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Potential role of boron-containing compounds as a therapeutic adjuvant in the treatment or management of certain chronic diseases: The role Polo-like kinase 1 (PLK1) modulation

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ABSTRACT

Polo-like kinase-1 (PLK1) is a crucial serine/threonine kinase that co-ordinates cell division. It also ensures genome stability by controlling centrosome maturation, spindle assembly, chromosome segregation, and cytokinesis. Therefore, overexpression of PLK1 can disrupts numerous essential biological functions, leading to the development or exacerbation of certain chronic diseases, including cancer, diabetes, cardiovascular disorders, and neurodegenerative diseases. The overexpression of PLK1 has a multifaceted mechanism in the pathogenesis of chronic diseases. Its overexpression induces dysregulation of NF-kB, leading to inflammation. It can also instigate NLRP3 inflammasome dysregulation, possibly through multiple pathways. This study suggests that PLK1 overexpression activates Never in Mitosis Gene A (NIMA)-related kinase 9 (NEK9), and subsequently NEK7, causing NLRP3 inflammasome dysregulation. Given that PLK1 contains a nucleophile amino acid residue in its ATP-binding pocket within the kinase domain, we suggest that a substance with electrophilic properties, such as boron, could be utilized as a targeted drug. Therefore, it is mechanistically plausible for a boron-containing compound to inhibit the activity of PLK1 by forming covalent bonds or through substrate binding mechanisms with a nucleophile amino acid residue. This could potentially slow down the progression of certain chronic diseases.

Keywords: Polo-like kinase 1, Boron, Inflammation, Chronic diseases.

1. INTRODUCTION

Health is wealth. This reflects a universal truth. However, many people do not know or value how important health is to wealth. Perhaps only sick people and caregivers

can truly understand and grasp this concept. While health is not wealth in a literal sense, poor health can lead to poverty due to medical costs and the inability to work. More importantly, wealth may become insignificant if health is compromised, emphasizing the need to prioritize better health above wealth (Abdulwaliyu et al., 2024).

Many people in different part of the world are suffering from chronic diseases (Garg, 2025; Lees et al., 2023). Unfortunately, chronic diseases are interconnected, with patients having one condition at a higher risk of developing others, a phenomenon known as comorbidity. Comorbidities often worsen individual conditions, leading to a higher treatment burden, and a loss of economic output (Li et al., 2021). Furthermore, some drugs could worsen an existing medical condition (Inglis et al., 2024), and this highlights the need for appropriate drug intervention.

Therefore, the aim of this study was to investigate the potential of using Boron-containing compounds (BCC) as a therapeutic regimen in the treatment and management of certain chronic diseases by modulating Polo-like protein kinase-1 (PLK1).

