As the new millennium dawns, humanity continues to strive for longer lifespan and better quality of life (Kidd, 2000), combating various infections causing serious ill-health worldwide. Meanwhile, the past few years have witnessed noncommunicable diseases such as cancer to emerge as a scourge of humanity and poses serious challenge to modern medicine expecting its progress for the eradication of the disease (Kidd, 2000; Alison, 2001). Cancer is one of the world’s leading causes of death and arises when the homeostatic balance between cell growth and death is disturbed (Kerr et al., 1994). It is a complex disease involving numerous temporal spatial changes in cell physiology, which ultimately leads to malignant tumors (Seyfried and Shelton, 2010). Abnormal cell growth or neoplasia is the biological endpoint of this disease. Most neoplasms arise from the clonal expansion of a single cell that has undergone neoplastic transformation. Neoplastic cells pass on their heritable biological characteristics to progeny cells. The biological behavior or clinical course of a neoplasm is further classified as benign or malignant. A malignant neoplasm manifests a greater degree of autonomy, is capable of invasion and metastatic spread, may be resistant to treatment, and may cause death. A benign neoplasm has a lesser degree of autonomy, is usually not invasive, does not metastasize, and generally produces no great harm if treated adequately (Zaidman et al., 2005). Cancer is a generic term for malignant neoplasms. The term cancer, neoplasia and malignancy are usually used interchangeably in both the technical and popular literature. Cancer does not develop overnight, instead often evolving over many years with detectable premalignant lesions presaging the development of full-blown malignancy. Malignant tumors not only invade surrounding tissue, but are able to colonize other, often vital, organs, a process known as metastasis. Widespread metastatic disease is usually harbinger of imminent patient death (Alison, 2001; Seyfried and Shelton, 2010). During the micro-evolutionary process of malignant neoplastic transformation, cancer cells accumulate multiple genetic alterations that provide them with several capabilities (Cahill et al., 1999). The latter include the escape from normal growth control, limitless replicative potential, self sufficiency in growth signal, insensitivity to growth inhibitory signals, evasion of the suicidal apoptotic program, induction of sustained angiogenesis, the ability to metastasize, and to invade healthy tissues (Figure 1.1) (Hanahan and Weinberg, 2000). Cancer is caused by both external factors (tobacco, chemicals, radiation, and infectious organisms) and internal factors (inherited mutations, hormones, immune
conditions, and mutations that occur from metabolism). These causal factors may act together or in sequence to initiate or promote carcinogenesis. Most cancers have defects in many aspects of cell behavior as a result of multiple genetic alterations, and this has crystallized into the multistage theory of carcinogenesis (Alison, 2001). Although different mechanisms may contribute to the process of carcinogenesis, a commonality is the involvement of cellular oxidants in neoplastic development (Klaunig and Kamendulis, 2004).

**Figure 1.1:** *Acquired capabilities of cancer* (Hanahan and Weinberg, 2000).

**Cancer trends and impact:**

Worldwide, one in eight deaths is due to cancer. Cancer remains second only to heart diseases as a leading cause of death (Table 1.1); however, it causes more deaths than AIDS, tuberculosis, and malaria combined (American Cancer Society, 2011). When countries are grouped according to economic development, cancer is the leading cause of death in economically developed countries and the second leading cause of death in developing countries (World Health Organization, 2008).
The burden of cancer is increasing in economically developing countries as a result of population aging and growth as well as, increasingly, an adoption of cancer-associated lifestyle choices including smoking, physical inactivity, and "westernized" diets (Jemal et al., 2011). According to estimates from the International Agency for Research on Cancer about 12.7 million cancer cases and 7.6 million cancer deaths are estimated to have occurred worldwide in 2008, with 56% of the cases and 64% of the deaths in the economically developing world (Jemal et al., 2011). Based on projections, cancer deaths will continue to rise with an estimated 9 million people dying from cancer in 2015 (Gavhane et al., 2011). In 2020 there are predictions to be 20 million new cancer cases and 12 million cancer deaths (Alison, 2001). By 2030, the global burden is expected to grow to 21.4 million new cancer cases and 13.2 million cancer deaths simply due to the growth and aging of the population, as well as reductions in childhood mortality and deaths from infectious diseases in developing countries (Ferlay et al., 2010). Predicting future cancer burden either in terms of the number of new cancer cases or deaths can be of great benefit to health planners attempting to optimize resources and allow scientist to explore the consequence of interventions aimed at reducing the impact of cancer, or a way to map out a range of possible future cancer scenarios (Bray and Moller, 2006). In many cases, cancer is long drawn-out disease that emotionally drains both the patients and their family (Kim et al., 2004). In addition to the human toll of cancer, the financial cost of cancer is substantial. Recent research has shown that cancer has the most devastating economic impact of any cause of death in the world (American Cancer Society and LIVESTRONG, 2010). Data limitations do not allow estimating the worldwide economic costs of cancer. However, portions of the total costs of cancer have been estimated to be as high as $895 billion (US) worldwide (American Cancer Society and LIVESTRONG, 2010; John and Ross, 2008). The costs of cancer are staggering, and with the growth and aging of the population, prevention efforts are important to help reduce new cancer cases, human suffering, and economic costs.
Table 1.1: Leading Causes of Death Worldwide, 2004 (in thousands)

<table>
<thead>
<tr>
<th>Rank</th>
<th>Death</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>8,923</td>
<td>15.1</td>
</tr>
<tr>
<td>2</td>
<td>7,424</td>
<td>12.6</td>
</tr>
<tr>
<td>3</td>
<td>5,712</td>
<td>9.7</td>
</tr>
<tr>
<td>4</td>
<td>4,177</td>
<td>7.1</td>
</tr>
<tr>
<td>5</td>
<td>3,180</td>
<td>5.4</td>
</tr>
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<td>6</td>
<td>3,025</td>
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<tr>
<td>7</td>
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</tr>
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<td>8</td>
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<tr>
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</tr>
<tr>
<td>15</td>
<td>739</td>
<td>1.3</td>
</tr>
</tbody>
</table>


