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SYNTHESIS, CHARECTERIZATION AND ANTI-INFLAMMATORY ACTIVITY OF SUBSTITUTED 5-(5-SULFANYL-1,3,4- OXADIAZOL-2-YL) BENZENE- 1,2,3-TRIOL DERIVATIVES

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Abstract:
Inflammation is a complex biological response of body tissues to harmful stimuli, including pathogens, damaged cells, or irritants. While inflammation is a protective mechanism, chronic inflammation is associated with various diseases, such as arthritis, cardiovascular disorders, and cancer. Several synthetic and natural compounds have been explored for their anti-inflammatory properties. Traditional nonsteroidal anti-inflammatory drugs (NSAIDs) and corticosteroids, though effective, often lead to adverse effects such as gastrointestinal toxicity, liver damage, and cardiovascular risks. As a result, there is an increasing demand for novel anti-inflammatory agents with improved safety and efficacy profiles. In this study, a series of 5-(5-sulfanyl-1,3,4-oxadiazol-2-yl) benzene-1,2,3-triol derivatives were synthesized and evaluated for their anti-inflammatory potential. The synthesis involved multiple steps, starting with preparing galloyl hydrazide from propyl gallate and hydrazine hydrate under reflux conditions. The reaction of galloyl hydrazide with carbon disulfide in an alkaline medium yielded oxadiazole derivatives, which were further functionalized using various aryl and alkyl halides in pyridine. The synthesized compounds were purified through recrystallization and characterized using thin-layer chromatography (TLC), melting point determination, FT-IR, mass spectrometry, and NMR spectroscopy.
Compared to synthetic methods, the described approach offers advantages such as higher yields, mild reaction conditions, and efficient purification. The synthesized derivatives were assessed for their anti-inflammatory activity, demonstrating promising results. These findings suggest the potential of novel oxadiazole derivatives as safer alternatives to conventional NSAIDs
Keywords: *Oxadiazole, Oxadiazole derivatives, Anti-Inflammatory activity*

1. INTRODUCTION:

Inflammation (Latin: *inflammo*, meaning "I ignite, set alight") is a complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells, or irritants. It is a protective immunovascular mechanism involving immune cells, blood vessels, and molecular mediators. The primary function of inflammation is to eliminate the initial cause of cell injury, clear necrotic cells and damaged tissues, and initiate tissue repair[1].
The classical signs of acute inflammation include pain, heat, redness, swelling, and loss of function. Inflammation is a fundamental aspect of innate immunity, providing a general defense mechanism, unlike adaptive immunity, which is pathogen-specific[2]. However, inflammation must be tightly regulated, as insufficient inflammation can lead to unchecked tissue destruction, while chronic inflammation is associated with various diseases, including fever, periodontitis, atherosclerosis, rheumatoid arthritis, and certain cancers such as gallbladder carcinoma[3].

1.1 Types of Inflammation

1. **Acute Inflammation**: This is the body's immediate response to harmful stimuli, characterized by increased movement of plasma and leukocytes (mainly granulocytes) to the affected area[4]. A cascade of biochemical events

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activates and regulates the inflammatory response, involving blood vessels, immune cells, and various cellular components.

2. **Chronic Inflammation**: Prolonged inflammation results in a progressive shift in cell types present at the site of injury, leading to simultaneous tissue destruction and repair. Chronic inflammation is often linked to severe pathological conditions.

Biological Activities of Oxadiazole Derivatives

Heterocyclic compounds, particularly 1,3,4-oxadiazoles, have gained significant attention due to their diverse biological activities [5, 6]. They exhibit antimicrobial, anti-tubercular, anti-inflammatory, anticonvulsant, hypnotic, and anesthetic properties. Oxadiazoles are structurally like well-known sulphonamide drugs and demonstrate potent antibacterial effects. The N=C-S linkage in the oxadiazole nucleus is responsible for various pharmacological activities[7,8].

Sulfone derivatives containing a heterocyclic moiety have been widely studied for their antifungal properties and have significant applications in both pesticide development and medicinal formulations. Numerous studies in the last three years have reported the synthesis and biological activities of these compounds, highlighting their importance in drug discovery and therapeutic applications[9,10].

1.2 Causes of inflammation[11]:

Table 1 Types and causes of Inflammation

Title	Physical	Chemical	Biological
Cause of Inflammation	➢ Burns ➢ Frostbite ➢ Physical injury, blunt or penetrating ➢ Foreign bodies, including splinters, dirt and debris, Trauma	➢ Infection by pathogens ➢ Immune reactions due to hypersensitivity ➢ Stress	➢ Chemical irritants ➢ Toxins ➢ Alcohol

Anti-inflammatory refers to the property of a substance or treatment that reduces inflammation or swelling. Anti-inflammatory drugs make up about half of analgesics[12],[13] remedying pain by reducing inflammation as opposed to opioids, which affect the central nervous system.

