1. Introduction

Herbal medicine offers and alternative therapeutic model for treating a patient. Approximately 25 percent of all prescription drugs are derived from trees, shrubs or herbal compounds (Pradhan et al, 2009). India is bestowed with an enormous wealth of medicinal plants by nature and often been referred as "medicinal garden of the world". "Saraca indica" is one of the foremost medicinal plants utilized since antiquity (Sharma et al, 2008). It is commonly known as Ashoka and it is one of the legendary sacred trees of India. 'Asoka' or 'Ashoka' is a sanskrit word which means “without sorrow” or "that removes grief". Ashoka tree was universally known by its binomial Latin name Saraca indica (Roxb.), it is also called as De.wild or Saraca indica belonging family Caesalpinaceae [web1, 2 and Akash Jain et al.,2013]. It is an evergreen tree and as per botanical nomenclature named as Saraca indica.

'Saraca indica' is named differently in different states of India as Asoka (in Sanskrit), Ashok(in Hindi), Asokam (in Tamil), Asokam (in Malayalam), Asokamu (in Telugu), Asokada (in Kannada), Ashok (in Marathi), Aspal (in Bengali), Ao palav (in Gujarati), Ashok (in Kashmiri), Ashoka (in Oriya), Ashoka (in Assamese), Ashok (in Punjabi)[web3].

Saraca indica tree is found throughout India. It is available up-to an altitude of 750 m in the central, in the eastern Himalayas, Khasi, Garo and Lushai hills, in Chittagong, Bihar, Orissa, Konkan, Deccan Ghats and Mysore. It has become quite scarce in several localities and is reported to be threatened in North Eastern Region of India. As part of a wild tree, it is a vulnerable species. It is becoming rarer in its natural habitat, but isolated wild Ashoka trees are still to be found in the foothills of central and
eastern Himalayas [web5], in scattered locations of the northern plains of India as well as on the west coast of the subcontinent near Mumbai.

1.1 Classification of the plant (Biswas et al, 1972), [web4]:

Kingdom: Plantae
Division: Magnoliophyta
Class: Magnoliopsida
Order: Fabales
Family: Caesalpinaceae
Genus: Saraca
Species: indica

Ashoka is native to India and Sri Lanka. Somehow, the name Ashok has stuck to north India, although the "real" Ashoka is that is called as Seetha Ashoka [web4]. Saraca (Ashoka) a very popular tree in India is commonly seen as a lofty column, very graceful with its downward-sweeping branchlets and shining, green foliage; but sometimes wide-spreadining slender branches issue from the straight trunk and form a compact symmetrical crown. The trees are grown in warm humid climates, and prefer a moist well-drained soil with plenty of organic matter.

The Saraca (Ashoka) which is a rain-forest tree is originally distributed in the central areas of the Deccan plateau, as well as the middle section of the Western Ghats in the western coastal zone of the Indian subcontinent (Bhaduria preeti et al, 2012) which can also be grown within greenhouses. The trees themselves are grown for their upturned flowers which have clusters in yellow, orange or red. The tree prized for its
beautiful foliage and fragrant flowers have no petals, but contain brightly colored sepals, and have stamens projecting up to eight inches long. It is a very beautiful, small, erect evergreen tree, with deep green leaves growing in dense clusters. The leaves are pinnate and have paired leaflets. Typically, these trees are habituated to the shade of other trees. Its flowering season is around February to April, for a short period of two to three weeks. The Saraca (Ashoka) flowers come in heavy, lush bunches. They are bright orange-yellow in color, turning red before wilting. Each flower, borne on a slim, green stem has a tiny calyx and six long, narrow, wavy petals arranged in two sets of three.[web6,web7,web8].

The bark is smooth and dark grayish-brown in color (Ghanshyam Yadav et al, 2013). The tree is covered with a profusion of delicate, star-like flowers, which, being palest-green in color, give the tree a peculiar hazy appearance. They grow in clusters from small protuberances all along the dark branchlets.

1.2 Description of the Plant:

*Saraca indica* tree is a small evergreen tree, 6-8 m high.

Leaves: Paripinnate; leaflets 4-6 pairs, oblong or oblong-lanceolate.

Flowers: Orange or orange-yellow, eventually turning vermillion, fragrant, in dense auxiliary corymbs.

Bark: Brownish red in color.

Pods: Flat, linear-oblung, leathery, 10-25cm long.

