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RESEARCH ARTICLE

Anticancer Potential of Zeatin in HepG2 Liver Cancer Cell Line

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ABSTRACT

Liver cancer remains a major global health challenge, necessitating the exploration of novel therapeutic agents with potent anticancer activity. Zeatin, a naturally occurring cytokinin, has demonstrated diverse biological properties, including antioxidant and anti-inflammatory effects. This study evaluates the biochemical properties and anticancer potential of zeatin against human hepatocellular carcinoma (HepG2) cells. Zeatin's cytotoxic effects were assessed using the MTT assay, while its impact on apoptosis was investigated through AO/EB and PI staining. Additionally, biochemical assays were performed to determine its influence on oxidative stress markers and key apoptotic regulators. Results revealed a dose-dependent reduction in HepG2 cell viability, along with enhanced apoptosis induction and alterations in oxidative stress parameters. These findings suggest that zeatin exerts significant anticancer activity against liver cancer cells, potentially through apoptotic and oxidative stress-mediated pathways. Further molecular studies are warranted to elucidate its precise mechanism of action and therapeutic applicability.

Keywords: Zeatin, HepG2 cells, Apoptosis, Oxidative stress, Cytotoxicity, Liver cancer

INTRODUCTION

Cancer

Cancer is a group of diseases characterized by the uncontrolled growth and spread of abnormal cells in the body. If the spread is not controlled, it can result in death. Cancer can develop in almost any tissue or organ, such as the lungs, breast, colon, skin, or bones. These abnormal cells grow beyond their usual boundaries and can invade adjoining parts of the body and spread to other organs, a process known as metastasis **(1).** Cancer arises from a complex interplay of genetic, environmental, and lifestyle factors. Mutations in DNA, often caused by exposure to carcinogens (like tobacco smoke or radiation), inherited genetic changes, or random errors during cell division, contribute to the transformation of normal cells into cancerous ones.

Liver Cancer

Liver cancer, particularly hepatocellular carcinoma (HCC), is one of the most prevalent and deadly cancers globally, ranking as the sixth most commonly diagnosed cancer and the third leading cause of cancer-related deaths **(2)**. HCC accounts for nearly 90% of primary liver cancers and is often diagnosed at an advanced stage, where curative treatments are limited and the prognosis remains poor. Conventional therapies such as surgical resection, liver transplantation, and chemotherapy offer limited success due to drug resistance, toxicity, and high recurrence rates **(3)**. HCC accounts for nearly 90% of primary liver cancers and is often diagnosed at an advanced stage, where curative treatments are limited and the prognosis remains poor. Conventional therapies such as surgical resection, liver transplantation, and chemotherapy offer limited success due to drug resistance, toxicity, and high recurrence rates **(4).** As such, the search for novel therapeutic agents, particularly those derived from natural sources with lower toxicity profiles, is gaining momentum in oncological research. In this context, phytochemicals, which are naturally occurring compounds in plants, have garnered significant attention for their potential anticancer properties. These bioactive molecules include polyphenols, flavonoids, alkaloids, and plant hormones such as cytokinins. Among cytokinins, zeatin, first isolated from maize (Zea mays), is a plant growth regulator involved in cell division, differentiation, and delay of senescence **(5).**

Chemically, zeatin is an adenine derivative with structural similarity to purines, which allows it to interact with various cellular pathways not only in plants but potentially in mammalian cells as well **(6).** Recent studies have demonstrated that zeatin and its derivatives may exhibit antioxidant, anti-inflammatory, and antiproliferative effects in vitro**.** These properties suggest a possible role in the modulation of cancer-related processes such as oxidative stress, apoptosis, and cell cycle regulation. In cancer cells, oxidative stress is often elevated, contributing to genomic instability and tumor progression. However, increased reactive oxygen species (ROS) can also be exploited to trigger apoptosis in cancer cells, and agents that modulate ROS levels are being investigated as potential chemotherapeutic agents **(7).** Furthermore, preliminary investigations into the cytotoxic effects of cytokinins on human cancer cell lines have revealed that zeatin can induce apoptosis, suppress proliferation, and affect mitochondrial function in a dose-dependent manner **(8).** However, the specific molecular mechanisms by which zeatin exerts its anticancer effects, particularly in human liver cancer cells, remain underexplored. Understanding its impact on cellular metabolism, ROS generation, and apoptotic markers could provide significant insights into its potential as an anticancer agent.

