2. Review of Literature

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2.1. Vegetables

Vegetables are edible portions of herbaceous plants that serve as food by providing energy and nutritional supplements such as vitamins, minerals, protein, carbohydrate and amino acids. Different parts of vegetable plants are used as food that include - root (Eg. Carrot, Radish); stem (Eg. Potato, Taro); leaf (Eg. Cabbage); flower (Eg. Cauliflower); fruit (Eg. Tomato, Okra) and young seeds (Eg. Pea). These diverse groups of vegetables do more than any other group of foods, by adding appetising colour, texture and flavour to our daily food.

Vegetable plants do provide multiple uses to human such as: the fibrous material from dried fruits of *Luffa* sp., is widely used as bath sponge, shock absorber and as excellent carrier for immobilization of microbial, plant and animal
cells (Liu et al., 1999; Roble et al., 2003; Chen et al., 2003a and Bal et al., 2004); oil from *Cucurbita pepo* seed is rich in omega-3-fatty acids and said to have medicinal properties (Hernandez and Guerra, 1997); betanin, a red pigment obtained from the peel of *Beta vulgaris* is used as food colourant (Grubben and Denton, 2004); purple pigment from *Solanum melongena* peel is an efficient natural sensitizer for dye-sensitized solar cells compared to synthetic pigments (Giuseppe and Gaetano, 2008) and the dried fruit of *Lagenaria siceraria* is used to make various musical instruments and used as bottle, utensil or pipe (Heiser, 1985). After harvesting the edible part of vegetable plants, all the remaining parts are used as livestock feed.

### 2.2. Recognition of vegetables in human health

Goldman (2003) characterizes the recognition of vegetables in human health in three stages. The first stage concerns the observation that many vegetable plants were originally domesticated for their medicinal property. Making their way into the diet for this purpose, vegetable plants remained on the fringe from a culinary point of view. The second stage began when the role of vitamins became more widely understood, and vegetable plants were quickly recognised as a rich source of certain vitamins, minerals and fiber. The third stage was characterized by the recognition that, vegetable plants contain compounds that have the potential to influence health beyond nutritional value and this is known as functional foods.

The term ‘Functional food’, was first used in 1980 in Japan. There is a government approval process for functional foods called Foods for Specified Health Use (FOSHU). Functional food is a natural or processed food that contains known biologically-active compounds which when in defined quantitative and qualitative amounts provide a clinically proven and documented health benefit, and thus, an important source in the prevention, management and treatment of chronic diseases of the modern age (Arai, 1996). For example, Lycopene, the primary carotenoid from tomatoes is associated with reduced cancer risk (Sloan, 2000). Clinical and *in vitro* data to some degree, supported the claims that certain foods have the potential to deter disease, however much research remains to be conducted in order to definitively answer specific dietary based questions about food and health.
2.3. Vegetable phytochemistry

Phytochemicals are naturally occurring, biologically active chemical compounds. Diverse variety of phytochemicals in vegetables represents the product of primary and secondary metabolism. Primary metabolites such as carbohydrates, amino acids, fatty acids and organic acids are involved in respiration, photosynthesis, hormone synthesis, protein synthesis and thus serving in growth and development. Secondary metabolites such as flavonoids, carotenoids, phytosterols, phytoestrogens, alkaloids, glucosinolates, saponins, tannins, terpenoids determine the colour of vegetables; protect plants against herbivores and microorganisms; attract pollinators and seed dispersing animals and act as signal molecules under stress conditions (Seiger, 1998 and Crozier et al., 2006). Besides the importance for the plant itself, such metabolites determine the nutritional quality of food, and pharmacological properties - antioxidative, anticarcinogenic, antihypertensive, anti-inflammatory, antimicrobial, immune stimulating, antihyperglycemic, cholesterol lowering property and so on (Hounsome et al., 2008).

2.4. Traditional medicinal values

Consuming a diet rich in vegetables will provide a milieu of phytochemicals that possess health protective effects. They are rich sources of vitamins, minerals, fibres and generally low in fat and proteins. Major contribution of vegetables to human health are the large quantity of vitamin C, A and folic acid. Folic acid helps in multiplication and maturation of cells, and its deficiency results in certain types of anemia, especially of infants and pregnant women (Forshaw and Harwood, 1963).

Many vegetables have been consumed traditionally to treat various diseases: *Momordica charantia* for antidiabetic (Raman and Lau, 1996), *Raphanus sativus* for antidiarrheal and to remove gallstones and kidney stones (Kala et al., 2005), *Glycine max* for anticancer (Shu et al., 2009), *Colocasia esculenta* for antidiarrheal (Prajapati et al., 2011).

Different medicinal properties have been attributed to south-Asian vegetables (Fig.1) by Pieroni et al. (2007).
Fig. 1-Perceived medicinal properties of the recorded traditional South-Asian vegetables (Pieroni et al., 2007).

2.5. Dietary transition and emergence of diseases

Diet and lifestyle are major factors that influence susceptibility to many diseases. Dietary transition or nutrition transition is occurring worldwide due to increased consumption of unhealthy food, which is rich in saturated fat of animal origin, more added sugars and industrially processed and packed foods resulting in malnutrition. This transition is accompanied by the sedentary life style, urbanisation and rapid economical development. Nutrition transition in conjunction with epidemiological transition, leads to health problems such as obesity, chronic diseases like, cardiovascular diseases, diabetes, cancer (Drewnowski and Popkin, 1997 and Popkin, 2001 & 2002). Unhealthy diet with nutritional deficiency is the root cause for the occurrence of both communicable and non-communicable diseases.

2.6. Non-communicable diseases

Non-communicable diseases (NCDs) are known as non-infectious, which are caused either by environmental factors, nutritional deficiencies or genetic inheritance. NCDs due to nutritional and environmental factors are the World’s biggest issue by causing 35 million deaths each year compared to genetic factor.
NCDs mainly cardiovascular diseases, various types of cancers, chronic respiratory diseases and diabetes mellitus represent a leading threat to human health (WHO, 2008).

According to World Health Organisation (WHO) report 2008, NCDs are emerging globally as a series of macroeconomic and development challenge because of the related loss of productivity, rapidly raising health care costs, and their links with poverty. It estimates that annually 8 million people die before the age of sixty around the globe from diseases which largely shared the risk factors like unhealthy diet, tobacco use, physical inactivity and the harmful use of alcohol. Total deaths from NCDs are projected to increase by further 17% over the next ten years. The worldwide burden of one of the NCDs, diabetes mellitus has also been increasing rapidly along with increase in obesity and cardiovascular diseases.