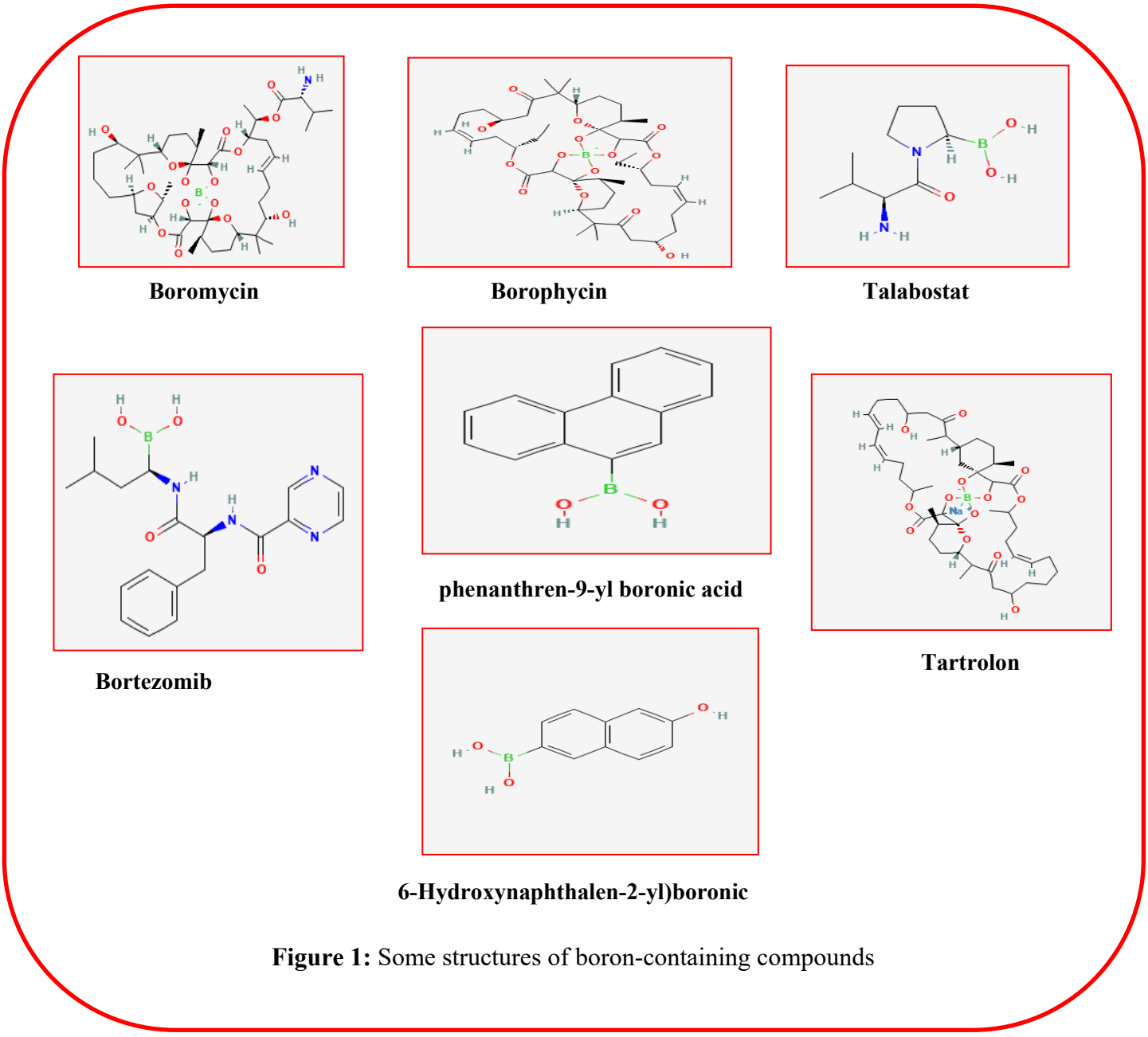

Figure 1: Some structures of boron-containing compounds

From an historical perspective, the element boron was derived from the Arabic and Persian words for borax. Boron, a metalloid in Group 13 was first isolated in 1808 (Gupta and Solanki, 2014). It is a strong electrophile. Boron can readily form covalent bonds (Parks

and Edwards, 2005) and form various compounds like borate minerals such as borax (tincal), kernite (rasorite), colemanite, and ulexite (Rendina, 2019). Boron also exists in natural form such as boromycin, borophycin, aplasmomycins, and tartrolons (Řezanka and Sigler, 2008). It can also be found in water, soil, marine algae, dairy products, and in foods such as apples, avocados, chicken breast, coffee, grapes, peanuts, raisins, and more (Khaliq, 2025).

Boron has diverse uses, particularly in the industrial, agricultural, food, and pharmaceutical sectors (Öcal et al., 2024; Sokmen and Buyukakinci, 2018). For instance, boron-based fertilizer has been used in the agricultural sector (Azaryar et al., 2024) as it serves as important ingredient for plant growth and yield (Bodeerath et al., 2024; Wu et al., 2024). It is also essential for the structural development of plant tissues (Songsriin et al., 2023).

Boron has been recognized as an important ingredient in the pharmaceutical industry and has been incorporated as key components in various drug candidates (Das et al., 2022; Fernandes et al., 2019). In fact various compounds of boron such as Bortezomib (a dipeptide boronic acid), talabostat (a methanesulfonate salt of L-valinyl-L-boroproline), asborin (a carbaborane analogue of aspirin), tavaborole (a benzoxaborole), pseudoaromatic hemiboronic naphthoids (benzoxaza- and benzodiazaborines), and Carboranes, a class of organometallic compounds containing carbon (C), boron (B), and hydrogen (H) have been used in the field of drug development. Other boron containing compounds include phenanthren-9-yl boronic acid, 6-hydroxynaphthalen-2-yl boronic acid and derivatives of borenium and borinium (Soriano-Ursúa et al., 2023). Structures of some boron containing compounds are shown in Figure 1.

Therapeutic role of boron-containing compounds (BCCs) against diseases has been revealed (Donoiu et al., 2018; Li et al., 2025). The ability of boron to inhibit specific enzymes can help slow or halt the progression of diseases (Del Prete et al., 2024; Pan and Kakeya, 2025).

Studies have reported that targeting NLRP3 inflammasome dysregulation has been a focal therapeutic target to halt inflammation and the progression of chronic diseases (Cabral et al., 2025; Karmakar et al., 2025). Furthermore, oxidative stress induces overexpression of Polo-like kinase 1 (PLK1) (Srinivas et al., 2019) and consequently increases inflammation and chronic diseases (Gheghiani and Fu, 2023). Hence, the anti-oxidant and anti-inflammatory role of boron in curtailing inflammation is critical, and has been highlighted in this study. Furthermore, its role against cancer, cardiovascular diseases, chronic kidney diseases, diabetes, and neurodegenerative diseases is underscored in this study. Additionally, this study posits that targeting Polo-like kinase 1 using boron-containing compounds may mitigate the progression of various chronic diseases.

