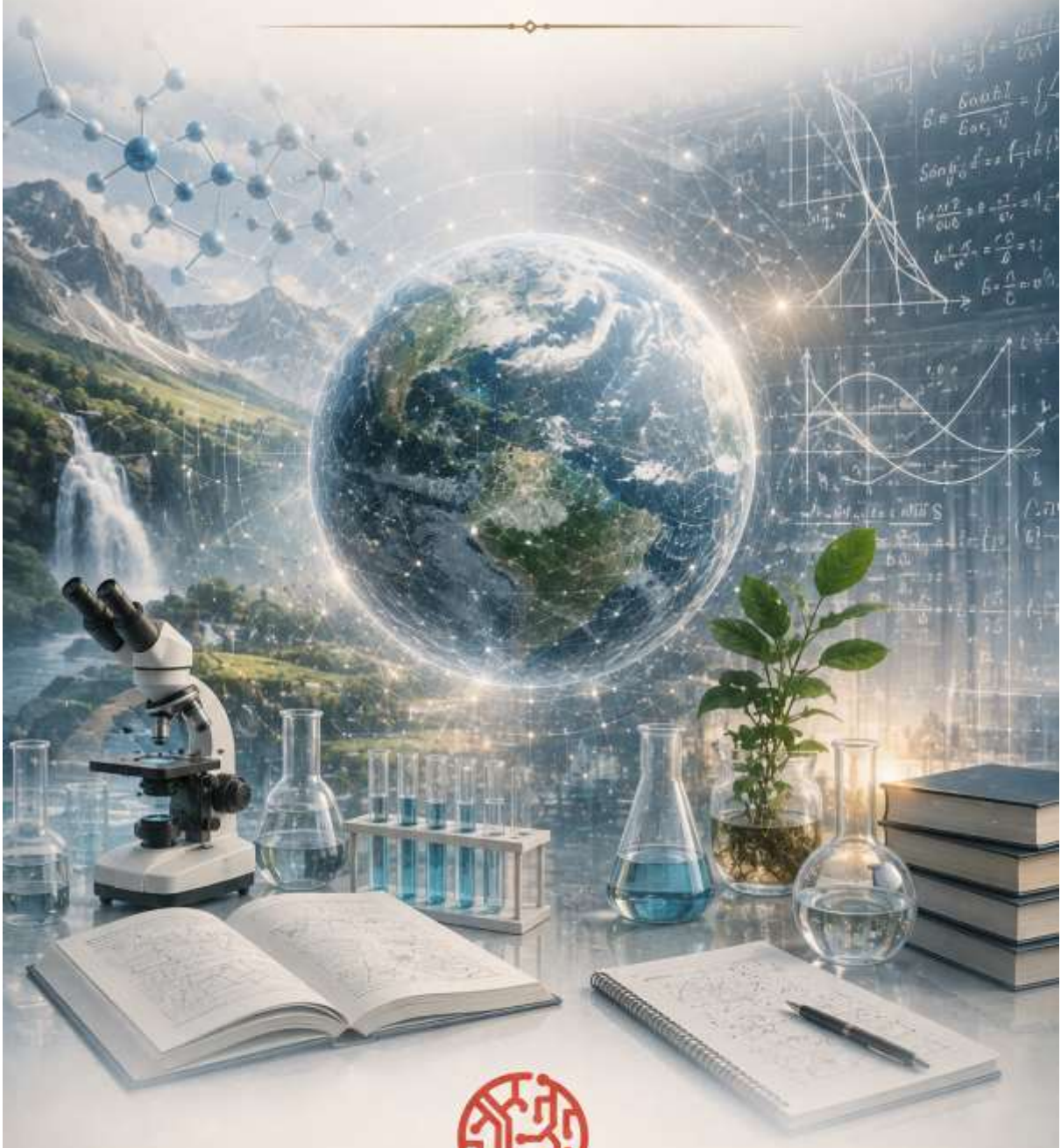
MODERN AND CONTEMPORARY RESEARCH IN NATURAL SCIENCE AND MATHEMATICS



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CONTENT

Drug-DNA Interactions: Binding Mechanisms, Analytical Techniques and Recent Advances

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ABSTRACT

DNA (Deoxyribonucleic acid) is a nucleic acid found in all cellular organisms and some viruses that stores and transmits genetic information and plays a fundamental role in the regulation of biological processes. Investigating the binding of drug molecules to DNA and their interactions with genetic material is of great importance for the development of new therapeutic agents and DNA detection and sensing strategies. In particular, the rapid and reliable evaluation of the interactions between DNA and potential drug candidates for the treatment of diseases such as cancer, diabetes, cardiovascular disorders, and neurodegenerative diseases provides valuable insights about the possible effects of these molecules on genetic material and contributes to the elucidation of their molecular binding mechanisms. This chapter provides an overview of DNA structure, drug-DNA binding mechanisms, analytical techniques employed to investigate these interactions, and recent advances in drug–DNA interaction studies.

Keywords – DNA-drug interactions, DNA, Binding Mechanisms, Analytical Techniques, Recent Advances.

INTRODUCTION

Structure of DNA

DNA and ribonucleic acid (RNA) are two important nucleic acids found in biological systems that are responsible for the transmission and storage of genetic information. DNA is a polymeric molecule composed of two long antiparallel strands formed by repeating units known as nucleotides. It contains the genetic information required for the survival, biological functions, and development of all living organisms and certain viruses. The primary function of DNA is the preservation and transmission of genetic information.

The structural framework of DNA consists of a polymer backbone composed of alternating sugar and phosphate groups. Each sugar molecule is attached to one of four nitrogenous bases, which determine the genetic sequence of the molecule (Swift & Golsteyn,2014). The arrangement of these bases along the two DNA strands encodes biological information. During protein synthesis, this genetic information is interpreted to determine the specific sequence of amino acids required for protein formation. In this process, the information stored within DNA is copied into RNA through a mechanism known as transcription (Goud et al., 2017.)

In cells, DNA is organized within chromosomes. In simple prokaryotic organisms, such as bacteria, DNA is located in the cytoplasmic region, whereas in complex eukaryotic organisms, including animals, plants, and fungi, it is primarily found inside the nucleus (Ng et al., 2016). The double-

helical model of DNA was proposed by Watson and Crick based on the X-ray diffraction studies of Rosalind Franklin and Maurice Wilkins. DNA exists as a double-stranded helix in the cell nucleus, and each strand is composed of four different nucleotides: adenine (A), thymine (T), cytosine (C), and guanine (G) (Kamal et al., 2016).

Drug-DNA Interactions

DNA–drug interactions occur through three basic binding mechanisms:

- **Electrostatic interaction:** Positively charged drug molecules interact electrostatically with the negatively charged phosphate groups of the DNA sugar–phosphate backbone.
- **Groove binding:** The drug molecule binds within the major or minor grooves formed by the double-helical structure of DNA.
- **Intercalation:** The drug molecule inserts itself between adjacent DNA base pairs. This type of interaction is generally observed with planar and aromatic molecules or ions, which can fit between the stacked base pairs of the DNA double helix (Sammriski et al., 2009; Lin et al., 2016).

Drug-DNA Interaction Types

Studies investigating the interactions between active pharmaceutical ingredients and DNA molecules provide valuable insights into molecular binding mechanisms and play a significant role in the development of new drug candidates. Interactions between drug molecules and DNA are generally classified into two main categories: covalent and non-covalent interactions. In covalent binding, the drug molecule forms irreversible covalent bonds with DNA, which may interfere with essential DNA-mediated processes and ultimately lead to cell death.

Cisplatin is one of the best-known platinum-based anticancer agents. Its biological activity arises after aquation, during which the platinum center forms coordination bonds predominantly with the N7 position of guanine residues in DNA. Drug molecules capable of forming covalent bonds with DNA include alkylating agents as well as platinum-based compounds. Alkylating agents exert their effects by transferring alkyl groups to DNA, whereas platinum compounds form coordination bonds with DNA bases and induce DNA cross-links. Alkylating agents primarily target guanine residues in DNA, especially the N7 and O6 positions, leading to DNA damage and disruption of replication and transcription (Altay et al., 2015; Sirajuddin et al., 2013).

Alkylating agents exert their effects through three main mechanisms:

a) They attach alkyl groups to DNA bases, leading to structural alterations of DNA.

b) Alkylating agents selectively bind to DNA and form covalent adducts with DNA bases, leading to DNA damage. These lesions disrupt normal base

pairing, impair DNA replication and transcription, and may destabilize hydrogen bonding between complementary DNA strands.
c) They may cause incorrect pairing of nucleotides, resulting in genetic mutations (Altay et al., 2015).

Through chemical interactions, alkylating agents establish covalent bonds with DNA. Consequently, processes such as DNA replication may be disrupted due to abnormal base pairing, elimination of functional groups, or structural rearrangements, which can ultimately trigger apoptosis.

In contrast, reversible interactions, also known as non-covalent interactions, can change DNA conformation and alter torsional tension, thereby affecting protein–DNA interactions and potentially causing structural deformation within genetic regions. Non-covalent DNA binding interactions can be divided into three major categories:

Binding to the major and minor grooves of DNA:

Molecules that interact with the major or minor grooves of the DNA double helix generally possess a curved or crescent-shaped structure complementary to these regions. These compounds interact with DNA through van der Waals forces and may also form hydrogen bonds with nitrogen atoms of adenine and oxygen atoms of thymine. Imidazole–pyrrole polyamides and synthetic lexitropsins are examples of molecules exhibiting this type of binding behavior (Rajendiran et al., 2012).

Binding through electrostatic interactions:

This type of interaction occurs when positively charged molecules associate electrostatically with the negatively charged sugar–phosphate backbone of nucleic acids. Certain positively charged ruthenium(II) complexes can interact with DNA through electrostatic interactions (Watson& Crick, 1953; Karacan &Okay, 2013). However, these interactions are generally considered to be non-specific.

Intercalation between DNA base pairs:

Intercalation involves the reversible insertion of a molecule or molecular group between adjacent DNA base pairs. This interaction may result in structural distortions within the DNA double helix, impairing DNA replication and RNA synthesis. Molecules with suitable chemical structures and geometries can insert between DNA base pairs and bind reversibly to nucleic acids; these molecules are generally referred to as ligands. DNA-intercalating ligands typically contain multiple aromatic rings and exhibit planar molecular structures, making them widely used as nucleic acid staining agents. Common examples of DNA intercalators reported in the literature include phenanthridine, proflavine, ethidium bromide, and acridine derivatives (Rajendiran et al., 2012).

A cationic intercalator is initially attracted to the polyanionic structure of DNA through electrostatic interactions in an aqueous environment. Transient local dissociation and structural fluctuations of adjacent DNA base pairs create sufficient space for the intercalator to insert

between them. This process results in local structural changes in DNA, such as partial unwinding and elongation of the double helix (Lin et al., 2016).

The incorporation of an intercalator between DNA base pairs is only possible after partial unwinding of the DNA double helix, which creates sufficient space for ligand insertion (Rajendiran et al., 2012; Karacan & Okay, 2013).

Recently developed synthetic intercalators are largely inspired by naturally occurring or clinically used intercalating agents employed in chemotherapy (Mukherjee et al., 2016). Furthermore, synergistic intercalating systems have been designed by incorporating two or more identical intercalating units within a single molecule (e.g., ditercalinium and bisacridine) or combining different functional groups with intercalating properties (e.g., cisplatin-based systems). These modifications aim to increase the affinity of intercalators toward DNA binding sites and prolong their residence time within the intercalation region (Hasanzadeh & Shadjou, 2016).

Interactions between intercalating molecules and biological macromolecules can induce various chemical and structural changes that can be monitored using spectroscopic (UV/Vis, Fluorescence, NMR) and chromatographic (HPLC). These approaches enable the characterization of intercalator functions and provide valuable information regarding DNA–ligand interactions. Currently, organic fluorescent intercalators capable of binding to DNA through insertion between base pairs are widely utilized not only in bioimaging applications but also in the development of intercalator-based therapeutic agents (Top et al., 2016).

RESULTS AND DISCUSSION

Recent Studies on Drug–DNA Interactions

The studies reviewed demonstrate that electrochemical, spectroscopic, and chromatographic methods provide complementary information for the characterization of drug-DNA interactions. While electrochemical methods stand out because of their low detection limits, short analysis times and cost-effectiveness, techniques such as UV–Vis spectroscopy, fluorescence spectroscopy and HPLC play a significant role in validating binding mechanisms. Nanomaterial-based biosensors developed in recent years have enabled the highly sensitive analysis of drug-DNA interactions and have been successfully applied to real samples. The combined use of molecular docking and molecular dynamics simulations with experimental data has made it possible to elucidate binding mechanisms at the atomic level. These developments contribute significantly to the evaluation of new drug candidates and the advancement of biosensor technologies.

Electrochemical, spectroscopic, and chromatographic methods have been employed to investigate and characterize DNA–drug interactions and their underlying binding mechanisms (Zareh et al., 2014; Kara, 2014).

The interaction between calf thymus double-stranded DNA (ctDNA) and daunorubicin (DNR), an anthracycline-based anticancer drug, was investigated using differential pulse voltammetry (DPV). Carbon quantum dot (CQD)-modified disposable pencil graphite electrodes were used. Several experimental parameters were optimized to improve the analytical performance of the proposed sensor. The detection limits for DNR and ctDNA were 0.02 and 0.89 μg/mL, respectively. The interaction mechanism between DNR and ctDNA was evaluated by monitoring the variations in the oxidation responses of both DNR and guanine during different interaction periods. Furthermore, the drug–DNA binding process was investigated and confirmed through electrochemical impedance spectroscopy (EIS) measurements (Eksin et al., 2020).

The electrochemical behavior of bendamustine (BND), an anticancer drug widely used in the treatment of various hematological malignancies, was investigated. For the first time, the interaction between BND and DNA was evaluated in the presence and absence of QRCT, and the protective effect of QRCT on drug–DNA binding was investigated. The LOD and LOQ values of BND were determined as 6.0 and 20.0 μg/mL, respectively, using the calibration equation $I = 0.029 \times C_{BND} + 1.197$ ($R^2 = 0.997$). The results demonstrated that QRCT significantly reduced the interaction between BND and DNA, which was attributed to its strong binding affinity for DNA molecules (Erol et al.,2021).

Anthracyclines represent one of the most extensively investigated classes of anticancer agents used in clinical applications. However, the equilibrium constants (K) associated with the interactions between anthracyclines and DNA, reported in both in vitro and in vivo studies, remain inconsistent in the literature. Reported K values span a broad range from 10^4 to 10^8 M^{-1}, indicating considerable variability and suggesting an approximately 1000-fold uncertainty in the estimation of the drug concentration required for effective DNA complex formation (Zhou et al., 2020).

The interactions between peramivir (PMV) and various DNA molecules—single-stranded DNA (ssDNA), double-stranded DNA (dsDNA), polyguanilic acid (PolyG), and polyadenylic acid (PolyA)—were studied using square-wave voltammetry (SWV), spectrophotometry, plasmid unwinding assays, and density functional theory (DFT). The study found that peramivir significantly affected the unwinding of the DNA double helix. The DFT studies further supported these observations by demonstrating that hydrogen bonding between PMV and nucleobases, together with interactions between PMV and the sugar–phosphate backbone of DNA, plays a significant role in stabilizing the DNA–PMV complex. In addition, plasmid relaxation experiments showed that PMV does not induce considerable DNA

damage, such as single- or double-strand breaks, nor does it exhibit a protective effect against oxidative DNA damage. These results suggest that the biological activity of PMV is primarily associated with its molecular interactions with DNA rather than disruption of DNA structural integrity (Wypych et al., 2025).

The influence of an antioxidant compound on the interaction between DNA and the anticancer drug daunorubicin (DNR) was investigated. DNR is an anthracycline-based chemotherapeutic agent widely used in the treatment of several malignancies, including leukemia. The use of antioxidant-containing supplements during chemotherapy may affect the therapeutic response of cancer patients, making the treatment process more complex. The interaction was evaluated by monitoring the oxidation responses of guanine (1.0 V) and DNR (0.5 V) within the same potential range, allowing the effects of caffeic acid on the DNA–drug interaction to be determined (Muti & Muti,2018).

For the electrochemical determination of 6-thioguanine, disposable graphite pencil electrodes were employed as a low-cost sensing platform. Differential pulse voltammetry (DPV), cyclic voltammetry (CV), and electrochemical impedance spectroscopy (EIS) were used. The interaction occurring at the electrode surface was assessed by monitoring changes in the oxidation responses of both 6-thioguanine and adenine. Under the optimized conditions, the sensor exhibited a linear response over the concentration range of 1–4 μM for 6-thioguanine, with a detection limit of 0.24 μM The adenine oxidation signal, which decreased after hybridization, decreased further following interaction with 6-thioguanine and reached its lowest value after a 10-minute incubation. These findings indicated the interaction between 6-thioguanine and DNA (Unal et al., 2018).

Phenylalanine-functionalized copper nanoclusters (Phe/CuNCs) were introduced for the first time as a fluorescent sensing platform to investigate the interaction between abiraterone acetate (ATA) and DNA. The synthesized Phe/CuNCs were comprehensively characterized by transmission electron microscopy (TEM), dynamic light scattering (DLS), X-ray photoelectron spectroscopy (XPS), UV–Vis spectroscopy, and fluorescence spectroscopy. The synthesis conditions were optimized to achieve optimal fluorescence performance. The developed fluorescent probe was successfully applied to evaluate the ATA–DNA interaction, yielding a binding constant (K_a) of 1.03×10^4 (Bilkay & Kara Satana, 2024).