Seeds: 4-8, ellipsoid-oblung, compressed.
1.3 History and Reference from classical ayurvedic text:

*Saraca* bark has been used by Ayurvedic physicians from time immemorial to relieve pain and cure all kinds of gynecological disorders. Many formulation of Asoka bark such as Asoka medicated wine, Asoka Ghee, Asoka milk are prepared keeping in mind the benefits and the health conditions of the female [web5, web10,web11].
1.3.1 Religious importance:

The Saraca (Ashoka) tree is held sacred by all the Hindus, Jainis and Buddhists.

1.3.2 Mythological significance:

Saraca is regarded as sacred, and also as a symbol of 'Kama Deva', God of Love. Some of the festivals were especially associated with this flower. The Ashoka tree is worshipped in first month of the Hindu calendar (Chaitra Masam). It has exotic fragrance flowers which were used in temple decorations. In Ramayana, Ashoka Vatika was a garden in Lanka (now known as Sri Lanka) where the Goddess Sita used to sit and remember the God Rama under Ashoka tree. This is the reason why this plant is also called after her name as 'Seetha Ashoka'.

1.3.3 In Buddhist mythology:

Lord Buddha (c.563-483 B.C.) was said to have been incarnated as Gautama Siddhartha beneath the Ashoka tree while doing tapsya and therefore it is a must to plant this tree in Buddhist monasteries [web4]. The plant's bark is reputed for keeping a woman healthy and youthful as popularly known to be said by Buddha.

1.3.4 Jainism:

Ashoka tree is regarded with veneration in Jainism. In the Jain tradition Mahavira is said to have renounced the world under this kind of tree in Vaishali. The earliest chronicle mentioned in Ayurvedic treatise like 'The Charaka Samhita' (100 A.D.), in which Asoka plant was recommended in medicinal formulations for pain
management related to Uterus (Gynecological) as Anodyne. The 'Bhavprakash Nighantu', commonly known as the 'Indian Materia Medica' (1500 A.D.), cites the plant as a uterine tonic that is effective in regularizing the menstrual disorders (web9, web10).

1.4 Chemical Constituents:

Stem bark of Asoka contains tannins and catechin. Asoka bark is also used in tea preparation; giving a beautiful red color to the tea and is very tasty. The Saraca indica tree contains glycosids, non-phenolic, sapogenetic glycoside, aliphatic alcohols, tannin, catechol, sterol, and organic calcium compounds. Its methanol fraction contains haematoxylene, tannin, and water-soluble glycoside. The powdered bark ash of Asoka contains silica, sodium, potassium, phosphate, magnesium, iron, calcium, strontium and aluminium [web5].

1.5 Pharmacological Activities of Saraca Indica Bark:

Bark of Saraca tree is known for its medicinal value having spasmogenic, oxytocic, uterotonic, antibacterial, anti-implantation, anti-tumor, anti-pro gestational, anti-estrogenic activity against menorrhagia and anticancer. In India, it is said to be useful in several ailments, including menstrual cramps, some cases of uterine bleeding, uterine fibroids, hemorrhoids, and internal bleeding. A famous remedy prepared from Ashoka bark is 'Ashokarishta', which is highly efficacious on diseases in women. Bark of Saraca ashoka is used to mitigate excessive bleeding, leucorrhoea and headache.
Even a complete cure of leucorrhoea is achieved with the prolonged use of this medicine.

Some preparations made by using bark of Saraca Ashoka in treating:

a) **Leucorrhoea**: is cured by taking preparation made from powdered bark of the tree with the equal quantity of candy (mishri), ½ teaspoonfuls in the morning and ½ in the evening to cure leucorrhoea.

b) **Stone**: excessive urination and stone problem in kidneys are treated by taking 1-2 spoonful of 1-2 gm of ground seeds of Ashok tree with water twice daily.

c) **Pimples**: Pimples are relieved by the Ashoka bark boiled in one cup of water and mixing with ½ cup of mustard oil into it.

d) **Respiratory complications**: the respiratory complications relieved by taking 'big betel leaf' stuffed with 'Ashoka tree seed powder' twice a day, morning and evening.

e) **Other Diseases**: the Saraca ashoka is used to obtain relief from pain caused by scorpion sting, burning sensations on the skin and also improve skin complexion. The Ashoka herb is also effective in purifying the blood and helps to detoxify the body naturally and also helps in preventing skin allergies (web4).