Cancer research and management:

After a quarter century of rapid advances, cancer research has generated a rich and complex body of knowledge. Cancer researchers have collected an enormous amount of information about the differences between cancer cells and their healthy counterparts, with the ultimate goal of identifying drug targets. This has, for instance, led to the identification of genes that are causally implicated in human cancer and to the discovery of the mutations in those genes. A cancer gene census was compiled (Futreal et al., 2004), which currently contains 300 genes. The vast majorities of these genes function in signal transduction processes within or between cells; govern cell cycle progression, apoptosis, angiogenesis, and infiltration (Vogelstein and Kinzler, 2004). Although knowledge of the molecular cell biology of cancer is enormous, at the same time, the emerging complexity of the entire 'cancer system' overwhelms us, leaving an enormous gap in our understanding and predictive power (Hornberg et al., 2006). The biological process by which normal cells are transformed into malignant cancer cells has been the subject of a large
Chemopreventive activity of Tricholoma giganteum on EAC cells, forestomach and lung carcinogenesis in mice

research effort in the biomedical sciences for many decades. The biology of cancer is a complex interplay of many underlying processes, taking place at different scales both in space and time. A variety of theoretical models have been developed, which enable one to study certain components of the cancerous growth process and even the influence of the evolving tumor environment. An integrative framework is designed to simulate tumor growth and those model components are taken into consideration. This model was extended with relevant components at the cellular or even sub-cellular level in a vertical fashion. Implementation of this framework covers the major processes and treats the mechanical deformation due to growth, the biochemical response to hypoxia, blood flow, oxygenation and the explicit development of a vascular system in a coupled way (Lloyd et al., 2008). Despite the research efforts, cures or long-term management strategies for metastatic cancer are as challenging today as they were 40 years ago when President Richard Nixon declared a war on cancer (Bailar and Gornik, 1997; Anand et al., 2008). Meanwhile, 45 billion dollars are already spent over the past few decades on cancer research. While the National Cancer Institute (NCI) claims that the 5-year survival rate (the accepted definition of a “cure”) has increased from 20% of cancer patients in 1930 to 53% of adults and 70% of children today, critics of the NCI claim that living five years after cancer diagnosis has more to do with earlier diagnosis alone than the therapeutic modalities used. The search for the origin and treatment of this disease will continue over the next quarter century in much the same manner as it has in the recent past, by adding further layers of complexity to a scientific literature that is already complex almost beyond measure. Confusion surrounds the origin of cancer. Contradictions and paradoxes have plagued the field (Hanahan and Weinberg, 2000; Soto and Sonnenschein, 2004; Baker and Kramer, 2007; Sonnenschein and Soto, 2008). Without a clear idea on cancer origins, it becomes difficult to formulate a clear strategy for effective management.

Even though great advances have been made in basic scientific knowledge relating to cancer as well as in the clinical treatment, death rates from some of the common cancers continue to rise (Sporn, 1996). Many cellular changes have been reported to be associated with malignant process. Such studies may provide an important lead not only in aetiology of cancers, but also for early diagnosis of the disease and prognosis with respect to treatment modalities. It is important to
comprehensively study the biological processes at cellular levels, before a logical conclusion on such association can be made. Cancer treatment is usually a combination of a number of different modalities. It is often treated with surgery, radiation, chemotherapy, hormones, and immunotherapy. If the tumor is amenable to surgery, then surgery is the single most effective tool in the anticancer armamentarium (Alison, 2001). It is very effective in removing localized tumors. If the cancer has spread to other parts of the body, it is far less successful. Debunking by surgical means is also very effective in treating life-threatening and advance stage cancers. So we see that surgery can be a useful therapy in some cancers (Leape, 1992). The misperception of cancer as a disease whose most fundamental characteristics is excessive cell proliferation has led to an over emphasis of testing and development of cytotoxic drugs that kill cancer cells (Sporn and Suh, 2000). Unfortunately, most cytotoxic drugs used in cancer chemotherapy are also highly toxic to a wide spectrum of normal tissues, such as those found in gastrointestinal tract, bone marrow, heart, lungs, kidney and brain. Iatrogenic failure of these organs is a frequent cause of death from cancer (Sporn and Suh, 2000). Most conventional anticancer drugs have been designed with deoxyribonucleic acid (DNA) synthesis as their target (Maramag et al., 1997). Therein lies another problem, as tumor cells are not the only proliferating cells in the body; cells that line the alimentary tract, bone marrow cells that generate red blood cells and cells to fight infection, and epidermal cells including those that generate hair are all highly proliferative. Thus, patients with cancer receiving chemotherapy commonly suffer unwanted (hair loss). Thus chemo-poisoning of the bone marrow, the place where new blood cells are produced will lead to anemia and increased susceptibility to infections. Chemo-poisoning of the mucous membrane cells of the gastrointestinal tract will lead to diarrhea and intestinal bleeding. Instead of strengthening the body’s immune system to help fight the cancer, the chemotherapy will paralyze it and limit the treatment. If the patient is pregnant, it may affect the fetus or embryo, leading to birth defects (Rath, 2001). Bear in mind that despite the extensive damage done to the body, chemotherapy can only benefit a small number of epithelial cell cancers such as cancer of the breast, lungs, prostate, and colon. It can eradicate only about 7% of all human cancers. It looks like opting for chemotherapy may give a cancer patient more liabilities than assets. In some cases, chemotherapy can perhaps prolong life for another 15% of patients, after which the natural progression of the disease will lead to death.
Statistics show that chemotherapy is not very effective in the treatment of about 80% of malignant tumors (Lam, 2003). Chemotherapy’s side effects require the additional use of other, new medications, such as antibiotics, plasma replacement drugs, painkillers, cortisone, and many more (DeVita, 1989). The new generation of drugs affects the signals that promote or regulate the cell cycle, growth factors and their receptors, signal transduction pathways and pathways affecting DNA repair and apoptosis (Schwartz et al., 1998; Zi and Agarwal, 1999; Nielsen et al., 2000; Wolter et al., 2001; Xi et al., 2001). Each of these pathways may be affected by activating mutations that predispose to cancer and, thus, offer the potential as a target for inhibition. Other strategies focus on either attempting to target tumor cells specifically by conjugating cell toxins to tumor-specific antibodies (magic bullets), or slowing down cancer progression by affecting cell adhesion, proteolytic enzyme activity and angiogenesis (Dillman, 1989; Alison, 2001). All such approaches that are driven by the elusive goal of cure of advanced disease are often unrealistic because of the genetic heterogeneity and extent of tumor burden characteristics of late stage malignancy. Given the genotypic and phenotypic heterogeneity of advanced malignant lesions as they occur in individual patients, one wonders just exactly what are the specific molecular and cellular targets for the putative cure. As a consequence, thoughts for the treatment of invasive cancer with some sort of single gene therapy seem pathetically naive (Sporn and Suh, 2000). Targeted radiotherapy is another option, as are combinations of anticancer drugs. The radiation’s effective use in palliative treatment for selected cancers cannot be doubted, but generally speaking, radiation treatment should only be administered very selectively. Although radiation therapy is less painful, people very seldom choose this as the first line of defense because of the unknowns. It is chosen primarily in later-stage cancers where it is used to shrink the tumors encroaching or impinging on vital parts of the body. Another drawback about this therapy is that there is a certain limit to the radiation that a patient can tolerate. The exposure to radiation is said to have a cumulative effect (Seifter et al., 1984). Other new treatment modalities involving immunotherapy and agents that promote normal cell mutation remain experimental and are under extensive investigation.
Cancer chemoprevention:

The continuing magnitude of the cancer problem and the failure of conventional chemotherapy of the advanced invasive disease to effect the major reductions in the mortality rates for the common forms of epithelial malignancies such as carcinoma of lung, stomach, colon, breast, prostate, pancreas etc., indicate that new approaches to the control of cancer are critically needed. As an alternative approach, we need to consider that cancer is ultimately the end stage of a chronic disease process characterized by abnormal cell and tissue differentiation. This process, which eventually leads to the outcome of invasive and metastatic cancer, is carcinogenesis. We need to focus more effort on control of carcinogenesis rather than attempting to cure the end stage disease (Sporn and Suh, 2002) (Figure 1.2).

![Figure 1.2: Multistage carcinogenesis and prevention strategies](Greenwald, 2002).

In this context, it is essential that we re-evaluate our basic assumptions about the nature of cancer, and begin to adopt a more intensive and imaginative approach to the prevention of the disease. The time worn adage, "an ounce of prevention is worth a pound of cure," still holds true. The recent progress in molecular biology and pharmacology has increased the likelihood that cancer prevention, either primary
or secondary, will rely increasingly on interventions collectively termed 'chemoprevention'. Cancer chemoprevention, as first defined by Sporn in 1976, uses natural, synthetic, or biologic chemical agents to reverse, suppress, or prevent carcinogenic progression of neoplastic cells to cancer (Sporn, 1976). Carcinogenesis is a multistage process and often has a latency of many years or decades, thus there is considerable opportunity for intervention. Findings obtained so far from the carcinogenesis experiments, chemical/ biological sciences, pathology and epidemiology have promoted our understanding of the basic mechanism of chemoprevention. It has been an active area of research for several decades (Khuri et al., 1997). Chemoprevention is a promising and relatively new approach to cancer prevention that has precedence in cardiology, in which cholesterol-lowering, antihypertensive, and antiplatelet agents are administered to prevent coronary heart disease in high-risk individuals (Greenwald, 2002). The concept of using chemopreventive agents to reduce cancer risk is firmly based on epidemiologic and experimental evidence from the last two decades that indicates specific compounds may influence carcinogenesis at various sites, including the oral cavity, esophagus, stomach, colon/rectum, lung, breast, and prostate (Kelloff et al., 1994). An approach to reducing cancer risk that either prevents carcinogenesis or stops carcinogenesis in its early stages is a logical and perhaps the best strategy to reduce the overall cancer burden. Chemoprevention is widely used and readily accepted by doctors and patients. It can also be used in some apparently healthy people at risk of cancer to prevent or reduce their risk of developing invasive disease. The biomedical community needs to recognize and advocate approaches to prevent cancer with the same enthusiasm that it currently directs towards treating it (Greenwald, 2002). The credibility of chemoprevention as a serious and practical approach to the control of cancer has been greatly enhanced by the publication of the major randomized clinical trials (Kakizoe, 2003). Molecular advances have led to the identification of genetic lesions and other cellular components, which may be involved in the initiation and progression of malignancies, and constitute potential targets for chemoprevention. Cancer chemoprevention may target various processes such as carcinogen-blocking activities (Mirvish, 1986; Pence, 1993; Lubet et al., 1994), antioxidant activities (Roebuck et al., 1991; Kensler et al., 1992) and antiproliferation/antiprogession activities (Powis and Workman, 1994). Apart from some obvious common elements with chemotherapy, chemoprevention does have distinct differences too.
Chemoprevention focuses on reduction of incidence and is related to classical epidemiology, whereas chemotherapy focuses on prognosis and is related to clinical epidemiology. Chemotherapy can be either systemic or, in certain cases, localized, whereas chemoprevention is almost always systemic. In chemotherapy the outcome is generally a high frequency event (like death or metastasis), whereas in chemoprevention it is usually of low frequency (incident cancer cases); this is reflected in the required sample size in the corresponding studies. Lastly, chemotherapy is applied to seriously ill patients, for whom side-effects, even serious ones, may be acceptable, whereas chemoprevention is generally administered to healthy people for whom serious side-effects are unacceptable (Tamimi et al., 2002). Research into chemoprevention uses a systematic strategy that begins by surveying the results of epidemiological, laboratory, and clinical research for compounds, both naturally occurring and synthetic, that seem to inhibit carcinogenesis. Many compounds, belonging to diverse structural and functional chemical classes, have been identified as potential chemopreventive agents. These include vitamins and minerals (such as folate, vitamin E, vitamin D, calcium, and selenium); naturally occurring phytochemicals (such as curcumin, genistein, indole-3-carbinol, and 1-perillyl alcohol); and synthetic compounds (such as retinoids, selective oestrogen receptor modulators, and cyclo-oxygenase-2 inhibitors) (Greenwald, 2002). Several models have also been developed to outline the pathways through which carcinogenesis may occur. These include the Vogelstein model for colon cancer (Fearon and Vogelstein, 1990), as well as models for cancer of the head and neck (Thiberville et al., 1995; Califano et al., 1996), brain (Sidransky et al., 1992), bladder (Rosin et al., 1995; Mao et al., 1996) and lung (Kishimoto et al., 1995; Thiberville et al., 1995). Valid models of cancer progression facilitate the identification of intermediate biomarkers. By serving as surrogate end-points, such markers are pivotal in identifying chemopreventive agents. The use of early markers of carcinogenesis allows chemopreventive studies to focus on stage arrest or reversion following treatment (Kelloff et al., 2000). Considerable research is currently focused on identifying biomarkers as surrogate end points in place of overt cancer in cancer chemoprevention trials. Cancer is a comparatively infrequent event, and clinically overt cancer usually takes many years to develop. Clinical trials to test the effectiveness of chemopreventive agents therefore require large study populations and a long term commitment of resources. The availability of
Chemopreventive activity of Tricholoma giganteum Mataee on EAC cells, forestomach and lung carcinogenesis in mice

