving diuresis through activation of the renin- angiotensin system, and possibly suppressing the immune system.[20]

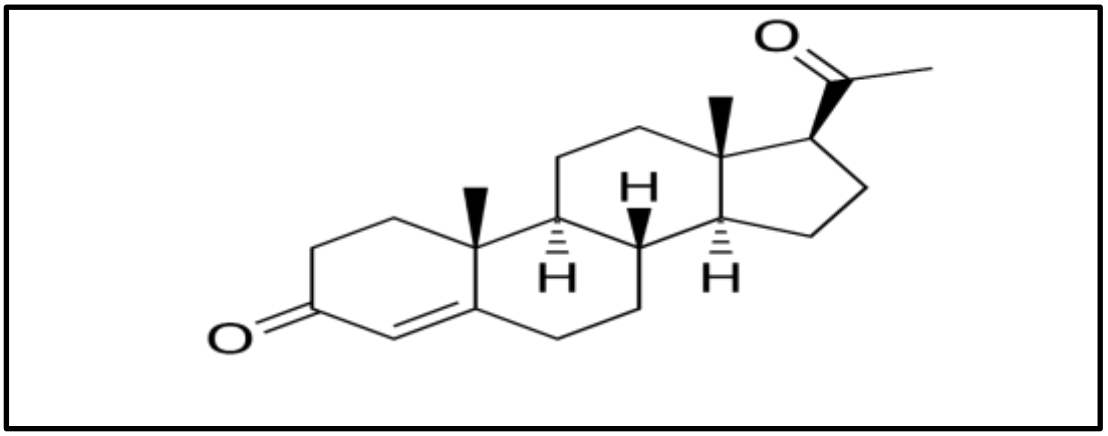

Fig-Structure of Progesterone

Mechanism of Progesterone Action -

The expression of enzymes unique to each of these organs determines the type and quantity of steroid hormones that are made and secreted, but the biosynthetic pathway remains the same regardless of the organ that generates the steroids—the ovary, testis,adrenal cortex, brain, and placenta. The ovaries lack 21 α-hydroxylase and 11 α-hydroxylase, which prevents them from producing glucocorticoids or mineralcorticoids. The female gonad,albeit a complete steroidogenic gland, varies from other organs of this type in terms of enzymatic provision and subsequently hormones generated.[21]

All steroid hormones share cholesterol as their common precursor. Beginning with the interconversion of two acetyl-CoA molecules, its production creates the two intermediate products, squalene and lanesterol.[22] Low-density lipoprotein (LDL) cholesterol 6 interacts with membrane receptors, internalizes in vesicles, and fuses with lysosomes to transfer cholesterol in plasma. Free form cholesterol can be released into thecell by the lysosomal hydrolases. Once within the mitochondria, the cholesterol is transformed into pregnenolone. When pregnenolone is released from mitochondria by cytochrome P450, it can proceed through two different metabolic pathways: the Δ5-hydroxy steroid pathway, which is the main metabolic pathway in adrenal glands and does not occurin luteinized follicles; or the Δ4-ketosteroid pathway, which is characteristic of corpus luteumgranulosa cells and leads to the synthesis of 17 α-hydroxyprogesterone andandrostenedione.[23] Progesterone originating from the adrenal glands is primarily transformed into glucocorticoids and androgens, while progesterone produced from the gonads is primarily transported in the circulation to carry out its biological activity. Progesterone is circulating in the bloodstream attached to serum albumin and 10% of globulin that binds cortisol. Progesterone's half-life in the body is comparatively brief—it is about five minutes. Urine contains the metabolites of reduced derivatives of glucuronides andsulfates, which are mostly generated in the liver. Renal 21-hydroxylation transforms circulating progesterone into the powerful mineralocorticoid desoxycorticosterone (DOC). This system is responsible for the majority of circulating DOC during the luteal phase, duringpregnancy, and after exogenous progesterone therapy. This can be an undesirable side-effect.[24]