The interaction between levetiracetam (LEV) and double-stranded fish sperm DNA (dsDNA) was investigated using spectroscopic methods, viscosity analysis, and molecular docking simulations to clarify the underlying binding mechanism. It was observed that a LEV–dsDNA complex formed when LEV bound to DNA through groove binding. In addition, an electrochemical biosensor for the LEV-DNA interaction was developed using differential pulse voltammetry (DPV) method. The sensing

strategy was based on monitoring the decrease in the oxidation current of deoxyguanosine (dGuo) after its interaction with LEV in acetate buffer (pH 4.80). The concentration range of LEV was 2.5–20 μM. The LOD and LOQ values were 0.70 and 2.31 μM, respectively (Al Faysal et al., 2025).

A chromatographic method was developed and validated to determine mirtazapine (MIR) in pharmaceutical formulations. The chromatographic conditions, including the mobile phase composition, pH, column type, flow rate, and column temperature, were systematically optimized. The interaction between MIR and dsDNA was investigated using UV–Vis spectroscopy, viscosity analysis and HPLC. The concentration range of MIR was 0.5–15 ppm (R^2 = 0.9998). The LOD and LOQ values were 0.013 and 0.044 ppm, respectively. The recovery values were between 98.82% and 100.97%. The binding behavior was characterized by DNA thermal denaturation experiments. Molecular docking and molecular dynamics simulations were performed to clarify the molecular basis of the interaction. Computational analyses demonstrated that MIR preferentially binds within the minor groove of dsDNA, where the complex is stabilized through hydrogen bonding as well as electrostatic interactions between the aromatic ring of MIR and the phosphate oxygen atoms of the DNA backbone. The developed HPLC method was applied to commercial MIR tablet formulations, confirming its suitability for investigating MIR–DNA interactions (Kuzpınar et al., 2024).

The interaction between 6-mercaptopurine (6MP) and dsDNA was analyzed. Two biosensors, (GSPE/dsDNA) and (GSPE/Pt-Cys-dsDNA) were proposed. The concentration ranges of the GSPE/dsDNA and GSPE/Pt-Cys-dsDNA were found to be 200-1000 nM and 2-75 nM, respectively. Selectivity experiments demonstrated that the proposed biosensors could effectively distinguish 6MP from common biological interferents as well as other drugs that may be administered concurrently. The results obtained with the platinum nanoparticle-based biosensor showed excellent agreement with those generated by the reference HPLC method, with deviations of less than ±7%, confirming the accuracy and reliability of the proposed approach. These findings demonstrate that the selective interaction between a specific dsDNA sequence and 6MP can be successfully utilized to construct a sensitive and robust electrochemical biosensor for the determination of the drug at nanomolar concentrations (Sołdatowska et al., 2026).

An electrochemical DNA biosensor based on Pt^{2+}-doped $ZnCo_2O_4$ was developed to quantify sunitinib (STB) in acetate buffer. The electrochemical characteristics of the nanocomposite were investigated using cyclic voltammetry (CV) and electrochemical impedance spectroscopy (EIS), revealing that Pt^{2+} doping markedly enhanced the electrical conductivity and accelerated the electron-transfer kinetics of $ZnCo_2O_4$. The resulting biosensor promoted efficient interaction between STB and immobilized dsDNA, while changes in the guanine oxidation peak were

utilized as the analytical signal for drug determination. The concentration range was between 0.01 and 80.0 μM. The LOD was 0.007 μM. The proposed method was applied to determine STB in pharmaceutical preparations and urine samples. The recovery values were between 97% and 103%. In addition, spectrophotometric analyses together with molecular docking simulations confirmed a strong interaction between STB and DNA and indicated that the drug predominantly binds within the DNA groove (Pour et al., 2026).

The interaction between adriamycin (AD) and DNA was investigated by differential pulse voltammetry (DPV). The binding mechanism of AD was evaluated using modified glassy carbon fiber electrodes. The decrease in the guanine oxidation peak current at +0.9 V was used to monitor the drug-DNA interaction mechanism in acetate buffer (pH 4.5). This interaction at the charged electrode surface resembles the in vivo process, where DNA interacts with charged phospholipid membranes and proteins. As a result, the biosensor facilitates a better understanding of the in vivo mechanism of action of this anticancer drug. The results demonstrated that AD intercalated into double-stranded DNA, underwent both oxidative and reductive reactions, and specifically interacted with guanine residues, leading to the formation of the mutagenic 8-oxo-G lesion (Tiwari & Pitre,2008).

The interaction between the antipsychotic drug aripiprazole (ARP) and calf thymus double-stranded DNA (ct-dsDNA) was investigated using differential pulse voltammetry (DPV) and UV–Vis spectroscopy. A glassy carbon electrode (GCE) modified with ct-dsDNA was employed to evaluate the ARP–DNA interaction by monitoring changes in the oxidation signals of ARP and DNA bases. These changes in the DNA base signals were further confirmed through solution-phase interaction studies. A binding constant (K) of approximately 3×10^5 M^{-1} was determined spectrophotometrically in 0.1 M acetate buffer at pH 4.7. A good correlation was observed between the electrochemical and UV–Vis spectrophotometric data in terms of the ARP signal decrease. The formation of the ARP-DNA complex was further confirmed by differential pulse (DP) voltammetric and spectrophotometric analyses of the ARP–polyguanine (polyG) and ARP–polyadenine (polyA) systems in solution. The interaction of ARP with damaged ct-dsDNA was analyzed using DPV (Kurbanoglu et al., 2015).

A new pyridine derivative, 1-(2,6 dichlorobenzyl)-4-(2-(2-4-hydroxybenzylidene)-hydrazinyl)-pyridinium chloride (DHPC), was synthesized as a candidate drug molecule. The interaction between DHPC and DNA was investigated using differential pulse voltammetry (DPV), cyclic voltammetry (CV), and electrochemical impedance spectroscopy (EIS) to evaluate its effect on DNA. The study revealed that the oxidation signal of guanine bases in DNA was significantly reduced, while the oxidation signal of DHPC increased upon interaction. The LOD and LOQ

values were 1.5 and 4.9 μg/mL, respectively. The toxicity value (S%) for DHPC was determined to be 31%, indicating its toxic effect on DNA (Karasakal et al., 2021).

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Geometric Algebraic Structure of Multiplicative Dual Numbers

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ABSTRACT

This study introduces multiplicative dual numbers, a novel algebraic structure that synthesizes the nilpotent properties of dual numbers with the scale-invariant framework of geometric (multiplicative) calculus. Unlike classical dual numbers defined by the additive nilpotent unit ε ($\varepsilon^2 = 0$), our system is constructed upon a multiplicative dual unit ε_* satisfying $\varepsilon_*^{2_*} = 1$ where 1 is the zero element of the geometric real field $\mathbb{R}_*$. We demonstrate that this set forms a commutative ring with unity and constitutes a two-dimensional vector space over $\mathbb{R}_*$. Furthermore, we establish a consistent matrix representation and define a multiplicative inner product, norm, and absolute value. This new framework offers a distinct geometric interpretation compared to its classical counterpart and opens potential avenues for applications in scale-invariant kinematics and differential geometry.

Keywords: Multiplicative dual numbers, multiplicative inner product, multiplicative norm, multiplicative semi-inner product. multiplicative semi-norm, multiplicative dual unit sphere.

INTRODUCTION

Since its foundations were laid by Isaac Newton and Gottfried Leibniz, mathematical analysis has been a field of continuous reinterpretation and extension to model dynamic systems. Two significant and independent branches of this evolution form the theoretical basis of this study. On one hand, there are the dual numbers, introduced by W.K. Clifford in 1871, which are defined by a nilpotent unit ε satisfying $\varepsilon^2 = 0$ (while $\varepsilon \neq 0$) (Clifford 1871:381). This additive algebraic structure has become an indispensable tool in fields such as automatic differentiation, instantaneous kinematics, and robotics, owing to its ability to directly encode the derivative of a function through the Taylor series expansion $f(x + \varepsilon h) = f(x) + hf'(x)\varepsilon$ (Pennestrì and Stefanelli 2007:310, Veldkamp 1976:141). The power of dual numbers stems from this inherently additive nilpotent property.

On the other hand, there is multiplicative (or geometric) calculus, also known as "Non-Newtonian Calculus," developed by Grossman in the late 1960s (Grossman 1979:525). This framework replaces standard addition and subtraction with multiplication and division, respectively, offering a scale-invariant analytical lens for phenomena characterized by exponential growth or decay, such as in finance, biology, and image processing (Bashirov, Kurpınar and Özyapıcı 2008:36). This system, which is isomorphic to standard calculus via a logarithmic transformation, has enabled the

development of its own distinct differential and integral geometry (Aslan, Bekar and Yayli 2023: 2350151, Georgiev 2022).

While these two powerful theoretical frameworks have developed on parallel tracks, a gap exists in the literature for an algebraic structure that systematically merges them. This raises a central question: How can the capacity for infinitesimal analysis, characteristic of dual numbers, be synthesized with the scale-invariant perspective of geometric calculus? This inquiry not only inspires theoretical curiosity but also opens new avenues for potential applications where both relative scale and instantaneous motion are of primary importance.

This paper addresses this precise gap by introducing a novel number system we term multiplicative dual numbers. The cornerstone of our approach is the translation of the nilpotent unit's definition from an additive to a multiplicative framework. Whereas the classical dual unit ε is additively nilpotent ($\varepsilon^2 = 0$), we define the multiplicative dual unit ε_* to be nilpotent with respect to multiplicative multiplication ($\cdot_*$).

This leads to the relation $\varepsilon_* \cdot_* \varepsilon_* = 0_*$, where $0_* = 1$ is the zero element of the multiplicative field ($\mathbb{R}_*$) of positive real numbers. Consequently, the seemingly paradoxical identity $\varepsilon_*^{2_*} = 1$ emerges as a natural and consistent consequence of translating the concept of nilpotency from an additive to a multiplicative domain.

In this study, we systematically construct and investigate the algebraic and geometric properties of this new number system. First, we prove that the set of Multiplicative Dual Numbers forms a commutative ring with unity but not a field. We then demonstrate that this structure constitutes a two-dimensional vector space over the field $\mathbb{R}_*$ and present an isomorphic matrix representation. Finally, we develop an inner product, norm, and absolute value compatible with this multiplicative structure, thereby establishing a consistent foundation for geometric analysis. This work introduces an original mathematical framework poised to unlock potential applications in fields such as scale-invariant kinematics or the differential geometry of exponential transformations.

1. PRELIMINARIES

In this section, we outline the fundamental algebraic and analytical structures of geometric (or multiplicative) calculus that form the basis of our work. These structures are derived by reinterpreting the operations of standard real analysis through a logarithmic map, a concept that provides the intuition behind all subsequent algebraic definitions.

The set of geometric real numbers is defined as the set of positive real numbers $\mathbb{R}_* = (0, \infty)$. On this set, the standard operations of addition and multiplication are redefined as follows (Bashirov, Kurpınar and Özyapıcı 2008:36, Grossman 1979:525). For any $x,\ y \in \mathbb{R}_*$

Geometric Addition: Corresponds to the standard sum $ln\,(x) + ln\,(y)$mapped back by the exponential function.

$$x +_* y = \exp(ln\,(x) + ln\,(y)) = xy\,.$$

Geometric Multiplication: Corresponds to the standard product $ln\,(x) * ln\,(y)$ mapped back by the exponential function.

$$x \cdot_* y = \exp(ln\,(x) \cdot_* ln\,(x)) = x^{ln\,(y)}\,.$$

Analogously, geometric subtraction ($x -_* y = x/y$) and division ($x /_* y = \exp\big(\ln(x)/\ln(y)\big)$) are also defined. This framework includes geometric counterparts to the 0 and 1 elements of standard real numbers:

Geometric Zero (Multiplicative Zero) $0_* = \exp(0) = 1$.

Geometric Identity (Multiplicative Identity) $1_* = \exp(1) = e$.

Under these operations, the triple $(\mathbb{R}_*,\ +_*,\ \cdot_*)$ forms a complete field that is isomorphic to the standard field of real numbers $(\mathbb{R},\ +,\ \cdot)$ (Georgiev, Zennir and Boukarou 2022).

The field $\mathbb{R}_*$ serves as the field of scalars for the $n-$ dimensional geometric vector space $\mathbb{R}_*^n$. The set $\mathbb{R}_*^n$ consists of $n-$tuples whose components are in $\mathbb{R}_*$, $\mathbb{R}_*^n = \{v = (v_1,\ v_2,\ \dots,\ v_n) | v_i \in \mathbb{R}_*\}$. The primary operations in this space are defined as follows for any vectors $u,\ v \in \mathbb{R}_*^n$ and any scalar $c \in \mathbb{R}_*$

Vector Addition:

$$\begin{aligned} u +_* v &= (u_1 +_* v_1, u_2 +_* v_2, \dots, u_n +_* v_n) \\ &= (u_1 v_1, u_2 v_2, \dots, u_n v_n). \end{aligned}$$

Scalar Multiplication:

$$\begin{aligned} c \cdot_* v &= (c \cdot_* v_1, c \cdot_* v_2, \dots, c \cdot_* v_n) \\ &= \Big(\exp\big(ln(c) ln(v_1)\big), \exp\big(ln(c) ln(v_2)\big), \dots, \exp\big(ln(c) ln(v_n)\big)\Big) \end{aligned}\quad .$$

Equipped with these operations, $\mathbb{R}_*^n$ is a vector space over the field $\mathbb{R}_*$.

The space $\mathbb{R}_*^n$ can be endowed with an inner product that serves as the geometric analogue of the standard Euclidean inner product. For any $u, v \in \mathbb{R}_*^n$:

Geometric Inner Product

$$\langle u, v\rangle_* \quad = u_1 \cdot_* v_1 +_* u_2 \cdot_* v_2 +_* \ldots +_* u_n \cdot_* v_n.$$

Using the logarithmic map, this can be expressed more explicitly as

$$\langle u, v\rangle_* = \exp\left(\sum_i \left(ln(u_i) \cdot_* ln(v_i)\right)\right)$$

Geometric Norm: The inner product naturally induces a norm on the space

$$\|v\|_* = sqrt_*(\langle v, v\rangle_*) = \exp\left(sqrt\left(ln(\langle v, v\rangle_*)\right)\right).$$

which is equivalent to

$$\|v\|_* = \exp\left(sqrt\left(\sum_i ln\,(v_i)^2\right)\right)$$

Within this framework, orthogonality of two vectors is defined by the condition $\langle u, v\rangle_* = 0_* = 1$, and a vector is a unit vector if $\|v\|_* = 1_* = e$ (Aslan, Bekar and Yayli 2023: 2350151, Georgiev 2022).

To complete the analytical framework, the multiplicative derivative of a function $f: \mathbb{R} \to \mathbb{R}_*$ measures its instantaneous relative rate of change and is defined as follows (Aydin, Has and Yilmaz 2023):

$$f^*(x) = \exp\left(xf'(x)/f(x)\right)$$

where $f'(x)$ is the standard Newton-Leibniz derivative of f. This definition will be utilized in subsequent sections to analyze functions of multiplicative dual variables.

2. MULTIPLICATIVE DUAL NUMBERS

This section we will introduce the multiplicative dual number system and give some of its basic properties.

Definition 2.1 The set of multiplicative dual numbers is defined as follows

$$\mathbb{D}_* = \left\{\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}:\ \zeta, \mathring{\zeta} \in \mathbb{R}_*,\ \varepsilon_* \neq 0_* = 1, \qquad \varepsilon_*^{2_*} = 0_* = 1\right\}$$

where $\varepsilon_* = e^{ln\varepsilon}$, ε is unit dual number.