The present study is designed to biochemically evaluate the effects of zeatin on human liver cancer cells focusing on its influence on cell viability, oxidative stress markers (such as lipid peroxidation, glutathione levels, and antioxidant enzyme activity), mitochondrial membrane potential, and apoptotic proteins (e.g., caspases, Bcl-2 family proteins). Through this investigation, we aim to establish a clearer understanding of the anticancer potential of zeatin, laying the groundwork for its future development as a natural therapeutic or adjunctive agent in liver cancer treatment. The MTT assay is used to assess cell viability and metabolic activity following treatment with zeatin, providing a quantitative measure of cytotoxicity. To determine whether cell death is due to apoptosis, Acridine Orange/Ethidium Bromide (AO/EB) dual staining is applied, which allows visual differentiation between viable, apoptotic, and necrotic cells under fluorescence microscopy. Additionally, Propidium Iodide (PI) staining coupled with flow cytometry is

performed to analyze the distribution of cells across different phases of the cell cycle and detect any zetain-induced arrest at G1, S, or G2/M phases. Together, these methods offer a comprehensive evaluation of zeatin anticancer potential and its mechanisms of action in liver cancer cells.

Natural Plant Hormones as Emerging Anticancer Agents

While plant hormones are traditionally associated with plant development, many of them exhibit biological activity in mammalian cells. Cytokinins, such as zeatin, have shown anti-proliferative, anti-angiogenic, and pro-apoptotic effects in cancer cells. Their mechanisms may include DNA intercalation, inhibition of kinases, and interference with mitochondrial function. In a study by zeatin riboside was found to induce apoptosis in leukemia and colon cancer cell lines, causing nuclear condensation, cytochrome c release, and caspase activation. These observations suggest that cytokinins may act through both intrinsic and extrinsic apoptosis pathways **(9).** Zeatin may also inhibit angiogenesis, a key process in tumor growth and metastasis, by downregulating VEGF expression. This multi-targeted approach makes cytokinins promising candidates for cancer therapeutics **(10).**

Zeatin

Zeatin is a naturally occurring plant hormone that belongs to the cytokinin group. It plays a vital role in promoting cell division, stimulating shoot formation, delaying leaf aging (senescence), and enhancing overall plant growth and development. First discovered in maize (corn) kernels, zeatin is also found in other plant tissues and coconut milk. Among its two forms—cis-zeatin and trans-zeatin—the trans form is considered more biologically active. Zeatin not only supports seed germination and fruit development but also helps in nutrient mobilization within the plant. Because of these properties, it is widely used in plant tissue culture and agricultural practices to improve crop quality and yield **(11**).

Pharmacology of Zeatin

Zeatin, a cytokinin plant hormone, exerts its pharmacological effects primarily by interacting with cytokinin receptors in plant cells, leading to the activation of signaling pathways that regulate gene expression associated with cell division, differentiation, and organogenesis. It binds to specific histidine kinase receptors such as AHKs (Arabidopsis Histidine Kinases), initiating a phosphorelay system that ultimately modulates transcription factors like ARR (Arabidopsis Response Regulators). These changes promote mitotic activity, delay senescence, and stimulate protein synthesis. Zeatin also influences chloroplast development and enhances resistance to stress by modulating antioxidant enzyme activities. In tissue culture, zeatin is widely used to induce shoot proliferation and maintain callus growth. Its stability and ability to resist rapid degradation make it a preferred cytokinin in experimental and commercial plant propagation systems **(12).**

