BULLETIN OF STOMATOLOGY AND MAXILLOFACIAL SURGERY
Volume 21, Issue 3

DOI: 10.58240/1829006X-2025.3-55

RESEARCH ARTICLE

EVALUTION OF ANTICANCER POTENTIAL OF PIPERINE IN HUMAN ORAL SQUAMOUS CELL CARCINOMA-AN IN VITRO EXPERIMENTAL STUDY

Afrin Nisha Mansoor Ali[1], Priyadharshini Ranganathan[2*]

[1]Undergraduate, Department of Pathology, Saveetha Dental College and Hospital, Saveetha Institute of medical and technical Sciences (SIMATS), Saveetha university, Chennai-600 077, Tamil Nadu, India. email ID: 152001076.sdc@saveetha.com
[2*]Assistant Professor, Department of Oral Pathology, Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences (SIMATS), Saveetha University, Chennai-600 077, Tamil Nadu, India. email ID: priyadharshinir.sdc@saveetha.com

[*]**Corresponding author:** Priyadharshini Ranganathan Assistant Professor, Department of Oral Pathology, Saveetha Dental College & Hospitals, Saveetha Institute of Medical and Technical Sciences, Chennai, Tamilnadu, India.Mail Id: priyadharshinir.sdc@saveetha.com

Received: *Feb. 24, 2025;* ***Accepted:*** *Mar. 14, 2025;* ***Published:*** *Mar. 24, 2025*

ABSTRACT

Background: Oral squamous cell carcinoma (OSCC) is one of the most prevalent and aggressive malignancies, characterized by uncontrolled cell proliferation and high recurrence rates. Despite advancements in therapy, resistance to conventional treatments remains a major challenge. *Piperine,* a bioactive alkaloid from black pepper, has demonstrated anticancer potential in various cancer models through apoptosis induction and cell cycle arrest. However, its specific effects on oral cancer cell lines remain underexplored. This study evaluates the anticancer activity of Piperine in KB (Human oral cancer) cell line, focusing on its impact on cell viability and apoptotic gene expression.

Material and Method: KB oral cancer cells were cultured and treated with Piperine at 25 µM, 50 µM, and 100 µM. Cell viability was assessed using the MTT assay, while morphological changes were observed under an inverted microscope. The expression of key apoptotic genes (Bcl-2, Bax, and p53) was analyzed using quantitative real-time PCR (qRT-PCR). Data were statistically analyzed using one-way ANOVA, and results were expressed as mean ± SEM.

Results: Piperine exhibited a dose-dependent cytotoxic effect, significantly reducing cell viability at higher concentrations (50 µM and 100 µM). Microscopic observations revealed cell shrinkage, detachment, and membrane blebbing, indicating apoptotic cell death. Gene expression analysis showed downregulation of Bcl-2 (anti-apoptotic gene) and upregulation of Bax and p53 (pro-apoptotic genes), confirming Piperine induced apoptosis.

Conclusion: These findings suggest that Piperine exerts anticancer effects on KB oral cancer cells by inducing apoptosis via the mitochondrial pathway. Its ability to modulate Bcl-2, Bax, and p53 expression highlights its potential as a therapeutic agent for OSCC. Further preclinical and clinical studies are warranted to explore its bioavailability and translational applications in oral cancer therapy.

Keywords: Apoptosis, Anti-cancer, Cell line, Oral squamous cell carcinoma, Piperine

INTRODUCTION

Oral squamous cell carcinoma (OSCC) is a disease characterized by uncontrolled and abnormal cell growth, which can progress slowly if not managed. When cancer infiltrates a single cell or a group of cells, its progression rate doubles over time[1].

This type of cancer can affect various tissues in and around the mouth, including bones, muscles, and nerves, leading to both benign and malignant growths. It is a significant global health concern due to its high incidence and mortality rates[2]. The complexity of treatments and the economic burden further challenge patient survival. Despite post-surgical treatment, there is an increased risk of local and regional recurrence[3]. Patients diagnosed early have a 90% survival rate, while those with late-stage cancer face a mortality rate of around 60%[4].