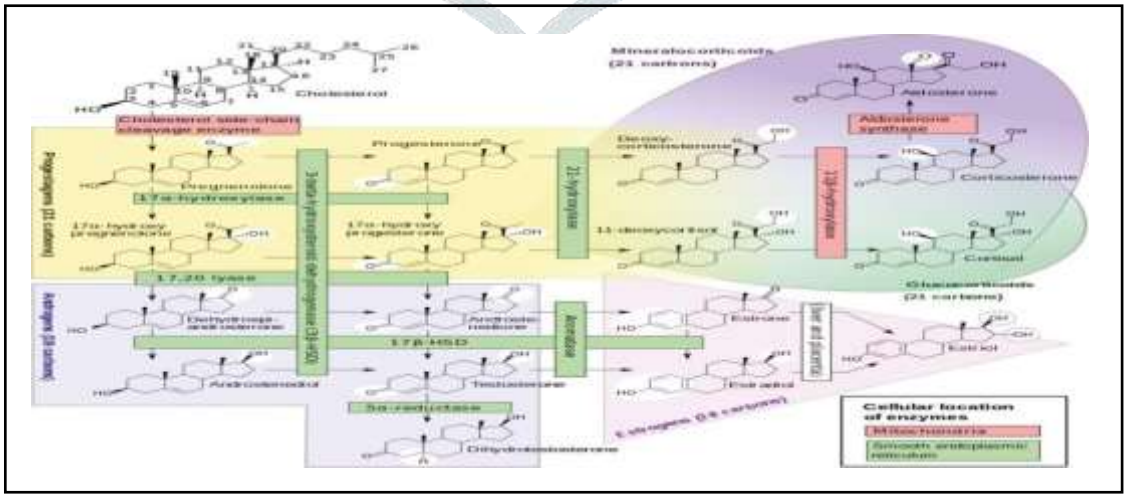

Fig-Mechanism action of Progesterone

Medicinal Plants With Notable Anti-Fertility Properties -

Though they have less potential for use by humans, some herbal contraceptives have also been produced. People are now searching for herbal remedies to control fertility and prevent numerous ailments as a result of these issues.[25] There are a number of preventive and corrective contraceptive methods available thanks to modern science, but none of them are particularly safe or free from major negative effects. Drugs having synthetic or chemical bases may disrupt the endocrine system and have impacts on the body's metabolism, development, reproduction, and nervous system. These substances might negatively impact natural hormone production, secretion, transport, and action. They either obstruct hormone function or hinder hormone synthesis and metabolism, which upsets the natural hormone balance. Several instances are Phthalates, pesticides, plasticizers, heavy metals, alkylphenols, bisphenol A, dioxins, heavy metals, fungicides, and insecticides stopthe synthesis of estrogen and progesterone, which affects the female sexual development. These chemicals have also been shown to have other negative effects on the reproductive system, such as endometriosis, toxicity to the gonads, testicular germ cell cancer, breast/prostate cancer, and temporary or permanent infertility.[26] It is imperative, for these reasons, to create a high-effective, entirely herbal medication that won't compromise the reproductive system. Human societies all throughout the world use more than 35,000 different plant species for medical purposes. The majority of traditional medicines used to treat basic health issues in about 80% of the world's population employ plant extracts. [27,28]People have been using plants to treat illnesses and soothe bodily pain since ancient times. The usefulness of many traditional medicines is now acknowledged due to their improved cultural acceptance, improved compatibility with the human body, decreased side effects, andimproved effectiveness. The need for the development of certain safe and effective natural contraceptives Even the most basic members of ancient civilizations employed herbal contraceptives to manage their fertility and avoid getting pregnant. Despite the fact that mainstream medicine has identified a number of significant female contraceptives (contraceptives), their efficacy and appeal among women are limited because of several unfavorable side effects. Common adverse effects include gastrointestinal distress, obesity, cholelithiasis, cervix and breast cancer, asthma, and venous thromboembolism.[29] Even with the variety of contraceptive methods available, one of the most difficult tasks in the fieldof pharmaceutical and medical sciences is the search for newer, more effective, Explorations into the hidden treasure of medicinal plants for use as contraceptives have recently been undertaken. For the majority of people worldwide, herbal medicine is still a widely used kind of therapy for both illness treatment and health maintenance. Information about the screening of plants with antifertility efficaciousness has been steadily accumulating.[30-33] The antifertility program can benefit from the knowledge about plants and herbs found in folklore and historical books. Recent years have seen the identification of several plants, and numerous studies have assessed extracts and active ingredients from diverse plant parts, such as seeds, roots, leaves, flowers, stems, or stem barks. [34-36]

Risks And Side Effects Of Current Birth Control Methods -

There are dangers connected to several contraceptive methods. The risk ofcardiovascular disease is associated with oral contraceptives. The intrauterine device is linkedto uterine perforation, pelvic inflammatory illness, spontaneous abortion, septic abortion, and tubal infertility. Tubal sterilization is related with surgical risks such as anesthesia andbleeding.[37] Abdominal pain, headaches, and menstrual cycle irregularities are just a few of the negative effects that emergency contraceptives can cause[38] Weight gain, acne, headaches, alterations in the menstrual cycle, and mood changes brought on by the presence of hormones are among the many negative effects connected to hormonal contraceptives.[39] Synthetic steroidal contraceptives can cause serious negative effects like breast cancer and cervical cancer. These side effects are linked to the use of synthetic estrogen and progesterone.[40]

Fertility And Infertility-

Being able to start a clinical pregnancy is a sign of fertility.[41] The ability to have children is what is meant by the term "fertility." Although the chance of conception fluctuates little from cycle to cycle within individuals, in the population as a whole, it usually peaks during the first few months of unprotected sexual activity and then steadily decreases after that.[42] Monthly fecundability, or the likelihood of becoming pregnant each month, significantly declines among those who continue trying to conceive if there isn't a pregnancy after three months.[42] Compared to women in their early 20s, women in their late 30s have a roughly 50% lower relative fertility rate.[43,44]

The inability to conceive successfully after 12 months or more of consistent, unprotected sexual activity is known as infertility, which is a medical condition.[45] Basedon medical history and physical findings, earlier evaluation and treatment may be warranted. For women over 35, it is necessary to repeat the evaluation and treatment after six months.[45] Although both men and women's fertility declines as they get older, the effectsof aging on women's fertility are much more pronounced.[43] Male fertility does not appear to decline significantly before roughly age 50, despite the fact that men's semen parameters

also start to decline detectably after the age of 35.[44] Some medical professionals confuse infertility and subfertility by using the same terms. But for the proper therapy of reproductive problems, formal classifications are crucial. Infertility affects >186 million persons worldwide, the majority of whom live in underdeveloped nations.[45] While the age of the mother at conception is the strongest negative predictor of fertility other factors, such as lifestyle choices and environmental factors, are thought to play an increasing influence. Gender-specific or non-specific fertility-affecting factors will be discussed.[46]

Phases Of Pregnancy -

Phase 1: Insertion of a "open wound" represent-

Seminal plasma has a crucial immunoregulatory role in the post-coitus period, establishing pregnancy and inducing an inflammatory response in the endometrium. As a result, a variety of immune

effector cells are drawn to the implantation site. While the exact mediators of these inflammatory responses are still unknown, it is known that cytokines workin tandem with prostaglandins and other steroid-binding proteins to produce certain effects.[47] One of the main factors allegedly controlling this inflammatory condition is TGF-b1 in serum plasma. Numerous cytokines are present in human semen, such as IL-2, IL-4, IL-1a/b, IL-6, IL-10, IL-8, IL-11, IL-12, TNFα, IFNγ, MIF (monocyte chemotactic and activating factor), VEGF (vascular endothelial growth factor) regulated on activation, RANTES (normal T cell expressed and secreted), macrophage colony-stimulating factor(CSF1), granulocyte colony stimulating factor (G-CSF), macrophage–CSF, stem cell factor, etc.[47] The postcoital uterus may also be the site of immunoregulatory functions for eotaxin, macrophage inflammatory proteins, monocyte chemotactic protein (MCP)-1, keratinocyte- derived chemokine, and IL-9.[48] Apart from helping sperm survive and subsequently be removed from the female reproductive system, these inflammatory reactions [49] act as mediators of immunosurveillance in support of embryo implantation and the establishment ofpregnancy. They also control uterine immune processes like granulocyte trafficking, recruitment, activation, and presentation of paternal antigens.[47]