Definition 3.2 The addition and multiplication of multiplicative dual numbers are defined as

Follows

$$+_* : \mathbb{D}_* \times_* \mathbb{D}_* \to \mathbb{D}_*$$

$$\left(\tilde{\zeta}, \tilde{\gamma}\right) \to \tilde{\zeta} +_* \tilde{\gamma} = \left(\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right) +_* \left(\gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}\right)$$

$$= (\zeta +_* \gamma) +_* \varepsilon_* \cdot_* \left(\mathring{\zeta} +_* \mathring{\gamma}\right)$$

$$= e^{ln\zeta + ln\gamma} +_* \varepsilon_* \cdot_* e^{ln\mathring{\zeta} + \mathring{\gamma}}$$

and

$$\cdot_* : \mathbb{D}_* \times_* \mathbb{D}_* \to \mathbb{D}_*$$

$$\left(\tilde{\zeta}, \tilde{\gamma}\right) \to \tilde{\zeta} \cdot_* \tilde{\gamma} = \left(\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right) \cdot_* \left(\gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}\right)$$

$$= (\zeta \cdot_* \gamma) +_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\gamma} +_* \mathring{\zeta} \cdot_* \gamma\right)$$

$$= e^{ln\zeta\, ln\gamma} +_* \varepsilon_* \cdot_* e^{ln\zeta\mathring{\gamma} + ln\mathring{\zeta} ln\gamma}$$

Definition 2.3 Let $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta},\ \tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma} \in \mathbb{D}_*$, we have

$$\tilde{\zeta} = \tilde{\gamma} \text{ if and only if } \zeta = \gamma,\ \mathring{\zeta} = \mathring{\gamma}.$$

The multiplicative additive identity element is

$$0_* = e^{ln1} +_* \varepsilon_* \cdot_* e^{ln1}.$$

The additive inverse of $\tilde{\zeta}$ is

$$-_* \tilde{\zeta} = -_* \zeta -_* \varepsilon_* \cdot_* \mathring{\zeta}\ .$$

The multiplicative identity element with respect to the operation $\cdot_*$ is

$$e +_* 0_* \cdot_* \varepsilon_*.$$

It is obvious that the multiplicative real number

$$e = e^{lne + ln1 ln\varepsilon} = e +_* 1,$$

is the multiplicative unit element in $\mathbb{D}_*$. The multiplicative number

$$\varepsilon_* = e^{ln\varepsilon} = e^{ln1 + lnelne} = 1 +_* \varepsilon_*,$$

is also referred to as a multiplicative dual unit in $\mathbb{D}_*$. Furthermore, we compute

$$\begin{aligned}\varepsilon_*^{2_*} &= \varepsilon_* \cdot_* \varepsilon_* \\ &= (e^{ln1+lnelne\varepsilon}) \cdot_* (e^{ln1+lnelne\varepsilon}) \\ &= e^{ln1ln1+ln\varepsilon(ln1lne+lneln1)}, \\ &= e^{ln1+ln\varepsilon ln1} \\ &= e^{ln1} \\ &= 1.\end{aligned}$$

Let $\varepsilon_* \neq 0_* = 1$, we have

$$0_* \cdot_* \epsilon_* = \epsilon_* \cdot_* 0_* = 0_*,$$

$$1_* \cdot_* \epsilon_* = \epsilon_* \cdot_* 1_* = \epsilon_*,$$

$$\epsilon_*^{2_*} = \epsilon_*^{3_*} = \cdots = \epsilon_*^{n_*} = 0_*.$$

Theorem 2.4 For any multiplicative dual number $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \in \mathbb{D}_*$, this can be represented as

$$\tilde{\zeta} = (\zeta, \mathring{\zeta})$$

where

$$e = (e, 1),$$

$$\varepsilon_* = (1, e).$$

Proof. For any $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$, we have

$$\begin{aligned}\tilde{\zeta} &= \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \\ &= \zeta \cdot_* (e, 0_*) +_* (0_*, e) \cdot_* \mathring{\zeta} \\ &= (\zeta, 0_*) + (0_*, \mathring{\zeta}) \\ &= (\zeta, \mathring{\zeta}).\end{aligned}$$

Definition 2.5 The multiplicative dual number $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$, ζ is called multiplicative real part of $\tilde{\zeta}$ and $\mathring{\zeta}$ is called multiplicative dual part of $\tilde{\zeta}$, denoted by $Re_*(\tilde{\zeta})$ and $Du_*(\tilde{\zeta})$ respectively. Accordingly, a multiplicative dual number $\tilde{\zeta}$ can be written as

$$\tilde{\zeta} = Re_*(\tilde{\zeta}) +_* \varepsilon_* \cdot_* Du_*(\tilde{\zeta}).$$

ε_* is dual number unit. The multiplicative real part of $\tilde{\zeta}$ is

Theorem 2.6 For any $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}, \tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}$ and $\tilde{\xi} = \xi +_* \varepsilon_* \cdot_* \xi \in \mathbb{D}_*$ then we have the following

i. $\tilde{\zeta} +_* \tilde{\gamma} = \tilde{\gamma} +_* \tilde{\zeta},$

ii. $(\tilde{\zeta}+_*\tilde{\gamma})+_*\tilde{\xi} = \tilde{\zeta}+_*(\tilde{\gamma}+_*\tilde{\xi})$,

iii. $\tilde{\zeta} \cdot_* \tilde{\gamma} = \tilde{\gamma} \cdot_* \tilde{\zeta}$,

iv. $(\tilde{\zeta} \cdot_* \tilde{\gamma}) \cdot_* \tilde{\xi} = \tilde{\zeta} \cdot_* (\tilde{\gamma} \cdot_* \tilde{\xi})$,

v. $(\tilde{\zeta}+_*\tilde{\gamma}) \cdot_* \tilde{\xi} = \tilde{\zeta} \cdot_* \tilde{\xi}+_*\tilde{\zeta} \cdot_* \tilde{\xi}$,

vi. $\tilde{\zeta} \cdot_* (\tilde{\gamma}+_*\tilde{\xi}) = \tilde{\zeta} \cdot_* \tilde{\gamma}+_*\tilde{\zeta} \cdot_* \tilde{\xi}$.

Proof.

i.

$$\begin{aligned}
\tilde{\zeta}+_*\tilde{\gamma} &= (\zeta+_*\varepsilon_* \cdot_* \mathring{\zeta})+_*(\gamma+_*\varepsilon_* \cdot_* \mathring{\gamma}) \\
&= e^{ln\zeta+ln\gamma}+_*\varepsilon_* \cdot_* e^{ln\mathring{\zeta}+ln\mathring{\gamma}} \\
&= e^{ln\gamma+ln\zeta}+_*\varepsilon_* \cdot_* e^{ln\mathring{\gamma}+ln\mathring{\zeta}} \\
&= \tilde{\gamma}+_*\tilde{\zeta}.
\end{aligned}$$

ii.

$$\begin{aligned}
(\tilde{\zeta}+_*\tilde{\gamma})+_*\tilde{\xi} &= \left[(\zeta+_*\varepsilon_* \cdot_* \mathring{\zeta})+_*(\gamma+_*\varepsilon_* \cdot_* \mathring{\gamma})\right]+_* (\xi+_*\varepsilon_* \cdot_* \xi) \\
&= e^{ln\zeta+ln\gamma}+_*\varepsilon_* \cdot_* e^{ln\mathring{\zeta}+ln\mathring{\gamma}} \\
&= \left(e^{ln\zeta}+_*\varepsilon_* \cdot_* e^{ln\mathring{\zeta}}\right)+_*\left[e^{ln\gamma+ln\xi}+_*\varepsilon_* \cdot_* e^{ln\mathring{\gamma}+ln\xi}\right] \\
&= \tilde{\zeta}+_*(\tilde{\gamma}+_*\tilde{\xi}).
\end{aligned}$$

iii.

$$\begin{aligned}
\tilde{\zeta} \cdot_* \tilde{\gamma} &= e^{ln\gamma ln\zeta}+_*\varepsilon_* \cdot_* e^{ln\mathring{\gamma} ln\zeta+ln\gamma ln\mathring{\zeta}} \\
&= e^{ln\zeta ln\gamma}+_*\varepsilon_* \cdot_* e^{ln\mathring{\zeta} ln\gamma+ln\zeta ln\mathring{\gamma}} \\
&= \tilde{\gamma} \cdot_* \tilde{\zeta}.
\end{aligned}$$

iv.

$$\begin{aligned}
(\tilde{\zeta} \cdot_* \tilde{\gamma}) \cdot_* \tilde{\xi} &= \left[e^{ln\gamma ln\zeta}+_*\varepsilon_* \cdot_* e^{ln\mathring{\gamma} ln\zeta+ln\gamma ln\mathring{\zeta}}\right] \cdot_* (\xi+_*\varepsilon_* \cdot_* \xi) \\
&= e^{ln\zeta ln\gamma ln\xi}+_*\varepsilon_* \cdot_* e^{ln\zeta ln\gamma ln\xi+ln\zeta ln\mathring{\gamma} ln\xi+ln\mathring{\zeta} ln\gamma ln\xi}.
\end{aligned}$$

Conversely, we have

$$\begin{aligned}
\tilde{\zeta} \cdot_* (\tilde{\gamma} \cdot_* \tilde{\xi}) &= \left(e^{ln\zeta}+_*\varepsilon_* \cdot_* e^{ln\mathring{\zeta}}\right) \cdot_* \left[e^{ln\gamma ln\xi}+_*\varepsilon_* \cdot_* e^{ln\gamma ln\xi+ln\mathring{\gamma} ln\xi}\right] \\
&= e^{ln\zeta ln\gamma ln\xi}+_*\varepsilon_* \cdot_* e^{ln\zeta ln\gamma ln\xi+ln\zeta ln\mathring{\gamma} ln\xi+ln\mathring{\zeta} ln\gamma ln\xi}.
\end{aligned}$$

Hence, $iv.$ holds.

v.

$$\begin{aligned}
(\tilde{\zeta}+_*\tilde{\gamma})\cdot_*\tilde{\xi} &= \left[e^{ln\zeta+ln\gamma}+_*\varepsilon_*\cdot_* e^{ln\mathring{\zeta}+ln\mathring{\gamma}}\right]\cdot_*\left(e^{ln\xi}+_*\varepsilon_*\cdot_* e^{ln\xi}\right)\\
&= e^{ln\zeta+ln\gamma}\cdot_* e^{ln\xi}+_*\varepsilon_*\cdot_* e^{ln\mathring{\zeta}+ln\mathring{\gamma}}\cdot_* e^{ln\xi}+_*\varepsilon_*\cdot_* e^{ln\xi}\cdot_* e^{ln\zeta+ln\gamma}\\
&= e^{ln\zeta ln\xi+ln\gamma ln\xi}+_*\varepsilon_*\cdot_* e^{ln\mathring{\zeta}ln\xi+ln\mathring{\gamma}ln\xi}+_*\varepsilon_*\cdot_* e^{ln\zeta ln\xi+ln\gamma ln\xi}\\
&= e^{ln\zeta ln\xi}+_*e^{ln\gamma ln\xi}+_*\varepsilon_*\cdot_*\left(e^{ln\mathring{\zeta}ln\xi}+_*e^{ln\zeta ln\xi}+_*e^{ln\mathring{\gamma}ln\xi}+_*e^{ln\gamma ln\xi}\right)\\
&= \left(e^{ln\zeta}+_*\varepsilon_*\cdot_* e^{ln\mathring{\zeta}}\right)\cdot_*\left(e^{ln\xi}+_*\varepsilon_*\cdot_* e^{ln\xi}\right)+_*\left(e^{ln\gamma}+_*\varepsilon_*\cdot_* e^{ln\mathring{\gamma}}\right)\cdot_*\left(e^{ln\xi}+_*\varepsilon_*\cdot_* e^{ln\xi}\right)\\
&= \tilde{\zeta}\cdot_*\tilde{\xi}+_*\cdot_*\tilde{\xi}.
\end{aligned}$$

Similarly, we can provide the conclusion $iv.$

Example 2.7 (Addition and Multiplication) Let $\tilde{\zeta}_1 = 2+_*\varepsilon_*\cdot_* 3$ and $\tilde{\zeta}_2 = 4+_*\varepsilon_*\cdot_* 5$ be two multiplicative dual numbers. Then, the multiplicative addition and multiplication of the numbers are given below

$$\begin{aligned}
\tilde{\zeta}_1 +_*\tilde{\zeta}_2 &= e^{ln2+ln4}+_*\varepsilon_*\cdot_* e^{ln3+ln5}\\
&= e^{3ln2}+_*\varepsilon_*\cdot_* e^{ln15}\\
\tilde{\zeta}_1 \cdot_*\tilde{\zeta}_2 &= e^{ln2ln4}+_*\varepsilon_*\cdot_* e^{ln2ln5+ln3ln4}\\
&= e^{2(ln2)^2}+_*\varepsilon_*\cdot_* e^{ln2ln5+2ln3ln2}.
\end{aligned}$$

Definition 2.8 Let $\tilde{\zeta} = \zeta+_*\varepsilon_*\cdot_*\mathring{\zeta}$ and $\tilde{\gamma} = \gamma+_*\varepsilon_*\cdot_*\mathring{\gamma}$, any two multiplicative dual numbers, with $\gamma \neq 0_*$, we can define the division of multiplicative dual numbers as the division operation is defined as

$$\frac{\tilde{\zeta}}{\tilde{\gamma}}* = \frac{\zeta+_*\varepsilon_*\cdot_*\mathring{\zeta}}{\gamma+_*\varepsilon_*\cdot_*\mathring{\gamma}}* = \left(\frac{\zeta}{\gamma}*\right)+_*\varepsilon_*\cdot_*\left(\frac{\gamma\cdot_*\mathring{\zeta}-_*\zeta\cdot_*\mathring{\gamma}}{\gamma^2}*\right).$$

Theorem 2.9 The triple $(\mathbb{D}_*, +_*, \cdot_*)$ is not a field.

Proof. Although $(\mathbb{D}_*, +_*, \cdot_*)$ is a commutative ring with identity, it is not a field because, in order for it to be a field, the set $\mathbb{D}_* \setminus \{1\}$ must form an abelian group under multiplicative $\cdot_*$. However, for any multiplicative element $\tilde{\zeta} = \zeta+_*\varepsilon_*\cdot_*\mathring{\zeta} \in \mathbb{D}_*$, there does not exist an multiplicative inverse element $\tilde{\zeta} = e^{lnx}+_*\varepsilon_*\cdot_* e^{lny} \in \mathbb{D}_*$ such that

$$\tilde{\zeta}\cdot_*\tilde{\zeta}^{-1_*} = \tilde{\zeta}^{-1_*}\cdot_*\tilde{\zeta} = e.$$

From the multiplicative multiplication operation, we obtain the following identity

$$e^{ln\zeta lnx}+_*\varepsilon_*\cdot_* e^{lnalny+ln\mathring{\zeta}lnx}=e. \tag{1}$$

Solving the equation *(*1*),* we have

$$e^{lnx}=\frac{e}{\zeta}*\ \text{and}\ e^{lny}=-_*\frac{\mathring{\zeta}}{\zeta^2}*.$$

Multiplicative inverse element

$$\tilde{\zeta}^{-1_*}=\frac{e}{\zeta}*-_*\varepsilon_*\cdot_*\frac{\mathring{\zeta}}{(\zeta)^2}*.$$

Theorem 2.10 The subspace $\widetilde{\mathbb{D}_*}=\{\tilde{\zeta}=\zeta+_*0_*\cdot_*\varepsilon_*:\zeta\in\mathbb{R}_*\}\subset\mathbb{D}_*$ is isomorphic to the space $\mathbb{R}_*$.

Proof. Define the multiplicative mapping

$$\begin{array}{rcl} f_* & : & \widetilde{\mathbb{D}}_*\to\mathbb{R}_* \\ \tilde{\zeta} & \to & f_*(\tilde{\zeta})=f_*(\zeta+_*0_*\cdot_*\varepsilon_*)=\zeta. \end{array}$$

If $\tilde{\zeta}=\zeta+_*0_*\cdot_*\varepsilon_*$ and $\tilde{\gamma}=\gamma+_*0_*\cdot_*\varepsilon_*$, then we have

$$\begin{array}{rcl} f_*(\tilde{\zeta}+_*\tilde{\gamma}) & = & f_*\big((\zeta+_*0_*\cdot_*\varepsilon_*)+_*(\gamma+_*0_*\cdot_*\varepsilon_*)\big) \\ & = & \zeta+_*\gamma \\ & = & f_*(\zeta+_*0_*\cdot_*\varepsilon_*)+_*f_*(\gamma+_*0_*\cdot_*\varepsilon_*) \\ & = & f_*(\tilde{\zeta})+_*f_*(\tilde{\gamma}). \end{array}$$

In addition, for multiplicative scalar multiplication we get

$$\begin{array}{rcl} f_*(d\cdot_*\tilde{\zeta}) & = & f_*(d\cdot_*\zeta+_*d\cdot_*0_*\cdot_*\varepsilon_*) \\ & = & d\cdot_*\zeta=d\cdot_*f_*(\zeta+_*0_*\cdot_*\varepsilon_*) \\ & = & d\cdot_*f_*(\tilde{\zeta}). \end{array}$$

Thus, f_* preserves both multiplicative addition and multiplicative scalar multiplication. Suppose $\zeta+_*0_*\cdot_*\varepsilon_*\neq\gamma+_*0_*\cdot_*\varepsilon_*$, i.e., $\zeta\neq\gamma$. Then

$$f_*(\zeta+_*0_*\cdot_*\varepsilon_*)\neq f_*(\gamma+_*0_*\cdot_*\varepsilon_*).$$

Hence, f_* is injective. For any $\zeta\in\mathbb{R}_*$, there exists a unique multiplicative dual number $\zeta+_*0_*\cdot_*\varepsilon_*\in\mathbb{D}_*$, such that

$$f_*(\zeta +_* 0_* \cdot_* \varepsilon_*) = \zeta.$$

Thus, f_* is surjective.

3. CONJUGATE, INNER PRODUCT, NORM AND ABSOLUTE VALUE IN MULTIPLICATIVE DUAL NUMBERS

Definition 3.1 The conjugate of a multiplicative dual number $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \in \mathbb{D}_*$ is denoted by $\tilde{\zeta}^{-_*}$ and is defined as

$$\tilde{\zeta}^{-_*} = \zeta -_* \varepsilon_* \cdot_* \mathring{\zeta} \in \mathbb{D}_*.$$

Theorem 3.2 The following conjugation properties hold for multiplicative dual numbers

i. $\left(\tilde{\zeta}^{-_*}\right)^{-_*} = \tilde{\zeta}$

ii. $\left(\tilde{\zeta} +_* \tilde{\gamma}\right)^{-_*} = \tilde{\zeta}^{-_*} +_* \tilde{\gamma}^{-_*}$

iii. $\left(\tilde{\zeta} \cdot_* \tilde{\gamma}\right)^{-_*} = \tilde{\zeta}^{-_*} \cdot_* \tilde{\gamma}^{-_*}$,

iv. $For\ \tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$ we have $\tilde{\zeta} \cdot_* \tilde{\zeta}^{-_*} = \zeta^{2_*}$.