Black pepper, a member of the Piperaceae family, produces mature but unripe fruits used as a popular spice. Many spices, particularly pepper, contain compounds with insulin resistance properties and antioxidant potential[5,6]. *Piperine*, a component of black pepper, has been traditionally used as a flavour enhancer and in traditional medicine[7]. Studies have shown that *Piperine* induces cell death by depleting Matrix Metalloproteinase (MMP) and activating caspase-3. It also reduces DNA levels and arrests cell growth in G2/M phase, as demonstrated in cell cycle studies. These findings suggest that *Piperine* effectively induces cell death by reducing MMP levels and Reactive Oxygen Species (ROS) release, subsequently triggering caspase-3 activation and cell cycle arrest[8]. *Piperine* has displayed a wide range of physiological effects, including anti-hypertensive, anti-coagulant, antioxidant, anti-cancer, antispasmodic, anti-asthmatic, antidepressant, and anxiolytic properties[9].

Most studies on *Piperine's* anticancer potential have focused on cancers such as breast, lung, colon, and prostate, with limited research on its effects in oral cancer. Furthermore, variations in dosages and exposure times across different studies make it difficult to establish a standardized therapeutic dose. While some research has explored *Piperine's* cytotoxic properties, where there is still a lack of in-depth mechanistic studies specifically targeting oral cancer cell lines. This study aimed to analyse the anticancer potential by observing the expression analysis of *Piperine* in Oral cancer cell line.

MATERIALS AND METHODS

Chemicals

For this study, *Piperine* was procured from Sigma-Aldrich. Essential cell culture reagents, including trypsin-EDTA, fetal bovine serum (FBS), antibiotic-antimycotic solution, Dulbecco's Modified Eagle's Medium (DMEM), and phosphate-buffered saline (PBS), were obtained from Gibco, Canada. The JC-1 dye(5,5,6,6-tetrachloro-1,1,3,3-tetraethyl benzimidazole carbocyanine iodide) and real-time PCR kits (MESA Green) were sourced from Invitrogen, USA. All chemicals utilized in the study were of high analytical grade to ensure precision and reliability.

Procurement and Culture of Oral Cancer Cells

The Oral cancer cell line used in this study was acquired from the National Centre for Cell Science (NCCS), Pune, India. The cells were cultured following standard protocols, utilizing Minimum Essential Medium (MEM) supplemented with 10% FBS, and maintained at 37°C in a humidified atmosphere with 5% CO_2 to support optimal growth conditions.

Cell Viability Assay

Oral cancer cells were seeded in 96-well plates at a density of 5×10^5 cells per well and allowed to adhere overnight. Following incubation, the cells were exposed to varying concentrations of *Piperine* in triplicate and maintained for 24 hours at 37°C in a humidified incubator with 5% CO_2 . Subsequently, 3- (4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) was added to each well, and the cells were incubated for an additional four hours at 37°C. The resulting formazan crystals were dissolved in 200 µL of dimethyl sulfoxide (DMSO), and the optical density (OD) was measured. The standard deviation (SD) was also calculated for each set of replicates. The entire experiment was conducted three times to ensure reproducibility. The percentage of cell growth inhibition was calculated using the following formula:

*%Growth Inhibition= (1−OD of extract-treated cells/OD of negative control)*100*

Gene Expression Analysis by Real-Time PCR

The expression levels of target mRNA were quantified using real-time PCR. Total RNA was extracted using Tri Reagent (Sigma), and cDNA synthesis was performed using the Superscript III First-Strand cDNA Synthesis Kit (Invitrogen, USA) according to the manufacturer's instructions. For

gene expression analysis, real-time PCR reactions were carried out on an MX3000p PCR system (Stratagene, Europe) using the MESA Green PCR Master Mix (Eurogentec, USA), which contains all essential PCR components along with SYBR Green dye. The specificity of the amplified products was confirmed through melting curve analysis for each primer pair. Data analysis was conducted using the comparative Ct method (ΔΔCt approach), and fold-change expression was determined using the $2^{-\Delta\Delta Ct}$ method, as described by Venkata et al. (2022)[11].