1.6 Other uses of *Saraca indica*:

The Ashoka tree is a healer for deep seated sorrow, sadness, grief, and disharmony in one's inner being due to events such as bereavement, failure, suffering, disease, and isolation. On using this essence, a profound inner state of joy, harmony and well-being is produced. It works very gently, in that it changes one's perception of the sorrow (web4).
Asoka bark: It is bitter, astringent and sweet in taste.

Asoka Fresh Flowers: It is an excellent uterine tonic and is used in cervical adenitis, biliousness, syphilis, hyperdipsia, burning sensation, hemorrhagic dysentery, piles, scabies in children and inflammation.

Dried flowers are used to treat diabetes.

Seeds are used in treating bone fractures, strangury and vesicle calculi.

1.7 Pharmacokinetic of the tree with recent modern parameters:

Oxytocin activity of the bark (Saraca indica) was seen in rat and human isolated uterine preparations. Estrogen-primed or gravid uterus was more sensitive to the action of the alcoholic extract. Pentolinium bitartrate completely blocked the oxytocic action. Seed extract is found effective against dermatophytic fungi.

1.8 BREAST CANCER:

Breast cancer is a malignant tumor that occurs in the breast tissue. A malignant tumor is a group of cancer cells that can grow into surrounding tissues or spread (metastasize) to distant areas of the body (Chaffer Christine *et al.*, 2011). Mammary glands consist of two types of tissue with glandular and ductal. Lobular tissue produce milk and the ducts that carry milk produced in lobules to the nipple. The supporting tissue of breast consists of fatty, fibrous connective tissue with lymphatic tissue network embedded (Clark Wallace *et al.*, 1969).
There are two types of breast cancer:

- Ductal carcinoma starts in the tubes (ducts) that carry milk from the glandular lobules to the nipple. Most breast cancers are of this type.
- Lobular carcinoma occurs in glandular lobules that produce milk.

Breast cancer may be invasive or noninvasive. Invasive Breast cancer spread from the milk duct or lobule to other surrounding connective tissues in the breast. Noninvasive breast cancer is called as “in situ” (Berger Karen et al., 1998).

In lobular carcinoma in situ (LCIS) cells that look like cancer cells grow in the lobules of the milk-producing glands of the breast. LCIS (also called lobular neoplasia) is sometimes grouped with ductal carcinoma in situ (DCIS) as a non-invasive breast cancer, but it differs from DCIS in that it doesn’t seem to become an invasive cancer if it isn’t treated.

Lobular cancers (cancers of the milk glands), common in women, are extremely rare in men since male breast tissue does not normally contain lobules. Other uncommon types of cancers of the breast that have been reported in men include ductal carcinoma in situ. Cancer in the ducts that has not spread beyond the ducts themselves such as cystosarcoma, phyllloides and Paget's disease of the breast (a cancer involving the skin of the nipple).

1.8.1 Factors influencing breast cancer

A risk factor is anything that affects a healthy person having a chance of getting a disease, such as cancer. Most women are prone to breast cancer, due to one or more risk factors.
Following are the factors which contribute to the risk of developing breast cancer are:

- **Family history of breast cancer** – A person may also have a higher risk for breast cancer if a subject parental history has a breast, uterine, ovarian, or colon cancers. About 20 - 30% of women with breast cancer had a family history of the disease.

- **Age and gender** – A person’s risk of developing breast cancer increases as one gets older. Most advanced breast cancer cases are diagnosed in women aged over 50. Woman population possesses a greater risk for developing breast cancer compared to men. Breast cancers are 100 times more common among women than men due to higher percentage of the female hormones like estrogen and progesterone found in women, which promote breast cancer cell growth.

- **Menstrual cycle** – Women attained early puberty (before age 12) or went through menopause late (after age 55) have an increased risk of breast cancer.

- **Race/Ethnicity** – Caucasian women have a higher incidence of breast cancer than African American women beginning at age 45. In contrast, African American women have a higher incidence rate before age 45 and are more likely to get affected from breast cancer at every age. Asian, Hispanic, and Native-American women have a lower risk of developing and affected by breast cancer.

- **Genetic factors / Gene defects** – About 5% to 10% of breast cancer cases are thought to be hereditary, resulting directly from gene defects (called mutations) passed on hereditary. Certain populations having defective genes that make them more susceptible to develop breast cancer. The most common gene defects are found in the BRCA1 and BRCA2 genes. These genes normally produce
proteins that protect from cancer. Women with one of these gene defects have an 80% chance of getting breast cancer sometime later during their life.