Cell Cycle Regulation in Cancer and Potential Intervention by Zeatin

The cell cycle is a tightly regulated process that ensures the accurate replication and division of cells. It involves sequential phases—G1, S, G2, and M—controlled by cyclins and cyclin-dependent kinases (CDKs)**(13).** Checkpoints at G1/S and G2/M monitor DNA integrity and cell readiness for division, halting progression when damage is detected. In cancer, including hepatocellular carcinoma (HCC), these checkpoints are frequently bypassed due to mutations or deregulation of key proteins like p53, p21, and cyclin D1, leading to uncontrolled cell proliferation and tumor progression. Recent research has highlighted the potential of plant-derived compounds to modulate the cell cycle and suppress tumor growth. Zeatin, a naturally occurring cytokinin, has shown promising anticancer effects in various in vitro studies. Evidence suggests that zeatin and its derivatives can induce cell cycle arrest, particularly at the G2/M and S phases, by modulating the expression of cyclins, CDKs, and CDK inhibitors. It has also been observed to upregulate p21 and downregulate proliferative markers like PCNA and Ki-67, thereby slowing down the replication machinery in cancer cells.

In liver cancer cells, zeatin's ability to interfere with cell cycle regulation may offer a novel therapeutic avenue. Its structural similarity to purines may enable it to disrupt DNA synthesis or activate DNA damage response pathways, halting cell division and promoting apoptosis. Further studies using flow cytometry, Western blotting, and gene

expression analysis can clarify its exact mechanism. If validated, zeatin could serve as a potential lead compound or adjuvant in the treatment of liver cancer, offering a natural and less toxic alternative to conventional chemotherapeutics.

MATERIALS AND METHODS

Cell Culture

HepG2 liver cancer cell line was purchased from NCCS, National Centre for Cell Sciences, Pune, India. The cells were maintained in DMEM medium containing 100 mg/L penicillin and 100 mg/L streptomycin supplemented with 10% foetal bovine serum at 37 C in a 5% C02 atmosphere. subculturing was done after the cells reached 80%-90%confluency. Prior to passaging, the media was discarded, and the cells were washed using 2 ml of PBS. The cells were then trypsinized with 2 ml of trypsin and incubated in a CO_2 incubator in a CO_2 incubator for 2 minutes to encourage cell detachment. 2 ml of serum containing media was added to the flask to inactivate the trypsin. The cells were centrifuged at 900 rpm for 3 minutes. The supernatant was discarded and the pellet was collected by gentle aspiration. The supernatant was discarded, and the pellet was collected by gentle aspiration. The pellet was suspended in growth media supplemented with 10% FBS. The cells were then seeded into 6 well and 96 well plates and incubated for 24 hours. Figure 1 represents the HepG2 cells on a 96 well plate.

Stock Preparation of Zeatin

ZEATIN (white crystalline powder form) was purchased from Himedia. The drug stock was prepared by dissolving 10mg of zeatin in 100 ul of ethanol. 10 ul from this stock is taken and dissolved in 1ml of DMEM medium and rapidly vortexed. This stock was then stored at 4c for future use (figure 2).

Assessment of Cytotoxicity of Zeatin –Mtt Assay in Hepg2 Cell Line

HepG2 liver cancer cells were plated in 96-well microplates with DMEM culture medium supplemented with 10% FBS and incubated at 37 c and 5% CO2, overnight. The next day, the cells were subject to treatment with zeatin for periods of 24 and 48 hours in varying concentrations from 5 to 50 ug/ml. The MTT dye was prepared by mixing 5 mg/ml of MTT in PBS. 50 µlof MTT solution was added to each well of the 96 well plate, The plate was wrapped in aluminium foil and incubated at 37 C for 3 hours Over the formazan precipitates formed, DMSO was added to each well to solubilize the crystals. The absorbance was taken at 570 nm concentration on the x axis and % inhibition on the y axis. The serves as a control to check the comparative effect of zeatin on liver cancer as well as normal cells (TOLOSA, DONATO AND GOMEZ-LECHON, 2015)

Assessment of Anticancer Activity of Zeatin –Mtt Assay on Hepg2 Cell Line

HepG2 liver cancer was plated in 96-well microplates, with DMEM culture medium supplemented with 10% FBS and incubated at 37 C and 5% CO2 overnight. The next day, the cells were subject to treatment with zeatin, for periods of 24 and 48 hours in varying concentrations from 5 to 50 ul /ml. The MTT dye was prepared by mixing 5 mg/ml of MTT in PBS. 50 ul of MTT solution was added to each well of the 96 well plate. The plate was wrapped in aluminium foil and incubated at 37 C for 3 hours. Over the formazan precipitates formed and DMSO was added to each well to solubilize the crystals. The absorbance was taken at 570 nm using a microplate reader. The data obtained were then plotted in a graph with concentration on the x axis and % inhibition on the y axis and the IC50 value was found (TOLOSA, DONATO, GOMEZ-LECHON, 2015).