2. REVIEW METHODS

In this study, major databases such as PubMed, Scopus, Google Scholar, and the Directory of Open Access Journals were searched to obtain relevant information. Key words and combined keywords such as Polo-like kinase-1 and biological function, Polo-like kinase-1 and inflammation, overexpression of Polo-like kinase-1 and chronic diseases, boron and Polo-like kinase-1, boron and inflammation, boron and cancer, boron and cardiovascular diseases, boron and diabetes, boron and chronic kidney disease, boron and neurodegenerative disease, were used.

3. RESULTS & DISCUSSION

Polo-like kinase 1 (PLK1) dysregulation: Implication for chronic diseases

Polo-like kinase 1 (PLK1), also known as serine/threonine-protein kinase 13 (STPK13), is a crucial regulator of the cell cycle (Colicino and Hehnly, 2018). It plays important roles in DNA damage response and phosphorylates multiple substrates to regulate events such as mitotic entry, chromosome condensation, spindle formation, and cytokinesis. And dysregulation of PLK1 may cause cancer, making it a promising target for anti-cancer therapies. Additionally, the overexpression of PLK1 may induce inflammation and chronic diseases.

Polo-like kinase 1 and asthma

PLK1 dysregulation may cause asthma through proliferation and contraction of airway smooth muscle cells ((Pan et al., 2025). It has been reported that microRNAs are downregulated in asthma (Hernández-Díazcouder et al., 2024) and the downregulation of the microRNAs in asthmatic episodes may be associated with PLK1. However, the relationship between PLK1 and microRNAs is complex. While PLK1 expression may be suppressed by microRNAs (Xu et al., 2016), PLK1 regulates the biogenesis of a specific subset of miRNAs (Fletcher et al., 2023).

A study suggests that microRNAs may contribute to asthma by increasing PLK1 expression and activates ERK1/2 pathway (Liao et al., 2018) (Figure 2). In fact, the relationship between activation of the ERK1/2 pathway and asthma has been documented in studies (El-Hashim et al., 2017; Shah et al., 2023). The ERK1/2 plays a significant role in the developments of Th2 cells (Ohnishi et al., 2009). Th2 cells derive the immune response in many asthma cases (Harker and Lloyd, 2023). Therefore, the involvement of PLK1 in asthma may be associated with activation of ERK1/2 and subsequent stimulation of Th2 cells.