Proof.

i) For $\tilde{\zeta}$ and $\tilde{\gamma} \in \mathbb{D}_*$, we have

$$\begin{aligned} \left(\tilde{\zeta}^{-_*}\right)^{-_*} &= \zeta -_* \left(-_* \varepsilon_* \cdot_* \mathring{\zeta}\right) \\ &= \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} = \tilde{\zeta}. \end{aligned}$$

ii) We have

$$\tilde{\zeta} +_* \tilde{\gamma} = (\zeta +_* \gamma) +_* \varepsilon_* \cdot_* \left(\mathring{\zeta} +_* \mathring{\gamma}\right).$$

Then,

$$\begin{aligned} \left(\tilde{\zeta} +_* \tilde{\gamma}\right)^{-_*} &= (\zeta +_* \gamma) -_* \varepsilon_* \cdot_* \left(\mathring{\zeta} +_* \mathring{\gamma}\right) \\ &= \left(\zeta -_* \varepsilon_* \cdot_* \mathring{\zeta}\right) +_* (\gamma -_* \varepsilon_* \cdot_* \mathring{\gamma}) \\ &= \tilde{\zeta}^{-_*} +_* \tilde{\gamma}^{-_*}. \end{aligned}$$

iii) Given

$$\tilde{\zeta} \cdot_* \tilde{\gamma} = (\zeta \cdot_* \gamma) +_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\gamma} + \mathring{\zeta} \cdot_* \gamma\right),$$

then we have

$$\begin{aligned}\left(\tilde{\zeta} \cdot_* \tilde{\gamma}\right)^{-_*} &= (\zeta \cdot_* \gamma) -_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\gamma} + \mathring{\zeta} \cdot_* \gamma\right) \\ &= (\zeta -_* \varepsilon_* \cdot_* a) \cdot_* (\gamma -_* \varepsilon_* \cdot_* \mathring{\gamma}) \\ &= \tilde{\zeta}^{-_*} \cdot_* \tilde{\gamma}^{-_*}.\end{aligned}$$

iv)

$$\begin{aligned}\tilde{\zeta} \cdot_* \tilde{\zeta}^{-_*} &= (\zeta \cdot_* \zeta) +_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\zeta} - \mathring{\zeta} \cdot_* \zeta\right) \\ &= \zeta^{2_*}.\end{aligned}$$

Definition 32.3 Multiplicative inner product of two multiplicative dual number, $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$ and $\tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma} \in \mathbb{D}_*$, is defined by

$\langle , \rangle_* : \mathbb{D}_* \times_* \mathbb{D}_* \to \mathbb{R}_*$

$$\left\langle \tilde{\zeta}, \tilde{\gamma} \right\rangle_* = \left(\tilde{\zeta} \cdot_* \tilde{\gamma}^{-_*} +_* \tilde{\zeta}^{-_*} \cdot_* \tilde{\gamma}^*\right) = e^{ln\zeta ln\gamma}.$$

Definition 3.4 Multiplicative norm of a multiplicative dual number $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \in \mathbb{D}_*$ is defined by

$$\left\|\tilde{\zeta}\right\|_* = \|\zeta\|_*.$$

Example 3.5 (Multiplicative inner product and norm) Let $\tilde{\zeta}_1 = 2 +_* \varepsilon_* \cdot_* 3$ and $\tilde{\zeta}_2 = 4 +_* \varepsilon_* \cdot_* 5$ be two multiplicative dual numbers. Then, the multiplicative inner product of the numbers are given below

$$\left\langle \tilde{\zeta}, \tilde{\gamma} \right\rangle_* = e^{ln2ln4} = e^{2(ln2)^2}.$$

In addition, the norm of $\tilde{\zeta}$ is

$$\left\|\tilde{\zeta}\right\|_* = \|\zeta\|_* = 2.$$

Definition 3.6 Multiplicative semi-inner product of two multiplicative dual number, $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$ and $\tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma} \in \mathbb{D}_*$, is defined by

$\langle , \rangle_{\mathbb{D}_*} : \mathbb{D}_* \times_* \mathbb{D}_* \to \mathbb{D}_*$

$$\left\langle \tilde{\zeta}, \tilde{\gamma} \right\rangle_{\mathbb{D}_*} = \tilde{\zeta} \cdot_* \tilde{\gamma}^{-_*} = (\zeta \cdot_* \gamma) +_* \varepsilon_* \cdot_* \left(\mathring{\zeta} \cdot_* \gamma - \zeta \cdot_* \mathring{\gamma}\right).$$

Definition 3.7 Multiplicative semi-norm of a multiplicative dual number $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \in \mathbb{D}_*$ is denoted by $\left\|\tilde{\zeta}\right\|_*$ and is defined as

$\|.\|_{\mathbb{D}_*} : \mathbb{D}_* \times_* \mathbb{D}_* \to \mathbb{D}_*$

$$\left\|\tilde{\zeta}\right\|_{*} = \|\zeta\|_{*} +_{*} \varepsilon_{*} \cdot_{*} \frac{\langle \zeta, \mathring{\zeta} \rangle_{*}}{\|\zeta\|_{*}} *, \ \|\zeta\|_{*} \neq 1$$

Example 3.8 (Multiplicative semi-inner product) Let $\tilde{\zeta}_1 = 2 +_{*} \varepsilon_{*} \cdot_{*} 3$ and $\tilde{\zeta}_2 = 4 +_{*} \varepsilon_{*} \cdot_{*} 5$ be two multiplicative dual numbers. Then

$$\left\langle \tilde{\zeta}_1 , \tilde{\zeta}_2 \right\rangle_{\mathbb{D}_{*}} = (2 \cdot_{*} 4) +_{*} \varepsilon_{*} \cdot_{*} (3 \cdot_{*} 4 - 2 \cdot_{*} 5) = e^{3ln2} +_{*} \varepsilon_{*} \cdot_{*} e^{ln3ln4 - ln2ln5}.$$

Definition 3.9 The inverse of a nonzero multiplicative dual number $\tilde{\zeta} = \zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta} \in \mathbb{D}_{*}$ is denoted by $\tilde{\zeta}^{-1_{*}}$ and is defined as

$$\tilde{\zeta}^{-1_{*}} = \frac{\zeta^{-_{*}}}{\|\tilde{\zeta}\|_{*}^{2_{*}}} * = \frac{e}{\zeta} * -_{*} \varepsilon_{*} \cdot_{*} \frac{\mathring{\zeta}}{\zeta^{2_{*}}} *.$$

For a function $f(\tilde{\zeta})$ of a multiplicative dual variable, we have

$$f(\tilde{\zeta}) = f(\zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta}) = f(\zeta) +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta} \cdot_{*} f^{*}(\zeta),$$

where $f^{*}(\zeta)$ is the multiplicative derivative of f at ζ.

If the real part of the multiplicative dual number ζ is positive ($\zeta > 0$), we have

$$\sqrt[*]{\zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta}} = \sqrt[*]{\zeta} +_{*} \frac{\varepsilon_{*} \cdot_{*} \mathring{\zeta}}{e^{2} \cdot_{*} \sqrt[*]{\zeta}} *.$$

Then $\zeta \neq 0_{*}$. We have

$$\left(\zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta}\right)^{2_{*}} = (\zeta)^{2_{*}} +_{*} e^{2} \cdot_{*} \varepsilon_{*} \cdot_{*} \zeta \cdot_{*} \mathring{\zeta}.$$

This implies that

$$\begin{aligned} \sqrt[*]{\tilde{\zeta}^{2_{*}}} &= \sqrt[*]{\zeta^{2_{*}}} +_{*} \frac{e^{2} \cdot_{*} \varepsilon_{*} \cdot_{*} \zeta \cdot_{*} \mathring{\zeta}}{e^{2} \cdot_{*} \sqrt[*]{\zeta^{2_{*}}}} * \\ &= |\zeta|_{*} +_{*} \frac{\varepsilon_{*} \cdot_{*} \zeta \cdot_{*} \mathring{\zeta}}{|\zeta|_{*}} *. \end{aligned}$$

Definition 3.10 Absolute value of a multiplicative dual number $\left|\tilde{\zeta}\right|_{*} = \sqrt[*]{\left(\tilde{\zeta}\right)^{2_{*}}}$, it can be written

$$\left|\tilde{\zeta}\right|_{*} = \left|\zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta}\right|_{*} = \begin{cases} \zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta} & \zeta > 1, \\ -_{*}\left(\zeta +_{*} \varepsilon_{*} \cdot_{*} \mathring{\zeta}\right) & \zeta < 1. \end{cases}$$

Theorem 3.11 Suppose that $\tilde{\zeta},\ \tilde{\gamma} \in \mathbb{D}_*$. Then,

1. $\left|\tilde{\zeta}\right|_* = 0_* = 1$ if and only if $e^{ln\zeta} = 1$,
2. $\left|\tilde{\zeta} \cdot_* \tilde{\gamma}\right|_* = \left|\tilde{\zeta}\right|_* \cdot_* |\tilde{\gamma}|_*$,
3. $\left|\tilde{\zeta} +_* \tilde{\gamma}\right|_* \leq_* \left|\tilde{\zeta}\right|_* +_* |\tilde{\gamma}|_*$.

Proof.

1. By the Definition 7 we have

$$\left|\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right|_* = 1 \Leftrightarrow \zeta = 1$$

2. We have

$$\tilde{\zeta} \cdot_* \tilde{\gamma} = (\zeta \cdot_* \gamma) +_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\gamma} +_* \mathring{\zeta} \cdot_* \gamma\right).$$

Then,

$$\left|\tilde{\zeta} \cdot_* \tilde{\gamma}\right|_* = \left|(\zeta \cdot_* \gamma) +_* \varepsilon_* \cdot_* \left(\zeta \cdot_* \mathring{\gamma} +_* \mathring{\zeta} \cdot_* \gamma\right)\right|_*.$$

Thus, we get

$$\begin{aligned} \left|\tilde{\zeta} \cdot_* \tilde{\gamma}\right|_* &= |\zeta \cdot_* \gamma|_* = |\zeta|_* \cdot_* |\gamma|_* \\ &= \left|\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right|_* \cdot_* |\gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}|_* \\ &= \left|\tilde{\zeta}\right|_* \cdot_* |\tilde{\gamma}|_*. \end{aligned}$$

3. We have

$$\begin{aligned} \left|\tilde{\zeta} +_* \tilde{\gamma}\right|_* &= \left|\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} +_* \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}\right|_* \\ &= \left|(\zeta +_* \gamma) +_* \varepsilon_* \cdot_* \left(\mathring{\zeta} +_* \mathring{\gamma}\right)\right|_* \\ &= |\zeta +_* \gamma|_*. \end{aligned}$$

On the other hand, from the triangle inequality of the absolute value we,

$$\begin{aligned} \left|\tilde{\zeta} +_* \tilde{\gamma}\right|_* &\leq_* |\zeta|_* +_* |\gamma|_* \\ &\leq_* \left|\tilde{\zeta}\right|_* +_* |\tilde{\gamma}|_* \end{aligned}$$

obtained.

Theorem 3.12 Suppose that $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta},\ \tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma} \in \mathbb{D}_*$, where $\zeta, \mathring{\zeta}, \gamma, \mathring{\gamma} \in \mathbb{R}_*$. Then we have

$$\tilde{\zeta} \cdot_* \tilde{\gamma}^{-_*} +_* \tilde{\gamma} \cdot_* \tilde{\zeta}^{-_*} = e^2 \cdot_* \zeta \cdot_* \gamma$$

which is a multiplicative real number.

Definition 3.13 Let $\mathbb{P}$ and $\mathbb{Q}$ any two points in the multiplicative dual plane, we can define the multiplicative distance between $\mathbb{P}$ and $\mathbb{Q}$ as

$$\begin{aligned} d_*(\mathbb{P},\mathbb{Q}) &= \left\|\tilde{\gamma}-_*\tilde{\zeta}\right\|_* \\ &= \left\|(\gamma+_*\varepsilon_*\cdot_*\mathring{\gamma})-_*(\zeta+_*\varepsilon_*\cdot_*\mathring{\zeta})\right\|_* \\ &= \left\|\gamma-_*\zeta+_*\varepsilon_*\cdot_*\mathring{\gamma}-_*\mathring{\zeta}\right\|_* \\ &= |\gamma-_*\zeta|_*. \end{aligned}$$

4. MATRIX REPRESENTATION IN MULTIPLICATIVE DUAL NUMBERS

The set $\mathbb{M}_*$ define as

$$\mathbb{M}_* = \left\{\begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} : \zeta, \mathring{\zeta} \in \mathbb{R}_*\right\}.$$

This set is called the matrix representation of multiplicative dual numbers, where the operations $+_*$ and $\cdot_*$ are defined as multiplicative matrix addition and multiplication, respectively. The multiplicative matrix additive identity element is

$$\begin{bmatrix} 0_* & 0_* \\ 0_* & 0_* \end{bmatrix}.$$

The matrix additive inverse of A is

$$-_*\mathrm{A}_1 = \begin{bmatrix} -_*\zeta & -_*\mathring{\zeta} \\ 0_* & -_*\zeta \end{bmatrix}.$$

The multiplicative matrix identity element with respect to the operation $\cdot_*$ is

$$\begin{bmatrix} 1_* & 0_* \\ 0_* & 1_* \end{bmatrix}.$$

Theorem 4.1 Let $A_1 = \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix}, A_2 = \begin{bmatrix} \gamma & \mathring{\gamma} \\ 0_* & \gamma \end{bmatrix}$ and $A_3 = \begin{bmatrix} \xi & \mathring{\xi} \\ 0_* & \xi \end{bmatrix} \in \mathbb{D}_*$ then we have the following

i. $A_1+_*A_2 = A_2+_*A_1$,

ii. $(A_1+_*A_2)+_*A_3 = A_1+_*(A_2+_*A_3)$,

iii. $A_1\cdot_* A_2 = A_2\cdot_* A_1$,

iv. $(A_1 \cdot_* A_2) \cdot_* A_3 = A_1 \cdot_* (A_2 \cdot_* A_3)$,

v. $(A_1 +_* A_2) \cdot_* A_3 = A_1 \cdot_* A_3 +_* A_2 \cdot_* A_3$,

vi. $A_1 \cdot_* (A_2 +_* A_3) = A_1 \cdot_* A_2 +_* A_1 \cdot_* A_3$.

Proof.

i.

$$\begin{aligned} A_1 +_* A_2 &= \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} +_* \begin{bmatrix} \gamma & \mathring{\gamma} \\ 0_* & \gamma \end{bmatrix} \\ &= \begin{bmatrix} \zeta +_* \gamma & \mathring{\zeta} +_* \mathring{\gamma} \\ 0_* +_* 0_* & \zeta +_* \gamma \end{bmatrix} \\ &= \begin{bmatrix} \gamma +_* \zeta & \mathring{\gamma} +_* \mathring{\zeta} \\ 0_* +_* 0_* & \zeta +_* \zeta \end{bmatrix} \\ &= A_2 +_* A_1. \end{aligned}$$

ii.

$$\begin{aligned} (A_1 +_* A_2) +_* A_3 &= \begin{bmatrix} \zeta +_* \gamma & \mathring{\zeta} +_* \mathring{\gamma} \\ 0_* +_* 0_* & \zeta +_* \gamma \end{bmatrix} +_* \begin{bmatrix} \xi & \xi \\ 0_* & \xi \end{bmatrix} \\ &= \begin{bmatrix} \zeta +_* \gamma +_* \xi & \mathring{\zeta} +_* \mathring{\gamma} +_* \xi \\ 0_* +_* 0_* +_* 0_* & \zeta +_* \gamma +_* \xi \end{bmatrix} \\ &= \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} +_* \begin{bmatrix} \gamma +_* \xi & \mathring{\gamma} +_* \xi \\ 0_* +_* 0_* & \gamma +_* \xi \end{bmatrix} \\ &= A_1 +_* (A_2 +_* A_3). \end{aligned}$$

iii.

$$\begin{aligned} A_1 \cdot_* A_2 &= \begin{bmatrix} \zeta \cdot_* \gamma & \zeta \cdot_* \mathring{\gamma} +_* \mathring{\zeta} \cdot_* \gamma \\ 0_* \cdot_* 0_* & \zeta \cdot_* \gamma \end{bmatrix} \\ &= \begin{bmatrix} \gamma \cdot_* \zeta & \mathring{\gamma} \cdot_* \zeta +_* \gamma \cdot_* \mathring{\zeta} \\ 0_* \cdot_* 0_* & \gamma \cdot_* \zeta \end{bmatrix} \\ &= A_2 \cdot_* A_1. \end{aligned}$$

iv.

$$\begin{aligned} (A_1 \cdot_* A_2) \cdot_* A_3 &= \begin{bmatrix} \zeta \cdot_* \gamma & \zeta \cdot_* \mathring{\gamma} +_* \mathring{\zeta} \cdot_* \gamma \\ 0_* \cdot_* 0_* & \zeta \cdot_* \gamma \end{bmatrix} \cdot_* \begin{bmatrix} \xi & \xi \\ 0_* & \xi \end{bmatrix} \\ &= \begin{bmatrix} \zeta \cdot_* \gamma \cdot_* \xi & \zeta \cdot_* \gamma \cdot_* \xi +_* \zeta \cdot_* \mathring{\gamma} \cdot_* \xi +_* \mathring{\zeta} \cdot_* \gamma \cdot_* \xi \\ 0_* \cdot_* 0_* \cdot_* 0_* & \zeta \cdot_* \gamma \cdot_* \xi \end{bmatrix}. \end{aligned}$$

Conversely, we have

$$
\begin{aligned}
A_1 \cdot_* (A_2 \cdot_* A_3) &= \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} \cdot_* \begin{bmatrix} \gamma \cdot_* \xi & \gamma \cdot_* \xi +_* \mathring{\gamma} \cdot_* \xi \\ 0_* \cdot_* 0_* & \gamma \cdot_* \xi \end{bmatrix} \\
&= \begin{bmatrix} \zeta \cdot_* \gamma \cdot_* \xi & \zeta \cdot_* \gamma \cdot_* \xi +_* \zeta \cdot_* \mathring{\gamma} \cdot_* \xi +_* \mathring{\zeta} \cdot_* \gamma \cdot_* \xi \\ 0_* \cdot_* 0_* \cdot_* 0_* & \zeta \cdot_* \gamma \cdot_* \xi \end{bmatrix}.
\end{aligned}
$$

Hence, $iv.$ holds.

v.

$$
\begin{aligned}
(A_1 +_* A_2) \cdot_* A_3 &= \begin{bmatrix} \zeta +_* \gamma & \mathring{\zeta} +_* \mathring{\gamma} \\ 0_* \cdot_* 0_* & \zeta +_* \gamma \end{bmatrix} \cdot_* \begin{bmatrix} \xi & \xi \\ 0_* & \xi \end{bmatrix} \\
&= \begin{bmatrix} \zeta \cdot_* \xi +_* \gamma \cdot_* \xi & \mathring{\zeta} \cdot_* \xi +_* \mathring{\gamma} \cdot_* \xi +_* \zeta \cdot_* \xi +_* \gamma \cdot_* \xi \\ 0_* \cdot_* 0_* \cdot_* 0_* & \zeta \cdot_* \xi +_* \gamma \cdot_* \xi \end{bmatrix} \\
&= \begin{bmatrix} \zeta \cdot_* \xi & \mathring{\zeta} \cdot_* \xi +_* \zeta \cdot_* \xi \\ 0_* \cdot_* 0_* & \zeta \cdot_* \xi \end{bmatrix} +_* \begin{bmatrix} \gamma \cdot_* \xi & \mathring{\gamma} \cdot_* \xi +_* \gamma \cdot_* \xi \\ 0_* \cdot_* 0_* & \gamma \cdot_* \xi \end{bmatrix} \\
&= \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} \cdot_* \begin{bmatrix} \xi & \xi \\ 0_* & \xi \end{bmatrix} +_* \begin{bmatrix} \gamma & \mathring{\gamma} \\ 0_* & \gamma \end{bmatrix} \cdot_* \begin{bmatrix} \xi & \xi \\ 0_* & \xi \end{bmatrix} \\
&= A_1 \cdot_* A_3 +_* A_2 \cdot_* A_3.
\end{aligned}
$$

Theorem 4.2 $\mathbb{M}_*$ is isomorphic to the set of multiplicative dual numbers $\mathbb{D}_*$.