Apoptosis Detection – Ao/Eb Staining

Acridine Orange (AO) and ethidium bromide (EB) staining is a widely used technique for evaluating cell viability and assessing apoptotic and necrotic cell populations within a sample. In liver cancer cell response to treatment. AO permeates both live and death cells, emitting green fluorescence upon integrity, emitting red fluorescence upon binding to nucleic acids. Live cells typically exhibit green fluorescence, whereas apoptotic cells may display a mix of green and orange fluorescence, and necrotic cells appear predominantly red or yellow. By analysing the relative

proportions of these fluorescent population under a fluorescence microscope we can gain insights into the efficacy of anti cancer treatments, mechanism of cell death, and overall cell viability in liver cancer cells.

***In vitro* Studies**

Assessment of anti

cancer activity of zeatin –MTT assay on HepG2 liver cancer

50 ul of MTT solution was added to each well of the 96 well plate. The plate was wrapped in aluminium foil and incubated at 37 C for 3 hours, over the formazan precipitates formed. DMSO was added to each well to solubilize the crystals. The absorbance was taken at 570 nm using a microplate reader. The data obtained were then plotted in a graph with concentration on the X axis and % inhibition on the y axis and the IC_{50} value was found. The IC_{50} value was found to be 14 ug/ml after 24 hours of drug treatment and 25 ug/ml after 48 hours of drug treatment.

Apoptosis Detection –AO/EB Staining

AO/EB dual staining was done on the HepG2 liver cancer after 24 hrs and 48 hours of drug treatment and images were taken in fluorescence microscopy. AO permeates both live and dead cells, emitting red fluorescence while EB only enters cells with compromised membrane integrity emitting red fluorescence upon binding to nucleic acid. The bright green dot represents cells in the early apoptotic stage with fragmented nuclei while the necrotic cells appear yellow.

RESULTS AND DISCUSSION

MTT assay

The cytotoxic potential of Zeatin against human hepatocellular carcinoma (HepG2) cells was evaluated using the MTT assay, a widely accepted method for assessing cell metabolic activity as an indicator of cell viability, proliferation, and cytotoxicity. HepG2 cells were treated with increasing concentrations of Zeatin (ranging from low to high doses) for 24 hours, and the resulting cell viability was measured by the reduction of MTT to formazan crystals by mitochondrial dehydrogenases in viable cells. The results demonstrated a clear dose-dependent decrease in cell viability, indicating that Zeatin exerts cytotoxic effects on HepG2 cells in a concentration-dependent manner. Lower concentrations exhibited mild inhibitory effects, while higher concentrations showed a substantial decline in viability. The absorbance values measured at 570 nm showed a progressive reduction with increasing concentrations of Zeatin, supporting its inhibitory impact on the proliferation of HepG2 cells. Statistical analysis confirmed the significance of the observed effects, with notable reductions in viability starting from mid-range concentrations. The half-maximal inhibitory concentration (IC_{50}) of Zeatin was calculated to be approximately 32.5 μg/mL, highlighting its potent cytotoxic action at this concentration. Morphological changes in Zeatin-treated HepG2 cells were also visually observed under an inverted phase-contrast microscope, showing typical signs of cellular stress, including cell shrinkage, rounding, and detachment from the surface—further supporting the MTT findings. These changes were more pronounced at concentrations near and above the IC_{50} value, correlating with the decreased viability observed in the colorimetric assay.