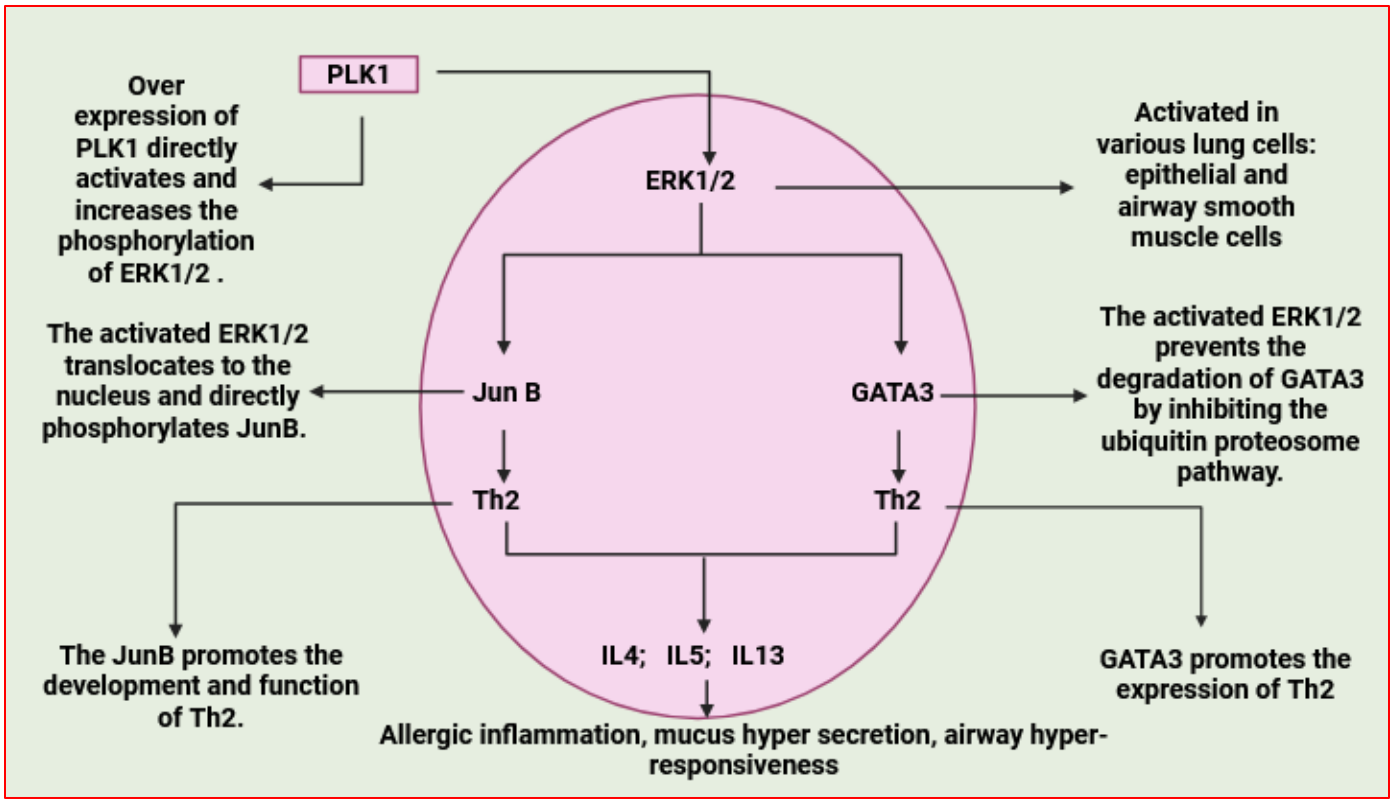

Figure 2: Illustration showing the role of PLK-1 overexpression in the pathogenesis of asthma. ERK1/2 – Extracellular signal regulated kinases 1 and 2; PLK-1 – Polo-like Kinase 1; Jun B – Jun B proto – oncogene, AP-1 transcription factor subunit. GATA 2 - Guanine – adenine – thiamine – adenine 2 binding factor 2.

PLK1 and cardiovascular disease

The relationship between PLK1 and cardiovascular disease is complex. A decrease in PLK1 levels has been observed during cardiac myocyte development (Coxon et al., 2009). On the contrary, an increase has been found to reduce ischemia reperfusion-induced myocardial damage (Mao et al., 2021). Furthermore, the levels of PLK1 have been found to decrease in atherosclerosis mice model (Hu et al., 2022). Overexpression of PLK1 has been found to be associated with excessive growth of pulmonary artery smooth muscle cells (PASMCs), and narrowing of the pulmonary arteries (Chen et al., 2023). It can also cause hyperplastic growth, as evidence in vascular smooth muscle cells of patients with pulmonary arterial hypertension (Wilson et al., 2019). Polo-like kinase 1 (PLK1) expressed in smooth muscle cells (SMCs) has been shown to play a role in regulating contraction (Moraes et al., 2025). It has been reported that sustained contraction coupled with thickening of the blood vessels in the lungs contribute substantially to pulmonary hypertension (Antigny et al., 2020), a risk factor for heart failure (Itelman et al., 2021). A study suggests that PLK1 overexpression may activate the NF-κB signaling pathway (Gao et al., 2023), a crucial transcription factor involved in inflammation and immune responses. Dysregulation of the NF-κB signaling pathway may lead to atherosclerosis, hypertension, and heart failure (Matsumori, 2023).

PLK1 and Lupus

Polo-like kinase 1 (PLK1) overexpression may be associated with lupus through Aurora-A/PLK1/mTOR signaling pathway. Since PLK1 may contribute to the pathogenesis of lupus via the Aurora-A/PLK1 signaling cascade, Wu et al. (2018) and Lindblom et al. (2023) suggest that it could be a therapeutic target for systemic lupus.

PLK1 and Diabetes

The expression of PLK1 may be elevated in diabetic states through thrombin-induced endothelial dysfunction, as demonstrated in experimental Type-2 Diabetes Mellitus (T2DM) (Hao et al., 2018). The underlying mechanism involves transcriptional activation and overexpression of PLK1 due to thrombin-induced overexpression of Krüppel-like factor 14 (KLF14). KLF14 plays a significant role in glucose and lipid homeostasis (Yang et al., 2015).

Just as Krüppel-like factor 14 (KLF14) plays a significant role in glucose regulation, phosphatidylinositol 3-kinase (PI3Ks) also play an important role in this process, with the possibility of being involved in the onset of diabetes mellitus (Maffei et al., 2018) potentially mediated by PLK1. It has been observed that insulin signaling activates the PI3K/AKT pathway, leading to the upregulation of PLK1. This upregulation may result in type 2-diabetes (Wu et al., 2023).

PLK1 and chronic kidney disease

The role of PLK1 in the pathogenesis of CKD may be attributed to its ability to regulate ATPase H^+ transporting V1 subunit A (ATP6V1A) phosphorylation (Du et al., 2023). ATP6V1A maintains the acid-base balance in the kidneys, and if compromised, it could lead to severe consequences for CKD progression (Wesson et al., 2020).

Phosphoglycerate kinase 1 (PGK1) plays a significant role in the pathogenesis of CKD. It inhibits glutathione peroxidase 1 (GPX1) and activates the NLRP3 inflammasome (Sun et al., 2025). Mechanistic insights suggest that the activity of the PGK1 in the pathogenesis of CKD is regulated by PLK1 (Jiang et al., 2025).