The inflammatory response to "an open wound" is similar to what happens during implantation, placentation, and the first and early second trimesters of pregnancy. The endometrial tissue is harmed as the blastocyst invades the uterine epithelial lining to implant itself during the first stage, and the trophoblast of the blastocyst replaces vascular smooth muscle in the mother's blood vessels and the uterine endothelium to adjust to an appropriate blood supply.[50] Numerous invading cells as well as dying and repairing cells infiltrate as a result of these processes. Therefore, in order to remove cellular debris and repair the damaged uterine epithelium, an inflammatory response is required. Along with other physiological changes like changes in endocrine regulation, the mother's body is in the process of becomingimmunologically acclimated to the presence of the fetus. [51-53] Several cytokines, includingIL-6, EGF, and basic fibroblast growth factor (bFGF), are produced by pre-implanted embryos and help with embryo–maternal amalgamation at the implantation sites. Accordingto reports, heparin binding EGF via the uterine epithelium during blastocyst attachment to theuterine wall is caused by steroid-hormone sensitive secretion. This process also results in blastocyst outgrowth. Critical cytokines like leukemia inhibitory factor (LIF)-like cytokines and IL-1 system components also play a role in mediating implantation. [54] Cytokines (IL11, LIF, activin A, and monoclonal non-specific suppressor factor-b) are involved in the decidualization process because they promote tissue differentiation.[55]

Phase 2: Becoming Accustomed To A Semi-Allogenic Fetus-

It is thought that the mother experiences a calming pregnancy during the second immunological phase. During this stage, the fetus grows quickly, forming a symbiotic relationship with the maternal components. As a result, the fetus, placenta, and mother are all adapted to one another, and the dominant immune system works to reduce inflammation. The traditional theory behind this anti-inflammatory state is that regulation of NK and lymphokine-activated cells causes a cytokine shift from inflammatory Th1 toantiinflammatory Th2 cytokines.[56] Th1-type responses are reduced by modulatory cytokines (IL-3, IL-4,

IL-5, and IL-10) that are seen at the foetomaternal interface.[57] and the no lymphoid trophoblast and placental/decidual tissues' predominant contribution of Th2- type cytokines during this second stage of pregnancy[58] During this stage, cytokines notonly help to develop immune tolerance to the fetal allograft but also encourage uterine growth. The mechanical strain that the expanding gestational sac places on the uterine wall may cause the production of transforming growth factor (TGF-β3), a cytokine that could speed up the growth of the uterus.[59,60] During this phase of growth, the endometrium also produces IL-1a, IL-1b, IL-6, and IL-8 to support the development of the uterine and fetal tissue as well as an anti-inflammatory state.[60] Through regulatory processes like epithelial- mesenchymal transformation, many other cytokines (like TGF-) are also linked to fetal and embryonic development. Axial patterning, craniofacial morphogenesis, cardiovascular and skeletal development, and erythropoiesis are other conditions they oversee. [61-63]

Phase 3: Inflammation Renews In Preparation For Parturition-

The fetal development process ends during the final immunological phase of pregnancy. The mother must now go through the parturition process, which is facilitated by new inflammation, in order to deliver the baby. Thus, after labor begins, the anti- inflammatory immune state is reversed.[56] It is acknowledged that the precise immune mechanisms regulating labor are not entirely clear, but that a coordinated production of prostaglandins and cytokines can cause an sequence.[64] First off, IL-6 and GM-CSF during the latter stages of pregnancy may aid in the fetus's final stages of lung maturation.[60] Increased levels of surfactant protein-A (SP-A) and phospholipid release (e.g., arachidonic acid for prostaglandin synthesis) are caused by a decrease in maternal progesterone and pulmonary maturation. Fetal amniotic macrophages activated by SP-A migrate to the uterine wall and produce IL-1b, which sets off an inflammatory/prostaglandin cascade that causes uterine contractility.[65] The inflammatory and chemotactic chemicals IL-1b, IL-6, and IL-8 are also produced as a result of myometrial leukocytic incursion and relocation.[60] It is clearthat cytokines play a role in uterine smooth muscle contraction because they have been shown to raise intracellular calcium, phospholipase activity, and cause the production of prostaglandin and oxytocin receptors in human myometrial cells in vitro.[60] which are necessary to induce labor in the uterus during parturition. Furthermore, cytokines (TNF-α andIL-1) facilitate the release of arachidonic acid to promote additional prostaglandin synthesis.[66] This stage of the inflammatory process also affects the membranes surroundingthe chorioamniotic valve.[67] The synthesis of cytokines during the gestational membrane leads to an increase in matrix metalloproteinase (MMP) expression, which in turn causes the extracellular matrix to remodel and weaken. This process is caused by the breakdown of collagen, which causes the fetal membranes to rupture and dissociate.[68] Although the cervical epithelium still responds to exogenous IFN-γ and TNF-α, it also releases a variety of cytokines, including M-CSF (microphage-colony stimulating factor), TGF-β1, IL-1a/b, IL-6, IL-7, IL-8, and RANTES.[60] Throughout this stage, the fibroblasts mediate the remodeling of the cervix through inflammatory progression by upregulating cytokines (IL-6, IL-8), matrix metalloprotease-1 (MMP-1) and MMP-3, and inhibiting their tissue inhibitors (tissue inhibitors of metalloprotease, or TIMPs).[68] Therefore, active immune cells infiltrate the myometrium to trigger an inflammatory process that leads to uterine contraction, baby expulsion, and placenta rejection,

which controls parturition.[51,64]