The MTT assay results provide compelling evidence that Zeatin effectively inhibits HepG2 cell growth and viability in vitro. Compared to untreated control cells, which maintained high metabolic activity and normal morphology, the treated cells demonstrated significant alterations in viability, suggesting that Zeatin's cytotoxicity is specific and impactful within a defined concentration range. These findings serve as a preliminary yet promising indication of Zeatin's anticancer potential and warrant further investigation through mechanistic studies, including apoptosis assays, oxidative stress analyses, and gene expression profiling. Additionally, the consistent and reproducible nature of the MTT results adds to the reliability of Zeatin as a potential therapeutic candidate. The clear dose-response relationship observed suggests that Zeatin may engage with specific intracellular pathways that regulate cell proliferation and survival, and its IC_{50} value of 32.5 μg/mL (Figure 3) serves as a benchmark for downstream experiments assessing molecular mechanisms of cell death. Taken together, these results emphasize that Zeatin exerts

substantial cytotoxic effects on liver cancer cells in vitro, thereby supporting its role as a viable candidate for further preclinical evaluation in hepatocellular carcinoma therapy **(14).**

Morphological analysis

The morphological assessment of HepG2 liver cancer cells**(15)** following treatment with Zeatin at its IC_{50} concentration (32.5 µg/mL) revealed distinct and significant alterations in cellular structure when compared to untreated control cells. Under normal conditions, control HepG2 cells displayed a typical epithelial-like morphology, characterized by a uniform monolayer of polygonal cells with intact cell membranes, well-defined borders, and strong adherence to the culture surface. These cells maintained normal nuclear integrity and cytoplasmic clarity, appearing healthy and proliferative with minimal signs of stress. In contrast, cells treated with Zeatin at the IC_{50} concentration 32.5 µg/mL for 24 hours exhibited marked morphological changes indicative of cytotoxic and apoptotic effects. These included a noticeable reduction in cell density and surface adherence, cell shrinkage, rounding of cells, and loss of the characteristic polygonal shape. Treated cells also showed membrane blebbing and cytoplasmic condensation—classic hallmarks of early apoptotic processes. In several treated fields, cellular debris and apoptotic bodies were evident, suggesting progression toward late apoptosis. These visual observations were consistent across multiple microscopic fields and were captured using an inverted phase-contrast microscope at 20× magnification. The overall architecture of the cell monolayer was disrupted following Zeatin treatment, and treated cells appeared more isolated, with diminished cell-cell contact, in stark contrast to the confluent monolayer observed in the control group.

The presence of these apoptotic features supports the findings of the MTT assay and further confirms the cytotoxic and anti-proliferative nature of Zeatin at its IC_{50} concentration 32.5 µg/mL. Notably, the visual evidence of nuclear condensation and membrane disruption aligns with the cellular events typically associated with programmed cell death. Moreover, these observations suggest that Zeatin may impair cellular adhesion and cytoskeletal integrity, leading to morphological deformation and detachment from the extracellular matrix. The contrast between treated and untreated cells provides strong visual confirmation of Zeatin's bioactivity against HepG2 cells, reinforcing the biochemical and viability assay data. In addition, no signs of necrotic swelling or lysis were observed, indicating that the mode of cell death was more likely apoptotic rather than necrotic**.** These results offer important qualitative evidence of Zeatin's impact on cancer cell structure and survival, thereby validating its role in inducing cytomorphological changes consistent with apoptosis. The observed morphological alterations at the IC_{50} concentration serve as crucial visual biomarkers of drug efficacy and may guide further studies investigating the ultrastructural and molecular mechanisms of Zeatin-induced cell death. Collectively, the morphological data underscore the anticancer potential of Zeatin in hepatocellular carcinoma by demonstrating profound structural changes in liver cancer cells upon treatment, thus highlighting its value as a candidate compound for therapeutic development (figure 4).