Proof. Define the multiplicative mapping

$$
\begin{aligned}
h_* &: \mathbb{D}_* \to \mathbb{M}_* \\
\zeta &\to h_*(\zeta) = \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix}
\end{aligned}
$$

where $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$. If $\tilde{\zeta} = \zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$ and $\tilde{\gamma} = \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}$ are then we have

$$
\begin{aligned}
h_*(\tilde{\zeta} +_* \tilde{\gamma}) &= \begin{bmatrix} \zeta +_* \gamma & \mathring{\zeta} +_* \mathring{\gamma} \\ 0_* +_* 0_* & \zeta +_* \gamma \end{bmatrix} \\
&= \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix} +_* \begin{bmatrix} \gamma & \mathring{\gamma} \\ 0_* & \gamma \end{bmatrix} \\
&= h_*(\tilde{\zeta}) +_* h_*(\tilde{\gamma}).
\end{aligned}
$$

In addition, for multiplicative scalar multiplication we get

$$
\begin{aligned}
h_*(d \cdot_* \tilde{\zeta}) &= h_*(d \cdot_* \zeta +_* \varepsilon_* \cdot_* d \cdot_* \mathring{\zeta}) \\
&= \begin{bmatrix} d \cdot_* \zeta & d \cdot_* \mathring{\zeta} \\ 0_* & d \cdot_* \zeta \end{bmatrix} \\
&= d \cdot_* h_*(\tilde{\zeta}).
\end{aligned}
$$

Thus, h_* preserves both multiplicative addition and multiplicative scalar multiplication. Suppose $\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta} \neq \gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}$, i.e., $\zeta \neq \gamma$ and $\mathring{\zeta} \neq \mathring{\gamma}$. Then

$$h_*\left(\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right) \neq h_*(\gamma +_* \varepsilon_* \cdot_* \mathring{\gamma}).$$

Hence, h_* is injective. For any $\zeta \in \mathbb{R}_*$, there exists a unique multiplicative dual number $\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}$, such that

$$h_*\left(\zeta +_* \varepsilon_* \cdot_* \mathring{\zeta}\right) = \begin{bmatrix} \zeta & \mathring{\zeta} \\ 0_* & \zeta \end{bmatrix}.$$

Thus, h_* is surjective.

5. THE INNER PRODUCT AND NORM OF MULTIPLICATIVE DUAL VECTORS

The term $\mathbb{D}_*^3$, is defined as the collection of all triples of multiplicative dual numbers, which is represented as

$$\begin{aligned} \mathbb{D}_*^3 &= \mathbb{D}_* \times_* \mathbb{D}_* \times_* \mathbb{D}_* \\ &= \left\{\vec{\zeta} = \left(\zeta_1 +_* \varepsilon_* \cdot_* \mathring{\zeta}_1,\ \zeta_2 +_* \varepsilon_* \cdot_* \mathring{\zeta}_2,\ \zeta_3 +_* \varepsilon_* \cdot_* \mathring{\zeta}_3\right)\right\}, \\ &= \left\{\vec{\zeta} = (\zeta_1,\ \zeta_2,\ \zeta_3) +_* \varepsilon_* \cdot_* \left(\mathring{\zeta}_1,\ \mathring{\zeta}_2,\ \mathring{\zeta}_3\right)\right\} \\ &= \left\{\vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \mathring{\tilde{\zeta}},\ \tilde{\zeta}, \mathring{\tilde{\zeta}} \in \mathbb{R}_*^3\right\} \end{aligned}$$

The elements within $\mathbb{D}_*^3$ are identified as multiplicative dual vectors. The multiplicative dual vector, denoted as $\vec{\zeta}$, can be expressed as $\vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \mathring{\tilde{\zeta}}$, where $\tilde{\zeta}$ and $\mathring{\tilde{\zeta}}$ represent vectors within the multiplicative real space $\mathbb{R}_*^3$, mirroring the concept of multiplicative dual number.

For any multiplicative vectors $\vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \mathring{\tilde{\zeta}}$ and $\vec{\gamma} = \tilde{\gamma} +_* \varepsilon_* \cdot_* \mathring{\tilde{\gamma}}$ in $\mathbb{D}_*^3$, $d \in \mathbb{R}_*$, multiplicative addition and multiplicative scalar multiplication are defined by

$$\begin{aligned} \vec{\zeta} +_* \vec{\gamma} &= \left(\tilde{\zeta}_1,\ \tilde{\zeta}_2,\ \tilde{\zeta}_3\right) +_* (\tilde{\gamma}_1,\ \tilde{\gamma}_2,\ \tilde{\gamma}_3) \\ &= \left(\tilde{\zeta}_1 +_* \tilde{\gamma}_1,\ \tilde{\zeta}_2 +_* \tilde{\gamma}_2,\ \tilde{\gamma}_3 +_* \tilde{\gamma}_3\right) \\ &= \left(\tilde{\zeta}_1\tilde{\gamma}_1,\ \tilde{\zeta}_2\tilde{\gamma}_2,\ \tilde{\gamma}_3\tilde{\gamma}_3\right), \end{aligned}$$

and

$$\begin{aligned} d \cdot_* \vec{\zeta} &= \left(d \cdot_* \tilde{\zeta}_1,\ d \cdot_* \tilde{\zeta}_2,\ d \cdot_* \tilde{\zeta}_3\right), \\ &= \left(e^{lndln\tilde{\zeta}_1},\ e^{lndln\tilde{\zeta}_2},\ e^{lndln\tilde{\zeta}_3}\right). \end{aligned}$$

Thus, $\mathbb{D}_*$ is a vector space.

Definition 5.1

$$\langle , \rangle_* : \mathbb{D}_*^3 \times_* \mathbb{D}_*^3 \to \mathbb{R}_*$$

$$\left\langle \vec{\zeta}, \vec{\gamma} \right\rangle_* = \left(\tilde{\zeta} \cdot_* \tilde{\gamma}^{-*} +_* \tilde{\zeta}^{-*} \cdot_* \tilde{\gamma}^* \right) = e^{ln\zeta_1 ln\gamma_1 + ln\zeta_2 ln\gamma_2 + ln\zeta_3 ln\gamma_3}$$

is called Multiplicative inner product in $\mathbb{D}_*^3$, where $\vec{\zeta} = \left(\tilde{\zeta}_1, \tilde{\zeta}_2, \tilde{\zeta}_3\right)$ and $\vec{\gamma} = (\tilde{\gamma}_1, \tilde{\gamma}_2, \tilde{\gamma}_3)$.

Definition 5.2 *For* any multiplicative vectors $\vec{\zeta}$ and $\vec{\gamma}$ in $\mathbb{D}_*^3$, the multiplicative *semi*-inner product of multiplicative dual vector is defined by

$$\langle , \rangle_{\mathbb{D}_*} : \mathbb{D}_*^3 \times_* \mathbb{D}_*^3 \to \mathbb{D}_*$$

$$\left\langle \vec{\zeta}, \vec{\gamma} \right\rangle_{\mathbb{D}_*} = \langle \tilde{\zeta}, \tilde{\gamma} \rangle_* +_* \varepsilon_* \cdot_* \left(\langle \tilde{\zeta}, \mathring{\tilde{\gamma}} \rangle_* +_* \left\langle \mathring{\tilde{\zeta}}, \tilde{\gamma} \right\rangle_* \right).$$

From the definition we have equality

$$\left\langle \vec{\zeta}, \vec{\gamma} \right\rangle_{\mathbb{D}_*} = e^{\sum_{i=1}^3 ln(\tilde{\zeta}_i) ln(\tilde{\gamma}_i)} +_* \varepsilon_* \cdot_* e^{\sum_{i=1}^3 ln\left(\mathring{\tilde{\zeta}}_i\right) ln(\tilde{\gamma}_i) - \sum_{i=1}^3 ln\left(\tilde{\tilde{\zeta}}_i\right) ln\left(\mathring{\tilde{\gamma}}_i\right)}.$$

Example 5.3 Let $\vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \mathring{\tilde{\zeta}} = (2,3,1) +_* \varepsilon_* \cdot_* (5,1,2)$ and $\vec{\gamma} = \tilde{\gamma} +_* \varepsilon_* \cdot_* \mathring{\tilde{\gamma}} = (1,4,2) +_* \varepsilon_* \cdot_* (1,3,4)$ in $\mathbb{D}_*^3$, be two multiplicative dual vectors. The multiplicative semi-inner product is computed as

$$\left\langle \vec{\zeta}, \vec{\gamma} \right\rangle_{\mathbb{D}_*} = \langle \tilde{\zeta}, \tilde{\gamma} \rangle_* +_* \varepsilon_* \cdot_* \left(\langle \tilde{\zeta}, \mathring{\tilde{\gamma}} \rangle_* +_* \left\langle \mathring{\tilde{\zeta}}, \tilde{\gamma} \right\rangle_* \right)$$

$$= (2 \cdot_* 1 +_* 3 \cdot_* 4 +_* 1 \cdot_* 2) +_* \varepsilon_* \cdot_* \left((5 \cdot_* 1 +_* 1 \cdot_* 4 +_* 2 \cdot_* 2) -_* (2 \cdot_* 1 +_* 3 \cdot_* 3 -$$

$$= e^{(\ln 2 \ln + \ln 3 \ln 4 + \ln 1 \ln 2)} +_* \varepsilon_* \cdot_* e^{\left((\ln 5 \ln 1 + \ln 1 \ln 4 + \ln 2 \ln 2) - (\ln 2 \ln + \ln 3 \ln 3 + \ln 1 \ln 4)\right)}$$

$$= e^{\ln 3 \ln 4} +_* \varepsilon_* \cdot_* e^{(\ln 2)^2 - (\ln 3)^2}$$

Definition 5.4 Multiplicative semi-norm of a multiplicative dual number $\vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \mathring{\tilde{\zeta}}$ in $\mathbb{D}_*^3$, is defined as

$$\|.\|_{\mathbb{D}_*} : \mathbb{D}_*^3 \to \mathbb{R}_*$$

$$\left\|\vec{\zeta}\right\|_{\mathbb{D}_*} = \left\|\tilde{\zeta}\right\|_* +_* \varepsilon_* \cdot_* \frac{\left\langle \tilde{\zeta}, \overset{\circ}{\tilde{\zeta}} \right\rangle_*}{\left\|\tilde{\zeta}\right\|_*} *, \quad \left\|\tilde{\zeta}\right\|_* \neq 1.$$

Example 5.5 The semi-norm of $\vec{\zeta} = (2,3,1) +_* \varepsilon_* \cdot_* (5,1,2)$

$$\left\|\vec{\zeta}\right\|_{\mathbb{D}_*} = \left\|\tilde{\zeta}\right\|_* +_* \varepsilon_* \cdot_* \frac{\left\langle \tilde{\zeta}, \overset{\circ}{\tilde{\zeta}} \right\rangle_*}{\left\|\tilde{\zeta}\right\|_*} *$$

$$\left\|\tilde{\zeta}\right\|_* = e^{\sqrt{(\ln 2)^2 + (\ln 3)^2 + (\ln 1)^2}} = e^{\sqrt{(\ln 2)^2 + (\ln 3)^2}}$$

$$\left\langle \tilde{\zeta}, \overset{\circ}{\tilde{\zeta}} \right\rangle_* = e^{\ln 2 \ln 5 + \ln 3 \ln 1 + \ln 1 \ln 2} = e^{\ln 2 \ln 5}$$

$$\left\|\vec{\zeta}\right\|_{\mathbb{D}_*} = e^{\sqrt{(\ln 2)^2 + (\ln 3)^2}} +_* \varepsilon_* \cdot_* \frac{e^{\ln 2 \ln 5}}{e^{\sqrt{(\ln 2)^2 + (\ln 3)^2}}}.$$

Definition 5.6 If $\left\|\vec{\zeta}\right\|_* = 1_* +_* 0_* \cdot_* \varepsilon_*$, then $\vec{\zeta}$is called a multiplicative dual unit vector.

Definition 5.7

$$\|.\|_* : \mathbb{D}_*^3 \to \mathbb{R}_*$$

$$\left\|\vec{\zeta}\right\|_* = \left\|\tilde{\zeta}\right\|_* = e^{(ln\zeta_1 ln\gamma_1 + ln\zeta_2 ln\gamma_2 + ln\zeta_3 ln\gamma_3)^{\frac{1}{2}}}$$

is called Multiplicative norm in $\mathbb{D}_*^3$.

6. MULTIPLICATIVE DUAL UNIT SPHERE

Definition 6.4 The set of multiplicative dual unit vectors, formally represented as

$$\mathbb{D}_* S^2 = \left\{ \vec{\zeta} \in \mathbb{D}_*^3 \mid \left\|\vec{\zeta}\right\|_* = 1_* \right\},$$

is defined as the multiplicative dual unit sphere.

In other words, the multiplicative dual unit sphere is

$$\mathbb{D}_* S^2 = \left\{ \vec{\zeta} = \tilde{\zeta} +_* \varepsilon_* \cdot_* \overset{\circ}{\tilde{\zeta}} \mid \left\| \tilde{\zeta} \right\|_* = (1_*, 0_*),\ \tilde{\zeta},\ \overset{\circ}{\tilde{\zeta}} \in \mathbb{R}_*^3 \right\}$$

Since $1_* = e^1$ in the geometric real field, the condition $\left\| \tilde{\zeta} \right\|_* = 1_*$ is equivalent to

$$ln\left(\tilde{\zeta}_1\right)^2 + ln\left(\tilde{\zeta}_2\right)^2 + ln\left(\tilde{\zeta}_3\right)^2 = 1 \text{ and } \sum_{i=1}^{3} ln\left(\tilde{\zeta}_i\right) ln\left(\overset{\circ}{\tilde{\zeta}}_i\right) = 0.$$

A typical element is $\vec{\zeta} = (\mathrm{e}^x,\ \mathrm{e}^y,\ \mathrm{e}^z) +_* \varepsilon_* \cdot_* \left(\mathrm{e}^a,\ \mathrm{e}^b,\ \mathrm{e}^c\right)$ satisfying

$$x^2 + y^2 + z^2 = 1 \text{ and } ax + by + cz = 0.$$

Example 6.2 (Multiplicative Dual Unit Vector) Consider the multiplicative dual vector

$$\vec{\zeta} = \left(\mathrm{e}^{\frac{1}{\sqrt{3}}},\ \mathrm{e}^{\frac{1}{\sqrt{3}}},\ \mathrm{e}^{\frac{1}{\sqrt{3}}} \right) +_* \varepsilon_* \cdot_* (\mathrm{e}^3,\ \mathrm{e}^2,\ \mathrm{e}^{-5})$$

Its norm is

$$\left\| \vec{\zeta} \right\|_* \quad = \quad 1_* +_* \varepsilon_* \cdot_* 0_*,$$

so $\vec{\zeta} \in \mathbb{D}_* S^2$.