Statistical analysis

Cell viability at varying concentrations of *Piperine* was assessed, and the fold change in the expression levels of Bcl-2, Bax and p53 was calculated, and the corresponding mean ± SEM values were determined. To evaluate statistical significance, one-way analysis of variance (ANOVA) was performed to compare cell viability across the three different concentrations of *Piperine*, assessing potential differences in fold change at various treatment levels.

RESULTS

The effect of *Piperine* on KB oral cancer cells was analyzed at different concentrations (25 µM, 50 µM, and 100 µM) using an inverted light microscope at 10× magnification. In the control group (untreated cells), the cells exhibited normal morphology, with a high density, adherent nature, and intact cellular structure, indicating healthy proliferation. However, upon treatment with *Piperine* at 25 µM, a slight reduction in cell density was observed, with some cells displaying rounding and detachment, suggesting early apoptotic changes.

At 50 µM, the cytotoxic effects of *Piperine* became more pronounced, with a marked decrease in cell density and a higher number of detached cells. Many cells exhibited membrane shrinkage and irregular morphology, indicating an increased rate of apoptosis. The 100 µM concentration of *Piperine* demonstrated the most substantial cytotoxic effect, as the majority of cells appeared shrunken and detached, with notable membrane blebbing and irregular structures, suggesting a combination of apoptotic and necrotic cell death. These findings suggest a dose-dependent cytotoxic effect of *Piperine* on KB oral cancer cells, with higher concentrations (50 µM and 100 µM) inducing significant morphological alterations associated with apoptosis and loss of viability (Figure 1).

Figure 1. The images show morphological changes in KB oral cancer cells treated with *Piperine* (25 µM, 50 µM, and 100 µM) compared to the untreated control. A dose-dependent reduction in cell density was observed, with cell shrinkage, rounding, and detachment, indicating apoptosis. At 100 µM, extensive cell death, membrane blebbing, and loss of adherence confirm *Piperine*-induced cytotoxicity.

The MTT assay results demonstrate a dose-dependent cytotoxic effect of *Piperine* on KB oral cancer cells, as indicated by the progressive decline in cell viability with increasing concentrations of *Piperine*. In the untreated control group, cell viability was the highest, approximately 90%, indicating normal cell proliferation in the absence of treatment. However, upon exposure to 25 µM of *Piperine*, a slight reduction in viability (~75–80%) was observed, suggesting mild cytotoxicity at this concentration.

At 50 µM, a significant decline in cell viability (~50–55%) was noted, indicating an increased cytotoxic response, likely due to the induction of apoptosis or cell death mechanisms. The most substantial reduction in cell viability was observed at 100 µM, where only ~40% of cells remained viable, highlighting the potent anticancer potential of *Piperine* at higher concentrations. These findings confirm that *Piperine* effectively inhibits KB oral cancer cell proliferation in a dose-dependent manner, with higher concentrations (50 µM and 100 µM) inducing strong cytotoxic effects. The results suggest that *Piperine* may exert its anticancer activity by triggering apoptosis or other cell death pathways (Figure 2).

Afrin Nisha Mansoor Ali, Priyadharshini Ranganathan.Evaluation of the Anticancer Potential of Piperine in Human Oral Squamous Cell Carcinoma- An In Vitro Experimental Study.Bulletin of Stomatology and Maxillofacial Surgery. 2025;21(3)55-63. doi:10.58240/1829006X-2025.3-55