1.3 Limitations of Current Anti-Inflammatory Therapies:

Table 2 Detailed description of current autoinflammatory therapies

Sr no	Limitation	Description
1	Cardiovascular Risk[14, 15]	NSAIDs, especially COX-2 inhibitors, increase the risk of heart attack, stroke, and hypertension
2	Renal Toxicity[16, 17]	Long-term NSAID use can reduce renal perfusion, causing nephrotoxicity and electrolyte imbalance.
3	Delayed Wound Healing [18]	Corticosteroids suppress immune response and tissue repair, delaying wound healing processes.
4	Immunosuppression[19]	Corticosteroids can cause systemic immunosuppression, increasing the risk of infections.
5	Hepatotoxicity[20]	Some NSAIDs (e.g., diclofenac) are associated with liver enzyme elevations and hepatocellular injury.
6	Drug Interactions[21, 22]	NSAIDs can interfere with antihypertensives, anticoagulants, and other chronic medications
7	Tolerance and Dependency (in corticosteroids)[23]	Long-term corticosteroid use can lead to dependency and adrenal suppression upon withdrawal.

1.4 Non-steroidal Anti-Inflammatory Drugs (NSAIDs)- non-steroidal anti-inflammatory Drugs (NSAIDs) are a widely used class of medications that alleviate pain and inflammation by inhibiting the activity of the cyclooxygenase (COX) enzyme. Under normal physiological conditions[24], the COX enzyme catalyzes the conversion of arachidonic

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acid into prostaglandins, which mediate inflammatory responses such as pain, swelling, and fever. By blocking COX activity, NSAIDs effectively reduce the production of prostaglandins, thereby diminishing inflammation and pain[25]. Common examples of NSAIDs include aspirin, ibuprofen, and naproxen. In contrast, selective COX-2 inhibitors, such as celecoxib, are not classified together with traditional NSAIDs despite sharing a similar mechanism of action, as they specifically target the COX-2 isoenzyme while sparing COX-1[26, 27].

1.4 Mechanism of action:

The therapeutic efficacy of NSAIDs stems from their ability to inhibit cyclooxygenase enzymes, which exist in two major isoforms:

1. **Cyclooxygenase-1 (COX-1):** This isoenzyme is constitutively expressed in various tissues and plays a crucial role in maintaining physiological functions, such as gastric mucosal protection, platelet aggregation, and renal perfusion. Inhibition of COX-1 can lead to adverse effects, including gastrointestinal irritation, ulcer formation, and impaired renal function[28].
2. **Cyclooxygenase-2 (COX-2):** Unlike COX-1, COX-2 is primarily induced in response to inflammatory stimuli such as cytokines, growth factors, and endotoxins. It mediates the synthesis of prostaglandins that promote inflammation, pain, and fever. By selectively inhibiting COX-2, NSAIDs effectively reduce inflammation while minimizing adverse effects associated with COX-1 inhibition[29].

   Most traditional NSAIDs act as **non-selective** inhibitors, blocking both COX-1 and COX-2 isoenzymes to varying degrees. Their inhibition is **competitively reversible**, meaning their effects diminish once the drug is cleared from the system. However, aspirin, a widely used NSAID, differs in its mechanism by **irreversibly** acetylating the serine residue of the COX enzymes, leading to prolonged inhibition of prostaglandin synthesis. This irreversible action also underlies aspirin's antithrombotic properties, as it permanently prevents thromboxane A_2-mediated platelet aggregation, reducing the risk of cardiovascular events[30].

   Selective COX-2 inhibitors, such as celecoxib, were developed to retain the anti-inflammatory and analgesic benefits of NSAIDs while reducing gastrointestinal side effects associated with COX-1 inhibition. However, concerns have been raised regarding their potential cardiovascular risks due to the imbalance between prostacyclin and thromboxane A_2 levels, which may predispose individuals to thrombotic events[31,32].