**2.6.1. Incidence of Diabetes**

In 1985, it was estimated that approximately 30 million people worldwide had diabetes (International Diabetes Federation, 1999), a decade later this estimate had risen to 135 million (King *et al.*, 1998). In the year 2000, the global burden of diabetes was estimated as 171 million and this is projected to increase to at least 366 million by the year 2030 (WHO, 2003). A large proportion of this increase is expected to occur in developing countries due to population growth, aging.
physical inactivity, obesity and mainly unhealthy diet. The impact is expected to be greatest in China and India (Lydia, 2005). World diabetes day is observed on 14th of November every year to create awareness and control the disease rate.

2.6.2. Diagnosis and treatment

Diagnosis is done by testing glucose level in the blood during fasting. Normal glucose level is below 100 mg per decilitre; 100 to 125 mg per decilitre is prediabetic and above 125 mg per decilitre indicates diabetes. Other than glucose test, C-peptide test, thyroid blood test, oral glucose test are used to confirm different types of diabetes. Glycosylated haemoglobin test is used to assess long term control of glucose (Peters et al., 1996 and Samreen, 2009). In conventional therapy, type I diabetes is treated with exogenous insulin and type II with oral hypoglycaemic drugs (Table-1). Though different types of oral hypoglycaemic drugs are available along with insulin for the treatment of diabetes, there is an increased demand by patients to use natural products with antidiabetic property, as drugs used in the treatment end up with side effects in the body (Jack, 2003).

Table 1 – Antidiabetic drugs, their mechanism and side effects (Jack, 2003)

<table>
<thead>
<tr>
<th>S.no.</th>
<th>Antidiabetic drug</th>
<th>Function/mechanism</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Insulin</td>
<td>Functions as substitute for the endogenous hormone</td>
<td>Hypoglycaemia, headache, redness at injection site, rapid heart rate, weakness.</td>
</tr>
<tr>
<td>2</td>
<td>Sulfonylureas</td>
<td>Stimulates insulin secretion</td>
<td>Hypoglycaemia, weight gain, hypersensitivity, heart attack.</td>
</tr>
<tr>
<td>3</td>
<td>Biguanides (Metformin)</td>
<td>Decrease production of glucose</td>
<td>Diarrhea, nausea, abdominal pain, lactic acidosis, vitamin B12 deficiency.</td>
</tr>
<tr>
<td>4</td>
<td>Acarbose, Miglitol inhibitor</td>
<td>Alpha glucosidase inhibitor</td>
<td>Flatulence, diarrhea, nausea, gastrointestinal symptoms, hepatitis.</td>
</tr>
<tr>
<td>5</td>
<td>Thiozolidinediones (Pioglitazone, Troglitazone)</td>
<td>Agonists of peroxisome proloferator activated receptor gamma</td>
<td>Hepatotoxicity, coronary heart disease, bladder cancer, edema.</td>
</tr>
</tbody>
</table>
2.7. Communicable diseases

Communicable diseases (CDs) are transmissible, caused by pathogenic organisms in host with characteristic symptoms of the disease. Infectious pathogens include some viruses, bacteria, fungi, protozoa and multicellular parasites. CDs cause 15 million deaths each year worldwide (WHO, 2006). Though pathogenic virulent factors are the major cause of CDs, the baseline cause for all is low level of immunity, due to unhealthy diet habits.

Nutritional deficiencies generally reduce the capacity of the host to resist the consequences of infection and suppress it. An aggravation of disease is the expected result in man whenever nutritional deficiency is sufficiently severe. Such effects were also observed in most studies with laboratory animals, particularly when infection is due to bacteria, protozoa and intestinal helminths (Nevin et al., 1968).

2.7.1. Treatment

CDs are prevented by vaccination and also treated by antibiotic and antimicrobial agents. Vaccines work by infecting the host with a weakened or avirulent strain of the pathogen, which induce the host body to produce antibodies to fight against diseases.

Several hundreds of compounds with antibiotic activity have been isolated from microorganisms and other natural sources over the years, but only a few are clinically useful due to selective toxicity. A drug must be highly effective against the pathogen and not to human, i.e. known as selective toxicity. Most of the antibiotics in clinical usage are directed against either pathogen’s cell wall synthesis, protein synthesis or nucleic acid syntheses, which are unique in some ways to pathogens. The mechanism of action of different types of antimicrobial drugs against bacterial, fungal and mycobacterial pathogens and their side effects are given in Table-2 a, b & c.
Antibiotics are screened for any negative effects on human or other mammals before approval for the clinical use and are considered as safe drugs. However, some drugs are associated with a range of adverse effects in the host, like fever, nausea and major allergic reactions even leading to death (Slama et al., 2005). Apart from these aspects, the treatment of CDs is worsening by the evolution in pathogens of acquiring resistance to antimicrobial drugs.
Table 2b – Mechanism of antifungal drugs with their side effects (Mahmoud and Louis, 1999)

<table>
<thead>
<tr>
<th>S.no.</th>
<th>Antifungal drug</th>
<th>Mechanism</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Amphotericin B, Nystatin</td>
<td>Interrupts membrane permeability</td>
<td>Hypotension, anorexia, nausea, vomiting, headache, dyspnea, drowsiness, hepatotoxic, nephrotoxic, cardiac failure.</td>
</tr>
<tr>
<td>2</td>
<td>Azoles (Ketaconazole, Fluconazole, Clotrimazole)</td>
<td>Blocks synthesis of ergosterol</td>
<td>Liver-damage, affecting estrogen levels, anaphylaxis, inhibitors of the cytochrome P450, calcium channel blockers, immune suppressants.</td>
</tr>
<tr>
<td>3</td>
<td>Amorolfine</td>
<td>Inhibits lanosterol synthesis</td>
<td>Skin irritation with redness and itching, slight transient burning sensation, hypersensitivity reactions.</td>
</tr>
<tr>
<td>4</td>
<td>Echinocandins (Caspofungin, Micafungin, Anidulafungin)</td>
<td>Inhibits beta (1,3) D-glucan synthesis</td>
<td>Nausea, diarrhea, headache, fever, thrombo phlebitis, increased plasma creatinine, hypokalemia, increased liver enzymes, rashes, facial edema, pruritus.</td>
</tr>
</tbody>
</table>

Table 2c – Mechanism of antituberculosic drugs with their side effects (Ma et al., 2007)