- **Child birth** – Women who never had children or who had them only after age 30 have an increased risk for breast cancer. Being pregnant more than once or becoming pregnant at an early age reduces your risk of breast cancer.

- **DES** – Women administered with diethylstilbestrol (DES) to prevent miscarriage has an increased risk of breast cancer after age 40.

- **Hormone replacement therapy (HRT)/Estrogen Therapy (ET)** – One has higher risk for breast cancer if received hormone replacement therapy with estrogen for several years or more. In fact, some research has suggested that women who previously had Hysterectomy are at higher risk of breast cancer. Women taking estrogen seem to have more problems with strokes and other blood clots. Also, when used long term (for more than 10 years), ET has been found to increase the risk of ovarian cancer in some studies. At this time there appear to be few strong reasons to use post-menopausal hormone therapy (either combined HT or ET), other than possibly for the short-term relief of menopausal symptoms. Along with the increased risk of breast cancer, combined HT also appears to increase the risk of heart disease, blood clots, and strokes.

- **Combined hormone therapy (CHT)** – Combined hormone therapy after menopause increases the risk of getting breast cancer. This increase in risk can be seen with as little as 2 years of use. Combined HT also increases the likelihood that the cancer may be found at a more advanced stage. The word bio identical is sometimes used to describe versions of estrogen and progesterone.
used in CHT with the same chemical structure as those found naturally in people. It is important to realize that although there are few studies comparing bio identical or natural hormones to synthetic versions of hormones, there is no evidence that they are safer or more effective. The use of these bio identical hormones should be assumed to have the same health risks as any other type of hormone therapy (Nayfield Susan et al., 1991).

- Obesity – Obesity has been linked to breast cancer, although this co-relation is controversial. It is attributed that obese women produce more estrogen, which fuel the development of breast cancer.

- Alcohol use – Consuming alcohol on a daily record may also increase risk of breast cancer.

- Radiation – Radiation therapy may increase the risk of developing breast cancer. The younger a subject undergoing radiation treatment and the higher the dose used the greater the risk prevail for breast cancer during breast development (Le Monique G et al., 1984).

1.8.2 Genes associated with Breast cancer:

Populations having some gene defects make them more susceptible to develop breast cancer. The most common gene defects found in breast cancer are BRCA1 and BRCA2 genes. These genes normally produce proteins that protect from cancer. Women with one of these defects have up to an 80% chance of getting breast cancer sometime later during their life span. Mutations in several other genes, including TP53, ATM, PTEN, STK11/LKB1, CDH1, CHEK2, MLH1, and MSH2, ER-alpha and ER-beta
polymorphism in exon 7 codon 392 (C1176G) have been associated with hereditary breast and/or ovarian tumors. These gene mutations are much rarer and often do not increase the risk of breast cancer as much as the BRCA genes.

1.9 Review of literature on estrogen action and its mechanism:

Estrogens are steroid hormones that target broad range of tissues to regulate growth and differentiation in humans. 17β estradiol being the most potent and dominant estrogen found in humans along with other estrogens like estrone and estriol are also present at lower concentrations compared to 17β estradiol. Estrogens exert their function through estrogen receptors (ER) which are the members of a large superfamily of nuclear receptors. Two forms of the ER have been identified; the earlier described ERα (Walter et al., 1985) and the more recently discovered ERβ (Kuiper et al., 1996; Mosselman et al., 1996; Tremblay et al., 1997). The DNA binding domains of these two receptors are highly conserved with 96% identity containing nearly identical zinc finger motifs, P- and D-boxes, which are involved in ERE sequence recognition and dimerization of the ER DNA binding domains, respectively. These receptors act as ligand activated transcription factors. The classical mechanism of ER action involves estrogen binding to receptors in the nucleus, after which the receptors dimerize and bind to specific response elements known as estrogen response elements (EREs) located in the promoters of target genes (Nilsson S et al., 2011). This induces a conformational change thereby recruiting co activator proteins (Rosenfeld MG et al., 2001). However recent studies have proven that estrogen receptors are also present in caveoli regions of plasma membrane (Chambliss KL et al., 2000 and Kim HP et al., 1999). The action of
mechanism by which ERs regulate transcription through ERE are mediated in two ways, one is genomic and the other non-genomic action.