1.5 Oxadiazole Linked with Natural Anti-inflammatory

The concept of hybrid molecules has emerged as a powerful approach in modern medicinal chemistry to enhance therapeutic efficacy by combining two or more pharmacophores into a single molecular entity33. Oxadiazole derivatives, particularly 1,3,4-oxadiazoles, are recognized for their significant anti-inflammatory potential due to their ability to inhibit cyclooxygenase (COX) enzymes, modulate prostaglandin synthesis, and suppress cytokine-mediated inflammatory responses. On the other hand, various natural compounds such as curcumin, flavonoids, coumarins, and phenolic acids exhibit potent anti-inflammatory effects through different mechanisms, including the inhibition of nuclear factor kappa B (NF-κB) pathway[34], reduction in pro-inflammatory cytokines like IL-1β, IL-6, TNF-α, and inhibition of oxidative stress mediators such as nitric oxide (NO) and reactive oxygen species (ROS)[35]. Hybrid molecules incorporating an oxadiazole core with a natural anti-inflammatory scaffold can potentially offer synergistic effects, leading to superior pharmacological profiles. Such conjugates not only provide enhanced bioavailability and dual-site activity but may also exhibit reduced side effects compared to traditional non-steroidal anti-inflammatory drugs (NSAIDs)[36],[37]. For instance, curcumin–oxadiazole hybrids have demonstrated increased COX-2 inhibitory activity, improved stability, and better free radical scavenging properties. Similarly, flavonoid–oxadiazole hybrids have shown enhanced anti-edematous effects and antioxidant potential, Coumarin–oxadiazole conjugates are also being studied for their dual inhibition properties against COX and lipoxygenase (LOX) enzymes[38,39]. These hybrids are generally synthesized through molecular fusion or linker-based approaches, ensuring structural compatibility and retention of bioactivity[40,41]. The design of such hybrid molecules is increasingly supported by structure-activity relationship (SAR)

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studies, molecular docking simulations, and pharmacokinetic evaluations[42]. Hence, oxadiazole-based hybrid molecules with natural scaffolds represent a promising class of multi-target anti-inflammatory agents, warranting further exploration in drug development programs[43].

2 MATERIAL & METHODS:

Materials chosen for synthesis are Oxadiazole Moiety, Substituted Phenyl Ring at the 2nd Position, Substituted Phenyl Ring at the 5th position (substitution) and 'Sulphur bridge'.

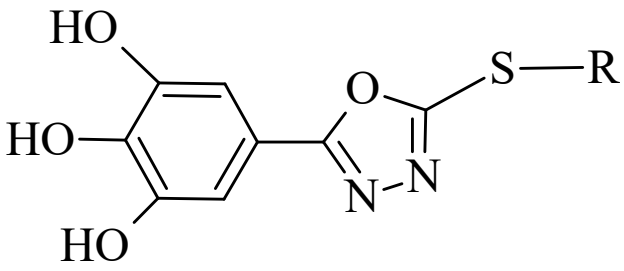

Figure 1 Target molecule

Methodology:

A mixture of 5-(5-sulfanyl-1,3,4-oxadiazol-2-yl) benzene-1,2,3-triol (0.97 g, 0.005 mol) and 0.005 mol of various aryl or alkyl halides was refluxed in 25 mL of pyridine for 3.5 hours. After completion, the reaction mixture was cooled and poured into crushed ice, leading to the precipitation of a solid product, which was then filtered, dried, and recrystallized from ethanol[44,45].

Following this procedure, different aryl or alkyl halides were used to synthesize a series of derivatives: 5-{5-[(4-methyl phenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVA) using 4-methylbromobenzene, 5-{5-[(4-methoxyphenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVB) using 4-methoxybromobenzene, and 5-{5-[(4-hydroxyphenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVC) using 4-hydroxybromobenzene. Similarly, 4-nitrobromobenzene yielded 5-{5-[(4-nitrophenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVD), 4-ethylbromobenzene gave 5-{5-[(4-ethylphenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVE), and bromobenzene resulted in 5-[5-(phenylsulfanyl)-1,3,4-oxadiazol-2-yl] benzene-1,2,3-triol (IVY). Additionally, methyl-bromo, ethyl-bromo, and propyl-bromo were employed to synthesize alkyl derivatives: 5-[5-(methylsulfanyl)-1,3,4-oxadiazol-2-yl] benzene-1,2,3-triol (IVF), 5-[5-(ethylsulfanyl)-1,3,4-oxadiazol-2-yl] benzene-1,2,3-triol (IVG), and 5-[5-(propylsulfanyl)-1,3,4-oxadiazol-2-yl] benzene-1,2,3-triol (IVH), respectively. Lastly, 4-amino bromobenzene was used to obtain 5-{5-[(4-aminophenyl) sulfanyl]-1,3,4-oxadiazol-2-yl} benzene-1,2,3-triol (IVY).

Solvent system for TLC- ethyl acetate: n-hexane (65:35).