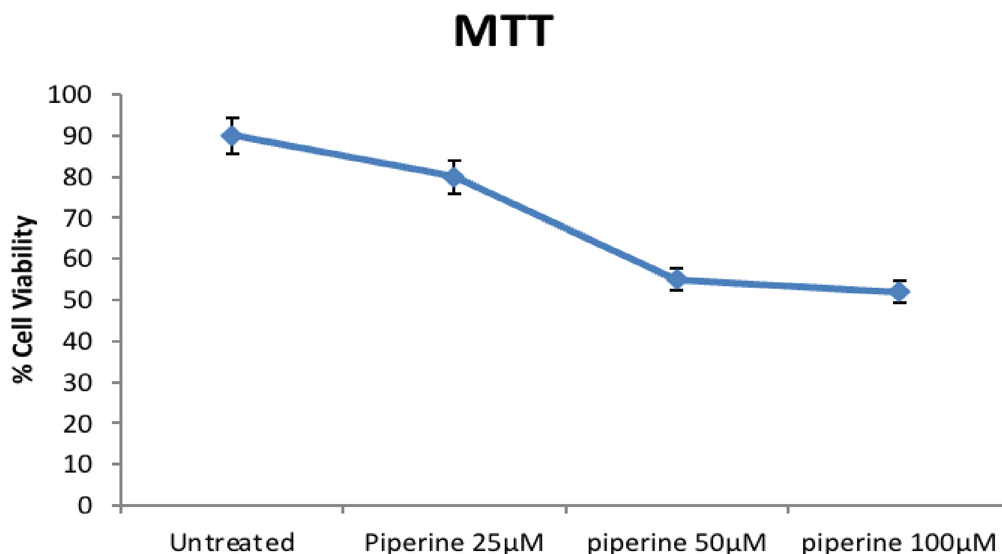

Figure 2. The graph shows the percentage cell viability of KB oral cancer cells treated with *Piperine* (25 µM, 50 µM, and 100 µM), assessed by the MTT assay. A dose-dependent decline in viability indicates *Piperine's* cytotoxic effect, with a significant reduction at 50 µM and 100 µM, suggesting

The expression levels of Bcl-2, an anti-apoptotic gene, in KB oral cancer cells following treatment with different concentrations of *Piperine* (25 µM, 50 µM, and 100 µM) compared to the untreated control group. The results indicate a dose-dependent reduction in Bcl-2 expression, suggesting that *Piperine* induces apoptosis by downregulating this survival promoting gene. In the untreated control group, Bcl-2 expression was the highest, set as baseline (1.0 mean expression value), indicating that the cancer cells maintain a strong survival mechanism in the absence of treatment. Upon exposure to 25 µM of *Piperine*, Bcl-2 expression decreases to 0.74 ± 0.09, indicating the initiation of apoptotic signalling. A further reduction was observed at 50 µM (0.60 ± 0.05) and 100 µM (0.59 ± 0.05), suggesting a significant suppression of Bcl-2 with increasing *Piperine* concentrations (table 1).

Table 1. Expression analysis of *Piperine* in Oral carcinoma cell line

Expression analysis	**Control (untreated KB Cells)**	**Oral carcinoma cells + *Piperine* (50µM)**	**Oral carcinoma cells + *Piperine* (100µM)**	**Oral carcinoma cells + *Piperine* (200µM)**
Bcl2 mRNA	1±0	0.74±0.09	0.60±0.05	0.59±0.05
Bax mRNA	1±0	1.1±0.07	1.3±0.06	1.4±0.06
p53	1±0	1.2±0.09	1.6±0.12	1.6±0.11

**Values are given as: Mean ± S.E.M*

The decline in Bcl-2 expression confirms that *Piperine* promotes apoptosis in KB oral cancer cells by inhibiting anti-apoptotic pathways, thereby making cells more susceptible to programmed cell death. This trend aligns with the MTT assay results, which showed a dose-dependent reduction in cell viability, further supporting *Piperine's* anticancer potential (Figure 3).