<table>
<thead>
<tr>
<th>S.no.</th>
<th>Antituberculosic drug</th>
<th>Mechanism</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Ethambutol</td>
<td>Inhibits arabinosyl transferase</td>
<td>Optic neuritis, Red-green color blindness, peripheral neuropathy, Arthralgia, hyperuricaemia, Vertical nystagmus.</td>
</tr>
<tr>
<td>2</td>
<td>Isoniazid</td>
<td>Inhibits cell wall macolic acid synthesis</td>
<td>Rashes, abnormal liver function tests, hepatitis, sideroblastic anemia, high anion gap metabolic acidosis, peripheral neuropathy, phenytoin (Dilantin), disulfiram (Antabuse) levels and intractable seizures.</td>
</tr>
<tr>
<td>3</td>
<td>Rifampsin</td>
<td>Inhibits RNA polymerase</td>
<td>Hepatitis, jaundice, liver failure in severe cases, breathlessness, flushing, pruritus, rash, redness and watering of eyes, nausea, vomiting, abdominal cramps with or without diarrhea, fever, headache, arthralgia, malaise and dysphoria.</td>
</tr>
<tr>
<td>4</td>
<td>Aminosalicyclic acid</td>
<td>Inhibits folic acid synthesis</td>
<td>Nausea, vomiting, diarrhoea, hepatitis, haemolysis, thyroid goitre.</td>
</tr>
<tr>
<td>5</td>
<td>Cycloserine</td>
<td>Inhibits peptidoglycan synthesis</td>
<td>Headache, irritability, depression, psychosis convulsions, toxic.</td>
</tr>
</tbody>
</table>
2.7.2. Emergence of drug resistant pathogens

The treatment of microbial infections is increasingly complicated by the ability of pathogens to develop resistance to antimicrobial agents. They acquire resistance by *de novo* mutation or *via* the acquisition of resistant genes from other organisms through horizontal or vertical gene transfers. Acquired resistance enables the pathogen: to produce enzymes that destroy the antimicrobial drugs; to express efflux system to prevent the drug from reaching its intracellular target; to modify the drug’s target site; to produce an alternative metabolic pathway that bypasses the reaction of the drug; or up regulates the target enzyme to reduce the concentration of the drug (Fred, 2006).

Antibiotic resistant organisms such as *Trichosporon beigeli* (Walsh et al., 1990); *Cryptococcus neoformans* (Alves et al., 1997); *Candida albicans* (Marichal et al., 1999); *Candida dubliniensis* (Sanglard and Bille, 2002); *Pseudomonas aeruginosa* (Schweizer, 2003); *Staphylococcus aureus* (Severin et al., 2004); *Escherichia coli* (Pitout et al., 2004; Woodford et al., 2004); *Candida parapsilosis* (Pfaller and Diekema, 2004); *Klebsiella pneumonia* (Ananthan and Subha, 2005); *Candida tropicalis* (Pfaller et al., 2005); *Mycobacterium tuberculosis* (Ormerod, 2005); *Aspergillus flavus* (Sabatelli et al., 2006); *A.fumigatus* (Messer et al., 2006); *Fusarium* sp., (Sabatelli et al., 2006); and *Candida krusei* (Pfaller and Diekema, 2007) are reported for their widespread occurrence and their adaptability to antibiotics by resistant mechanism(s).

2.7.3. Impact of drug resistance with special reference to *S.aureus* and *M.tuberculosis*

*S.aureus* is an opportunistic pathogen often carried asymptptomatically in the human body. It was first reported in 1961 for resistance to the antibiotic methicillin, which was used to treat Staphylococcal infections. Prior to methicillin, penicillin was used to treat such infections, but this organism
developed resistance by producing beta lactamase, after which methicillin was the only hope. But the pathogen acquired a gene called Mec A that codes for penicillin binding proteins, which gave them resistance to methicillin and essentially all other beta-lactum antibiotics. (Fitzgerald et al., 2001; Duquette and Nuttal, 2004; Weese, 2005). The methicillin resistant strain is called as Methicillin Resistant Staphylococcus aureus (MRSA). About 32 % MRSA infections was reported in India (Arakere et al., 2005).

Until recently vancomycin is the only antibiotic available for treating MRSA infections (Fitzgerald et al., 2001). But vancomycin resistant MRSA strains have increasingly been reported (Tenover and Goering, 2009).

**Tuberculosis** (TB) is among the top three causes of death from a single infectious agent along with malaria and HIV. One third of the world’s population is infected with *M. tuberculosis* (Raviglione et al., 1995). India alone accounted for 23.24% of global burden of tuberculosis (World Health Report, 2006). TB is treated with an initial intensive two month regime comprising of multiple antibiotics- rifampicin (RIF), isoniazid (INH), pyrazinamide (PZA) and ethambutol (EMB) or streptomycin (SM), to ensure that mutants resistant to a single drug do not emerge (MMWR,1993). In the next four months, only RIF and INH were used to eliminate any persisting tubercule bacilli. INH and RIF, the two most potent antituberculous drugs, kills more than 99% of bacilli within two months of initiation of therapy (Mitchison, 1985; Iseman and Madsen, 1989). Along with these two drugs, PZA with a high sterilizing effect appears to act on semidormant bacilli not affected by any other drugs (Heifets and Lindholm, 1992).

Using these drugs in conjunction with each other reduces the antitubercular therapy from 18 months to 6 months. Therefore, the emergence of strains resistant to either of these drugs causes a major concern, as it leaves only
drugs that are far less effective, have more toxic side effects, and result in high death rates.

*M. tuberculosis* developed resistance to multi drugs - RIF and INH simultaneously (Vareldzis *et al*., 1994). The main causes involved in the development of such multidrug resistant tuberculosis (MDR-TB) are: single use of a drug; inadequate and incomplete treatment; noncompliance; variations in bioavailability of drug and failure to identify pre-existing resistance (Espinal *et al*., 2001). Mechanism of resistance was reported as, due to: the mycobacterial cell wall decreases the permeability to many drugs; drug efflux systems, mutation in the target enzyme or in the gene and by titration of the drug through overproduction of the target (Davies, 1994).

Working out the exact biochemical details of drug–target interaction acquires considerable attention in the era of multidrug resistance where rational structure and mechanism based approaches to inhibitor design is reported to have possible results in drug discovery (Rattan *et al*., 1998). The action and mechanism of resistance to the important drugs are still not fully understood. However, current molecular evidence indicates that routine application of rapid molecular test for drug resistant TB is essential (Somoskovi *et al*., 2001)

### 2.8. Need for new leads

Rapid emergence of drug resistant pathogens and widespread occurrence of non-communicable diseases, in addition to adverse effects of drugs and lesser efficacy of drugs put us under pressure in searching for new potent leads to combat against such problems. In empirical method, developing a new drug needs more time and cost, that would be immense and at unexpectable range. This kind of drug development (Fig.2), is estimated to involve costs of about $880 million and 14 years of research before it is introduced in the market (Dickson and Gagnon, 2004).