Involvement of PLK1 in the pathogenesis of nephronophthisis has been reported in a study. The study showed that PLK1 can phosphorylate the Nephronophthisis 1 gene (NPHP1), which is the most common cause of nephronophthisis (Seeger-Nukpezah et al., 2012), and the most common genetic cause of chronic kidney disease (CKD) among children and can ultimately lead to end-stage renal disease (Shah et al., 2025).

PLK1 and Cancer

Polo-like kinase 1 (PLK), is often overexpressed in many types of cancer (Weiß and Efferth, 2012). In fact, the overexpression of Polo-like kinase 1 in several cancer types and poor response to treatment makes it a unique target for cancer treatment (Feng et al., 2023). The overexpression of PLK1 in cancer compromises cell cycle checkpoints, resulting in genomic instability and tumor formation (Cunningham et al., 2020).

During cellular stresses, such as DNA damage, PLK1 is phosphorylated and inactivated by ataxia telangiectasia mutated (ATM), leading to arrest at the G2/M phase. ATM requires the phosphorylation of p53 at the key regulatory site, Ser15 (Smith et al., 2017). One of the p53's functions as a tumor suppressor is to maintain the G2/M checkpoint, and also provides protection against tumor development (McKenzie et al., 2010). Therefore, p53 and PLK1 often exhibit an opposing relationship. While PLK1 can inhibit p53 function, the loss of p53 in cancer cells could elicit higher PLK1 expression (Jung et al., 2021).

PLK1 and neurodegenerative diseases

Polo-like kinase1 has been observed to be highly expressed in spinal motor neurons which correlate greatly with the loss of nuclear fused in Sarcoma (Szewczyk et al., 2025). Increased PLK1 overexpression has been observed in the brains of AD patients, as evidenced by the increased phosphorylation signal of p150Glued at Ser179 starting at interphase (Song et al., 2011). This phosphorylation event may facilitate nuclear envelope breakdown at the prophase of the cell cycle. The breakdown of the nuclear envelope compromises nuclear integrity and has been linked to neurodegenerative diseases through the disruption of nucleocytoplasmic transport (Hachiya et al., 2021).

Inhibition of PLK1 has been found to induce cell apoptosis and DNA damage in glioma stem cells (GSCs) through the phosphorylation of Y-box-binding protein 1 (YBX1) (Li et al., 2023). YB-1 has the ability to enter the brain, and has been shown to inhibit the formation of toxic β-amyloid fibrils, improved memory and neuron morphology in animal models of Alzheimer's (Bobkova et al., 2015). Over expression of PLK-1 is also associated with Parkinson's disease (PD) through its interactions with alpha-synuclein and Parkin (Dzamko et al., 2014).

PLK1 overexpression and inflammation

The overexpression of PLK1 has a multifaceted mechanism in the pathogenesis of chronic diseases. Its overexpression induces dysregulation of NF-kB, leading to inflammation (Figure 3). It can also instigate NLRP3 inflammasome dysregulation, possibly through

multiple pathways. However, this study suggests that, PLK1 overexpression activates NEK9, and subsequently NEK7 activation, causing NLRP3 inflammasome dysregulation (Figure 3). NEK9 has been identified as a kinase activated by PLK1 through a two-step process involving an initial phosphorylation by CDK1. CDK1 phosphorylates Nek9 at multiple sites, creating a binding site for PLK1's polo-box domain (PBD), and sets the stage for PLK1 to activate NEK9 (Bertran et al., 2011). The phosphorylation of NEK9 directly impacts the activation of other kinases such as kinase NEK7 through NEK7's C-terminal domain (Belham et al., 2003).

NEK7 is essential for the assembly and activation of the NLRP3 inflammasome, which means that, the dysregulation of NEK7 can lead to NLRP3 inflammasome dysregulation, and consequently, excessive inflammatory responses and inflammatory diseases (Xiao et al., 2025).

While dysregulation of the NLRP3 inflammasome plays a significant role in inflammatory events, there are many pathways that can lead to inflammation independently of the inflammasomes. The Nuclear Factor kappa-light-chain-enhancer of activated B cell (NF-kB) pathway is another pathway that mediates inflammatory events. Additionally, PLK1, a known mediator of NLRP3 inflammasome dysregulation, may also mediate inflammation through the PLK1/PARP10/NF-kB pathway. This suggests that targeting PLK1 may mitigate be more inflammatory diseases, associated with both NLRP3 and the NF-kB deregulations.

Effects of boron-containing compounds on certain chronic diseases

Targeting PLK1 with boron-containing compounds could have a multifaceted impact on mitigating the progression of chronic diseases. Even more intriguing is the speculation that targeting PLK1 could also prevent comorbidity. Boron compounds are primarily electrophiles with an incomplete octet and a vacant p-orbital. This allows them to form a covalent bond with the oxygen atom in an OH group of the target substrate, blocking the OH group from interacting with a kinase enzyme or phosphate. Additionally, compounds containing boric acid may also inhibit the active site by binding to catalytic serine or threonine residues. This action can block the catalytic function of PLK1, preventing it from phosphorylating its target substrate. Targeting PLK1 with boron-containing compounds may slow down abnormal expression or activity, genomic instability, chromosomal defects, tumorigenesis, and inflammatory diseases.