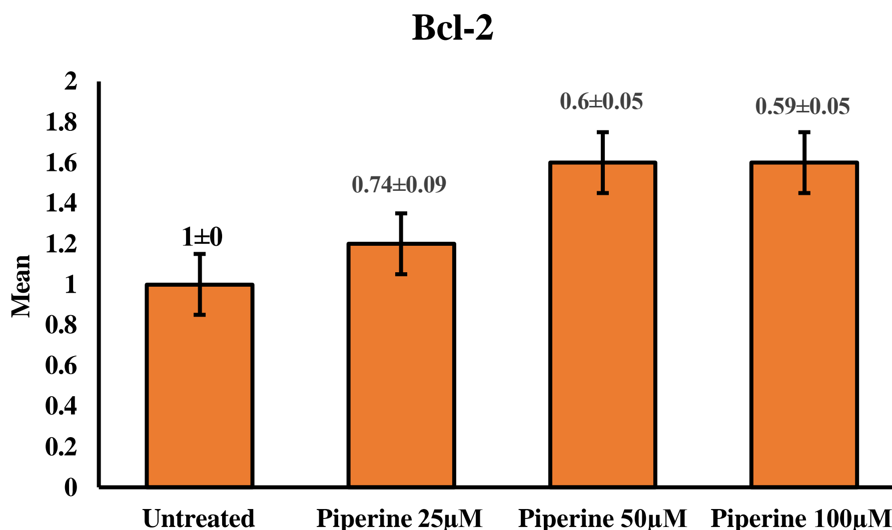

Figure 3. The y-axis represents the mean Bcl-2 expression levels, while the x-axis denotes the different treatment groups, with error bars indicating the standard error of the mean (SEM). A dose-dependent decrease in Bcl-2 expression was observed, with the highest reduction at 100 µM (0.59 ± 0.05), indicating that *Piperine* suppresses anti-apoptotic signalling. These findings suggest that *Piperine* induces apoptosis in KB cells by downregulating Bcl-2, thereby shifting the balance toward cell death pathways.

Bax, a pro-apoptotic gene, in KB oral cancer cells treated with different concentrations of *Piperine* (25 µM, 50 µM, and 100 µM) compared to the untreated control group. The results indicate a dose-dependent upregulation of Bax expression, suggesting that *Piperine* induces apoptosis by promoting the activation of this pro-apoptotic pathway. In the untreated control group, Bax expression was at its baseline (~1.0 mean expression value), indicating a normal level of apoptotic signaling in cancer cells. Following treatment with 25 µM *Piperine*, Bax expression increases to 1.1 ± 0.07, suggesting the initiation of apoptosis. At 50 µM, Bax expression rises further to 1.3 ± 0.06, and at 100 µM, it reaches its highest level at 1.4 ± 0.06, indicating a significant induction of apoptosis at higher *Piperine* concentrations.

The progressive increase in Bax expression with increasing *Piperine* concentrations confirms that *Piperine* triggers apoptosis in KB oral cancer cells by activating the pro-apoptotic pathway. This trend was consistent with the Bcl-2 expression results, which showed a dose-dependent downregulation of Bcl-2, leading to a higher Bax/Bcl-2 ratio a critical factor for apoptosis activation. These findings reinforce *Piperine's* anticancer potential and suggest that its apoptotic effect may be mediated through the mitochondrial (intrinsic) pathway (Figure 4).

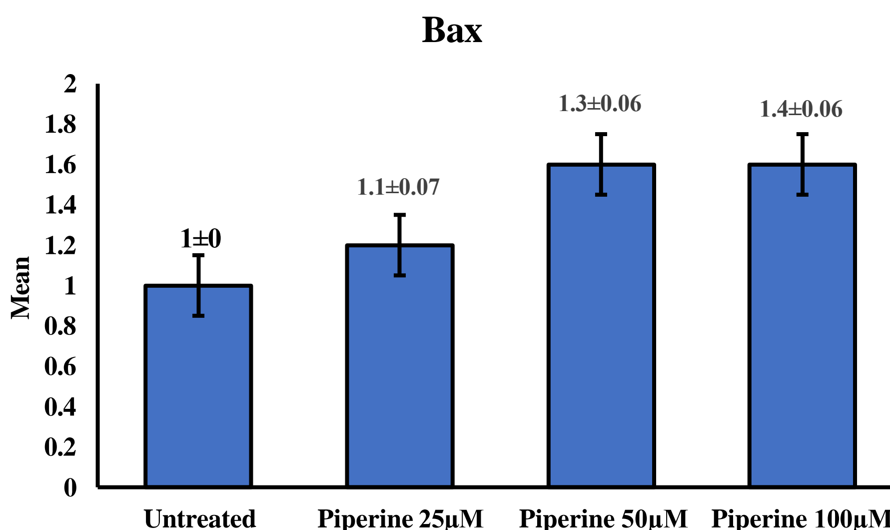

Figure 4. The expression of Bax, a pro-apoptotic gene, was analyzed in KB oral cancer cells after treatment with