Necrosis Assay (PI staining)

Propidium iodide (PI) staining was employed to assess the morphological changes associated with late apoptosis and cell death in HepG2 liver cancer cells following treatment with Zeatin at its IC_{50} concentration (32.5 µg/mL). In the untreated control group, HepG2 cells displayed normal nuclear morphology, with minimal or no PI staining, indicating intact cell membranes and viable cells. The nuclei in these cells appeared faint and uniform, with no signs of chromatin condensation or nuclear fragmentation. In contrast, cells treated with Zeatin exhibited significant morphological alterations consistent with apoptotic cell death. PI staining revealed intense red fluorescence in the nuclei of treated cells, indicating compromised membrane integrity—a hallmark of late-stage apoptosis or necrosis. These cells also displayed classical apoptotic features such as nuclear condensation, fragmentation, and irregular nuclear shapes. A marked increase in the number of PI-positive cells was observed in the treated group compared to the control, confirming enhanced cell death upon exposure to Zeatin. The high-intensity nuclear staining observed in Zeatin-treated cells further supports the disruption of membrane permeability, which is typically observed during apoptosis. Additionally, treated cells appeared more rounded and detached, with clear loss of normal cellular morphology, further emphasizing the cytotoxic effect of Zeatin. The stark contrast between control and treated

groups demonstrates that Zeatin induces significant nuclear damage and loss of membrane integrity in HepG2 cells at IC_{50} concentration. These findings correlate well with MTT assay results and reinforce the pro-apoptotic activity of Zeatin**(16)**. The PI staining results provide strong visual confirmation of Zeatin-induced cell death, underscoring its potential as a therapeutic agent targeting hepatocellular carcinoma through membrane-disruptive and apoptosis-mediated mechanisms (figure 5)

AO/EB Staining

Acridine orange/ethidium bromide (AO/EB) dual staining was performed to assess the morphological features associated with apoptotic and necrotic cell death in HepG2 liver cancer cells following treatment with Zeatin at its IC_{50} concentration (32.5 µg/mL), in comparison with untreated control cells. In the control group, cells exhibited bright green fluorescence with round, intact nuclei, indicating a predominance of viable cells with intact membranes and no evidence of nuclear damage. These control cells showed typical morphology with normal cytoplasmic and nuclear architecture, suggesting healthy proliferation and minimal spontaneous apoptosis under standard conditions.

In contrast, HepG2 cells treated with Zeatin displayed marked fluorescence changes indicative of apoptosis and compromised membrane integrity. AO/EB staining allows for the differentiation between viable, early apoptotic, late apoptotic, and necrotic cells based on differential uptake and fluorescence color: acridine orange stains both live and dead cells green by intercalating into DNA, while ethidium bromide only enters cells with damaged membranes and stains their nuclei orange to red. Upon Zeatin treatment, a significant population of HepG2 cells exhibited yellow-orange to reddish fluorescence with condensed or fragmented nuclei, signifying early and late apoptotic stages. Many cells also showed membrane blebbing, chromatin condensation, and apoptotic body formation—classic morphological features of apoptosis. The appearance of orange to red nuclei with dense chromatin in treated cells confirmed the progression of apoptosis and disruption of membrane integrity. Furthermore, a visible reduction in the number of viable green-fluorescent cells was noted, with a corresponding increase in apoptotic and necrotic cells, suggesting Zeatin-induced cytotoxicity is primarily mediated via apoptosis **(17).**

The proportion of early apoptotic cells (greenish-yellow with condensed chromatin) and late apoptotic/necrotic cells (bright orange-red with fragmented nuclei) increased significantly compared to the control, reflecting the dose-specific activity of Zeatin at IC_{50} concentration. These observations were consistent across multiple microscopic fields and were captured under a fluorescence microscope using a green-red filter combination. The treated cells appeared shrunken, with disrupted morphology, loss of adherence, and reduced intercellular contact—further supporting the induction of programmed cell death. The distinct shift in nuclear morphology and fluorescence intensity clearly distinguished apoptotic cells from viable cells, confirming that Zeatin triggers significant apoptotic cell death in HepG2 cells (18).

The AO/EB staining results complement the findings of the MTT and PI assays, providing visual and qualitative evidence of Zeatin's pro-apoptotic action **(19)**. The clear morphological distinction between untreated and treated cells underscores the potent cytotoxic nature of Zeatin and its ability to activate apoptotic pathways in liver cancer cells. These results collectively demonstrate that Zeatin induces apoptosis in HepG2 cells through morphological and nuclear alterations detectable by AO/EB staining, and further support its potential as an effective anticancer compound targeting hepatocellular carcinoma. The consistency of apoptotic features across treated fields strengthens the conclusion that apoptosis, rather than necrosis, is the primary mode of cell death induced by Zeatin in this experimental setting (20).