Anti Implantation Activity-

The notable decline in implantation locations suggested that suppression of implantation might be one mechanism at work. The disruption of endocrine-endometrial synchronization, which depends on the balance of progesterone and estrogen, may be the cause of this extract's anti-implantation impact.[69] Andrographis paniculata (Acanthaceae) and Curcuma longa (Zingiberaceae) Rats' implantation and estrus cycles were used to measure the antifertility effects of curcumin and andrographolide. When compared to the usual control group, the combination dramatically decreased the number of implants and the size of the litters in the rats. Additionally, the combination significantly changed the lengthsof each estrus cycle phase and combined their effects to reduce the number of ovarian follicles.[70]

The women of the Indian state of Chhattisgarh have long used Michelia champaca L. (family: Magnoliaceae), also referred to as champa [Hindi], to control fertility. The extract's antifertility properties were assessed in two experimental animal models, namely the anti-implantation activity in female wistar rats and the estrogenic/antiestrogenic activity in female ovariectomized rats, at dose levels of 100 and 200 mg/kg body weight, po. At 100 and 200 mg/kg doses, respectively, the extract demonstrated significant (po0.01) 49.95% and 71.03% anti-implantation activities. Along with increased body weight, uterine weight, endometrial thickness and height, vaginal cornification, and significant (po0.01) increases in estrogen, cholesterol, alkaline phosphate, and triglycerides at higher doses when given alone, the extract also demonstrated significant (po0.01) estrogenic activity.[71]

Anti Ovulation Activity-

The primary hormone that prevents pregnancy is progesterone. Their primary mode of action involves inhibiting follicular growth in order to prevent ovulation.(Source:) The hypothalamus uses progesterone negative feedback to lower the gonadotropin-releasing hormone's pulse frequency.[72] Hydropiper polygonum Belonging to the Polygonaceae family, linne (marsh pepper) is prized for its leaves and roots, which also contain active components like oxymethyl-anthraquinones, tannin, beldianic acid, acetic acid, and formic acid. It is applied to cases of dyspepsia, hemorrhoids, diarrhea, and skin issues. It has anti-rheumatic and anti-cancer properties in traditional medicine. These ingredients may have biological effects on human fertility, antimicrobial, anti-inflammatory, and antioxidant properties. Kapoor et al. (7) reported on this plant's anti-ovulatory properties in one study. Their investigation into the antifertility properties of this specific plant involved the use of three different extract types: petroleum, aqueous, and alcohol. The antifertility activity was observed in rabbits whose ovulation was induced by copper. Root extract in petroleumether.[73]

Anti Spermatogenic Activity-

Anti spermatogenic drugs to stop the formation of sperm and to stop them from maturing, traveling through the vas deferens, and depositing.[74] Oftentimes, conventional medications used as contraceptives are insufficient [74] Azadirachta indica It belongs to family Meliaceae and also known as Neem. Seed oil of Neem is used as anti- diabetic, spermicidal, anti-fertility, anti-bacterial [75] Apium graveolens, commonly known as celery, is a member of the Apiaceae family and is a species of parsley (Umbelliferae). It is approximately 100 cm tall, has fleshy stems, and a strong scent.[76] According to reports that are currently available, plants like celery contain phytoestrogens, which may have an impact on reproductive health and fertility.[77] I.e. Celery has also been shown in a study to protect the testes from sodium valproate and di (2-ethylhexyl) phthalate. [78,79] .. Research showed that celery shields sperm from atrazine and quinine sulfate-induced toxicity and testes from structural and functional damage. [80,81] When 200 and 300 mg/kg were given to rats for 60 days, the number of sperms, sertoli cells, and primary spermatocytes was significantly higher in the treated group than in the control group. Celery appears to boost spermatogenesis in male rats without negatively impacting testicular tissue.[82]

Folk medicine has described ashwagandha (Withania somnifera), also called "Indian ginseng," as an aphrodisiac and geriatric tonic because of its revitalizing effects.[83] Ashwagandha has a wide range of medicinal properties that can either directly or indirectly prevent and treat a number of diseases because it is rich in chemical compounds like amino acids, neurotransmitters, ergostane steroids, and alkaloids.[84-86] Various researchers have reported that ashwagandha is helpful in treating male infertility.[87,88] In one study, oligospermic males treated for 90 days with a high-concentration, full-spectrum root extract of ashwagandha (225 mg/kg) showed significantly improved semen parameters along with improved and regulated levels of sexual hormones.[89]

Abortificient Activity-

An abortifacient is any substance that is used to terminate a pregnancy. Historically, lead and quinine have been used as abortifacients, but now over-the-counter preparations such as acetaminophen, aspirin, iron, and herbal preparations are more commonly used to induce abortion. The greatest dangers of abortifacients are the effect of thetoxin on the mother and the potential teratogenic effect on the fetus. Most attempts to chemically abort a fetus are made in the first trimester. Acetaminophen and multidrug ingestions are the most common exposures, with minor maternal toxicity reported in most cases. Given the gravity of an attempted abortion and the frequently ambiguous clinical presentation of ingestion, a high degree of suspicion is needed to determine whether this has happened in gravid patients. Therefore, a pregnancy test ought to be conducted onany female overdose patient who is of childbearing age. Women who exhibit heavy vaginal bleeding should also be asked if they have ever used an abortion pill.[90] The plant Cannabis sativa (Cannabinaceae) is said to have antifertility properties in folk medicine. Cannabis sativa extracts in alcoholic, chloroform, and aqueous forms showed notable abortificient activity (9% to 42%). It was

discovered that the strongest abortifacient activity was produced by the alcoholic extract when administered at a dose of 400 mg/kg body weight. Additionally,the extract extended the estrous cycle in the test animal and demonstrated estrogenic activity. While the body weight increased non-significantly, the ovarian and uterine weights significantly decreased as a result of the Cannabis sativa extract. Serum progesterone levels increased and serum estrogen levels slightly decreased, but the levels of LH and FSH were found to be significantly lower.[91]