different concentrations of *Piperine* (25 µM, 50 µM, and 100 µM) in comparison to the untreated control. The y-axis denotes mean Bax expression levels, while the x-axis represents the treatment groups. A dose-dependent increase in Bax expression was observed, with the highest level at 100 µM (1.4 ± 0.06), confirming the apoptotic potential of *Piperine*. These results suggest that *Piperine* induces apoptosis in KB cells by upregulating Bax, promoting a shift towards programmed cell death.

A dose-dependent elevation in p53 expression was observed following *Piperine* treatment. Compared to the untreated control, where p53 expression was at its lowest, an increase to 1.2 ± 0.09 was noted at 25 µM, suggesting the initiation of p53-mediated apoptotic signaling. A further substantial upregulation was detected at 50 µM and 100 µM with Mean ± S.D as 1.6 ± 0.12 and 1.6 ± 0.11, indicating that *Piperine* induced a sustained activation of p53-dependent cell death pathways. Since p53 played a pivotal role in DNA damage response, cell cycle arrest, and apoptosis, its upregulation supported the notion that *Piperine* triggered apoptotic mechanisms to inhibit oral cancer cell survival.

These findings suggested that *Piperine* effectively enhanced p53 expression, reinforcing its role in tumour suppression by potentially promoting apoptosis and inhibiting uncontrolled proliferation. The consistency in p53 expression levels at 50 µM and 100 µM implied that beyond a certain threshold, *Piperine* may have maximized its stimulatory effect on p53 activation (Figure 5).

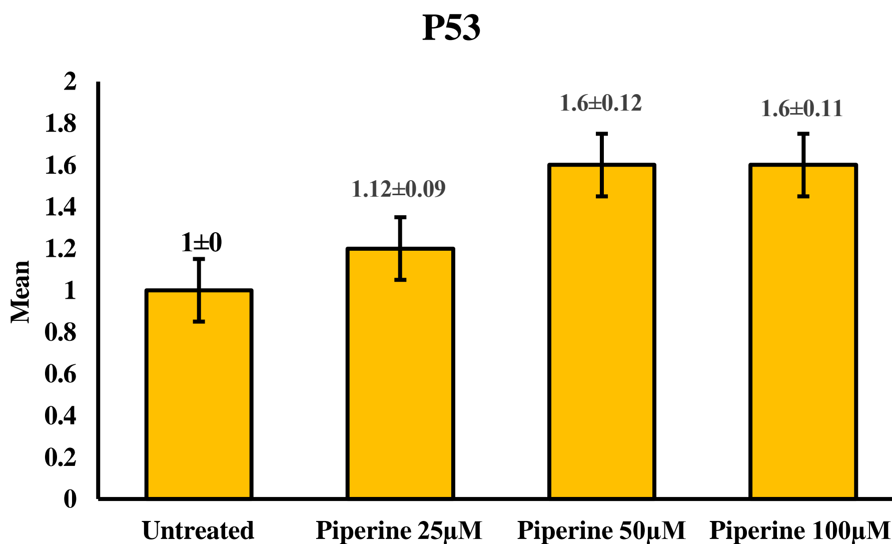

Figure 5. The y-axis denoted the mean p53 expression levels, while the x-axis represented the treatment groups. A dose-dependent increase in p53 expression was observed, with the highest levels at 50 µM (1.6 ± 0.12) and 100 µM (1.6 ± 0.11), suggesting that *Piperine* enhanced p53-mediated apoptotic signaling. These results indicated that *Piperine* upregulated p53 expression, promoted apoptosis, and potentially contributed to tumour suppression in KB oral cancer cells.