This study suggests that, the effect of boron-containing compounds on PLK1 may prevent downstream activation of NEK9 and NEK7, consequently mitigating NLRP3 inflammasome dysregulation and inflammation (Figure 3). Although information on the effects of boron-containing compounds on PLK1 is limited, its role in the NLRP3 inflammasome has been studied. A boron-containing compound, 2-aminoethoxy diphenylborinate, has been shown to inhibit the NLRP3 inflammasome (Baldwin et al., 2017). Similarly, a synthetic boron compound, Oxazaborine, has been shown to inhibit the NLRP3 inflammasome (Baldwin et al., 2018).

This study suggests that the effects of boron-containing compounds on the NLRP3 inflammasome may be associated with the PLK1/NEK9/NEK7 pathway. Specifically, because PLK1 promotes NEK9 phosphorylation and the subsequent activation of NEK7 enhances the NEK7-NLRP3 binding, a critical step for inflammasome assembly (Sharif et al., 2019). It has been reported that targeting NEK7 can disrupt its interaction with NLRP3 complex and mitigate inflammation (Ni et al., 2025). Targeting PLK1 may also retard the interaction between NEK and NLRP3, and may offset the risk of inflammation. A boron-containing compound (ZCL-082), and boron nitride nanotubes functionalized with curcumin have been shown to down-regulate the NF-κB pathway (Ma et al., 2022; Mansoori et al., 2024). Similar observation has been reported for boron and selenium (Cengiz et al., 2024). The effects of boron on NF-κB signaling may be mediated through the regulation of PLK1 as shown in Figure 3.

Recent research has shown that boric acid and borax significantly reduces inflammation in the tissue and liver of male Sprague Dawley rats through the miR-21/PTEN/AKT pathway (Sevim et al., 2025). Additionally, boron-containing pyrazole compounds show potential for treating inflammation through JAK inhibition (Sabnis, 2022). The impact of boron containing compounds on oxidative stress, a known mediator of inflammation and chronic diseases has been investigated**.** Boron containing compound in low amount has been shown to improve the antioxidant system (Khaliq et al., 2018).

Boron-containing compounds may have the potential to activate and translocate Nrf2 to the nucleus. A study has shown that activated Nrf2 binds to antioxidant response elements (AREs) and produces antioxidant enzymes and other protective proteins (Tkaczenko and Kurhaluk, 2025). This indicates that boron-containing compounds may provide protection against chronic diseases like cancer, diabetes, and cardiovascular disease (CVD), primarily through anti-inflammatory and anti-oxidative mechanisms.

Boron-containing compounds and cancer

Boron containing compounds have a dual relationship with cancer. They can act as both preventive dietary agent and a key component in advanced cancer therapies, particularly Boron Neutron Capture Therapy (BNCT) (Cheng et al., 2022). Although, BNCT uses has some limitation such as insufficient tumor targeting, rapid *in vivo* metabolism, high doses due to poor solubility and accumulation (Li

et al., 2025). Miyabe et al. (2019) suggests that the use of oligopeptide transporters, particularly peptide transporter 1 (PEPT1) may enhance sufficient tumor targeting by BNCT.

Anticancer mechanism of BNCT is associated with cell cycle arrest in the G2/M phase and apoptosis via the mitochondrial pathway (Sun et al., 2013). A decrease in the quantity of cells in the G0/G1 and G2/M phases, along with a significant increase in the quantity of DNA fragmented cells following boron neutron capture therapy, has been observed (Faião-Flores et al., 2011).