To overcome the side effects and toxicity of the currently available drugs, new effective chemical entities have to be screened and identified in short duration. Traditionally developed or synthesized drugs have resulted in high attrition rates with failures attributed to poor pharmacokinetics (39%), lack of
efficacy (30%), animal toxicity (11%), adverse effects in humans (10%) and various miscellaneous factors. Such failure has to be avoided and their chances of success have to be implemented (Bharath et al., 2011).

To solve this problem, the process of rational drug discovery has been invented with the advent of genomics, proteomics, bioinformatics and efficient technologies through computational structure based drug designing method by rational approach.

2.9. Rational drug designing approach

Rational drug designing is used to discover and develop new drug compounds by applying computer based modeling technologies (Fig.3). A variety of computational methods is used to identify novel compounds; design compounds for selectivity, efficacy and safety; and develop them into clinical trial candidates. These methods fall into several categories such as – structure based drug design, ligand based drug design, de novo design and homology modeling depending on how much information is available about drug targets and potential drug compounds (Srinivasa and Srinivas, 2011). Computer aided drug discovery process comprises of three stages (Yun et al., 2006), as follows:

![Diagram of drug discovery process](image-url)
Stage 1: Identification of therapeutic target, lead molecule and building small molecular library to be tested against the target.

Stage 2: Selected hits or leads are checked for specificity by docking at binding sites of other known targets.

Stage 3: Toxicity study and pharmacokinetic analysis of leads and those molecules that pass these studies are termed as leads.

2.9.1. Target identification

Structure based drug discovery begins with the identification of the function of a potential therapeutic drug target and understanding its role in the disease process. Target is generally a protein or gene, which is involved in a particular metabolic or signalling pathway specific to a disease condition or survival of microbial pathogen. Either by upregulating or by downregulating the target using an appropriate drug, a disease can be cured.

Target is identified with the help of bioinformatics, chemoinformatics and data mining tools. Target of the lead can be identified based on the structural similarity of the lead with the target’s substrate or experimentally proved inhibitor of the target. In this approach, prediction of biological activity spectrum for substances (PASS) (www.ibmc.ru/PASS/) was developed as an online prediction server, which predicts more than 900 pharmacological effects and biochemical
mechanisms on the basis of the structural formula of a substance or lead. This may be efficiently used to find new targets for some ligands and conversely, to reveal new ligands for some biological targets (Filimonov and Poroikov, 1996).

PASS predictions were experimentally proved and reported as effective tool in target and lead discovery. Some examples of successful predictions are: cognition enhancers, anxiolytic and convulsion property (Geronikaki et al., 2003); antitumor property (Islyaikin et al., 1997; Pogrebnyak et al., 1998) and antibacterial property (Maiborode et al., 1998).

2.9.2. Target Validation

Target validation is about implication of a target in the disease process. In drug discovery process, the major bottleneck is target validation. If this process can be accelerated with the computational tools, the target validation step will speed up significantly. The target validation process includes determining the target’s function with desired clinical outcome, protein expression, potential binding sites, pharmacodynamic and in vitro assays (Bleicher et al., 2003).

2.9.3. Lead Identification

Identification of a lead molecule in rational drug discovery process is by Virtual High Throughput Screening (VHTS), and by pharmacophore search methods (Yun et al., 2006). In VHTS, protein targets are screened against a library of small molecules to analyse which molecule binds strongly to the target. If there is a “hit” with a particular compound, in can be extracted from the library for further testing (Satyajit et al., 2010).

Interaction of the target with the lead can be studied by using various bioinformatics software which is designed based on molecular simulations. Docking or interaction of target and lead results in “scoring” which determines strong or weak interaction between them. Molecular modeling technique helps to predict the position and orientation of a ligand when it is bound to a protein/target or receptor. Docking software used often in the analysis are - Auto dock (www.autodock.scripps.edu), Dock (www.dock.compbio.usf.edu), Gold (www.udc.cam.ac.ukl) and Accelrys - Discovery studio (Srinivasa and Srinivas, 2011).
Docking analysis was found to be effective for drug designing and few reported illustrations are: a novel terpenoid from *Elephatopus scaber* was analysed for antibacterial activity on *Staphylococcus aureus* using Hex docking software (Daisy *et al.*, 2008); T2384 was docked with peroxisome proliferator activating ribosome gamma protein (PPARγ) and compared with standard drug pioglitazone and proved as an antidiabetic agent (Li *et al.*, 2008); and salacinol derivatives were analysed for inhibition of α-glucosidase enzyme by docking analysis (Nakamura *et al.*, 2010).

### 2.9.4. Lead Optimization

When a promising lead candidate has been found, the next step is to optimize the structure and properties of the potential drug. This usually involves a series of modifications to the primary and secondary structure of the compound, based on Quantitative Structure Activity Relationship (QSAR). QSAR study correlates molecular chemical structure to biological activity. The underlying principle is that molecules with similar chemical structures will exhibit similar biological properties. QSAR is used to find out analogues for leads based on physico-chemical properties of the molecules and assists in optimizing the lead by altering the chemical structure of substitutes (Guha, 2008).

Based on QSAR analysis, drug candidates have been reported for their efficiency in drug discovery. A few examples reported are: sixty analogues of acylate derivatives were reported to have antimalarial relationship among them based on structural properties: hydrophobic, hydrogen bond acceptors and stearic property (Mohamed *et al.*, 2006); diverse classes of aromatase inhibitors used in the treatment of breast cancer were analysed and optimized by 3D QSAR study (Partha and Kunal, 2010); and betulinic acid derivatives were screened for their anti-HIV activity by structure activity relationship and revealed to have excellent potency (Ping *et al.*, 2011).

### 2.9.5. Preclinical testing

Preclinical studies and testing strategies with and without the use of animal testing methods have the purpose of limiting risks whenever a new active substance is to be used as a drug by humans. Most drug candidates fail in clinical
trials because of toxicity problems with metabolism even after many years of research and millions of dollars had been spent on them. The key characteristics for drugs are: absorption, distribution, metabolism, excretion and toxicity (ADMET) and efficacy, i.e., bioavailability and bioactivity. This kind of properties can be predicted using bioinformatics software modules such as C2, ADME, TOPKAT, CLOGP, drug matrix, Absolv, Bio print etc.(Srinivasa and Srinivas, 2011).

2.10. Need for Database

Development of a database and data mining are the base for the progress of drug discovery process. Compilation of the data on plants, their uses and their active substances with activity profile is necessary to retrieve meaningful informations from the random data by data mining.