DISCUSSION

The present study demonstrated that *Piperine* induces apoptosis in KB oral cancer cells in a dose-dependent manner, as indicated by a reduction in Bcl-2 expression and an upregulation of Bax and p53. These findings are consistent with recent research highlighting *Piperine's* anticancer properties in various malignancies, particularly its role in apoptotic pathway activation and tumour suppression mechanisms. A recent study by Guo et al. (2021) reported that *Piperine* induces apoptosis in gastric cancer cells by increasing Bax/Bcl-2 expression ratio, ultimately leading to mitochondrial membrane depolarization and caspase activation[12]. Similarly, Shaheer et al. (2024) found that *Piperine* inhibits proliferation and promotes apoptosis in breast cancer cells via p53-mediated signaling, further supporting the results obtained in this study[13]. Our findings align with these studies, as we observed a significant increase in Bax and p53 expression, confirming *Piperine's* role in intrinsic apoptotic pathway. Despite these promising results, limited research has

specifically investigated *Piperine's* effects on oral squamous carcinoma cell lines. A study by Siddiqui et al. (2017) examined the cytotoxic effects of *Piperine* on oral cancer cells and found that it induces G2/M cell cycle arrest and promotes apoptosis via ROS generation and caspase activation[14]. The current study extended these findings by demonstrating a dose-dependent decrease in Bcl-2 expression, further supporting *Piperine's* role in downregulating anti-apoptotic mechanisms in oral cancer.

In addition to apoptotic pathway activation, our study highlights *Piperine's* potential for clinical application. Recent evidence suggests that *Piperine* enhances the efficacy of conventional chemotherapeutic agents. Mohammed et al. (2024) reported that *Piperine* synergistically enhances the anticancer effects of cisplatin in tongue squamous cell carcinoma by increasing ROS production and apoptosis markers[15]. These findings suggest that *Piperine* could be a valuable adjunct in combination therapies for oral cancer treatment, reducing the required dose of conventional chemotherapy while mitigating side effects.

Furthermore, bioavailability remains a critical challenge in *Piperine*-based treatments. Bolat et al. (2020) emphasized the need for nano formulations and delivery systems to improve *Piperine's* pharmacokinetics and therapeutic efficacy[16]. Future studies should focus on nanoparticle-based *Piperine* formulations to enhance targeted drug delivery and maximize its anticancer potential in oral squamous cell carcinoma.

CONCLUSION

This study confirms that *Piperine* effectively induces apoptosis in KB oral cancer cells by regulating key apoptotic markers such as Bcl-2, Bax, and p53. The results suggest that *Piperine* has significant therapeutic potential and could be explored as a treatment strategy for oral cancer. However, further preclinical and clinical studies are required to assess its bioavailability, pharmacokinetics, and long-term therapeutic impact.

DECLARATIONS

Acknowledgement
The authors are thankful to Saveetha Institute of Medical and Technical Sciences, Saveetha Dental College and Hospitals, Saveetha University for giving a platform to conduct the study.

Conflict of interest
All the authors declare that there was no conflict of interest in the present study.

Source of funding
The present project is funded by

- Saveetha Institute of Medical and Technical Sciences,
- Saveetha Dental College and Hospitals and Saveetha University
- Saveetha University
- National Insurance building, Parry's, Chennai.