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Bibliography of Zerconid Mites (Acari: Zerconidae) of Türkiye (1979-2026)

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ABSTRACT

This study presents a comprehensive bibliography of research on the systematics, taxonomy, and ecology of zerconid mites (Acari: Zerconidae) in Türkiye covering the period from 1979 to 2026. It brings together the scientific literature accumulated over more than four decades and provides a consolidated source of information on the diversity, classification, distribution, and ecology of the Turkish zerconid fauna. Bibliographic records concerning the two genera, *Prozercon* and *Zercon*, and the 143 species recorded from Türkiye were systematically compiled from journal articles, book chapters, conference proceedings, and postgraduate theses. The literature was organized into three periods to illustrate the development of research on Zerconidae in the country. Between 1979 and 1990, one publication documenting six species was recorded. During 1991-2009, the literature expanded to 36 publications covering 63 species, while the period from 2010 to 2026 accounted for 116 publications and 74 species. In total, 153 publications documenting 143 species were identified. The collected literature encompasses species descriptions, taxonomic revisions, faunistic records, distributional data, and ecological preferences. In this bibliography, journal articles, book chapters, conference papers, and postgraduate theses are grouped separately and arranged alphabetically according to the surname of the first author(s). The present study constitutes the first comprehensive bibliographic compilation of the Zerconidae fauna of Türkiye and provides a reference base for future studies on the taxonomy, systematics, distribution, ecology, and biodiversity of the species concerned.

Keywords – Publication list, acarology, Mesostigmata, Prozercon, Zercon

INTRODUCTION

Zerconid mites, or briefly zerconids, are free-living members of the order Mesostigmata that predominantly inhabit soil, leaf litter, moss, decaying plant material, and the forest floor. Their frequent occurrence in moist and organic matter-rich microhabitats indicates that these mites may play important roles in soil ecosystems. They primarily feed on small soil invertebrates and other microarthropods, making them one of the key components of soil mesofauna. Members of the family Zerconidae have been recorded from various parts of Europe, Asia, North Africa, and North America and have been shown to occur under a wide range of climatic and ecological conditions. Nevertheless, compared with the extensive taxonomic research on the family, current knowledge of the biology, feeding behavior, life cycles, and habitat preferences of zerconids remains relatively limited.

Research on the family Zerconidae in Türkiye was initiated by the Polish acarologist Czesław Błaszak and significantly expanded from the

early 1990s onward through the taxonomic and faunistic studies conducted by Raşit Urhan, particularly in the northeastern part of the country. While early research focused primarily on the identification and classification of zerconid species recorded from Türkiye, subsequent studies focused on describing new species, determining their geographic distributions, comparing morphological characteristics, and investigating ecological traits such as altitudinal and habitat preferences. Findings obtained from various regions of Türkiye across different research periods revealed that the country harbors considerable species diversity within the family Zerconidae. However, the fact that these studies were published across different periods and in various types of scientific publications, including journal articles, books, conference proceedings, and theses, has made it difficult to comprehensively assess the literature on the Zerconidae of Türkiye. In this study, all available publications on the Zerconidae of Türkiye between 1979 and 2026 were compiled with the aim of providing an overview of the historical development of scientific research on the family and the overall scope of the existing literature.

METHODS AND CONVENTIONS

All research conducted on zerconid mites in Türkiye was grouped into three distinct periods (Table 1). In the bibliography section, all publications on the family Zerconidae were classified and presented under four main categories (journal articles, book chapters, conference papers, and postgraduate theses). In each category, studies published by the same author(s) in the same year are distinguished from one another by letters placed after the year in parentheses.

RESULTS AND DISCUSSION

Analysis of the bibliographic data reveals distinct patterns in the temporal development, publication types, author productivity, geographical distribution, research topics, and discovery of new species and records of Zerconidae in Türkiye.

Total number of publications by year

The annual distribution of publications included in the bibliography demonstrates a clear increase in Zerconidae research in Türkiye over time. The first study was published by Błaszak in 1979, followed by a limited but steady increase during the 1990s. Research activity intensified particularly during the 2010s, with the highest number of publications recorded in 2017. The years 2015 and 2024 also stand out for their relatively high publication output. Continued publication activity during 2020-2026 indicates that Zerconidae research remains an active and current field in Türkiye.

Table 1: Numerical distribution of Zerconidae publications across three periods

Part	Period	Researcher(s)	Number of Publications	Number of Recorded Species*
1	1979-1990	Czesław Błaszak	1	6
2	1991-2009	Raşit Urhan, Nusret Ayyıldız, Ali Nafiz Ekiz, Yusuf Katılmış, Ayşe Öksüz, Ayşe Toluk, Elif Koçoğlu, Abdülkadir Taşdemir, Sedat Per	36	63
3	2010-2026	Raşit Urhan, Mehmet Karaca, Elif Hilal Duran, Davut Rıza Bulut, Ayşenur Demirdöven, Büşra Keçeci/Aksu, Cevdet Bostancı, Esat Enis Karnak, Kamil Bilki, Murat Öztaş, Seyhan Güler, Şerif Naci Orman, Zhanerke Kassen, Esat Kızılkaya, Yusuf Katılmış, Gamze Karaca, Gürçay Kıvanç Akyıldız, Merve Tepe, Mehmet Urhan	116	74
		TOTAL	153	143

*Species recorded in previous periods were excluded from the counts for the second and third periods. Thus, each period represents only species recorded from Türkiye for the first time during that period.

Distribution of publication types

Journal articles constitute the largest publication category, with 65 records, followed by conference proceedings with 61 records. Book chapters account for 11 records, while postgraduate theses comprise 16 records, including 13 master's and 3 doctoral theses. This distribution indicates that Zerconidae research in Türkiye has been disseminated primarily through journal articles and conference proceedings, with postgraduate research also contributing to the development of the literature.

Author productivity

In terms of author productivity, R. Urhan ranks first with 78 publications, followed by M. Karaca with 61 and E. H. Duran with 28 publications (Figure 1). N. Ayyıldız, D. R. Bulut, and E. Kızılkaya show moderate levels of productivity, whereas the contributions of the remaining researchers are more limited. This distribution indicates that Zerconidae research in Türkiye has been concentrated around a relatively small group of

researchers, with R. Urhan and M. Karaca making particularly substantial contributions to the development of the field.

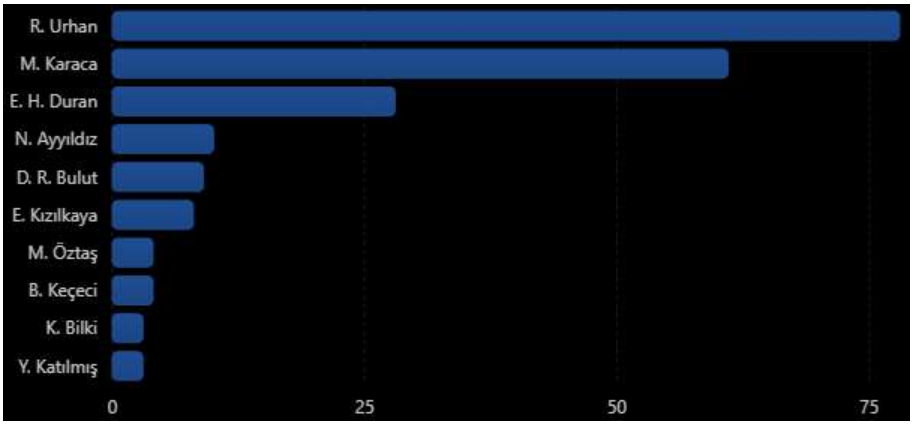

Figure 2: Author productivity in studies on Zerconidae in Türkiye

Research intensity by geographical region of Türkiye

In terms of geographical distribution, the Aegean Region ranks first with 22 publications, followed by the Marmara Region with 17 publications (Figure 2). The Black Sea Region, Central Anatolia, and the Mediterranean Region follow with 9, 7, and 6 publications, respectively. The Eastern Anatolia and Southeastern Anatolia regions have comparatively fewer studies. This distribution indicates that Zerconidae research is not geographically uniform across Türkiye, but is particularly concentrated in the Aegean and Marmara regions.

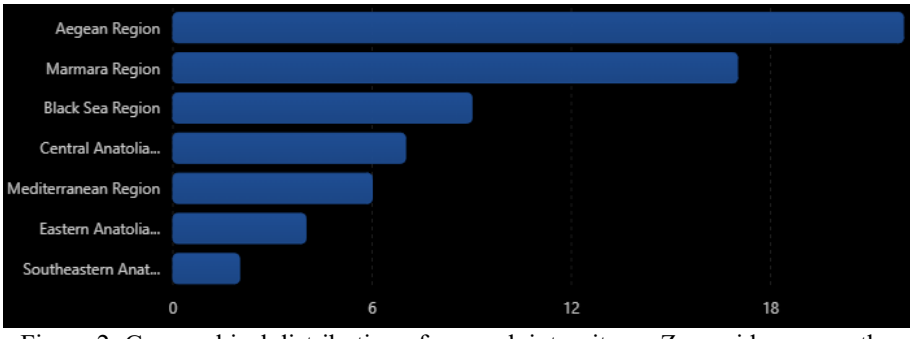

Figure 2: Geographical distribution of research intensity on Zerconidae across the geographical regions of Türkiye

Temporal changes in taxonomic, faunistic, and ecological studies

Zerconidae research in Türkiye has gradually evolved from a predominantly taxonomic focus toward a broader range of research topics. Taxonomic studies were dominant during the 1990s, while faunistic studies also increased substantially during 2001-2010. The period 2011-2020

represents the most intensive period for taxonomic, faunistic, and ecological studies combined. During 2021-2026, research in all three areas continued, with ecological studies becoming more prominent. This shift indicates that the scope of research has expanded beyond species descriptions to include habitat, elevation, and environmental factors.

Annual variation in new species descriptions and new records

New species descriptions became increasingly prominent from the mid-1990s onward, reaching their highest level in 1996. New species continued to be described regularly during the 2000s and, particularly, the 2010s. New records showed a more irregular distribution, with notable increases in 2015, 2017, and 2019. The simultaneous increase in new species descriptions and new records in some years indicates that research has contributed both to the description of taxa new to science and to documenting the distribution of previously known species in Türkiye.

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Single-celled Eukaryotic Human Disease Models: From Yeast To Human

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ABSTRACT

Single-celled eukaryotic model organisms, particularly *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe*, provide a versatile and experimentally accessible framework for investigating the molecular mechanisms underlying human diseases. Their short generation times, extensive genetic tractability, and the high evolutionary conservation of core cellular processes enable systematic functional analysis of disease-associated genes and variants. Yeast models offer a unique balance between biological relevance and experimental simplicity, making them especially valuable for dissecting genotype–phenotype relationships.

Yeast-based and humanized yeast systems have been widely applied to model disorders linked to protein homeostasis, mitochondrial dysfunction, metabolic imbalance, genome instability, and cell cycle dysregulation. Through heterologous expression and functional replacement strategies, pathogenic human variants can be evaluated using quantifiable cellular phenotypes, thereby providing direct functional evidence that complements clinical genomics and helps resolve variants of uncertain significance. In this context, yeast has emerged as a powerful platform for studying neurodegenerative diseases, mitochondrial and metabolic disorders, and cancer-associated mechanisms in a controlled and scalable manner.

Although yeast models cannot fully recapitulate the complexity of multicellular organisms, their experimental flexibility and evolutionary conservation make them an effective preclinical system for mechanism discovery, genetic interaction mapping, and therapeutic target prioritization. When integrated with high-throughput phenotyping, genome editing, and multi-omics approaches, yeast-based models continue to serve as a critical experimental bridge between simplified cellular systems and more complex disease models.

Keywords – Disease model, neurodegenerative disorders, cancer, metabolic disorders, yeast.

INTRODUCTION

Research aimed at elucidating the molecular basis of human diseases requires model systems that are both biologically meaningful and technically tractable for experimental investigation. In this context, model organisms are

employed as fundamental research tools for the experimental dissection of disease mechanisms. This broad spectrum, ranging from bacteria to unicellular eukaryotes and from invertebrates to mammalian models, offers distinct experimental advantages and limitations depending on the complexity of the research question and the biological processes under investigation. Consequently, the selection of an appropriate model organism necessitates careful consideration of the balance between physiological relevance and experimental accessibility and scalability (Johnston, 2020; Kunze and Malfatti, 2025).

Although multicellular animal models offer strong advantages in terms of recapitulating physiological context, they are not ideal for every research question due to long experimental timelines, high costs, ethical constraints, and limited flexibility in genetic manipulation. In this regard, unicellular eukaryotic model organisms, particularly yeasts, have emerged as an important experimental platform for dissecting the fundamental principles of disease biology (Wangler et al., 2017).

Yeasts, as unicellular eukaryotes, differ fundamentally from prokaryotic systems in that they possess a nucleated cell architecture, chromatin organization, an endomembrane system, and eukaryote-specific organelles such as mitochondria. These structural and functional features enable the investigation of processes directly implicated in human disease, including cell cycle regulation, DNA damage responses, epigenetic control, protein folding, and metabolic networks through evolutionarily conserved mechanisms. Indeed, the fact that a substantial proportion of human disease-associated genes have functional orthologs in unicellular eukaryotes renders these organisms not merely simplified systems, but evolutionarily informative models (Hoffman et al., 2015).

In conclusion, unicellular eukaryotic model organisms constitute simplified experimental systems that nevertheless retain the fundamental structural and functional features of complex eukaryotic cells such as human cells. In this respect, experimental approaches spanning ‘from yeast to human’ serve as a strategic bridge for elucidating the molecular pathogenesis of human diseases and for laying the groundwork for translational research. In this section, studies highlighting the use of yeasts as models for human diseases are presented.

Yeast as A Human Disease Model

Yeasts such as *Saccharomyces cerevisiae* and *Schizosaccharomyces pombe* have emerged as powerful model systems for the functional analysis of disease-associated genes due to their evolutionarily highly conserved cellular pathways, short generation times, and advanced genetic manipulation capabilities. Yeast models are widely used to elucidate disease mechanisms involving processes that are evolutionarily conserved between yeast and humans, including DNA repair, cell cycle control, mitochondrial function, protein homeostasis, RNA metabolism, and metabolic networks. The high degree of conservation of core components of RNA metabolism between yeast and humans enables the analysis of disease-relevant processes such as transcription, RNA processing, translation, and RNA decay. In particular, the roles of RNA-binding proteins, splicing factors, RNA quality control mechanisms, and RNA degradation pathways (e.g., the exosome and nonsense-mediated decay) in neurodegenerative diseases, cancer, and developmental disorders have been extensively investigated. Yeast-based RNA studies thus contribute not only to the understanding of disease mechanisms but also to the identification of potential therapeutic targets and RNA-based interventions (Gastelum et al., 2024).

In these organisms, functional models can be generated through gene deletion, point mutagenesis, overexpression, and CRISPR/Cas-based genome editing approaches, either by manipulating yeast homologs of human genes or by directly replacing homologous yeast genes with their human counterparts. This approach, referred to as 'humanized yeast,' enables the direct assessment of the cellular effects of genetic variants through quantifiable phenotypes such as loss of function, toxicity, growth defects, stress sensitivity, metabolic disturbances, and cell cycle abnormalities (Wangler et al., 2017; Cervelli and Galli, 2021; Guaragnella et al., 2025).

The humanized yeast approach is based on the genetic reengineering of yeast to recapitulate aspects of human biology, allowing the functional testing of human genes within a cellular context. The well-characterized molecular architecture and high genetic plasticity of yeast permit the expression of human proteins in place of their yeast orthologs. The scope of this approach is not limited to elucidating gene-function relationships, but also enables the system-level investigation of the roles of human proteins within evolutionarily conserved cellular networks (Kachroo et al., 2022). In addition, growth defects or cellular stress phenotypes induced by viral or

human-derived toxic proteins can be exploited using genome-wide deletion libraries or small-molecule libraries to identify suppressor or enhancer factors. Through such studies, yeast cells also become a valuable platform for the discovery of potential therapeutic targets (Cervelli and Galli, 2021).

Functional replacement experiments have proven to be important for revealing the extent to which human genes can support essential cellular processes within the yeast cell. The ability of human genes to substitute for their yeast orthologous demonstrates the evolutionary conservation of fundamental biological processes, whereas cases in which replacement fails point to species-specific regulatory divergence or incompatibilities within protein–protein interaction networks. In this context, humanized yeast is positioned not only as a disease model but also as an experimental platform for exploring the evolutionary boundaries of human biology (Kachroo et al., 2022).

Humanized yeast systems provide a powerful model for assessing the functional consequences of genetic variants associated with human diseases. In particular, quantifiable phenotypes elicited by mutations causing monogenic disorders in yeast cells offer direct functional evidence of the pathogenicity of these variants. This approach fills a significant methodological gap in diagnostic processes and rare genetic disease research by enabling the classification of variants of uncertain significance (VUS), frequently encountered in clinical genomic studies, as pathogenic or benign. In this regard, humanized yeast models emerge as a rapid, scalable, and ethically advantageous pre-screening platform that can substitute for experiments that are difficult or costly to perform in higher organisms (Kachroo et al., 2022).

The integration of humanized yeast approaches with multi-omics data represents one of the current advances that further enhance the translational potential of these systems. In this way, yeast has become a unique experimental tool that bridges classical model organisms and cellular systems in the study of human disease biology (Kachroo et al., 2022; Guaragnella et al., 2025).

An example of a ‘humanized’ model is provided by Ahammed and colleagues (2025) in their investigation of exosomopathies, a group of rare inherited disorders associated with the RNA exosome. In this study, core components of the yeast exosome were replaced with their human homologs, and the functional consequences of pathogenic or clinically ambiguous variants identified in affected individuals were assessed. The findings demonstrated that pathogenic variants induced pronounced growth defects

and disruptions in RNA metabolism in yeast cells, whereas benign variants largely preserved exosome function. These results indicate that *S. cerevisiae* is not merely a conservation-based model organism but also offers a rapid, low-cost, and reliable platform for the functional classification of human genetic variants. In particular, in the interpretation of VUS frequently encountered in clinical genomic studies, humanized yeast systems clearly illustrate the translational potential of unicellular eukaryotic disease models (Ahammed et al., 2025).