Many databases have been developed in this regard and facilitated as web interfaces for everyone to access. Such databases are: (1) Dr. Dukes phytochemical and ethnobotanical database, which comprises data on phytochemical, a limited number of phytochemical activities and ethnobotanical uses (www.ars-grin.gov/duke1); (2) Phytochemical database developed by United States Department of Agriculture (USDA) enables one to search phytochemicals and the data contains- Chemical structure, formula and molecular weight of the phytochemical (www.pl.barc.usda.gov/); (3) Phytochemical database specific to 240 Chinese herbal constituents with known target specificities designed to provide informations about plant constituents and their pharmacological mechanisms (Ehrman et al., 2007); (4) Phytochemical database based on the plants used in ayurvedic treatment for diabetes mellitus (Sushilkumar et al., 2009).

All these reported databases provide informations about plants, their phytochemical profile, molecular informations and a few pharmacological activities, which are already tested by in vitro and in vivo experiments. For the rapid progression of drug discovery process, a specific database has to be developed for phytochemicals with their unexplored biological activities that will aid in revealing the new targets and potential leads.
2.11. Review on vegetables included in the present study

2.11.1. Description of the plants (Gamble and Fischer, 1921; Pande et al., 1995)

Description of the twenty-seven vegetables included for the present study is given in alphabetical order of their biological name with family name, as follows:

*Abelmoschus esculentus* (L.) Moench. (Malvaceae)

Common name - bhendi, okra. It is an annual herb, sturdy and woody, branching and nearly glabrous. Leaves are alternate, cordate-ovate, flowers are yellow with reddish centre, fruit is pyramidal-oblong capsule, longitudinally furrowed, hirsute and becomes woody when ripe. Seeds are round and tuberculate.

*Amorphophallus konjac* K. Koch. (Araceae)

Common name – elephant yam, konjac. It is a perennial plant growing from a large corm up to 25cm in diameter. The single leaf is up to 1.3m across, bipinnate and divided into numerous leaflets. The flowers are produced on a spathe enclosed by a dark purple spadix. This is used in asian fruit jelly snack preparation.

*Benincasa hispida* (Thunb.) Cogn. (Cucurbitaceae)

Common name – wax gourd, ash gourd. It is a large climber, hispid all over. Leaves are large, hispid, flowers solitary, large and yellow. Fruits are round, broadly cylindric, hairy, mature fruit glaborous and ash coloured.

*Beta vulgaris* L. (Chenopodiaceae)

Common name - beet root. This a biennial with swollen, rounded or tapering roots, dark red or yellow-green leaves arising in cluster from a shortened stem. Flowers produced in the second year of growth, which are usually borne in axillary clusters. They are small, bracteates, bisexual, actinomorphic and perigynous. The fruit is one seeded utricle.
Brassica oleracea var. botrytis L. (Brassicaceae)

Common name - cauliflower. This is a biennial, although grown as an annual crop. It has a very short stem bearing a large condensed, compact, swollen hemispherical head consisting of tightly packed leaves. Morphologically the head consists of a tightly packed aggregation of undeveloped white or creamy flower buds borne on the thick hypertrophied branches of the much branched inflorescence.

Brassica oleracea var. capitata L. (Brassicaceae)

Common name - cabbage. This is a biennial herb, grown as an annual crop. It has a short and stout stem bearing a mass of closely packed, thick, fleshy, overlapping leaves.

Brassica oleracea var. gongylodes L. (Brassicaceae)

Common name – kohlrabi, knolkhol. It is a low, stout cultivar of cabbage. It has been created by artificial selection for lateral meristem growth of wild cabbage plant.

Brassica rapa var. Rapa L. (Brassicaceae)

Common name – turnip. It is a biennial herb with a purplish white enlarged napiform root. The stem is extremely reduced, bearing a cluster of leaves at its top. The flowers are typical cruciferous.

Capsicum annuum L. (Solanaceae)

Common name – capsicum, bell pepper. It is an evergreen perennial plant. Leaves broad, long, alternate, flowers bisexual, fruit green and turns red when ripe. Flowers small, solitary, axillary, white or greenish, five parted, fruit shiny, berry of various colours.

Colocasia esculenta (L.) Schott. (Araceae)

Common name – taro. This is a tropical plant grown for its rhizomes of different shapes and sizes. Leaves sprout from rhizome, dark green above and light green beneath, triangular-ovate, subrounded and mucronate at apex with long petiole. The surface of the corms is marked by a number of rings representing the
nodes of the stem. Inflorescence is a spadix consisting of a much thickened axis which bears numerous unisexual flowers, the female at the base and the male above.

*Cucurbita pepo* L. (*Cucurbitaceae*)

Common name – pumpkin. This plant is indigenous to warm and temperate regions. It is a annual climber, with green, hairy, creeping stem. Leaves are large, dark green with prominent veins, pointed, toothed lobes. Flower is large, yellow colour and axillary. Fruit is large, round, orange pumpkin with numerous ovate seeds.

*Daucus carota* L. (*Apiaceae*)

Common name – carrot. This plant is erect, biennial herb, producing a thickened conical tap root and a whorl of decompounds leaves. The inflorescence is a terminal compound umbel.

*Glycine max* (L.) Merr. (*Fabaceae*)

Common name – soybean. Stem, leaves and pods are covered with fine brown or gray hairs. Leaves are trifoliate having leaflets. Leaves fall before the seeds are mature. The inconspicuous self-fertile flowers are borne in the axis of the leaf and are white, pink or purple. Fruit is a hairy pod in clusters, containing 2-4 seeds.

*Lagenaria siceraria* (Molina.) Standl. (*Cucurbitaceae*)

Common name - bottle gourd. It is a tendril climber with hollow angular stems, with large, long petioled and palmately lobed leaves. The yellowish unisexual flowers arise in the leaf axil. The fruit is a pepo with a hard rind which develops from the receptacular tissue.

*Luffa aegyptiaca* Miller. (*Cucurbitaceae*)

Common name – luffa. This is large climber with tendrils. Leaves orbicular in outline, palmately angled or sublobate, flowers yellow and large, male flowers axillary in racemes, female flowers solitary. Fruit is long, elevate-oblong, tapering towards the base.
**Lycopersicon esculentum** L. (Solanaceae)

Common name – tomato. It is an erect or trailing herb with imparipinnate leaves and small yellowish flowers borne in clusters on the main axis and on lateral branches. The fruit is fleshy and juicy berry.

**Momordica charantia** Descourt. (Cucurbitaceae)

Common name – bitter melon, bitter gourd. This is a herb with tendril, bears simple alternate leaves, flowers-separate male and female, yellow in colour, fruit is oblong with warty exterior, a thin layer of flesh surrounding a central seed cavity filled with large flat seeds and pith.