REFERENCES

1. Reichal P, Ramani P, Kizhakkoottu S. Association of Site and recurrence in oral squamous cell carcinoma patients visiting private hospital in Chennai: a retrospective study. Cureus. 2024 Jan 23;16(1).
2. Badwelan M, Muaddi H, Ahmed A, Lee KT, Tran SD. Oral squamous cell carcinoma and concomitant primary tumors, what do we know? A review of the literature. Current Oncology. 2023 Mar 27;30(4):3721-34.
3. Mohandas R, Ramani P, Mohapatra S. Interobserver Audit of Surgical Margins in Patients with Recurrence of Oral Squamous Cell Carcinoma–A Retrospective Study. Oral & Maxillofacial Pathology Journal. 2023 Jul 1;14(2).
4. Yuktha A, Bandari SC, Fathima SJ, Selvaraj J, Veeraraghavan VP, Dasari AK, Patil SR. Determinants of Diagnostic Delays in Oral Squamous Cell Carcinoma: Insights from Demographic and Socio-Economic Factors. Asian Pacific Journal of Cancer Prevention. 2024 Nov 1;25(11):3997-4003.
5. Prasad M, Jayaraman S, Natarajan SR, Veeraraghavan VP, Krishnamoorthy R, Gatasheh MK, Palanisamy CP, Elrobh M. Piperine modulates IR/Akt/GLUT4 pathways to mitigate insulin resistance: Evidence from animal and computational studies. International Journal of Biological Macromolecules. 2023 Dec 31;253:127242.
6. Jayachandran P, Gayathri R, Jayaraman S, Vishnu Priya V, Kavitha S. Antioxidative stress potential of piperine in the gastrocnemius muscle of high fat diet and sucrose induced type 2 diabetic rats. HIV Nurs. 2023;23:81-9.
7. Ashokkumar K, Murugan M, Dhanya MK, Pandian A, Warkentin TD. Phytochemistry and therapeutic potential of black pepper [Piper nigrum (L.)] essential oil and piperine: A review. Clinical Phytoscience. 2021;7(1):52.
8. de Almeida GC, Oliveira LF, Predes D, Fokoue HH, Kuster RM, Oliveira FL, Mendes FA, Abreu JG. Piperine suppresses the Wnt/β-catenin pathway and has anti-cancer effects on colorectal cancer cells. Scientific reports. 2020;10(1):11681.
9. Stojanović-Radić Z, Pejčić M, Dimitrijević M, Aleksić A, V Anil Kumar N, Salehi B, C Cho W, Sharifi-Rad J. Piperine-a major principle of black pepper: a review of its bioactivity and studies. Applied Sciences. 2019;9(20):4270.
10. Benayad S, Wahnou H, El Kebbaj R, Liagre B, Sol V, Oudghiri M, Saad EM, Duval RE, Limami Y. The promise of Piperine in cancer chemoprevention.Cancers. 2023;15(22):5488.
11. Venkatakarthikeswari GV, Ranganathan Jr P, Palati S, Priyadharshini R. Tangeretin's anti-apoptotic signaling mechanisms in oral cancer cells: in vitro anti-cancer activity. Cureus. 2023;15(10).
12. Guo L, Yang Y, Sheng Y, Wang J, Ruan S, Han C. Mechanism of piperine in affecting apoptosis and proliferation of gastric cancer cells via ROS- mitochondria- associated signalling pathway. Journal of Cellular and Molecular Medicine. 2021;25(20):9513-22.
13. Shaheer K, Prabhu BS, Ali HS, Lakshmanan-M D. Breast cancer cells are sensitized by piperine to radiotherapy through estrogen receptor-α mediated modulation of a key NHEJ repair protein-DNA-PK. Phytomedicine.2024.Jan 1;122:155126.
14. Siddiqui S, Ahamad MS, Jafri A, Afzal M, Arshad M. Piperine triggers apoptosis of human oral squamous carcinoma through cell cycle arrest and mitochondrial oxidative stress.Nutrition andcancer.2017;69(5):791-9.
15. Mohamed MA, Elgayar SF, Abd Elaziz EA. Piperine Enhances the Anticancer Effects of Cisplatin on Tongue Squamous Cell Carcinoma Cell Line by Inducing Cell

Apoptosis. Asian Pacific Journal of Cancer Prevention: APJCP. 2024;25(9):3219.

16. Bolat ZB, Islek Z, Demir BN, Yilmaz EN, Sahin F, Ucisik MH. Curcumin-and piperine-loaded emulsomes as combinational treatment approach enhance the anticancer activity of curcumin on HCT116 colorectal cancer model. Frontiers in bioengineering and biotechnology. 2020 Feb 11;8:50.

Afrin Nisha Mansoor Ali, Priyadharshini Ranganathan.Evaluation of the Anticancer Potential of Piperine in Human Oral Squamous Cell Carcinoma- An In Vitro Experimental Study.Bulletin of Stomatology and Maxillofacial Surgery. 2025;21(3)55-63. doi:10.58240/1829006X-2025.3-55