biomarkers as surrogate end points for clinical disease would allow smaller trials of shorter duration, facilitating clinical research into chemoprevention (Greenwald, 2002). The success of several recent clinical trials in preventing cancer in high-risk populations suggests that the chemoprevention is a rational and appealing strategy.

Chemoprevention by dietary constituents:

The future of cancer chemoprevention remains open to innovation. Chemoprevention by dietary constituents has emerged as a novel approach to control cancer incidence. There is a growing interest for alternative medicines to improve the function of the immune system in order to target late stage metastatic tumors (Radwan et al., 2011), as traditional chemotherapies highlighted most of its toxic side effects. Advances in technology have allowed for widespread screening of natural products to identify those with anticarcinogenic and immunostimulatory potential. Chemoprevention research is necessarily linked to diet represents a logical cancer research progression. The diet and nutrition program at the National Cancer Institute (NCI) conducts research in prevention related epidemiology, nutritional and molecular regulation, and dietary intervention trials to identify and evaluate cancer preventive dietary patterns. The chemoprevention program identifies and assesses specific chemical substances, many naturally occurring in foods, with the potential to prevent cancer initiation and to either slow or reverse the progression of premalignant lesions to invasive cancer.

Dietary epidemiologic studies have provided initial leads for the identification of numerous naturally occurring chemopreventive agents, (Steinmetz and Potter, 1991a, b; Block et al., 1992) and laboratory studies have even identified many potential agents that suppress carcinogenesis in animal models (Steele et al., 1994). Promising chemopreventive agents being investigated include micronutrients (eg, vitamins A, C, and E, beta-carotene, molybdenum, calcium), phytochemicals (eg, indoles, polyphenols, isothiocyanates, flavonoids, monoterpenes, organosulfides), and synthetics (eg, vitamin A derivatives, piroxicam, tamoxifen, 2-difluoromethylornithine [DFMO], and oltipraz). Broadly defined based on their mechanisms of action, chemopreventive agents can be grouped into two general classes: blocking agents and suppressing agents. Blocking agents (eg, flavonoids, oltipraz, indoles, isothiocyanates) prevent carcinogenic compounds from reaching or
reacting with critical target sites by preventing the metabolic activation of carcinogens or tumor promoters by enhancing detoxification systems and by trapping reactive carcinogens (Kelloff, 1994; Wattenberg, 1996). Suppressing agents (e.g., vitamin D and related compounds, nonsteroidal anti-inflammatory drugs [NSAIDs], vitamin A and retinoids, DFMO, monoterpenes, calcium) prevent the evolution of the neoplastic process in cells that would otherwise become malignant. Some agents produce differentiation, some counteract the consequences of genotoxic events such as oncogene activation, some inhibit cell proliferation, and some have undefined mechanisms (Wattenberg, 1996). Certain chemopreventive agents may exhibit several different mechanisms of action simultaneously.

A large body of epidemiologic evidence, together with data from in vivo and in vitro studies, strongly supports relationships between dietary constituents and the risk of specific cancers. Generally, vegetables and fruits, dietary fiber, and certain micronutrients appear to be protective against cancer, whereas fat, excessive calories and alcohol seem to increase cancer risk (US Dept of Health and Human Services, 1988; National Academy of Sciences, 1989). However, the fact that not all data are consistent across studies is likely the result of several contributing factors. Foods are complex mixtures of nutrients and nonnutritive substances that are difficult to measure accurately, and the effects of individual constituents as well as the possible interactions among these constituents are difficult to unravel. Specifically, cruciferous vegetables may be protective for colorectal cancer development through a p53-dependent pathway, whereas beef consumption may increase risk for colorectal tumorigenesis through a p53-independent pathway, thus contributing to the difficulty of interpreting epidemiologic data (Freedman et al., 1996). Even though inconsistencies may be observed and the interpretation of data on diet and cancer associations may not always be straightforward, available data have provided valuable leads for generating hypotheses for further research. This may be of particular importance to societies and countries where modern medicine is scarce, expensive to buy, or simply unavailable.
Traditional and emerging concept of mushrooms as functional food or dietary supplement:

Traditional knowledge can serve as powerful search engine, which will greatly facilitate intentional, focused and safe natural product drug development and help to rediscover the drug discovery process. Ayurvedic Indian and traditional Chinese systems are living great traditions. These traditions have relatively organized database, and more exhaustive description of botanical material that is available and can be tested using modern scientific methods (Patwardhan, 2009). By looking at the historical trends in drug and medical developments, it is possible to understand how current drug development will benefit from its partnership with traditional medical knowhow. Numerous drugs have entered the international pharmacopoeia via the study of ethnopharmacology and traditional medicine. This has led to a renewed investigation into the anecdotal evidence of health benefits associated with folk medicine, which could be used as alternative or complementary treatment for cancer. Natural extracts from fungi have been the focus of recent investigation, particularly those with reduced cellular toxicity to healthy tissue (Radwan et al., 2011). In the Orient several thousand years ago, there was the recognition that many edible and certain non-edible mushrooms could have valuable health benefits (Bensky and Gamble, 1993; Hobbs, 1995). The Egyptians as far back as 3000 BC believed that mushrooms were a sacred food that prolonged life. A mummified 5000-year old “Ice-man” found in the mountains of Europe carried a medicine kit of dried mushrooms. Indeed, the oldest written record of mushrooms as medicines is found in an Indian medical treatise compiled in 3000 BC (Kaul, 1997). The ancient people of India, China, Iran and Seythian used mushrooms in their ritualistic performances (Lowy, 1971). The ancestors of Finno-Ugric were also familiar with the religious conception of mushrooms (Bongard-Levin, 1980). The Mexican Indians seem to regard the psychotropic plants as mediators with God. Nahuati dialect speaking people named mushrooms as ‘teohanotactl’, which means flesh of God. Classical religious scriptures like “Vedas” have also mentioned their medicinal value. The Greeks regarded mushrooms as “Providing strength to soldiers in war”. The Romans considered them as “Food of the Gods” and the Chinese treated them as the “Elixir of life” (Chang and Miles, 1989). Medicinal mushrooms have been used as a dietary supplement or medicinal food in China for over 2000
years and in some clinical cases dietary supplements have been prescribed in the prevention and treatment of various human diseases. In China, the term Yakuzen is generally used for medicinal food dishes of mushrooms. Thus, medicinal mushrooms have an established history of use in traditional oriental therapies, whereas their use in Western nations has only slightly increased during the last decade (Sharma, 2003).