Table 1-List Of Antifertility Medicinal Plant -

Plant	Type	Dose/body weight (mg/kg)	Activity	Refs
Abroma augusta	Petroleum ether	50	Anti-implantation	[92]
Abutilon indicum	50% aqueous methanolic extract	500	Anti-implantation	[92]
Juniperus phoenica	Ethanolic extract	Intraperitoneal injections of 400	Antifertility activity	[106]
Artemisia vulgaris	Methanolic extract	300 and 600	Anti-implantation	[93]
Aegle marmelos	50% ethanolic extract	100, 200, and 300	Antifertility effect	[94]
Albizzia lebbeck	Methanolic extract	50, 100, and 200	Antifertility effect	[95]
Aloe barbadensis	Aqueous extract	100	Anti-implantation	[96]
Cichorium intybus	50% ethanolic extract	50	Anti-implantation	[96]
Acalypha indica Linn	Ethanolic extract	600	Estrogenic activity	[97]
Cuscuta reflexa	Ethanolic extract	800	Anti-implantation	[96]
Curcuma longa	Petroleum ether	200	Anti-implantation	[96]
Cannabis sativa	Alcoholic extract	20	Antispermatogenic	[98]
Rubia cordifolia	Ethanolic extract	250	Anti-implantation	[96]
Striga orobanchioides	Ethanolic extract	200	Anti-implantation	[99]

Acalypha indica Linn	Ethanolic extract	600	Estrogenic activity	[100]
Abrus precatorius Linn	70% methanolic extract	20 and 40	Antifertility	[101]
Albizzia lebbeck	Methanolic extract	50, 100, and 200	Antifertility effect	[102]
Bacopa monnieri	Dry powder	250	Antispermatogenic	[103]
Fadogia agrestis	Aqueous extract	18, 50, and 100	Adverse effects on male rat testicular function	[104]
Leptadenia hastata	Aqueous extract	100, 200, 400, and 800	Antispermatogenic activity	[105]
Urtica diocia	Ethanolic extract	250	Anti-implantation	[96]
Striga	Ethanolic	200	Anti-implantation	97

Conclusion -

The list of medicinal plants used as antifertility agents presented in this review is useful to researchers, as well as practitioners. This list is best used only as a preliminary screening of potential antifertility plants, not as a definitive or complete list of antifertility plants.

Acknowledgement -

The authors would like to express their gratitude to the Swami Institute of Pharmacy, Abhona Tal. Kalwan, Dist. Nashik, as well as the entire college staff at the Institute, for their support with this review work.

References -

1] Kumar D, Kumar A, Prakash O. Potential antifertility agents from plants: a comprehensivereview. J Ethnopharmacol 2012;140:1-32.

2] Peri X, Nai W. Can ethnopharmacology contribute to the development of antifertilitydrugs? J Ethnopharmacol 1991;32:167-77.

3] Yegnanarayan R, Joglekar G. Antifertility effect of non-steroidal anti-inflammatory drugs. Jpn J Pharmacology 1978;28:909-17.

4] United Nations, Department of economic and social affairs, population division (2019). World

population prospects 2019. Highlights. (ST/ESA/SER.A/423).

5] Singh A, Singh SK. Evaluation of antifertility potential of Brahmi in male mouse. Contraception. 2009;79:71–9. [PubMed] [Google Scholar].

6] Ekor, M. (2013) The growing use of herbal medicines: issues relating to adverse reactionsand hallenges in monitoring safety. Front Pharmacology. 4, 177.

7] Roozbeh, N., Amirian, A., Abdi, F. & Haghdoost, S. (2021) A Systematic Review on Useof Medicinal Plants for Male Infertility Treatment. J Family Reprod Health.15(2), 74–81.

8] Williamson EM, Okpako DT, Evans FJ. Pharmacological methods in phytotherapy research: Selection preparation and pharmacological evaluation of plant material. John Wileyand Sons Ltd., London, 1996; 1: 191-212.

9] Brinker F. Inhibition of endocrine function by botanical agents, antigonadotropic activity. Br J Phytother, 1997; 4: 123-145.

10] Munson PL, Mueller RA and Breese R: Principles of Pharmacology. Basic concepts and clinical applications; 1996: 809-22.

11] Cassidy A, Bingham S and Carlson J: Biological effects of a diet rich in isoflavone on themenstrual cycle of premenopausal women. American Journal of Clinical Nutrition 1994; 60 (3): 333-40.

12] Couse JF, Lindzey J, Grandien K, Gustafsson JA, Korach KS. Tissue distribution andquantitative analysis of estrogen receptor-alpha (ERalpha) and estrogen receptor-beta (ERbeta) messenger ribonucleic acid in the wild-type and ERalpha-knockout
mouse. Endocrinology. 1997;138(11):4613–21. [PubMed] [Google Scholar].

13] Hardman Joel G, Limbird Lee E and Alfred Goodman Gilman: Pharmacological basis oftherapeutics. USA: Mc Graw-Hill Companies 10th Ed 2001; 1600-6.

14] Paranjape MH, Bothara KG and Patil VR: Fundamentals of pharmacology. NiraliPrakashan 1994: 2: 239.

15] Rajiv Rai and Vijendra Nath: Some lesser known orla herbal contraceptives in folk claims as anti-fertility and fertility induced plants in Bastar region of Chhattisgarh. Journal ofNatural Remedies 2005; (5/2): 153-9.

16] Chowdhury RR and Haq M: Review on anti-fertility activity of Indian Medicinal Plants. Bulletine of Medico Ethno Botanical Research 1980a; 1: 408.