**Figure 1.2: Schematic representation of estrogen mediated pathway** (Linda jornstrom and Maria Sjoberg, 2005)

Figure 2 illustrates the mechanisms of Estrogen exert functions by different pathways, 1. Estrogen (E2) act by classical mechanism by forming E2-ERs complex further these E2-ERs bind to EREs present in the gene promoters of estrogen (E2) target proteins. 2. Nuclear E2-ER complexes are tethered through protein-protein interactions to a transcription factor complex (TF) that contacts the target gene promoter.
In This E2 exerts its function through transcriptional cross talk. 3. Estrogen-independent genomic actions are exerted through Growth factors (GF) by activating protein-kinase cascades, leading to phosphorylation (P) and with further leads to activation of nuclear ERs at EREs. 4. In nongenomic actions, membrane E2-ER complexes activate protein-kinase cascades, leading to altered functions of proteins in the cytoplasm, e.g. activation of eNOS, or to regulation of gene expression through phosphorylation (P) and activation of a TF.

1.9.1 ERE-Independent Genomic Actions:

ERs functioning by ERE-Independent genomic mechanism regulate the expression of a large number of estrogen-responsive genes that do not contain EREs. The mechanism is commonly used by other members of the nuclear receptor superfamily and is often referred to as transcriptional cross talk (Gottlicher M et al., 1998).

1.9.2 Non-Genomic Actions of Estrogens:

Non genomic actions are a common property of steroid hormones and are frequently associated with the activation of various protein-kinase cascades (Losel R et al., 2003). Earlier studies on non-genomic actions of estrogens are mediated through a sub-population of the classical ERs, ER alpha and ER beta that is located at the plasma membrane (Razandi et al., 1999 and Papa TC et al., 1995). The plasma membrane ERs exist as functional dimers when activated by estrogens (Razandi et al., 2004).

Increased levels of estrogen are shown to be sensitive in causing cancer in female population compared to male. So, such increases in estrogen levels are treated
by Anti-estrogen ligands (Lawrence Riggs et al., 2003). This includes selective estrogen-receptor modulators (SERMs), chemically diverse non steroid compounds which have tertiary structures that bind to the estrogen receptor. These compounds have either selective agonist or antagonist effects, depending on the target tissue. The molecular basis of SERM action and the tissue-selective agonist–antagonist effects is very useful in studying about the treatment strategies for curing breast cancer among women. Tamoxifen, Raloxifene are the mainly used SERMs in treating cancers in recent years.

Breast cancer growth is majorly of two types like estrogen dependent breast cancer and HER2 dependent breast cancer.

1.9.3 Role of HER2 and ER in Breast Cancer:

In about 1 of every 5 breast cancers, the cancer cells make an excess of HER2 (Human Epidermal growth factor receptor 2) due to a gene mutation. These gene mutation that are activating mutations that includes G309A, D769H, D769Y, V777L, P780ins, V842I, and R896C (Bose R et al, 2013)) and the elevated levels of HER2 occur in many types of cancer not only breast cancer. This is a gene mutation that occurs only in the cancer cells. HER2 gene which encodes for tyrosine kinase receptor that mediates critical signaling functions in normal and malignant breast epithelial cells. An acquired alteration consisting of amplification and overexpression of this gene product occurs in approximately 20 to 25% of human breast cancers. Breast cancer cells have a higher than normal level of HER2 on their surface, which stimulates them to cancerous activity. Around 15–20% of invasive breast cancers are called HER2
positive. HER2-positive breast cancers tend to be more aggressive than other types of breast cancers. These are also less responsive to hormone treatment. Breast cancer has been associated with exposure to estrogen, not all breast cancers are responsive to this hormone and its analogs. The response of cells to estrogen depends on whether they have estrogen receptors or not. Breast cancers are classified by their estrogen receptor status (estrogen receptor positive (ER+ve) or estrogen receptor negative (ER-ve)). About 70-80% invasive breast cancers are ER +ve (Bo Huang et al, 2013). This classification is done to assist in the selection of appropriate therapies to which some ER +ve cancers respond favorably to hormone blockers while ER -ve cancers do not. Several studies have found that ER+ve and ER-ve breast cancers have distinctly different risk factors and therefore, possibly different etiologies. Majority of the breast cancers are mediated by receptors ER and HER2, of which HER2-positive breast cancers are less responsive to hormone treatment and estrogen positive breast cancers are cured by selective estrogen receptor modulators (SERMs).