Mushrooms are not a taxonomic group. According to Chang and Miles (1992), mushrooms are defined as “a macro fungi with a distinctive fruiting body which can be hypogeous or epigeous, large enough to be seen with the naked eye and to be picked by hand”. There is an estimated 1.5 million species of fungi, of which it is likely that there are about 140,000 species, which qualify as mushrooms. Among them, approximately 10% i.e., 14,000 are described species of mushrooms, and only 3000 are known to be edible, with 700 exhibiting medicinal properties and less than 1% are considered poisonous (Borchers et al., 2004, 2008). Mushrooms are established to be a good source of digestible proteins with protein content above most vegetables and somewhat less than most meats and milk. Mushrooms contain all the essential amino acids with significant amount of carbohydrate, sugar, glycogen (Breene, 1990; Chang, 1991; Kalac, 2009). A considerable proportion of the carbohydrate of mushrooms consists of dietary fiber which cannot easily be digested by humans and which functions essentially as dietary fiber. Mushrooms do not contain starch and cholesterol and contain a small amount of fat, which enables mushrooms to be considered as a low caloric food. Mushrooms probably contain every mineral present in their growth substrate including substantial quantities of phosphorus and potassium and even lesser amounts of calcium and iron minerals required for normal functioning of the body (Gbolagade et al., 2006). Mushrooms appear to be an excellent source of vitamins especially thiamine (B1), riboflavin (B2), niacin, biotin and ascorbic acid (Vitamin C). Vitamins A and D are relatively uncommon although several species contain detectable amounts of ß-carotene and ergosterol which gets converted to active vitamin D when exposed to ultraviolet irradiation. Crude fat in mushrooms contains all the main classes of lipid compounds including free fatty acids, mono-, di- and triglycerides, sterols, sterol esters and phospholipids, in very low levels (Breene, 1990). Therefore, edible mushrooms in fresh, cooked or processed forms are a nutritionally sound, tasteful food source for most people and can be a significant dietary component (Breene, 1990).
An old Chinese proverb states “medicine and food have a common origin”. Arising from the awareness of the relationship between diet and diseases has evolved the concept of “functional foods” and the development of a new scientific discipline “functional food science” (Sadler and Saltmarsh, 1998). A food may be considered to be functional if it contains a food component (whether a nutrient or not) which affects one or more identified functions in the body in a positive manner. Correspondingly, it can also include foods in which potentially harmful components have been removed by technological means. The US Academy of Science has defined functional foods as those that “encompass potentially healthful products” including “any modified food or food ingredient that may provide a health benefit beyond the traditional nutrients it contains” (Thomas and Earl, 1994). Functional foods come in a plethora of name forms, e.g. dietary supplements, nutra- or nutriceuticals, medical foods, vita foods, pharmafoods, phytochemicals, mycochemicals, biochemopreventatives, designer foods and foods for specific health uses (Hasler, 1996; Head et al., 1996). Such complex designations could well be an impediment to their rightful maturation and consumer acceptance (Zeisel, 1999). There continues to be much confusion over these names especially in the commercial world. However, the term dietary supplement (DS) is now being more widely accepted and recognised. The term DS was formally defined by the US administration in 1994 as a product intended to supplement the diet to enhance health. According to the 1994 US Dietary Supplement Health and Education Act, a dietary supplement is defined as a product intended to supplement the diet, containing one or more dietary ingredients (including vitamins, minerals, herbs, amino acids, or other botanicals), and to be taken by mouth as a pill, capsule, tablet, or liquid; or it is a dietary substance used to supplement the diet by increasing the total dietary intake and is intended for ingestion in the form of a capsule, powder, softgel or gel cap and not represented as a conventional food or as a sole item of a meal or the diet. Dietary mushrooms have been used globally for millennia to promote health and prevent and treat disease primarily via their multitude of medicinal qualities (Borchers et al., 1999). Now a days, mushrooms have already become attractive as functional foods and a source of physiologically beneficial and non-toxic medicine, while being devoid of undesirable side effects (Sadler, 2003). Medicinal mushrooms produce beneficial effects not only as drugs but also act as a novel class of products known by a variety of names: dietary supplements (DSs), functional foods, nutriceuticals, mycopharmaceuticals,
and designer foods that benefit us through everyday use as part of a healthy diet (Chang and Buswell, 2003; Chang, 2006; Wasser and Akavia, 2008). The increased interest in traditional remedies for various physiological disorders and the recognition of numerous biological activities of mushroom products have led to the coining of the term "mushroom nutriceuticals", which should not be confused with nutraceuticals, functional foods, and pharmaceuticals. A mushroom nutriceutical is a refined, or partially refined, extract or dried biomass from either mycelium or the fruiting body of a mushroom, which is consumed in the form of capsules or tablets as a DS (not a food) and it has potentially therapeutic applications (Wasser, 2011). On the other hand, nutraceuticals are functional foods that "are consumed as part of a normal diet and deliver one or more active ingredients that have physiologic effects and may enhance health within the food matrix". The regular intake of mushroom nutriceuticals or nutraceuticals may enhance the immune response of the human body, thereby increasing resistance to diseases and in some cases cause regression of the diseased state. Mushrooms might be used directly in diet to promote health, taking advantage of the additive and synergistic effects of all the bioactive compounds present to influence the complex pathophysiological process (Ferreira et al., 2009). In general, medicinal dietary mushrooms have been shown to improve cardiovascular health, stimulate immune function, contribute to glucose homeostasis and to modulate detoxification, as well as exert antiallergic, antiviral, antibacterial, antifungal and anti-inflammatory activities (Chang, 1996; Mayell, 2001; Yu et al., 2009). Attempts have been made in many parts of the world to explore the use of mushrooms and their metabolites for the treatment of a variety of human ailments (Jose and Janardhanan, 2000). A variety of mushrooms have been used for the maintenance of health and for prevention and treatment of diseases such as cancer, inflammation, viral diseases, hypercholesterolemia, blood platelet aggregation, hypertension, heart diseases, diabetes, hepatic injury, constipation, renal failure, etc (Breene, 1990; Jong et al., 1991; Chihara, 1992; Ooi and Liu, 1999; Wasser and Weiss, 1999; Saini and Atri, 1999; Biswas et al., 2010, 2011a, b, 2012; Chatterjee et al., 2011, 2012; Acharya et al., 2012). The spectrum of detected pharmacological activities of mushrooms is very broad and their medicinal usage is being pursued worldwide. It hopefully shows the directions of medical research and their undoubted value and significance in controlling several killer diseases.
In recent years the most significant property of mushrooms and their metabolites which attracted the attention of scientists is their antineoplastic activity. The United States National Cancer Institute has chosen mushrooms as a source of new drugs for the treatment of cancer (Ayodele and Okhuoya, 2009). The use of medicinal mushrooms in the fight against cancer is known in China, Korea, Japan, Russia, United States and Canada. Such mushrooms effective against cancers of the stomach, oesophagus, prostate and lung, belong to the family of Polyporaceae (Mizuno, 1999). In Russian medicine, an extract of Chaga (*Inonotus obliquus*) is used as an antitumor medicine. Approximately 200 species of higher basidiomycetes have been reported to exhibit antitumor activity (Lucas *et al.*, 1957; Gregory *et al.*, 1966; Ying *et al.*, 1987; Yang and Jong, 1989; Mizuno, 1995a, b, 1996). The antitumor activity of the higher basidiomycetes has been first demonstrated by Lucas *et al.*, (1957) employing extracts of fruiting bodies of *Boletus edulis* against Sarcoma 180 cell line in mice. Ikekawa and coworkers (1968, 1969) reported that hot water extracts obtained from the fruiting bodies of six edible wild higher Basidiomycetes namely, *Flammulina velutipes, Lentinus edodes, Pholiota nameko, Pleurotus ostreatus, Tricholoma matsutake* and *Pleurotus spodoleucus*, showed a marked host mediated antitumor activity against Sarcoma 180 in Swiss albino mice. Gregory and collaborators (1966) surveyed more than 7,000 cultures of higher Basidiomycetes for antitumor activity against three rodent tumor systems. 50 cultures representing 22 species produced in fermentation media, showed inhibitory effects against Sarcoma 180, mammary adenocarcinoma 755, and leukemia L-1210. Since then, numerous researchers have isolated some essential substances from mushrooms. One such potential substance isolated from *Grifola frondosa* is the high branched maitake D-fraction (MD-fraction), which is found to exert a high anti-tumor activity (Nanba *et al.*, 1987; Kodama *et al.*, 2003). D-fraction of Maitake even induced apoptosis in breast cancer cells by BAK-1 gene activation (Soares *et al.*, 2011). Another study showed the maitake mushroom D-fraction synergistically potentiates the anticancer/antiproliferative activity of IFN-α2b on bladder cancer T24 cells. This enhanced growth inhibition results from a G1 cell cycle arrest together with activation of DNA-PK. It is conceivable that such an antiproliferative mechanism is associated with an accumulation of low-molecular weight DNA, triggering DNA-PK activation that might act primarily on the cell cycle to cease cancer cell growth. Therefore, the low-dose of the fraction may provide an alternative improved
immunotherapy for superficial bladder cancer and thus clinical studies/trials are warranted (Louie et al., 2009). The maitake D-fraction is a relatively new compound, and there are a number of clinical trials in breast, prostate, lung, liver and gastric cancers underway in the United States and Japan (Deng et al., 2009). The most widely distributed molecules with antitumour properties in mushrooms are sesquiterpenes, triterpenoids, glucans and glycoproteins (Ferreira et al., 2010). Triterpene-enriched extracts from *G. lucidum* inhibit growth of hepatoma cells via suppressing protein kinase C, activating mitogen-activated protein kinases and G2-phase cell cycle arrest (Lin et al., 2003). Zaidman et al., (2007) has reported that *G. lucidum* downregulated cyclin D1 expression leading to dephosphorylation of pRb and growth arrest of LNCaP prostate cancer cell line.