17] Williams C L, Stancel G M. Elmsford, Oxford: Pergamon Press; 1996. Estrogens andProgestins; pp. 1411–1440. [Google Scholar].

18] Millart P, Paszkowski T. Progestageny w praktyee ginekologieznej. Ginekol Prakt. 2002;67:16–21. [Google Scholar.

19] Paszkowski T, Kozlowska J. Progesteron-druga mlodosc starego leku. Ginekol Prakt. 2003;70:52–57. [Google Scholar].

20] Paszkowski T. Renaissance of the clinical applications of a progesterone. Gin Politec Project. 2011;19:41–47. [Google Scholar] [Ref list]

21] Genazzani AR (ed). Endocrinologia ginecologica. Editeam, Cento (FE), Italy. 2004; 1–464.

22] Andersen JM, Dietschy JM. Relative importance of high and low density lipoproteins inthe regulation of cholesterol synthesis in the adrenal gland, ovary and testis in the rat. J Biol Chem. 1978; 253: 9024–6.

23] Brown MS, Goldstein JL. Receptor-mediated control of cholesterolmetabolism. Science. 1976; 191: 150–5.

24] Madori A, Cavallari C, Giacomucci E, Macrelli S, Mastronuzzi G, Ucci N. (eds). Fisiologia della Riproduzione. Bologna: CLUEB, 1994; 1–192

25] Patil SJ & Patil SB. Antiovulatory activity of petroleum ether extract of chromatographic fractions of Citrus medica seeds in albino rats. International Journal of Medical Sciences, 2013; 13(6): 410-417.

26] Schug TT, Janesick A, Blumber B & Heindela JJ. Endocrine disrupting chemicals and disease susceptibility. Journal of Steroid Biochemistry & Molecular Biology, 2011; 127: 204–15.

27] Shah GM, Khan MA, Ahmad M, Zafar M & Khan AA: Observations on antifertility and abortifacient herbal drugs. African Journal of Biotechnology, 2009; 8(9): 1959-64.

28] Kaur R, Sharma A, Kumar R & Kharb R: Rising Trends towards Herbal Contraceptives.Journal of Natural Products and Plant Resour, 2011; 1(4): 5-12.

29] Kumud Bala, Mahima Arya & Deepshikha Pandey Katare. Herbal Contraceptive: An Overview. World Journal of Pharmacy and Pharmaceutical Sciences, 2014; 3(8): 130526.

30] Chopra RN, Nayar SL & Chopra IC. Glossary of Indian Medicinal Plants, CSIR, New Delhi, 1956; pp222.

31] Casey RCD. Alleged antifertility plants of India, Indian J Med Sci, 1960; 14: 590-600.

32] Farnsworth NR, Bingel AS, Cordell GA, Crane FA & Fong HHS. Potential value of plants as source of new antifertility agents I, J Pharm Sci, 1975; 64: 535-49.

33] Farnsworth NR, Bingel AS, Cordell GA, Crane FA and Fong HHS, Potential value ofplants as source of new antifertility agents II, J Pharm Sci, 1975; 64: 717-36.

34] Henshaw PS. Physiological control of fertility, Science, 1953; 117: 572-82.

35] Orzechowski G. Nature against nature, Deut Apoth, 1972; 24: 277-78Orzechowski G.Nature against nature, Deut Apoth, 1972; 24: 277-78.

36] Brondegaard VJ. Contraceptive plant drugs, Planta Med, 1973; 23: 167-72. 37] National Research Council (US) Committee on Population. Contraception and
Reproduction: Health Consequences for Women and Children in the Developing World.
Washington (DC): National Academies Press (US); 1989. 4, Contraceptive Benefits andRisks.

38] World Health Organization Department of Reproductive Health and Research (WHO/RHR) and Johns Hopkins Bloomberg School of PublicHealth/Center for Communication Programs (CCP), Knowledge for Health Project. Family Planning: A GlobalHandbook for Providers (2018 update).Baltimore and Geneva: CCP and WHO; 2018.

39] Sabatini R, Cagiano R, Rabe T. Adverse effects of hormonal contraception. Journal für Reproduktionsmedizin und Endokrinologie-Journal of Reproductive Medicine and Endocrinology. 2011;8(1) :130-156.

40] Shukla A, Jamwal R, Bala K. Adverse effect of combined oral contraceptive pills. AsianJ Pharma & Clin Res. 2017;10;17-21.

41] F. Zegers-Hochschild The international glossary on infertility and fertility care, 2017Fertil. Steril. (2017).

42] Gnoth C, Godehardt D, Godehardt E, Frank-Herrmann P, Freundl G. Time to pregnancy:results of the German prospective study and impact on the management of infertility. Hum Reproductive 2003;18:1959–66.

43] Howe G, Westhoff C, Vessey M, Yeates D. Effects of age, cigarette smoking, and otherfactors on fertility: findings in a large prospective study. BMJ 1985;290:1697–700.

44] Dunson DB, Baird DD, Colombo B. Increased infertility with age in men and women.Am J Obstet Gynecol 2004;103:51–6.

45] R.D. Nachtigal International disparities in access to infertility services Fertil. Steril.(2006).

46] A. Maheshwari *et al.* Women's awareness and perceptions of delay in childbearingFertil. Steril. (2008).

47] Gopichandran N1, Ekbote UV, Walker JJ, Brooke D, Orsi NM (2006) Multiplexdetermination of murine seminal fluid cytokine profiles. Reproduction 131: 613-621.

48] Robertson SA1, Mau VJ, Hudson SN, Tremellen KP (1997) Cytokine-leukocyte networks and the establishment of pregnancy. Am J Reprod Immunol 37: 438-442.

49] Denison FC, Grant VE, Calder AA, Kelly RW (1999) Seminal plasma componentsstimulate interleukin-8 and interleukin-10 release. Mol Hum Reprod. 5:220-6.

