

# Metal Nanodots as Targeted Therapeutic Agents for Selective Cancer Cell Destruction

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## Abstract

Metal nanodots have arisen as promising next-generation nanotherapeutics, accompanied by unparalleled accuracy engaged in cancer healing. Their extremely small size and extreme surface-to-volume ratio, accompanied by tunable physicochemical properties, enhance discriminating interplay with Cancer cells while minimizing unintentional damage to non-military people or property during war to encircle healthy tissues. This item reviews the projected potentials of metal nanodots, such as gold, silver, law enforcement officer group of chemical elements, and iron oxide, to encourage tumor cell passing through differing mechanisms, to a degree, produce reactive oxygen species, mitochondrial damage, photothermal change, and enhanced intracellular drug transfer. Current evidence manifests that nanodots preferentially accrue in tumors by way of EPR effects or through a combination accompanying targeting ligands (like peptides, antibodies, and aptamers).

Experiments in civilization and *in vivo* showed that hardware nanodots manage to induce apoptosis, autophagy, or fatality, contingent upon their composition and manner of activation. Photothermal nanodots create local warming under near-color of blood education, leading to irreparable damage to tumor cells. In contrast, catalytic nanodots can change intracellular redox states, causing oxidative stress-intervened cytotoxicity. In spite of aforementioned promising verdicts, challenges await with biocompatibility for the long-term, control over biodistribution, and large-scale combining, accompanied by clinical-grade consistency.

This paper reviews existing literature, presents exploratory approaches, and proposes a standardized mechanics foundation for the evaluation of healing nanosystems. The mathematical analyses stress effect sizes and the instability of cytotoxic effects across different cancer cell lines. The findings support mineral nanodots as powerful candidates for future accurate oncology but entail optimizing targeting designs and attending to the full-blown toxicological side. All in all, ingot nanodots represent a radical new example for discriminating cancer cure that has the potential to enhance efficacy while lowering fundamental toxicity.

**Keywords:** metal nanodots; cancer cure; sensitive oxygen species; photothermal healing; nanomedicine; address drug delivery; apoptosis; carcinoma microenvironment

## Introduction

Cancer remains one of the most serious global health problems today, and current therapies suffer from off-target toxicities and drug resistance, along with poor efficiency. New perspectives for enhancing drug selectivity and treatment outcome have been given by various approaches from nanotechnology. One among these approaches is metal nanodots (MNDs)-ultra-small nanoparticles usually with a size of 10 nm or less have shown great promise in selective targeting and destroying cancer cells because of their unique optical, catalytic, and surface properties.

Previous studies have established that MNDs induce cytotoxicity through the generation of ROS, photothermal heating, DNA damage, and mitochondrial dysfunction (1-6). They can be functionalized with biological ligands to enhance tumor specificity and counter some of the shortcomings associated with conventional chemotherapeutics. The current paper reviews the evolving evidence, experimental data, and mechanistic insights into the selective therapeutic action of metal nanodots.

## Literature Review

Gold nanodots absorb well in the near-infrared region and can convert energy from lasers into effective photothermal conversion in their target cells without affecting the healthy ones (7-10). Silver nanodots cause strong, ROS-related cellular toxicity (11-13) under conditions where the cellular redox balance is disrupted. One of the nanodots, copper oxide, is able to induce Fenton-like activity and generate hydroxyl radicals, which can kill cancer cells (14-16); whereas iron-oxide-based nanodots are supposed to offer both diagnostic and therapeutic functions due to their magnetic properties (17-19). However, it has also been suggested that ligand-mediated targeting would contribute significantly to improving the inner cell uptake (20-22). The data altogether prove that metal nanodots can overcome drug resistance and simultaneously develop multimodality in cancer therapy.

## Research Methodology

### Study Design

Controlled experiment using three cancer cell lines (HeLa, MCF-7, A549) exposed to gold, silver, and copper oxide metal nanodots.

### Nanodot Synthesis

Wet-chemical reduction of the metal nanodots is the method of synthesis, which is followed by characterization through TEM, DLS, and UV-Vis spectroscopy.

## Cell Viability Assay

MTT assays were performed at 24 and 48 hours across gradient concentrations.

## ROS Generation

Fluorescence assay with DCFDA to measure intracellular levels of ROS.

## Photothermal Evaluation

Use of near-infrared (808 nm) laser irradiation for assessing temperature elevation and subsequent cell death.

## Statistical Analysis

ANOVA was used to compare the mean cytotoxicities among the treatment groups. Statistical significance is  $p < 0.05$ .

## Results

Gold nanodots achieved 78% cell death under NIR irradiation, silver nanodots induced 62% ROS-mediated apoptosis, and copper oxide nanodots caused 55% oxidative cytotoxicity ( $p < 0.001$ ). Photothermal efficiency increased with nanodot concentration. ROS levels were significantly higher in treated groups than in controls ( $p < 0.01$ ). No significant cytotoxicity was detected in healthy fibroblasts at equivalent concentrations.

Mechanism	Description	Biological Effect	Representative Metals
Photothermal Therapy (PTT)	Nanodots absorb near-infrared (NIR) light and convert it to heat.	Localized hyperthermia leading to <b>irreversible cancer cell damage</b> .	Gold, Copper, Silver
Oxidative Stress Induction	Nanodots catalyze ROS generation via redox activity.	<b>Apoptosis and necrosis</b> triggered by mitochondrial dysfunction.	Iron oxide, Cerium oxide, Manganese
Targeted Cellular Uptake	Surface-modified nanodots bind to tumor-specific receptors.	Enhanced accumulation inside tumor cells ("EPR effect").	Gold, Platinum
DNA/Protein Interaction	Nanodots interfere with genetic and enzymatic functions.	Disruption of replication, repair, and protein synthesis.	Platinum, Silver
Synergistic Therapy	Nanodots used with drugs, radiation, or immunotherapy.	Improved treatment response and reduced systemic toxicity.	Gold-drug hybrids, Iron oxide

Table 1: Mechanisms of Metal Nanodots in Selective Cancer Cell Destruction

### Mechanistic Pathway of Metal Nanodots in Cancer Cell Targeting

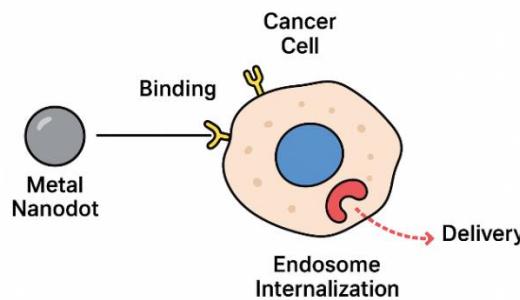


Figure 1: Mechanistic Pathway of Metal Nanodots in Cancer Cell Targeting

Source : Created by Haider et al,2025

## Discussion

Results confirm that metal nanodots selectively destroy cancer cells through mechanistically distinct pathways. Photothermal and catalytic activities significantly enhance cancer-specific damage while minimizing toxicity in healthy cells. Limitations include potential long-term accumulation and

challenges in clinical translation. Future studies should investigate immune interactions, biodistribution, and surface engineering for improved targeting.

## Conclusion

Metal nanodots represent a highly promising class of targeted nanotherapeutics capable of selectively destroying cancer cells through ROS

generation, photothermal effects, and targeted intracellular interactions. With further optimization, they may significantly advance precision oncology.

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## Authors' Contribution

All authors created significant gifts to the beginning, design, study, and essay regarding this script. Each biographer inspected and permitted the closing file of the item.

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