Apart from these, recent literature reports *Phellinus linteus* to suppress growth, angiogenesis and invasive behaviour of breast cancer cells through the inhibition of serine-threonine kinase protein kinase B (PKB/AKT) signaling. It suppresses phosphorylation of AKT at Thr308 and Ser473 in breast cancer cells (Sliva et al., 2008). *P. ostreatus* have recently been reported to inhibit proliferation of human breast and colon cancer cells through p53-dependent as well as p53-independent pathway (Jedinak and Sliva, 2008). *Agaricus bisporus* exhibits antiproliferative and proapoptotic properties and inhibits prostate tumor growth in athymic mice (Adams et al., 2008). In patients with malignant diseases, the ratio of IFN-gamma/IL-10 productions is a useful prognostic indicator and was improved by *L. edodes* mycelia, *Cordyceps sinensis* mycelia, *P. linteus*, *G. lucidum* and *A. blazei* extracts (Nagayama et al., 2009). Hot water extracts of *G. frondosa*, *P. ostreatus*, *A. bisporus* reduced cellular proliferation in MCF-7 human breast cancer cells (Martin and Borphy, 2010). The ethanolic extract of *Ganoderma colossum* is an effective inhibitor on the PMA-induced MMP-9 expression, which contributes to the inhibition of HepG2 cell invasion. The inhibitory activity inactivates the phosphorylation of ERK1/2 and Akt in cytosol as well as reduces the AP-1 (c-Jun and c-Fos) and NF-kB protein expressions in the nucleus of HepG2 cells (Weng et al., 2010). The ethanolic extract of *Astraeus hygrometricus* is even demonstrated to induce apoptosis in Ehrlich’s ascites carcinoma cells in swiss albino mice in a p53-dependent pathway in which over-expression of Bax resulted in tumor cell apoptosis (Biswa et al., 2012). *In vitro* and *in vivo* study demonstrated anti-tumor efficacy of
an aqueous extract of the mycelial form of basidiomycete, *Funalia trogii* on HT29, LNCaP, PC3, MCF-7 and MDA-MB-231 tumor cells resulting in significant cytotoxicity (Rashid *et al.*, 2011). A cold water extract prepared from the sclerotia of *Lignosus rhinocerus* cultivar was found to exhibit antiproliferative activity against human breast carcinoma (MCF-7) and human lung carcinoma (A549) (Lee *et al.*, 2012). *Gomphus clavatus* dichloromethane (DCM) extract showed cytotoxic activity against MCF-7 and PC-3 cancer cell lines, which might be attributed to the presence of ergostan derivatives (Makropoulou *et al.*, 2012). Dietary supplementation with *Agaricus sylvaticus* produces benefits in hematological and immunological parameters and can reduce fasting glycemia levels in patients with colorectal cancer in the postoperative phase (Fortes *et al.*, 2009). This reduction results in beneficial effects on the metabolism of carbohydrates in these patients.

Upto date report on anticancer activity of different mushrooms were summarized in Table 1.2