50] Dekel N, Gnainsky Y, Granot I, Mor G (2010) Inflammation and implantation. Am JReprod Immunol. 63:17-21.

51] Mor G, Abrahams V: Immunology of implantation. In Immunology and Allergy Clinics.Edited by Arici A. Philadelphia: W.B. Saunders Company; 2002: 545-65.

52] Dutta S, Sengupta P (2017) Yoga escalates female reproductive health during pregnancy.J Pregnancy Reprod. 1:1.

53] Sengupta P (2014) The bliss yoga inculcates during the different stages of pregnancy.International Journal of Pharmacy and Pharmaceutical Sciences 6:86-87.

54] Sharkey A (1998) Cytokines and implantation. Rev Reprod 3: 52-61.

55] Salamonsen LA, Hannan NJ, Dimitriadis E (2007) Cytokines and chemokines duringhuman embryo implantation: roles in implantation and early placentation. Semin ReprodMed. 25:437-44.

56] Raghupathy R (1997) Th1-type immunity is incompatible with successful pregnancy.Immunol Today. 18:478-82.

57] Carp H1 (2004) Cytokines in recurrent miscarriage. Lupus 13: 630-634.

58] Chaouat G, Cayol V, Mairovitz V, Dubanchet S (1999) Localization of the Th2 cytokinesIL-3, IL-4, IL-10 at the fetomaternal interface during human and murine pregnancy and lack of requirement for Fas/Fas ligand interaction for a successful allogeneic pregnancy. Am J Reprod Immunol. 42:1-13.

59] Shynlova O, Tsui P, Dorogin A, Langille BL, Lye SJ (2007) The expression of transforming growth factor beta in pregnant rat myometrium is hormone and stretchdependent. Reproduction 134: 503-511.

60] Orsi NM, Tribe RM (2008) Cytokine networks and the regulation of uterine function inpregnancy and parturition. J Neuroendocrinol 20: 462-469.

61] Barnett JV, Desgrosellier JS (2003) Early events in valvulogenesis: a signalingperspective. Birth Defects Res C Embryo Today. 69:58-72.

62] Serra R, Chang C (2003) TGF-beta signaling in human skeletal and patterning disorders.Birth Defects Res C Embryo Today. 69:333-51.

63] Nawshad A, LaGamba D, Hay ED (2004) Transforming growth factor beta (TGFbeta)signalling in palatal growth, apoptosis and epithelial mesenchymal transformation (EMT).Arch Oral Biol. 49:675-89.

64] Romero R, Espinoza J, Kusanovic JP, Gotsch F, Hassan S (2006) The preterm parturitionsyndrome. BJOG. 113 Suppl 3:17-42.

65] Condon JC, Jeyasuria P, Faust JM, Mendelson CR (2004) Surfactant protein secreted by the maturing mouse fetal lung acts as a hormone that signals the initiation of parturition. Proc Natl Acad Sci U S A. 101:4978-98.

66] Molnar M, Romero R, Hertelendy F (1993) Interleukin-1 and tumor necrosis factor stimulate arachidonic acid release and phospholipid metabolism in human myometrial cells.Am J Obstet Gynecol. 169:825-9.

67] Haddad R, Tromp G, Kuivaniemi H, Chaiworapongsa T, Kim YM, et al. (2006) Humanspontaneous labor without histologic chorioamnionitis is characterized by an acute inflammation gene expression signature. Am J Obstet Gynecol. 195:394 e1-24.

68] Cockle JV, Gopichandran N, Walker JJ, Levene MI, Orsi NM (2007) Matrix metalloproteinases and their tissue inhibitors in preterm perinatal complications. Reprod Sci.14:629-45.

69] Gupta S S. Prospects and perspectives of natural plants products in medicine. IndianJournal of Pharmacology. 1994;26:1–12. [Google Scholar].

70] Singh S, Sharma A.. Studies on ethnomedicinal plant of Baghicha Jashpur Chattisgarh. J Sci Lett. 2017;2:48–55. [Google Scholar] [Ref list].

71] Taprial S, Kashyap D, Mehta V, et al. Antifertility effect of hydroalcoholic leaves extractof Michelia champaca L. An ethnomedicine used by Bhatra women in Chhattisgarh state of India. J. Ethnopharmacol 2013;147:671-675.

72] Baird DT, Glasier AF. Hormonal contraception. N Engl J Med. 1993 May27;328(21):1543-9. [PubMed].

73] Kapoor M, Garg SK, Mathur V. Antiovulatory activity of five indigenous plants inrabbits. Indian J Med

Res, 1974; 62: 1225-7.

74] Emmanuel Mouafo Tekwu, ... Victor Kuete, in Medicinal Plant Research in Africa, 2013[Pub Med].

75] Jain S, Choudhary GP and Jain DK. Medicinal plants with potential antifertility activity: A review. Int J of Green Pharmacy. 2015;9(4):223-228.

76] Khare CP (2007) Indian Medicinal Plants: An Illustrated Dictionary. Springer Science,New York, USA.

77] Kooti W, Ghasemiboroon M, Asadi-Samani M, Ahangarpoor M, Zamani M, et al. (2014)He Eؤect of Halcoholic Extract of Celery Leaves on the Delivery Rate (Fertilization and Stillbirths), the Number, Weight and Sex Ratio of Rat oؤ Spring. Adv Environ Biol 10: 824-
30.

78] Hamza AA, Amin A (2007) Apium graveolens modulates sodium valproate-inducedreproductive toxicity in rats. J Exp Zool A Ecol Genet Physiol 307: 199-206.

79] Madkour NK (2014) %beneficial role of celery oil in lowering the di(2- ethylhexyl)phthalate-induced testicular damage. Toxicol Ind Health 30: 861-872.