**Moringa oleifera** Lam. (Moringaceae)

Common name – drumstick. It is the most widely cultivated crop in Asia, Africa, Indonesia, America and Srilanka. It is a small or medium sized tree, the bark is grey and thick and looks like cork, peeling in patches, pinnate opposite leaflets, flowers pale green to white in colour, long fruit with ridges, which is triangular in cross section, slender pods, oily black winged seeds.

**Musa paradisiaca** L. (Musaceae)

Common name – musa, banana. This is widely distributed throughout the tropical regions. This is a giant perennial with an underground rhizome, large smooth glossy and light green leaves are borne on a pseudostem consisting of clasping leaf bases. The inflorescence that pushes out of the pseudostem, is a spike consisting of a stout panicle on which flowers are borne in nodal clusters in two rows in the axils of brightly coloured bracts. The lower 5-15 basal nodes bear female flowers, followed by neuter and finally male flowers towards the tip. Fruit is parthenogenic, in clusters known as ‘hands’, is a long curved berry.

**Phaseolus vulgaris** L. (Fabaceae)

Common name - bean. It is an annual herb, erect and bushy or twining, with a taproot and nitrogenous nodules, trifoliate leaves, white or coloured flowers, slender pod with glabrous seeds. Beans are highly nutritive, relatively low cost protein food.
**Pisum sativum** L. (*Fabaceae*)

Common name – pea. This plant is a climbing or trailing annual herb with pinnately compound leaves and foliaceous stipules. Each leaf has up to three pairs of leaflets, and terminated by branched tendrils. Flowers are white-pinkish and are borne single or in few flowered axillary cymes. The pods are somewhat swollen, long (6-10 cm), contain 4-10 seeds. Seeds are angular or globose and smooth or wrinkled.

**Raphanus sativus** L. (*Brassicaceae*)

Common name – radish. It is an annual or biennial herb with a rosette of lyrate, pinnatifid leaves arising from a short stem. At the time of flowering, the condensed stem grows out into an elongated peduncle bearing white to lilac, cruciform flowers. The fruit is fleshy siliqua with a long conical beak.

**Solanum melongena** L. (*Solanaceae*)

Common name - eggplant, brinjal. It is a much branched perennial herb but usually grown as an annual. Leaves are simple, alternate, ovate-oblong with sinuately lobed margins. The light purple-bluish flowers are solitary. Fruit is large, usually ovoid-oblong, white, yellow to deep purple berry.

**Solanum tuberosum** L. (*Solanaceae*)

Common name – potato. It is an erect, branching and spreading annual, attaining a height of 1-1.25 m. The leaves are pinnately compound and densely hairy when young. Tubers are underground stems and bear buds in the axils of scale like leaves, which have shed soon, leaving a rudimentary leaf scar. Flowers are borne in terminal clusters and the fruit is berry.

**Tamarindus indica** L. (*Fabaceae*)

Common name – tamarind. It is a long-lived, medium height, bushy tree. Leaves are evergreen, elliptical, alternate, pinnately compound type, flowers elongated, red colour in small racemes. Fruit is an indehiscent pod, with hard, brown shell, fleshy acidulous pulp and contains 6-12 seeds which are flattened and glossy brown.
Review of Literature

*Trichosanthes anguina* L. (*Cucurbitaceae*)

Common name – snake gourd. It is a tropical or subtropical plant, climber, slender stem with tendrils, leaves long, orbicular-reniform, deeply lobed, male flowers in axillary racemes with long peduncle, solitary axillary female flowers white in colour. Fruit is long and contorted, green with white strips.

*Vicia faba* L. (*Fabaceae*)

Common name – broad bean. It is a rigid erect plant with stout stems with a square cross section. The leaves are long, pinnate. Flowers are long with five petals, the standard petal is white while the wing petals are white with a black spot. The fruit is broad, leathery pod, containing 3-8 seeds.

### 2.11.2. Phytochemical profile of the plants included in the study

Phytochemicals present in the selected vegetable plants include primary metabolites and an extensive range of secondary metabolites. Some common metabolites in the selected vegetable plants are: carbohydrate; protein; fibre; fatty acids (linoleic acid, linolenic acid, oleic acid, stearic acid, palmitic acid, erucic acid and arachidic acid); amino acids (methionine, thiamine, tryptophan, analnine, arginine, histidine, isoleucine, leucine, proline, serine, threonin, tyrosine, cysteine and valine); vitamins (A, B, C and E); minerals (calcium, potassium, magnesium, iron, copper, sulphur, phosphorous and chloride); organic acids; pigments; polyphenols, flavonoids; alkaloids; glycosides; terpenoids; phytoestrogens; phytosterols; saponins and tannins. The quantity of these metabolites and the part of the plant in which they occur differ (Duke and Atchley, 1984).

Along with these metabolites, vegetables are reported to possess some unique metabolites (Table-3).
Table 3 - List of some unique phytochemicals present in the vegetables included in this study