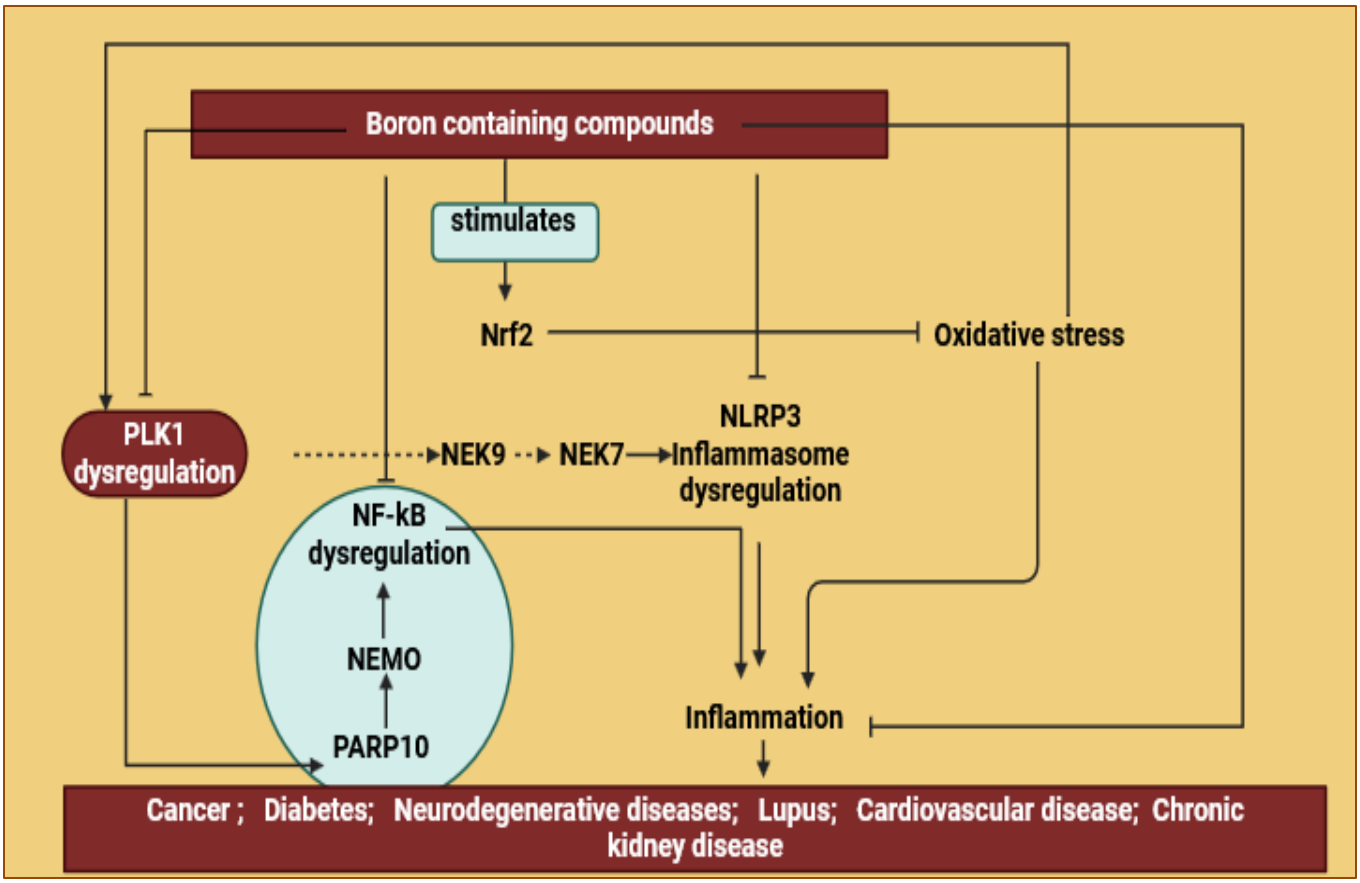

Figure 3: Schematic diagram showing the hypothetical mechanism of the role of boron containing compounds against various chronic diseases. PLK-1 – Polo like kinase-1; NrF$_2$- Nuclear factor erythroid 2- related factor 2; PARP10 – Poly [ADP-ribose] polymerase 10; NF-κB - **Nuclear** factor kappa – light – chain - enhancer of activated B cells; NLRP3 – NOD like receptor pyrin domain containing 3; NEMO - NF-κB essential modulator; NEK – NIMA related kinase 9. Created with BioRender.com.

Considering the role of PLK1 as a crucial regulator of the cell cycle, especially the G2/M phases, suggests that the cell cycle arrest by boron may be associated with its inhibition. Furthermore, boron can act as a bio-isosteric replacement for carbon, allowing its derivatives to interfere with the function of essential biological macromolecules (Hunter et al., 2009).

Studies suggest that boron glycine monoester, boron glycine diester, and hollow boron nitride spheres may be used for cancer treatments (Koldemir-Gündüz et al., 2021; Li et al., 2017). Certain enzymes including Pyrroline-5-carboxylate reductase-1 (PYCR1) are over expressed in cancer cells (Kay et al., 2022; Niu et al., 2025). For instance, PYCR1 has been observed to modulate cancer progression through the Akt/mTOR pathway (Wang and Liu, 2019). PYCR1 has been down-regulated using sodium pentaborate pentahydrate and sodium perborate tetrahydrate (Yanar et al., 2025). These enzymes are associated with PL. Boron containing compounds such as boric acid (BA), sodium pentaborate pentahydrate (NaB), and sodium perborate tetrahydrate (SPT) have been shown to induce cell cycle arrest in lung cancer cells (Cebeci et al., 2022).

Anti-diabetic properties of boron containing compounds

Studies suggest that boron-containing compounds may offer glycemic balance (Bakken and Hunt, 2003; Soriano-Ursúa et al., 2024). The anti-diabetic properties of two different boron-containing compounds, sodium pentaborate pentahydrate and boric acid, have been

investigated. They have shown a positive effect on pancreatic cells by increasing cell viability and insulin production (Aydın et al., 2019).