CONCLUSION

Zeatin exhibits significant anticancer activity against HepG2 liver cancer cells by inducing dose-dependent cytotoxicity and apoptosis. Morphological alterations and nuclear staining confirm Zeatins role in disrupting cell

structure and promoting programmed cell death. These findings highlight Zeatins potential as a promising natural compound for hepatocellular carcinoma therapy. Further studies are needed to explore its molecular mechanisms and therapeutic applicability.

Declaration of Competing Interest

The authors claim that no known conflicting financial interests or personal relationships appeared to have an impact on the work that was published in this paper.

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Figure 1. HepG2 Cells seeded on a 96 well plate.

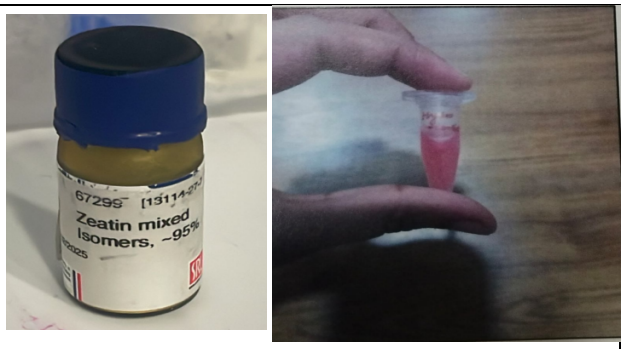

Figure 2. Zeatin and proper Stock preparation from Zeatin

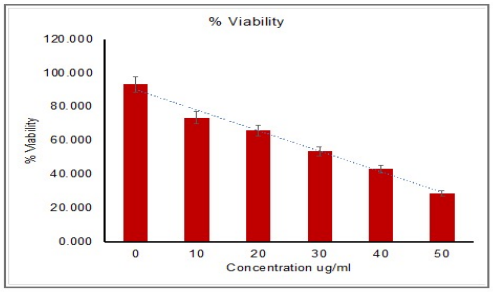

Figure 3. Proliferation rates based on the MTT assay for HepG2 liver cancer cells

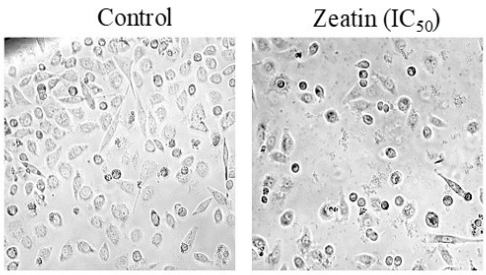

Figure 4. Morphological analysis of HepG2 liver cancer cells treated with IC_{50} concentrations of zeatin (32.5 µg/mL) for 24 hrs.

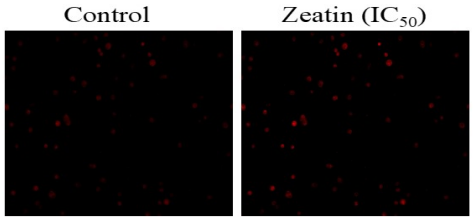

Figure 5. Morphological analysis by PI staining of HepG2 liver cancer cells treated with IC_{50} concentrations of zeatin 32.5 µg/mL for 24 hrs.

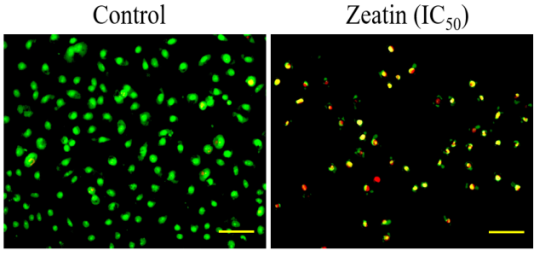

Figure 6 Morphological analysis by AO/EB staining of HepG2 liver cancer cells treated with IC_{50} concentrations of zeatin 32.5 µg/mL for 24 hrs.