<table>
<thead>
<tr>
<th>Vegetable name</th>
<th>Unique phytochemical name</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Abelmoschus esculentus</em></td>
<td>12,13-epoxyoleic acid; 9-hexadecenoic acid; gossypetin; gossypol; cyaniding-3-glucoside-4'-glucoside; cyanidine-4''-glucoside</td>
<td>Duke and Atchley, 1984; Lengsfeld <em>et al</em>., 2004; Adelekun <em>et al</em>., 2009</td>
</tr>
<tr>
<td><em>Amorphophallus konjac</em></td>
<td>Glucomannan; soluble oxalates</td>
<td>Duke and Atchley, 1984; Chattopadhyay <em>et al</em>., 2009.</td>
</tr>
<tr>
<td><em>Beta vulgaris</em></td>
<td>Allantoin; betaine; betaxanthine; vulgaxanthine I and II; coniferin; raphanol</td>
<td>Duke and Atchley, 1984; Medani <em>et al</em>., 2000.</td>
</tr>
<tr>
<td><em>Brassica oleracea</em> var. Botrytis*</td>
<td>Glucobrassicin; glucosinolates; glucoraphanin; glucoraphin; glucosaturtin; glucoraphin; progoitrin.</td>
<td>Duke and Atchley, 1984; Fowke, 2007.</td>
</tr>
<tr>
<td><em>Brassica oleracea</em> var. Capitata*</td>
<td>Glucosinolates; brassinin; brassitin; 1-methoxybrassinin; 4-methoxybrassinin; 1-methoxybrassinin; 1-methoxybrassinin A &amp; B; cyclo brassinin; brassicanal; camalexin.</td>
<td>Duke and Atchley, 1984; Muhammad <em>et al</em>., 2009.</td>
</tr>
<tr>
<td><em>Brassica oleracea</em> var. gongylodes</td>
<td>Sulforaphane; indole-3-carbinol; zeaxanthin.</td>
<td>Duke and Atchley, 1984.</td>
</tr>
<tr>
<td><em>Capsicum annuum</em></td>
<td>Cycloeicosane; capsaicin; dihydrocapsaicin; homocapsaicin; homohydrocapsaicin; nor dihydrocapsaicin; apiin; luteolin.</td>
<td>Duke and Atchley, 1984; Wesolowska <em>et al</em>., 2011.</td>
</tr>
<tr>
<td><em>Colocasia esculenta</em></td>
<td>Luteolin-7-O-glucoside; nonacosane; cyaniding-3-glucoside; 12,13-trihydroxy, (E)-10-octadecanoic acid; vitexin; isovitexin</td>
<td>Duke and Atchley, 1984; Iwashina <em>et al</em>., 1999; Prajapati <em>et al</em>., 2011</td>
</tr>
<tr>
<td><em>Cucurbita pepo</em></td>
<td>Vomifoliol; dehydrovomifoliol; neo-xanthin; rhamnoin-3-rutinoside</td>
<td>Duke and Atchley, 1984; Gohari <em>et al</em>., 2011.</td>
</tr>
<tr>
<td><em>Daucus carota</em></td>
<td>Carotene; daucic acid; heracelenin; falcarinol; falcarindiol-3-acetate; betaine; daucene; diosgenin; eugenin; umbelliferone; umbelliferose.</td>
<td>Duke and Atchley, 1984; Mehmet and Jean, 2007; Zaini <em>et al</em>., 2012.</td>
</tr>
<tr>
<td><em>Lagenaria siceraria</em></td>
<td>Cucurbitacin B, D, E, G and H; aglycones; fucosterols; lagenin; 22-deoxycurcurbitacin; flavones-C-glyco-sides; bryonolic acid</td>
<td>Duke and Atchley, 1984; Baranoswka and Cisowski, 1994; Shirwaikar and Srinivasan, 1996; Shah <em>et al</em>., 2010 and Shah and Seth, 2010.</td>
</tr>
</tbody>
</table>

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2.11.3. Pharmacological properties of the plants included in the study

Extensive reports on pharmacological properties of the selected vegetable plants are reviewed below. Most of the reports are on crude extracts of the plants with different solvents. A few isolated compounds that were screened for their biological property, which is based on traditional knowledge or trial and error method or based on literature.
Antioxidant (Tsuda et al., 1994; Sudheesh et al., 1999; Chu et al., 2002; Niwa et al., 2002; Irena and Magorzata, 2003; Ravindra and Narayan, 2003; Jessica et al., 2004b; Nkosi et al., 2006; Rasal et al., 2006; Fernandes et al., 2007; Yin et al., 2008; Adelakun et al., 2009; Gacche et al., 2010; Bhutkar and Bhise, 2011; Naresh et al., 2011; Leelaprakash et al., 2011; Ordonez et al., 2011; Ponnusha et al., 2011; Ramesh et al., 2011; Sangeetha, 2011; Spanou et al., 2012);

Antidiabetic (Keji, 1981; Yoshikawa et al., 1996; Grindley et al., 2002; Jenkins et al., 2003; Kar et al., 2003; Adolfo and Michael, 2005; Kwon et al., 2008; Vimala et al., 2008; Ali and Agha, 2009; Dolly et al., 2009; Rai et al., 2009; Banafsheh et al., 2010; Jaysree et al., 2011a; Pimple et al., 2011; Sabitha et al., 2012; Saha et al., 2012);

Antidiuretic (Shaw and Jana, 1982; Mahran et al., 1991; Vered et al., 1997; Maryam et al., 2003; Heber, 2004; Ghule et al., 2007; Jain et al., 2007; Jaysree et al., 2011b; Vaishali et al., 2011);

Analgesic (Biswas et al., 1991; Ghule et al., 2007; Choudhary et al., 2010; Ashish and Yadav, 2011; Naresh et al., 2011);

Antibacterial (Sofowora, 1993; Monica et al., 2003; Lim et al., 2004; Sabahat and Perween, 2005; Peyvast and Khorsandi, 2007; Hatil and Sana, 2009; Archana, 2011; Velicanski et al., 2011; Carla et al., 2011; Manju et al., 2011; Sureka et al., 2011);

Antidiarrheal (Emery et al., 1997; Rabbani et al., 1999; Mathad et al., 2005);

Antifungal (Dixit and Tripathi, 1975; Sisti et al., 2003; Yang and Yeh, 2005; Fagbemi et al., 2009; Tamanna, 2010);

Antihypertensive (Faizi et al., 1992; Orie, 1997; Muhammad and Anwarul, 2007; Vishal and Subhash, 2010);

Anticancer (Pal et al., 1968; DeLorenzo et al., 2001; Brown et al., 2005; Salman et al., 2007; Vaishampayan et al., 2007; Pranay et al., 2009; Tang et al., 2009; Dan and Gu, 2010; Shokrzadeh et al., 2010; Saha et al., 2011);
Antipyretic (Mutalik *et al.*, 2003; Izquierdo *et al.*, 2007; Zulfkar *et al.*, 2009);

Anti-inflammatory (Caceres *et al.*, 1992; Kolte *et al.*, 1997; Rimbau *et al.*, 1999; Shah *et al.*, 2007; Leticia *et al.*, 2009; Dave *et al.*, 2010; Karpagam *et al.*, 2011; Shubangi *et al.*, 2011);

Antiobesity (LeBerre *et al.*, 1997; Bin *et al.*, 2005; Vimala *et al.*, 2008; Farhat *et al.*, 2011);

Antihypercholesteromic (Venter *et al.*, 1987; Bonilla *et al.*, 1998; Guimaraes *et al.*, 2000; Mehta *et al.*, 2003; Takai *et al.*, 2003; Mallick *et al.*, 2007; Ozlem and Refiye, 2010; Afify *et al.*, 2011; Dosari *et al.*, 2011);

Antidepressant (Patroganesh *et al.*, 2009; Kalariya *et al.*, 2010; Millind and Suman, 2010; Prajapati *et al.*, 2011; Umadevi *et al.*, 2011);


Anthelminthic (Elisha *et al.*, 1987; Chiranjib *et al.*, 2010; Devraj *et al.*, 2011);

Wound healing (Agarwal *et al.*, 2009; Sharma *et al.*, 2010; Anita *et al.*, 2011; Abirami *et al.*, 2011; Mohammad *et al.*, 2012);

Antiulcerative (Ruckmani *et al.*, 1998; Matsuda and Yoshikawa, 1999; Chia *et al.*, 2006; Jain *et al.*, 2007; Jun *et al.*, 2009; Camilo *et al.*, 2011; Onasanwo *et al.*, 2011);

Cardioprotective (Fard *et al.*, 2008; Muralidharan *et al.*, 2008; Das *et al.*, 2011).