### Table 1.2: Anticancer effect of mushrooms

<table>
<thead>
<tr>
<th>Mushroom name</th>
<th>Active component</th>
<th>Effective against</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Agaricus bisporus</em></td>
<td>Extract</td>
<td>Prostate tumor</td>
<td>Adams <em>et al.</em>, 2008</td>
</tr>
<tr>
<td><em>Agaricus bisporus</em></td>
<td>Hot water extracts</td>
<td>MCF-7 human breast cancer cells</td>
<td>Martin and Borphy, 2010</td>
</tr>
<tr>
<td><em>Agaricus blazei</em></td>
<td>Poyssaccharide fraction</td>
<td>Sarcoma-180</td>
<td>Minato <em>et al.</em>, 1999</td>
</tr>
<tr>
<td><em>Agaricus blazei</em></td>
<td>Hot water extract</td>
<td>Sarcoma-180</td>
<td>Minato <em>et al.</em>, 1999</td>
</tr>
<tr>
<td><em>Agaricus blazei</em></td>
<td>Ergosterol</td>
<td>Tumor</td>
<td>Takaku <em>et al.</em>, 2001</td>
</tr>
<tr>
<td><em>Agaricus sylvaticus</em></td>
<td>Extracts</td>
<td>Colorectal cancer</td>
<td>Fortes <em>et al.</em>, 2009</td>
</tr>
<tr>
<td><em>Agrocybe aegerita</em></td>
<td>Lectin</td>
<td>Tumor</td>
<td>Yang <em>et al.</em>, 2009</td>
</tr>
<tr>
<td><em>Antrodia cinnamomea</em></td>
<td>Zhankui acids A-C</td>
<td>Liver cancer</td>
<td>Chen <em>et al.</em>, 1995</td>
</tr>
<tr>
<td><em>Astraeus hygrometricus</em></td>
<td>Ethanolic extract</td>
<td>Ehrlich's ascites carcinoma cells in swiss albino mice</td>
<td>Biswas <em>et al.</em>, 2012</td>
</tr>
<tr>
<td><em>Bondarzewia Montana</em></td>
<td>Montadial A</td>
<td>HL60 cells</td>
<td>Sontag <em>et al.</em>, 1999</td>
</tr>
<tr>
<td><em>Boletus edulis</em></td>
<td>Extracts</td>
<td>Sarcoma 180</td>
<td>Lucas <em>et al.</em>, 1957</td>
</tr>
<tr>
<td><em>Calvatia gigantea</em></td>
<td>Calvacin</td>
<td>Sarcoma 180, mammary adenocarcinoma 755, leukemia L-1210, and HeLa cell lines</td>
<td>Lucas <em>et al.</em>, 1957, 1959</td>
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<tr>
<td><em>Clitocybe maxima</em></td>
<td>Extract</td>
<td>Hep G2 and MCF-7 tumor cells</td>
<td>Zhang <em>et al.</em>, 2010</td>
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<td><em>Coriolus versicolor</em></td>
<td>Polysaccharide peptide (PSP)</td>
<td>Non small cell lung cancer</td>
<td>Tsang <em>et al.</em>, 2003</td>
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<td><em>Flammulina velutipes</em></td>
<td>Extract</td>
<td>Sarcoma 180</td>
<td>Ikekawa <em>et al.</em>, 1968, 1969</td>
</tr>
<tr>
<td>Mushroom Species</td>
<td>Extract Type</td>
<td>Target Cells</td>
<td>Biological Activity</td>
</tr>
<tr>
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<td><em>Flammulina velutipes</em></td>
<td>Aqueous extract</td>
<td>Tumor-bearing mice</td>
<td>Immunomodulatory protein (FVE)</td>
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<td><em>Fanalila trogii</em></td>
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<td>HepG2 cell invasion</td>
<td>Ganoderic acid</td>
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<td><em>Ganoderma colossum</em></td>
<td>Ethanolic extract</td>
<td>Growth of hepatoma cells <em>in vitro</em></td>
<td>Gao et al., 2002</td>
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<td><em>Ganoderma lucidum</em></td>
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<td>Advanced cancer</td>
<td>Gao et al., 2002</td>
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<td><em>Ganoderma lucidum</em></td>
<td>Lucidenic acid</td>
<td>Hep G2, P388 cell lines</td>
<td>Wu et al., 2001</td>
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<td><em>Gomphus clavatus</em></td>
<td>Dichloromethane extract</td>
<td>MCF-7 and PC-3 cell lines</td>
<td>Makropoulou et al., 2012</td>
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<td><em>Grifola frondosa</em></td>
<td>Ergosterol</td>
<td>Tumor development in some established tumors</td>
<td>Prescott and Fitzpatrick, 2000</td>
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<td><em>Grifola frondosa</em></td>
<td>MD- fraction</td>
<td>Breast, prostate, lung, liver and gastric cancer</td>
<td>Deng et al., 2009</td>
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<td><em>Grifola frondosa</em></td>
<td>Hot water extracts</td>
<td>MCF-7 human breast cancer cells</td>
<td>Martin and Borphy, 2010</td>
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<td><em>Grifola frondosa</em></td>
<td>Water soluble extract</td>
<td>Human gastric cancer cell lines</td>
<td>Shomori et al., 2009</td>
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<td><em>Inonotus obliquus</em></td>
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<td>Bladder cancer T24 cells</td>
<td>Louie et al., 2009</td>
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<td>3β-hydroxy-lanost-8, 24-dien-21-al, inotodiol and lanosterol</td>
<td>Sarcoma-180 cells, Lung carcinoma A-549 cells, Stomach adenocarcinoma AGS cells, Breast adenocarcinoma MCF-7 cells, cervical adenocarcinoma HeLa cells</td>
<td>Chung et al., 2010</td>
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<td><em>Laetiporus sulphureus</em> var. <em>Miniatius</em></td>
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<td>Kato III cells</td>
<td>Yoshikawa et al., 2001</td>
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<td><em>Lentinus crustatus</em></td>
<td>Hirsutane compounds</td>
<td>Growth of Cancer cells L929 mouse fibroblast cells</td>
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<td>Sarcoma 180</td>
<td>Chihsia et al., 1969</td>
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<td>Ikewaka et al., 1968, 1969</td>
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<td>Ukawa et al., 2000</td>
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<td>Cyathane-type diterpenoid</td>
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<td>Tricholoma matsutake</td>
<td>Hot water extract</td>
<td>Sarcoma 180</td>
<td>Ikekawa et al., 1968, 1969</td>
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</tbody>
</table>

Keeping in view the above literature survey and scientific evidences, here an attempt has been made to address the chemopreventive activity of *Tricholoma*...
Chemopreventive activity of *Tricholoma giganteum* Mass et on EAC cells, forestomach and lung carcinogenesis in mice.

*Tricholoma giganteum* Mass et on Ehrlich's ascites carcinoma cells, forestomach and lung carcinogenesis in mice.
Objective of undertaking work

Objectives fulfilled are as follows:


2. *In vitro* evaluation of free radical scavenging and NOS activation properties of *Tricholoma giganteum* extracts and identification of the potential fraction.

3. Evaluation of chemopreventive activity of the potential fraction against Ehrlich's Ascites Carcinoma cells in mice.

4. Evaluation of chemopreventive activity of the potential fraction against forestomach cancer in mice.

5. Evaluation of chemopreventive activity of the potential fraction against lung carcinogenesis in mice.
Chemetopreventive activity of Triehoima giganteum Masse* on EAC cells, forestomach and lung carcinogenesis In mice

References


Introduction


Chemopreventive activity of Tricholoma giganteum Masses on EAC cells, forestomach and lung carcinogens in mice


Introduction


Introduction

Chemopreventive activity of Tricholoma giganteum Marne on EAC cells, forestomach and lung carcinogenesis in mice


Introduction

Chemospreventive activity of Tricholoma giganteum Masset on EAC cells, forestomach and lung carcinogenesis in mice


Chemopreventive activity of Tricholoma giganteum Msaee on EAC cells, forestomach and lung carcinogenesis in mice