1.9.4 Breast cancer chemoprevention:

Several chemotherapeutic drugs have been studied for lowering breast cancer risk. Tamoxifen blocks the effect of estrogen on breast tissue; it acts like estrogen in the bones, heart, breast and uterine tissues. Survival rate of tamoxifen-treated women increases by as much as 50% compared to control breast cancer women. Contrastingly in the postmenopausal endometrium, tamoxifen appears to act as a weak ER (estrogen receptor) agonist, resulting in enhanced proliferation and an increased incidence of endometrial pathologies, including endometrial cancer. Raloxifene is similar to
tamoxifen in its effect on the breasts, bones, and heart. However, it has the opposite effect on the uterus that is, raloxifene blocks estrogen's effect in uterus & unlike tamoxifen it does not increase a woman's risk of endometrial cancer. Raloxifene can reduce a woman’s risk of breast cancer by 50 to 70 percent. However, tamoxifen is the only chemoprevention option available today for breast cancer since raloxifene is in its approval stages in treatment of breast cancer (Prichard et al., 2003). Hence, focus on the above two or three SERMs will help to treat breast cancer in a very effective manner. These drugs function effectively when prescribed in combinations rather than using as a single drug, which helps in giving right treatment after an appropriate diagnosis by clinical physician (Carol Fabian et al., 2007).

HER2 positive breast cancer is treated in a similar way to other breast cancers by targeted therapies using Trastuzumab (Herceptin). Herceptin locks onto the HER 2 protein. This blocks the receptor and stops the cells from dividing and growing.

1.9.4.1 Other drugs:

Some studies have found that women medicated with aspirin or non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen seem to have a lower risk of breast cancer. Studies have also found if drugs called bisphosphonates may lower the risk of breast cancer. Bisphosphonates are mainly used to treat osteoporosis but they are also used to treat breast cancer that has spread to the bone. These as well as several other drugs and dietary supplements are being studied to see if they can lower breast cancer risk.
1.9.4.2 Surgical Interventions:

For the few women who have a very high risk for breast cancer, the treatment option is to remove the breasts.

1.10 Role of Herbal Medicine:

From the past observations of the cancer studies till today there is no particular drug available for the breast cancer therapy and the drugs that are available today have many side effects and may lead to other cancers. Plant derived drugs have fewer side effects when compared with synthetic drugs (De smet et al., 1997).

Plants contain numerous biologically active compounds, many of which have been shown to have antimicrobial and other disease curing properties. Plant derived medicines have been part of traditional healthcare in most parts of the world for thousands of years and there is increasing interest in plants as sources of agents to fight several diseases.

Herbal medicine has such an extraordinary influence that numerous alternative medicine therapies treat their patients with herbal remedies like; Unani and Ayurveda and have gained importance in recent years. Approximately 25 percent of all prescription drugs are derived from plants. Recently the major pharmaceutical companies have moved into investing in natural product research. Because, natural products are still being developed into drugs. In fact natural products, natural product derivatives, and synthetic compounds containing a natural product pharmacophore make up 38% of the small molecule pharmaceutical companies out of which 52% are anticancer drugs.
1.11 Scope of the Present Study:

Estrogen is an important steroidal hormone which regulates the reproductive and physiology of female. The present study is aimed to extract, purify and characterize the steroid like bioactive compound from bark of Saraca indica and to analyze its functional role by in-silico (molecular docking and dynamics studies) and exvivo (MCF7 cell lines) analysis. Bark of Saraca indica is used in several traditional medications like ayurveda. This study provides a proof of evidence about steroid bioactive compound as probable anti-cancerous compound. SERMs and estrogen serves as positive and negative controls in analyzing the mechanism of action of bioactive steroid compound from bark of Saraca indica using Insilco and Ex-vivo studies.

Objectives of the Work:

1. Extraction and purification of bioactive compound/s from bark of Saraca indica.
2. To characterize the extracted bioactive compound/s using various biophysical methods for E2 like action.
3. To perform Insilco analysis of ketosterol and it’s binding possible interacting partners in cells. Analyzing ketosterol interactions with estrogen receptors and also known drugs like Tamoxifene and Raloxifene (SERMs).
4. To evaluate therapeutic significance of ketosterol in Breast cancer by analyzing its toxicity on MCF7 cells.