In recent studies, yeast model systems have been widely employed to investigate the molecular foundations of a broad range of human pathologies, most notably cancer, neurodegenerative diseases, mitochondrial disorders, and metabolic diseases. Owing to their evolutionarily conserved cellular processes, yeasts constitute an important model organism for dissecting the fundamental biological mechanisms underlying these diseases. In this context, protein folding and protein quality control mechanisms are linked to protein misfolding, aggregation, and proteostasis defects that contribute to the pathogenesis of neurodegenerative diseases, whereas metabolic pathways, stress responses, and the regulation of energy homeostasis facilitate the elucidation of the molecular mechanisms of mitochondrial dysfunctions and metabolic disorders. Similarly, conserved regulatory networks governing cell cycle control and proliferation enable the modelling of uncontrolled cell division and genomic instability, which form the basis of cancer biology (Hoffman et al., 2015; Guaragnella et al., 2025). In the following sections, representative studies conducted in recent years using yeast model systems are presented, grouped by cellular mechanisms and associated disease processes.

Modelling Protein Homeostasis Dysregulation and Neurodegenerative Disease

Disruptions in the folding, quality control, and degradation mechanisms responsible for maintaining protein homeostasis play a central role in the pathogenesis of neurodegenerative diseases. Neurodegenerative disorders are generally characterized by an increased propensity for protein misfolding and aggregation within cells. Experimental modelling of these processes is therefore of critical importance for understanding disease-specific molecular mechanisms. Numerous studies have employed heterologous expression of human proteins associated with

neurodegenerative diseases in yeast, including α-synuclein (Parkinson's disease), polyQ-expanded huntingtin (Huntington's disease), and TDP-43/FUS (amyotrophic lateral sclerosis, ALS). All of these proteins induce cellular toxicity due to their aggregation-prone nature and their capacity to disrupt protein homeostasis. Yeast systems enable the assessment of the regulatory roles of protein aggregates, molecular chaperones, and autophagic processes in modulating toxicity (Popova et al., 2015; Hofer et al., 2018; Sai Swaroop et al., 2023).

The role of protein homeostasis disorders in the pathogenesis of neurodegenerative diseases has been elucidated in detail through amyloid-β (Aβ) toxicity models developed using *Saccharomyces cerevisiae*. To investigate this role, Chen and colleagues (2020) modelled the toxicity induced by the Alzheimer's disease–associated amyloid-β (Aβ) peptide in *S. cerevisiae*. Expression of Aβ in yeast cells led to protein aggregation, increased oxidative stress, and metabolic imbalances. The authors reported that flavin mononucleotide (FMN), a riboflavin-derived cofactor, significantly alleviated this toxicity. It has been shown that the protective effect of FMN occurs not through the direct inhibition of Aβ aggregation, but rather through the improvement of cellular redox balance and the regulation of energy metabolism (Chen et al., 2020; 2022).

Randez-Gil et al. (2020) employed a *Saccharomyces cerevisiae*–based humanized yeast model to investigate the cellular regulation of tau proteinopathies. Expression of the human tau protein in yeast revealed a close association between tau phosphorylation levels and cellular lipid metabolism. In particular, alterations in signaling pathways linked to sphingolipids and inositol phosphates were shown to directly influence the phosphorylation status of tau. This study demonstrates that, in tau proteinopathies, not only protein kinases but also the cellular metabolic state plays a decisive role (Randez-Gil et al., 2020).

In another study, heterologous expression of the tau protein was also performed in *Schizosaccharomyces pombe*, and this model demonstrated that under distinct metabolic conditions induced by nutrient deprivation, changes in tau phosphorylation status and the induction of cellular stress responses occur (Yılmazer et al., 2025).

Nogueira et al. (2022) employed *Saccharomyces cerevisiae* as a model system to investigate how the N88S mutation in the *seipin* gene, which causes autosomal dominant motor neuron diseases known as seipinopathies, leads to cellular damage. Expression of the mutant human seipin protein in yeast cells induced endoplasmic reticulum stress, which was shown to

directly trigger oxidative damage. Using the yeast model, the relationship between ER stress and oxidative damage was delineated, and the impact of these processes on cellular viability was examined (Nogueira et al., 2022).

Magalhães et al. (2024) generated an ALS proteinopathy model in *Saccharomyces cerevisiae* by expressing wild-type and ALS-associated mutant forms of the human SOD1 protein and investigated the protective effects of trehalose. Expression of mutant SOD1 in yeast cells was shown to induce protein aggregation, increased oxidative stress, and reduced cellular fitness, whereas trehalose treatment markedly reduced aggregation and significantly improved cellular viability (Magalhães et al., 2024).

Similarly, de Holanda Paranhos et al. (2024) employed humanized *Saccharomyces cerevisiae* cells in which the endogenous Cu/Zn superoxide dismutase (SOD1) was replaced with its human ortholog to investigate the role of human SOD1 (hSOD1) in the regulation of aerobic glycolysis and its implications for amyotrophic lateral sclerosis (ALS), a neurodegenerative disease. By examining the effects of the familial ALS–associated A4V SOD1 mutant on cellular metabolism, the authors demonstrated that SOD1 proteinopathy is associated not only with protein aggregation but also with dysregulation of metabolic control. Their findings indicate that SOD1 mutations in ALS pathogenesis are linked not only to toxic protein accumulation but also to impaired regulation of energy metabolism (de Holanda Paranhos et al., 2024).

Yeast models based on the heterologous expression of viral capsid proteins have also been developed. Owing to their high structural complexity, propensity for misfolding, and extensive potential for interactions with cellular components, viral capsid proteins act as potent triggers of proteotoxicity and aggregation. Expression of viral capsid proteins in yeast has been shown to activate the ubiquitin–proteasome system, autophagy, and endoplasmic reticulum stress responses, accompanied by the accumulation of reactive oxygen species and mitochondrial dysfunction. These phenotypes closely overlap with early cellular events described in human neurodegenerative diseases (Martynova et al., 2018; Bayandina and Mukha, 2023).

When considered together, these studies demonstrate that yeast model systems can constitute an effective and controllable experimental platform for investigating conserved processes associated with neurodegenerative diseases, including protein homeostasis, cellular stress responses, redox balance, and metabolic networks. By focusing on the cellular consequences of proteinopathies rather than neuronal effects, these approaches contribute

to the elucidation of the molecular mechanisms underlying disease and enable the rapid and scalable pre-screening of potential therapeutic compounds.

Modelling Metabolic and Mitochondrial Disorders

Yeasts offer significant advantages for elucidating fundamental mitochondrial processes, including respiration, mitochondrial biogenesis, mitophagy, and oxidative stress responses. The high degree of conservation of respiratory and oxidative phosphorylation pathways, as well as mitochondrial genes and proteins, enables the functional analysis of genetic defects associated with human mitochondrial diseases in yeast models. When findings obtained from yeast systems are integrated with human cell models, the biological and disease-related consequences of mitochondrial dysfunction become more understandable (Ždralević and Giannattasio, 2022).

Mitochondrial diseases comprise a group of disorders arising from defects in core mitochondrial functions, including oxidative phosphorylation, mitochondrial protein import, and mitochondrial translation. A substantial proportion of these diseases result from mutations in nuclear genes encoding mitochondrial functions rather than in mitochondrial DNA (Thompson et al., 2020). Insufficient energy production leads to pronounced clinical phenotypes, particularly in tissues with high metabolic demands.

Saccharomyces cerevisiae provides a powerful unicellular eukaryotic system for modelling human mitochondrial diseases due to the high degree of conservation of mitochondrial biogenesis and respiratory pathways. The ability to model nuclear gene mutations in yeast homologs or to replace them with human genes enables functional testing of variants identified in clinical studies. Moreover, the ease with which respiratory defects in yeast cells can be correlated with readily observable growth phenotypes renders this system a rapid and reliable validation platform (Ceccatelli Berti et al., 2021).

The direct relationship between mitochondrial dysfunction and protein homeostasis, as well as cellular stress responses, has become increasingly clear. Studies based on integrated multi-omics analyses have demonstrated that impairments in oxidative phosphorylation modulate pathological processes such as protein aggregation. In an ALS-related study conducted using a yeast model, alterations in mitochondrial respiratory capacity were shown to influence levels of protein aggregation (Sai Swaroop et al., 2023).

Studies have demonstrated that human variants in genes associated with mitochondrial biogenesis, DNA repair, and mitochondrial dynamics, such as *POLG*, *OPA1*, and *MFN2*, can be modeled and functionally tested in their yeast homologs. These variants have been shown to induce respiratory defects, reduced ATP production, and alterations in mitochondrial morphology in yeast cells (Ceccatelli Berti et al., 2021).

Maffezzini et al. (2019) demonstrated that mutations in the mitochondrial tryptophanyl-tRNA synthetase gene (WARS2) cause a neurometabolic disorder characterized by growth retardation and progressive leukoencephalopathy. In their study, the authors linked the molecular basis of the disease to defects in mitochondrial translation by elucidating how impaired mitochondrial protein synthesis affects oxidative phosphorylation capacity and cellular energy homeostasis. They employed a yeast model for the functional analysis of WARS2 mutations. Owing to the high degree of conservation of mitochondrial aminoacyl-tRNA synthetases in *Saccharomyces cerevisiae*, disease-associated variants of the human WARS2 gene were tested in the yeast system. These pathogenic mutations were shown to cause respiration-dependent growth defects and mitochondrial dysfunction in yeast cells (Maffezzini et al., 2019).

Boonekamp et al. (2022) demonstrated that an entire human metabolic pathway can be functionally reconstituted in a unicellular eukaryote by replacing all enzymes of the glycolytic pathway in *Saccharomyces cerevisiae* with their corresponding human enzymes. The human glycolytic enzymes functioned properly in yeast cells and were able to sustain cellular energy production. Despite certain regulatory differences, yeast cells were capable of adapting to this newly established metabolic structure (Boonekamp et al., 2022).

Vignogna et al. (2022) employed a *Saccharomyces cerevisiae*-based model to investigate diseases associated with phosphomannomutase (PMM) deficiency in humans. Yeast mutants mimicking human PMM2 deficiency exhibited severe growth defects and impaired cellular functions. Under long-term culture conditions, these cells were shown to undergo evolutionary adaptation (evolutionary rescue), developing secondary genetic changes that compensated for the metabolic imbalances caused by PMM deficiency. These findings demonstrate that yeast provides a powerful model for understanding the mechanisms underlying phenotypic variability and partial recovery observed in human metabolic diseases. Moreover, they highlight the capacity of simple eukaryotic systems to yield important insights into cellular adaptation and compensatory pathways in response to disease-

associated metabolic defects. In this context, yeast models can also be utilized in experimental evolution to identify genes and pathways that compensate for alleles associated with human disease (Vignogna et al., 2022).

Model organisms with extensively characterized metabolism, such as *Saccharomyces cerevisiae*, constitute important systems for understanding the organization of intracellular metabolic reactions and cellular responses to environmental or genetic perturbations (Nielsen and Petranovic, 2025). Genome-scale metabolic models developed in yeast enable the prediction of the effects of specific genes or pathways on cellular metabolism; this approach contributes both to the elucidation of fundamental biological processes and to the interpretation of metabolic disruptions associated with metabolic diseases.

These studies demonstrate that yeast provides a powerful platform for evaluating the functional effects of genetic variants associated with mitochondrial function and metabolism in a rapid, reliable, and controllable experimental system, prior to transitioning to complex mammalian models.

Genome Stability and Cell Cycle Dysregulation: Modelling Cancer-Related Mechanisms

In cancer genomics studies, the functional impact of many identified gene variants remains uncertain, representing a major bottleneck in clinical interpretation. Owing to the high degree of conservation of pathways involved in cell cycle regulation, DNA repair, and genome stability, *Saccharomyces cerevisiae* has been widely used for the functional analysis of cancer-associated genetic variants. Beyond its utility in variant characterization, yeast has also played a pivotal role in the discovery of fundamental cellular mechanisms, including those regulating the cell cycle. High Mobility Group Box (HMGB) proteins constitute a highly conserved family of proteins involved in essential nuclear processes such as chromatin organization, DNA bending, transcription, replication, and DNA repair. The first functional characterizations of this protein family were carried out in simple eukaryotic model systems such as *Saccharomyces cerevisiae*, and these foundational studies continue to underpin contemporary cancer research (Lamas-Maceiras et al., 2023). While yeast-based studies of cell division are of historical significance, they remain an indispensable resource for modern cancer biology and translational research.

Point mutation analyses conducted in yeast homologs functionally related to the human tumor suppressor genes *BRCA1/2*, *TP53*, and *RAD51* have elucidated the cellular effects of these variants under DNA damage responses and replication stress. Growth defects, synthetic lethal interactions, and DNA damage sensitivity phenotypes observed in yeast cells provide valuable insights into the pathogenic effects of cancer-associated genetic variants at the cellular level (Cervelli et al., 2020).

Lee and colleagues (2015) evaluated the functional consequences of rare variants identified in human genes involved in the homologous recombination (HR) pathway, which plays a central role in the maintenance of genome stability, using a *Saccharomyces cerevisiae* model. Specific point mutations in DNA repair genes associated with cancer predisposition were modelled in yeast homologs, and the effects of these variants on the repair of DNA double-strand breaks, tolerance to replication stress, and cell viability were systematically analyzed. Impaired DNA damage sensitivity and reduced recombination efficiency revealed that certain variants reduce homologous recombination capacity, thereby weakening genome stability (Lee et al., 2015).

In another study focusing on DNA repair genes, a *Saccharomyces cerevisiae*–based experimental model was developed to assess the functional consequences of single-nucleotide polymorphisms (SNPs) identified in human DNA repair genes that are critical for genome stability. Cancer-associated variants across different DNA repair pathways were tested in the yeast model, and their effects on DNA damage responses, repair efficiency, and cell viability were systematically examined. Defects in DNA damage sensitivity and cellular stress tolerance demonstrated that certain SNPs reduce DNA repair capacity, indirectly impair cell cycle checkpoint control, and promote genomic instability (Kim et al., 2018).

Brown and Andrews (2021) described an innovative ˈmolecular trapˈ approach using *Saccharomyces cerevisiae* to elucidate the mechanisms of action of anticancer compounds targeting processes associated with genome stability and cell cycle regulation. In this strategy, the functional capacities of proteins involved in DNA replication, chromatin organization, and cell cycle checkpoints were deliberately constrained in yeast, enabling the effects of small molecules on these systems to be monitored through sensitive phenotypes. Growth defects and synthetic lethal interactions observed in these genetically sensitized yeast strains made it possible to identify the direct or indirect targets of anticancer drugs within pathways responsible for maintaining genome stability (Brown and Andrews, 2021).

Dolan and colleagues (2023) performed a genome-wide high-throughput screen in *Saccharomyces cerevisiae* to elucidate the genetic determinants underlying cancer-associated genome damage induced by environmental carcinogens. In their study, resistance and sensitivity profiles of yeast mutants exposed to the heterocyclic amine carcinogen 2-amino-3-methylimidazo[4,5-f]quinoline (IQ), commonly found in cooked meats, were systematically analyzed. The results revealed that numerous genes involved in DNA repair, DNA replication, chromatin organization, cell cycle control, and oxidative stress responses contribute to the cellular response to IQ-induced toxicity. Notably, a substantial proportion of the identified genes have human orthologs previously implicated in colorectal cancer, highlighting a direct link between exposure to environmental carcinogens, disruption of genome stability, and the molecular mechanisms that increase cancer risk (Dolan et al., 2023).

Coorey and colleagues (2025) investigated the cellular effects of the inhibitor methylthioadenosine-DADMe immucillin-A (MTDIA), which targets methylthioadenosine phosphorylase (MTAP)—an enzyme whose loss or dysregulation has been reported in multiple cancer types—by expressing human MTAP in *Saccharomyces cerevisiae*. Inhibition of human MTAP activity by MTDIA was shown to induce metabolic stress in yeast cells, which in turn triggered a cell cycle–associated autophagic response. These findings demonstrate that inhibition of metabolic enzymes can influence not only proliferative capacity but also cellular stress responses and survival mechanisms, thereby exerting indirect yet critical effects on genome stability and cell cycle regulation in cancer cells (Coorey et al., 2025).

Yeast models can also be used to identify cellular signalling pathways that may serve as therapeutic targets. RAS homologs in yeast regulate fundamental processes such as cell cycle progression, nutrient sensing, stress responses, and the maintenance of genome stability, and these functions exhibit a high degree of functional similarity to those of human RAS oncogenes. Disruption of RAS signalling in yeast models has been shown to lead to bypass of cell cycle checkpoints, impairment of DNA damage response mechanisms, and the development of genomic instability (Cazzanelli et al., 2018).

Components of the PI3K–PTEN–Akt signaling pathway, which plays a central role in regulating proliferation, cell growth, and survival in human cells, have also been heterologously expressed in *Saccharomyces cerevisiae*, resulting in the establishment of simplified yet functional signaling networks. In models developed by different research groups, PI3K activity

led to the accumulation of phosphoinositides at the yeast plasma membrane and subsequent activation of Akt. In contrast, expression of the tumor suppressor PTEN attenuated this signaling cascade, reduced Akt activity, and demonstrated that negative regulatory mechanisms described in human cells can be preserved within the yeast system. Collectively, these studies indicate that key regulators of complex signaling pathways can be analyzed independently of multicellular organization (Coronas-Serna et al., 2020).