Expression of NAD-dependent protein deacetylase sirtuin-1 (Sirt1) and Glycogen Synthase Kinase 3 (GSK-3α/β) have been shown to improve using sodium borate decahydrate and boric (Ozanso et al., 2020). The activity of NAD-dependent protein deacetylase sirtuin-1 is often reduced in individuals with diabetes (Hassaan et al., 2025). This strongly suggests that the improved expression of the enzyme may be necessary for glycemic balance. Furthermore, Şahin et al. (2023) suggests that topical application of sodium pentaborate gel may help treat diabetic foot ulcers and prevent their recurrence (Şahin et al., 2023).

Boron-containing compounds and cardiovascular diseases

Information on the relationship between boron-containing compounds and cardiovascular disease is limited. However, boron-containing compounds may improve certain risk factors such as hypercholesterolemia, inflammation, and oxidative stress, associated with CVD (Shen et al., 2021). It may also reduce tissue degeneration and cardiac fibrosis (Hoque et al., 2025; Bouchareb et al., 2020).

Boron-containing compounds and neurodegenerative diseases

Boron-containing compounds have been shown to retard the progression of neurodegenerative diseases (Barrón-González et al., 2023) through reducing oxidative stress and inflammation. They may also reduce amyloid-beta plaque buildup (Novo et al., 2018) which contributes to neuronal damage and cognitive decline. This suggests that suppressing the aggregation of amyloid-beta may be necessary.

Boron-containing compounds have been shown to inhibit peptide's ability to form β-sheets, which are crucial in the development of Alzheimer's disease (Das et al., 2024). A study found that boron nitride nanotubes (BNNT) provide structural stability to amyloid-β (Aβ) peptides by keeping them separated (Sorout and Chandra, 2020). In the absence of BNNT, Aβ (1–42) trimer aggregates, leading to a transition from α-helix to β-sheet formation (Sorout and Chandra, 2020).

Reports from studies show that boron-containing compounds have been shown to significantly inhibit acetylcholinesterase (Cacciatore et al., 2021; Küçükdoğru et al., 2020) which plays a substantial role in the pathogenesis of neurodegenerative diseases. Therefore, the use of boron-containing compounds, in addition to existing drugs, may improve the quality of life among sufferers of neurodegenerative diseases.

Effects of boron-containing compounds on Lupus

Studies indicate that various boron compounds exhibit potential immunomodulatory and anti-inflammatory effects. This may be relevant to autoimmune diseases like systemic lupus erythematosus (SLE). For instance, borax has been shown to significantly increase thymus-derived and bone marrow-derived cells populations (Routray and Ali, 2016). Also, boric acid and calcium fructoborate have been shown to increase the expression and activity of RAR-related orphan receptor gamma t (Ror-γt) in patients with SLE (Yapar et al., 2025). RORγT plays a significant role in the development of Th17 lymphocytes which are involved in the body's defense against pathogens (Pastwińska et al., 2023). In fact, direct impact of boric acid on Treg/Th17 population in the healthy patients has been reported (Yapar et al., 2025). The improve balance of Treg/Th17 population provides significant health benefits, as it may help prevents the development of chronic, low-grade inflammation and autoimmunity.

4. CONCLUSION

It is widely observed that despite increased awareness about well-being, global health is deteriorating as the prevalence of certain illnesses, particularly chronic diseases, is rising. This may be due to a complex of issues including poor healthcare delivery and the complex nature of diseases. To overcome limitations of existing drugs, the search for new drugs for chronic diseases, especially comorbidity, has become necessary. This study investigated the potential use of boron-containing compounds against chronic diseases through the inhibition of polo-like kinase 1. Hypothetically, inhibiting polo-like kinase 1 using boron-containing compounds may mitigate inflammation through the PLK1/PARP10/NEMO/NF-kB pathway and through the PLK1/NEK9/NEK7/NLRP3 inflammasome dysregulation pathways. Effectively targeting the enzyme (using boron) that drives disease development may serve as a strategy in modern medicine to significantly mitigate the progression of certain chronic diseases.

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Author Contributions
Conceptualization – BML and IA; Methodology - SOY and RAM; Formal Analysis- IA, SOA, and AMH; Data Curation – EHO, BML, and IA; Writing – Original Draft Preparation – BML, IA, EAM, RTD, and UB; Writing –Review & Editing – BML, IA, SOY, SOA, EAM, RTD, UB, OSO, and AIB; Visualization-SOY and SOA; Supervision- RAM and IA; Project Administration-BML and IA.

Informed consent
Not applicable.

Conflicts of interests
The authors declare that they have no conflicts of interest, competing financial interests or personal relationships that could have influenced the work reported in this paper.

Ethical approval & declaration
Not applicable. This article does not contain any studies with human participants or animals performed by any of the authors.

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Data and materials availability
Data that support the findings of this study are embedded within the manuscript.

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