Apart from these properties, some vegetable plants have been reported to exert unique pharmacological properties, which are listed in Table-4.
Table - 4 – Some unique pharmacological properties of vegetables included in this study

<table>
<thead>
<tr>
<th>Vegetable name</th>
<th>Pharmacological property</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phaseolus vulgaris</td>
<td>Osteoprotective</td>
<td>Shirke et al., 2009</td>
</tr>
<tr>
<td></td>
<td>Insecticidal</td>
<td>Janzen et al., 1976; Gatehouse et al., 1984</td>
</tr>
<tr>
<td>Beta vulgaris</td>
<td>Cerebroprotective</td>
<td>Aaishwarya et al., 2011</td>
</tr>
<tr>
<td>Abelmoschus esulentus</td>
<td>Pharmaceutical adjuvant</td>
<td>Ravikumar et al., 2009</td>
</tr>
<tr>
<td></td>
<td>Spermatorrhea</td>
<td>Sarfaraz et al., 2011</td>
</tr>
<tr>
<td>Solanum melongena</td>
<td>Antiasthmatic</td>
<td>Bello et al., 2005</td>
</tr>
<tr>
<td></td>
<td>Antiplatelet activity</td>
<td>Saima, 2011</td>
</tr>
<tr>
<td>Vicia faba</td>
<td>Antiparkinsonian activity</td>
<td>Kempster et al., 1993</td>
</tr>
<tr>
<td>Brassica oleracea var. Capitata</td>
<td>Antithyroid</td>
<td>Alihaery et al., 1992</td>
</tr>
<tr>
<td></td>
<td>Protection against gamma radiation</td>
<td>Enas and Atif, 2010</td>
</tr>
<tr>
<td></td>
<td>Antiasthmatic</td>
<td>Kalpana et al., 2011</td>
</tr>
<tr>
<td>Daucus carota</td>
<td>Antifertility</td>
<td>Garg, 1975</td>
</tr>
<tr>
<td></td>
<td>Lowers intraocular pressure</td>
<td>Renu et al., 2008</td>
</tr>
<tr>
<td></td>
<td>Spermatogenic</td>
<td>Mohammad et al., 2009</td>
</tr>
<tr>
<td>Luffa aegyptiaca</td>
<td>Larvicidal</td>
<td>Mullai et al., 2008</td>
</tr>
<tr>
<td>Momordica charantia</td>
<td>Abortifacient</td>
<td>Law et al., 1983; Tam et al., 1984; Chan et al., 1986.</td>
</tr>
<tr>
<td>Moringa oleifera</td>
<td>Nervous disorders</td>
<td>Gupta et al., 1997; Mekonnen, 1999.</td>
</tr>
<tr>
<td></td>
<td>Urinary tract infections treatment</td>
<td>Shaw and Jana, 1982.</td>
</tr>
<tr>
<td></td>
<td>Dental caries</td>
<td>Fuglie, 1999</td>
</tr>
<tr>
<td></td>
<td>Antianemic</td>
<td>Quisumbing, 1978; Fuglie, 1999</td>
</tr>
<tr>
<td></td>
<td>Snakebite, scorpion bite treatment</td>
<td>Fuglie, 1999</td>
</tr>
<tr>
<td></td>
<td>Aphrodisiac</td>
<td>Fuglie, 1999</td>
</tr>
<tr>
<td>Musa paradisiaca</td>
<td>Antisnake venom activity</td>
<td>Borges et al., 2005</td>
</tr>
<tr>
<td></td>
<td>Haemostatic</td>
<td>Weremfo et al., 2011</td>
</tr>
<tr>
<td>Pisum sativum</td>
<td>Oral contraceptive</td>
<td>Sanyal, 1956</td>
</tr>
<tr>
<td></td>
<td>Insecticidal</td>
<td>Wesley et al., 2004</td>
</tr>
<tr>
<td>Solanum tuberosum</td>
<td>Antiplatelet aggregant</td>
<td>Diana et al., 2007</td>
</tr>
<tr>
<td>Raphanus sativus</td>
<td>Antiurolithiatic</td>
<td>Vargas et al., 1999</td>
</tr>
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<table>
<thead>
<tr>
<th><strong>Trichosanthes anguina</strong></th>
<th>Purgative</th>
<th>Chandra and Sastry, 1990</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastroprotective</td>
<td></td>
<td>Arawwawala <em>et al.</em>, 2009</td>
</tr>
<tr>
<td>Antifertility</td>
<td></td>
<td>Devendra <em>et al.</em>, 2009</td>
</tr>
<tr>
<td><strong>Tamarindus indica</strong></td>
<td>Antisnakevenome</td>
<td>Ushananndini <em>et al.</em>, 2006</td>
</tr>
<tr>
<td><strong>Lycopersicon esculentum</strong></td>
<td>Radioprotective</td>
<td>Dhirhe <em>et al.</em>, 2011</td>
</tr>
<tr>
<td></td>
<td>Anticataract</td>
<td>Gupta <em>et al.</em>, 2003</td>
</tr>
<tr>
<td></td>
<td>Surfactant</td>
<td>Yamanaka <em>et al.</em>, 2008</td>
</tr>
<tr>
<td><strong>Benincasa hispida</strong></td>
<td>Gastroprotective</td>
<td>Rachchh and Jain, 2008</td>
</tr>
<tr>
<td></td>
<td>Anxiolytic</td>
<td>Nimbal <em>et al.</em>, 2011a</td>
</tr>
<tr>
<td></td>
<td>Antiurolithiatic</td>
<td>Patel <em>et al.</em>, 2011</td>
</tr>
<tr>
<td></td>
<td>Anticonvulsant</td>
<td>Nimbal <em>et al.</em>, 2011b</td>
</tr>
<tr>
<td></td>
<td>Anorectic</td>
<td>Vimalavathini and Kumar, 2012</td>
</tr>
</tbody>
</table>

### 2.12. Conclusion

Vegetables are considered as rich source of phytochemicals but only few pharmacological properties are evaluated for the particular phytochemicals. Since there is a rapid emergence of communicable and non communicable diseases worldwide, the rational drug designing method could be useful in finding out new leads from the vegetables. There is also a need for a database to explore the phytochemicals present in the vegetables to identify effective leads in a short time.