Defects in epigenetic regulation also contribute significantly to cancer development. Studies using yeast models have demonstrated how specific histone mutations disrupt epigenetic regulation and the resulting cellular consequences of these alterations. Owing to the high evolutionary conservation of the core functions of histone proteins, yeasts provide a powerful and tractable platform for dissecting cancer-associated epigenetic dysregulation in a simplified and interpretable model system. Mutations in genes encoding histone proteins that drive oncogenesis have been identified, leading to the expression of so-called oncohistones. In the human genome, histone genes are present in multiple copies, and to date, histone H3 isoforms represent the most frequently identified oncohistone variants. In contrast to humans, budding and fission yeasts include only two and three histone H3 genes, respectively. Moreover, yeast histones share approximately 90% sequence similarity with the human H3 protein. This genetic simplicity, together with their strong evolutionary conservation, makes yeast an excellent model for the functional characterization of oncohistones (Zhang et al., 2023).

Taken together, these studies demonstrate that yeast and humanized yeast models constitute powerful, reliable, and experimentally accessible systems for the functional evaluation of cancer-associated gene variants involved in cell cycle control, DNA repair, and genome stability, as well as for the systematic analysis of gene–environment interactions and the assessment of the cellular effects of metabolism-based anticancer strategies.

Limitations of Yeast Models

Although yeast is widely used as a model organism due to its ease of genetic manipulation, short generation time, and highly conserved core cellular processes, it also possesses certain structural and biological limitations. First, as a unicellular organism, findings obtained from yeast must be interpreted with caution in terms of their direct physiological relevance. As a single-cell model, yeast cannot reflect phenotypes arising

from cell–cell interactions, tissue-specific signalling, or organism-level homeostatic mechanisms. Consequently, it provides a limited model for the functional analysis of genes involved in processes that require multicellularity, such as development, differentiation, immune responses, and tissue specificity.

In addition, it should be considered that some human proteins expressed in model organisms may not fully reflect the functionality they exhibit in their native cellular context in human cells. One of the main reasons for this limitation is that certain protein variants interact with proteins that are absent in yeast or have diverged substantially over evolutionary time, or that such interactions cannot be faithfully reconstituted in the yeast cellular environment. Moreover, some variants may influence protein expression levels or activity by affecting alternative transcript usage or by altering binding sites for microRNAs or mRNA-binding proteins. Such transcriptional and post-transcriptional regulatory mechanisms can be insufficiently represented in yeast systems that rely predominantly on cDNA-based gene expression (Mohammadi et al., 2015; Bayandina and Mukha, 2023).

While yeast provides a powerful and tractable platform for functional analyses, it is important to consider that overexpression of heterologous proteins can provoke non-physiological cellular stress responses. Such effects can give rise to secondary consequences, including protein misfolding, endoplasmic reticulum stress, or perturbation of proteostasis, which may complicate the interpretation of phenotypic outcomes. For this reason, findings obtained from yeast-based functional analyses are generally strengthened when complemented by validation in mammalian cell culture systems or *in vivo* animal models, thereby enhancing their translational relevance (Cervelli and Galli, 2021; Bayandina and Mukha, 2023).

Nevertheless, the high degree of conservation of many human genes in yeast with respect to core cellular functions, together with the fact that their gene products ultimately operate at the level of individual cells, renders unicellular eukaryotic models powerful and informative systems for investigating fundamental biological processes such as DNA repair, cell cycle regulation, metabolism, and stress responses. Accordingly, yeast models continue to represent valuable research tools, particularly for elucidating the functional consequences of genetic variants, understanding their impact on cellular pathways, and performing preliminary assessments prior to transitioning to more complex model systems.

Future Perspectives in Yeast-Based Human Disease Modelling

Yeast remains one of the most accessible and experimentally versatile model systems for analyzing genotype–phenotype relationships in eukaryotic cells. Owing to its ease of genetic manipulation, short generation time, and extensively characterized cellular pathways, yeast enables systematic and comparative evaluation of the fundamental biological functions of human genes. Analysis of phenotypes arising from the expression of disease-associated human genes in yeast provides direct and quantitative insights into the functional consequences and pathogenic potential of genetic variants. In this context, yeast has emerged as a powerful platform for rapid, scalable, and cost-effective testing of variants underlying monogenic diseases, serving as an efficient pre-screening and hypothesis-generation system prior to the use of more complex cellular and animal models.

In the future, the integration of yeast-based approaches with CRISPR/Cas-mediated genome editing, high-throughput phenotyping, single-cell analyses, and computational biology tools is expected to further accelerate the functional classification of genetic variants. In particular, the continued development of humanized yeast models will enable the investigation of human proteins within yeast cells in contexts that more closely approximate physiological conditions, thereby enhancing the contribution of yeast models to elucidating the molecular bases of mitochondrial disorders, cancer, and other complex disorders. In this context, yeast is likely to remain a significant model organism for the functional interpretation of disease-associated genetic variants, the understanding of fundamental biological processes and disease mechanisms, and the identification of potential therapeutic targets.

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A Review on the Remediation of Textile Dye-Containing Wastewater

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ABSTRACT

The rapid growth of the textile industry has intensified global water pollution due to excessive water consumption and the exponential increase in dyes. The textile industry employs various dyes and chemicals during wet fabric processing, resulting in textile wastewater that contains high levels of toxic effluents and is difficult to treat. Improper management of these recalcitrant pollutants, suspended solids, and toxic metals not only diminishes the aesthetic value of water but also adversely affects aquatic habitats, marine life, and human health. The presence of these compounds in aquatic environments can prominently influence principal water quality parameters, including biochemical oxygen demand (BOD), chemical oxygen demand (COD), and pH. Additionally, untreated effluents from the textile industry can persist and accumulate in various life forms if not managed properly. They can alter food chain dynamics, resulting in widespread ecological imbalance. Therefore, sufficient textile dye treatment is a primary global environmental concern before discharging into water bodies. Selection of environmentally friendly treatments is crucial in the textile industry because conventional dyeing processes use a wide variety of chemicals and raw materials, which help minimize the negative impacts of dye-containing wastewater on the environment. Various treatment methods have been used to eliminate different contaminants and chemicals from textile wastewater, including mechanical, physical, chemical, biological, hybrid, advanced oxidation processes, membrane filtration, and nanotechnology-based approaches. This review focuses on recent advancements in methods for treating textile wastewater, highlighting their advantages and disadvantages while also considering operational costs and operating factors.

Keywords – Advanced oxidation processes, color removal, conventional methods, dyes, environmentally friendly, nanotechnology-based approaches, water treatment.

I. INTRODUCTION

The rapid expansion of the textile industry is considered a major source of pollution in modern civilization, primarily due to the excessive use of synthetic dyes and auxiliary chemicals involved in wet processing steps, such as de-sizing, scouring, bleaching, dyeing, and finishing. The improper treatment of textile wastewater, which contains residual dyes and chemicals such as ammonia, sodium, silicate, hydrogen peroxide, and surfactants, as well as heavy metals such as mercury, chromium, and lead. Dye levels above 1 mg/L in waterways, resulting from direct discharges of textile wastewater—whether treated or untreated—may cause public irritation. The release of untreated textile wastewater into water bodies blocks sunlight penetration due to its high dye content and dark color, disrupting photosynthesis and leading to oxygen deficiency. These effluents accumulate

in food chains, causing significant harm to aquatic life and human health [1-4]. Thus, developing and applying efficient treatment technologies for textile wastewater is crucial for mineralizing large amounts of highly stable organic compounds [5-7].

II. APPLIED TREATMENT METHODS FOR TEXTILE WASTEWATER

Physical, chemical, and biological methods have been utilized individually or in combination to eliminate toxic pollutants from textile wastewater. Each conventional method has specific advantages and disadvantages [8-10].

Common physical treatments for industrial textile wastewater treatment include adsorption, filtration, and ion exchange. Among these, adsorption is efficient and easy to implement for decolorizing textile dye effluents, which often contain a wide variety of dyes. Adsorption is a surface phenomenon, and the two primary types are physisorption and chemisorption. The adsorption of dye molecules can involve hydrogen bonds, van der Waals forces, electrostatic interactions, and hydrophobic interactions. Most widely used adsorbents include activated carbon, fly ash, bentonite clay, polymeric resins, ion exchangers, metal oxides, and zeolites. The adsorption process is an effective physical method for treating high dye concentrations, but it faces challenges in recovering the adsorbents, which limits scalability. Another drawback of the adsorption process is that it cannot fully degrade dye molecules [8,10-14].

Filtration is based on membrane separation methods, such as reverse osmosis, ultrafiltration, and nanofiltration, which enable the collection of reusable water and recycled dyes. Membranes facilitate the removal of hydrolyzed dye substances, reducing wastewater color, BOD, and COD. The type and porosity of the chosen membrane depend on the chemical composition of the wastewater and the specific operational temperature. This approach is preferred due to its resistance to temperature fluctuations, chemical toxicity, and microbial attacks. However, the concentrated residue left behind presents disposal challenges, including high start-up costs, clogging, and the need for membrane maintenance [8-10].

Chemical methods mainly include catalytic reduction, coagulation/flocculation, electrocoagulation, electrochemical reduction, and photolysis/photochemical reduction. Chemical treatments, such as Fenton oxidation and electrochemical processes, effectively decolorize dye molecules. However, these processes often generate secondary pollutants, including chemical sludge, complicating disposal [9,15].

Coagulation/flocculation is an effective, easy-to-operate, and low-cost method for decolorizing wastewater containing disperse dyes, while also reducing the fading of the water. It has been reported that high-molecular-weight dyes can be effectively removed using this process, which includes sedimentation, flotation, and filtration. The main drawback is its ineffectiveness in removing highly soluble, low-molecular-weight cationic

dyes. The type of coagulant used plays a crucial role in removing specific pollutants, and aluminium, iron, and their salts are frequently utilized as coagulants [15-17]. Electrocoagulation is an innovative technology that uses electric current to destabilize and eliminate contaminants while producing minimal sludge [2,18].

Biological treatment technologies can facilitate dye degradation through the adsorption of dyes by microorganisms, as well as the biodegradation of dyes by microbial cells. The biological methods represent viable, profitable, and environmentally friendly alternatives for biodegradation using aerobic, anaerobic, or combined techniques, whereas microorganisms, including bacteria, fungi, yeasts, algae, and enzymes, are utilized. In practice, to treat textile wastewater, an anaerobic process is initially employed to address COD, followed by an aerobic polishing process for the remaining low-COD wastewater. The treatment of dyes begins by breaking the azo linkages under anaerobic conditions, which produces amines. Further, the aromatic amines are decomposed into small, non-toxic molecules under aerobic conditions [9,11,16,17]. In biological treatment, it is crucial to maintain a balance of parameters such as oxygen content, temperature, pH, initial dye concentration, types of dye, and redox potential, keeping them within their optimal ranges to ensure an efficient dye reduction process [19].

Enzyme treatment can be performed at high dye concentrations and across a wide range of temperatures, pH levels, and salinity. Highly specific enzymes can effectively break down certain persistent dye molecules in wastewater [17]. The primary source of most bacteria is soil near textile factories, textile dye wastewater sludge, and areas contaminated by dye-polluted textile wastewater [9]. The most well-known method involves utilizing bacteria or fungi in combination with physicochemical processes. The benefit of using bacterial degradation is their rapid growth and ease of cultivation compared to other organisms. However, fungal treatment has been reported to be more effective than bacterial treatment due to the use of large and diverse fungal cultures. Fungi are considered versatile microorganisms that offer alternative strategies for studying biological systems related to aromatic compounds, which pose environmental issues [2,20].

Examples of physical, chemical, and biological treatment studies for textile dyeing wastewater are presented in Table 1.

Table 1. Selected physical, chemical, and biological treatment studies for textile dyeing wastewater

Physical Treatment			
Process	**Dye**	**Material**	**References**
Adsorption	Acid Blue 74, Direct Blue 80,	Activated carbon	[13]

	Methylene Blue, Crystal Violet		
Adsorption	Reactive Red 120, Reactive Orange 84, Reactive Blue 160	natural clay	[14]
Adsorption	Methylene Blue	Activated carbon	[21]
Adsorption	Cibacron reactive yellow	Modified immobilized activated alumina	[22]
Adsorption	Methylene Blue	Micro and nano-bentonite	[23]
Adsorption	Real textile wastewater	Bentonite-clay/carbon-nanotube	[24]
Adsorption	Malachite Green	para-aminobenzoic acid functionalized activated carbon	[12]
Adsorption	Methylene Blue	Fly ash	[25]
Adsorption	Reactive Orange 16	Fly ash modified magnetic chitosan-polyvinyl alcohol blend	[26]
Membrane filtration	Congo Red, Methylthionine chloride	Polypropylene	[27]
Membrane filtration	Reactive Black 5	Ultrafiltration with a ceramic membrane	[28]
Membrane filtration	Reactive Red 120, Direct Red 23	Nanofiltration membrane TFC-SR3, reverse osmosis membrane AG	[29]
Chemical Treatment			
Process	**Dye**	**Material**	**References**
Coagulation/flocculation	Reactive dye, RP-HE7B, Reactive dye, OP-HER	Moringa oleifera Lam seeds extracted from different saline solutions (NaCl and KCl))	[30]
Coagulation/flocculation	Real textile wastewater	Ferric Sulphate ($Fe_2(SO_4)_3$), Aluminium Chloride ($AlCl_3$), Aluminium Sulphate ($Al_2(SO_4)_3$) and Ferric Chloride ($FeCl_3$)	[31]
Electrocoagulation	Acid Red 336	Continuous photovoltaic	[32]

		electrocoagulation	
Biological Treatment			
Process	**Dye**	**Material**	**References**
Bacterial	Coomassie Brilliant Blue G-250, Indigo Carmine, Remazol Brilliant Blue R	*Bacillus aryabhatta*	[33]
Fungi	Acid Blue 161, Procion Red MX-5B	*Aspergillus niger, Aspergillus terreus, Rhizopus oligosporus*	[34]
Enzymatic	Methyl orange	Soybean peroxidase, *Luffa acutangula (luffa) peroxidase*	[35]
Hybrid Treatment			
Process	**Dye**	**Material**	**References**
Anaerobic biological method and chemical oxidation	Reactive Black 5, Procion Red MX-5B	Yeast extract, Catalytic wet peroxide oxidation	[36]
Bio-adsorbent and membrane technology	Real textile wastewater	Sweet orange seeds, Tubular ceramic (Al_2O_3) membranes	[37]

Advanced oxidation processes (AOPs) are an effective chemical treatment method for dealing with a diverse range of complex dyes and chemical compounds in textile wastewater. These processes rely on the generation of reactive and oxidizing free radicals, such as hydroxyl and sulfate radicals. Fenton, ozonation, heterogeneous photocatalysis, and Fenton-assisted by ultrasound are well-known methods of AOPs [38-42].

Photocatalysis is recognized as one of the most popular and promising chemical-based AOPs due to its economical, highly efficient, and environmentally friendly features. Metal oxide catalysts, especially TiO_2 and ZnO, are the most utilized photocatalysts [43-45]. Recent studies have investigated the addition of dopants to TiO_2 and ZnO to reduce their band gap energy and improve photoresponse to visible light [44,46-49]. Another strategy is preparing polymer-based composites for modifying the semiconductor surfaces to overcome rapid recombination of photogenerated charge carriers [50]. Conducting polymers, such as polyaniline, polypyrrole, and polythiophene, have been widely used to prepare novel composites based on TiO_2 and ZnO [50-52]. Numerous photocatalytic studies have been focused on synthesizing catalysts, including PANI-TiO_2, PANI-ZnO, CuO-PANI, and PANI-TiO_2-CuO composites for the treatment of textile

wastewater [51,53-56]. The selected examples of photocatalytic studies for textile dyeing wastewater are presented in Table 2.

Table 2. Selected photocatalytic studies for textile dyeing wastewater

Catalyst	Preparation Method	Dye	References
PANI-ZnO composite	*in-situ* chemical oxidation polymerization method under neutral conditions, hybridization method	Methylene Blue	[51]
PANI-TiO_2 composites	*ex–situ* chemical oxidative polymerization	Methylene Blue	[53]
PANI-TiO_2 composites	*ex–situ* chemical oxidative polymerization	Methylene Blue	[54]
PANI-CuO composite	*in-situ* chemical oxidation polymerization, mechano-chemical preparation	Reactive Orange 122	[56]
PANI-TiO_2-CuO composite	*in-situ* chemical oxidation polymerization, mechano-chemical preparation	Reactive Blue 198	[55]
self-doped yolk–shell structure TiO_2 mesospheres	solvothermal alcoholysis	Rhodamin B	[57]
hierarchical Sn-doped TiO_2 materials	sol-gel	Methylene Blue	[58]
Zn-doped TiO_2/C@SiO_2	sol-gel	Rhodamin B	[59]
three-dimensional hierarchical urchin-like hollow SiO_2@TiO_2 spheres	solvothermal	Rhodamin B	[60]
Manganese-doped ZnO	wet-chemical method	Basic Aniline, Methylene Blue	[61]
Carbon/nitrogen codoped ZnO	solvent-free mechano-thermal method	Methylene Blue, Brilliant Cresyl Blue	[62]

III. CONCLUSION

The increasing levels of contaminated dye effluents and toxic chemicals discharged into water bodies from inadequate industrial textile wastewater management are a global concern because of their highly carcinogenic nature, requiring urgent attention. This study reviews strategies for developing cost-effective and sustainable methods to treat textile wastewater generated from dyeing processes in the textile industry. Conventional methods, such as physical, chemical, and biological methods, have been discussed during this review. The challenge of recovering adsorbents limits scalability, while membrane filtration has a short lifespan. Although biological treatments have great potential, they also encounter limitations in real-world applications. Photocatalysis can be enhanced by designing new, efficient catalysts that are active under visible light. In this context, researchers have developed doped catalysts, polymer-based composites, and various hierarchical structural materials to enhance the absorption of visible light and reduce the recombination rate of photogenerated electrons and holes. Future research should focus on designing a waste-free method that minimizes risks to the environment and public health and applies from laboratories to pilot scales.

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