80] Abarikwu SO, Pant AB, Farombi EO (2012) He protective eؤects of quercetin on thecytotoxicity of atrazine on rat Sertoli-germ cell coculture. Int J Androl 4: 590–600.

81] Farombi EO, Ekor M, Adedara IA, Tonwe KE, Ojujoh TO, et al. (2012) Quercetin protects against testicular toxicity induced by chronic administration of therapeutic dose ofquinine sulfate in rats. J Basic Clin Physiol Pharmacol 1: 39–44.

82] Kooti W, Mansouri E, Ghasemiboroon M, Harizi M, Ashtary-Larky D, et al. (2014) He Eؤects of Hydroalcoholic Extract of Apium graveolens Leaf on the Number of Sexual Cellsand Testicular Structure in Rat. Jundishapur Journal of Natural Pharmaceutical Products. 4:e17532.

83] Weiner MA, Weiner J (1994) Ashwagandha “(Indian ginseng),” in Herbs Hat Heal.Quantum Books, Mill Valley, Calif, USA.

84] Mirjalili MH, Moyano E, %onfill M, Cusido RM, Palazón J (2009) Steroidal lactonesfrom Withania somnifera, an ancient plant for novel medicine. Molecules 14: 2373-2393

85] Bhattacharya SA, Bhattacharya A, Sairam K, Ghosal S (2000) Anxiolyticantidepressant activity of Withania somnifera glycowithanolides: an experimental study, Phytomedicine 7: 463–469.

86] Jayaprakasam B, Strasburg GA, Nair MG (2004) Potent lipid peroxidation inhibitors from Withania somnifera fruits. Tetrahedron 60: 3109–3121.

87] Mishra RK, Verma HP, Singh N, Singh SK (2012) Male infertility: lifestyle and orientalremedies. Journal of 6cientific Research 56: 93–101.

88] Ahmad MK, Mahdi AA, Shukla KK (2010) Withania somnifera improves semen qualityby regulating reproductive hormone levels and oxidative stress in seminal plasma of infertilemales. Fertility and Sterility 94: 989– 996.

89] Ambiye VR, Langade D, Dongre S, Aptikar P, Kulkarni M, et al. (2013) Clinical Evaluation of the Spermatogenic Activity of the Root Extract of Ashwagandha (Withaniasomnifera) in Oligospermic Males: A Pilot Study. Evidence-based Complementary and Alternative Medicine 57: 14-20.

90] TIMOTHY B. ERICKSON MD, ... VALERIE A. DOBIESZ MD, in Haddad and Winchester's Clinical Management of Poisoning and Drug Overdose (Fourth Edition), 2007.

91] Zade V, Wikhe M, Dabhadkar D, et al. Antifertility efficacy of Cannabis sativa leaves onfemale albino rats. Int J Sci Invent Today 2013;2(2):107-117.

92] R. Maurya, S. Srivastava, D. Kulshreshta Traditional remedies for fertility regulationCurr Med Chem, 11 (2004), pp. 1431-1450.

93] A. Shaik, R. Kanhere, R. Cuddapah, K. Nelson, P. Vara, S. Sibyala Antifertility activityof Artemisia vulgaris leaves on female Wistar rats Chin J Nat Med, 12 (2014), pp. 180-185.

94] Chauhan A, Agarwal M, Kushwaha S, Mutreja A. Antifertility studies of Aeglemarmelos Corr., an Indian medicinal plant on male albino rats. Egyp J Biol 2008;10:28e35.

95] Gupta RS, Kachhawa JB, Chaudhary R. Antifertility effects of methanolic pod extract of Albizzialebbeck (L.) Benth in male rats. Asian J Androl 2004;6:155e9.

96] Maurya R, Srivastava S, Kulshreshta D. Traditional remedies for fertility regulation. CurrMed Chem, 2004; 11: 1431-50.

97] Hiremath S, Rudresh K, Badami S, Patil S, Patil S. Post-coital antifertility activity ofAcalypha indica L. J Ethnopharmacol 1999;67:253e8.

98] Sailani MR, Moeini H. Effect of Ruta graveolens and Cannabis sativa alcoholic extract on spermatogenesis in the adult Wistar male rats. Indian J Urol 2007;23:257e60.

99] Hiremath S, Badami S, Swamy K, Patil S, Londonkar R. Antifertility activity of Strigaorobanchioides. Biol Pharm Bull 1994;17:1029e31.

100] Hiremath S, Rudresh K, Badami S, Patil S, Patil S. Post-coital antifertility activity ofAcalypha. indica L. J Ethnopharmacol 1999;67:253e8.

101] Bhatt N, Chawla SL, Rao MV. Contraception evaluation of seed extracts of Abrusprecatorius L. in male albino rats (Mus musculus). J Herb Med Toxicol 2007;1:45e8.

102] Gupta RS, Kachhawa JB, Chaudhary R. Antifertility effects of methanolic pod extract of Albizzia lebbeck (L.) Benth in male rats. Asian J Androl 2004;6:155e9.

103] Singh A, Singh SK. Evaluation of antifertility potential of Brahmi in male mouse. J Contracep 2009;79:71e9.

104] Yakubu MT, Akanji MA, Oladiji T. Effects of oral administration of aqueous extract of Fadogia agrestis (Schweinf. Ex Hiern) stem on some testicular function indices of male rats. J Ethnopharmacol 2008;115:288e92.

105] Bayala B, Telefo PB, Bassole IHN, Tamboura HH, Belemtougri RG, Sawadogo L, et al. Anti-spermatogenic activity of Leptadenia hastata M. Daniyal, M. Akram / Journal of the Chinese Medical Association Decne leaf stems aqueous extracts in male wistar rats. J Pharmacol Toxicol 2011;6:391e9.

106] Shkukani HG, Salhab AS, Disi AM, Shomaf MS, Al Quadan F. Antifertility effect ofethanolic extract of Juniperus phoenica (L.) in male albino rats. J Herb Pharmacother 2008;7:179e89.