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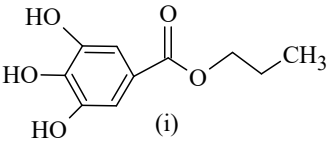
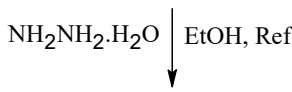


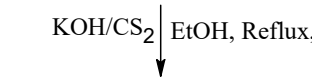
(i)

propyl 3,4,5-trihydroxybenzoate /(propyl gallate)



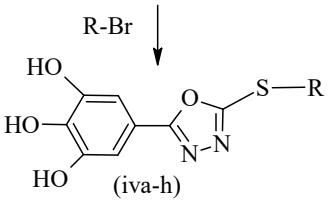
(ii)

3,4,5-trihydroxybenzohydrazide/(galloyl hydrazide)

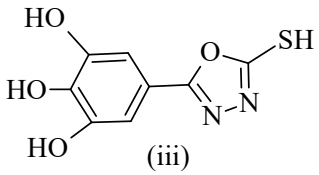


(iii)

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

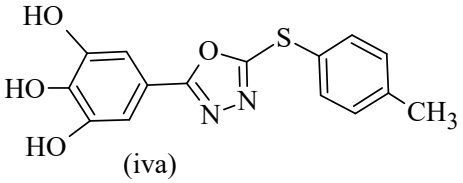


(iva-h)



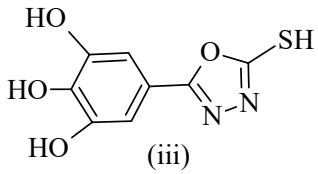
(iii)

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol



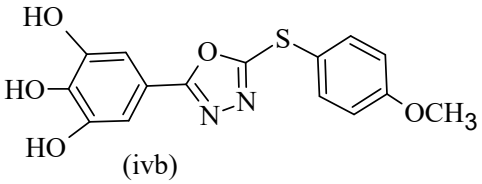
(iva)

5-{5-[(4-methylphenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol



(iii)

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol



(ivb)

5-{5-[(4-methoxyphenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol

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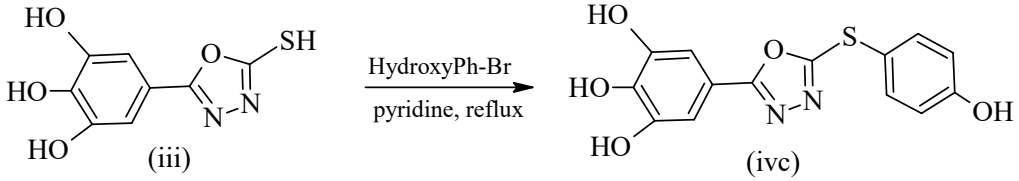
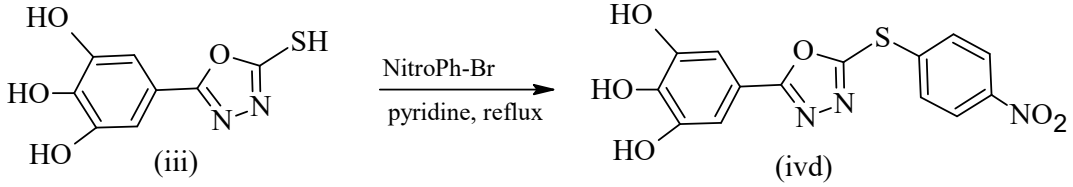


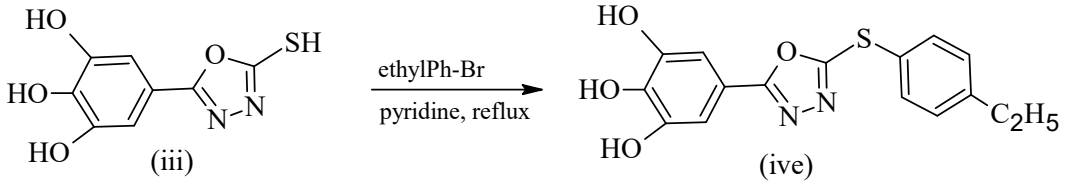
5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-{5-[(4-hydroxyphenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol



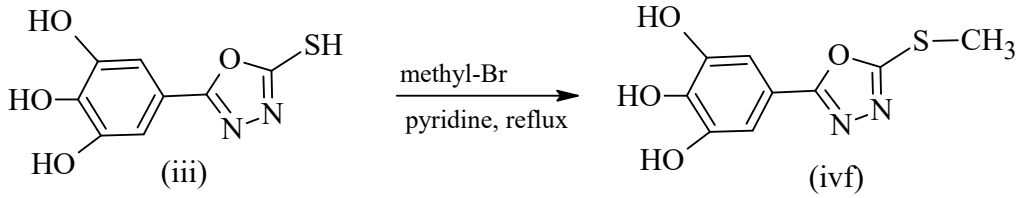
5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-{5-[(4-nitrophenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol



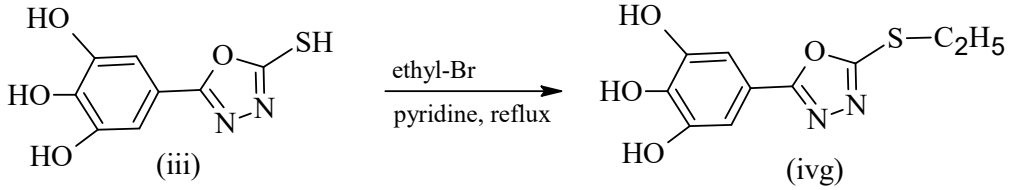
5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-{5-[(4-ethylphenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol



5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-[5-(methylsulfanyl)-1,3,4-oxadiazol-2-yl]benzene-1,2,3-triol



5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-[5-(ethylsulfanyl)-1,3,4-oxadiazol-2-yl]benzene-1,2,3-triol

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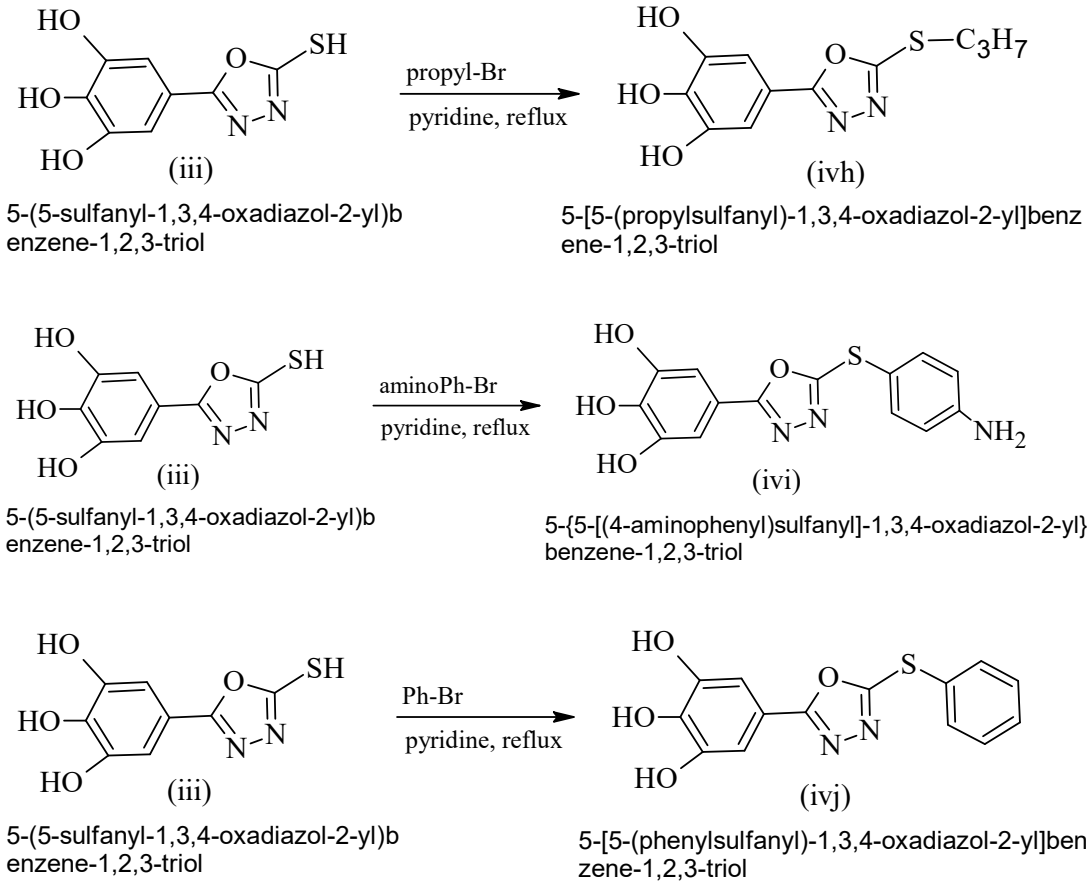

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-[5-(propylsulfanyl)-1,3,4-oxadiazol-2-yl]benzene-1,2,3-triol

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-{5-[(4-aminophenyl)sulfanyl]-1,3,4-oxadiazol-2-yl}benzene-1,2,3-triol

5-(5-sulfanyl-1,3,4-oxadiazol-2-yl)benzene-1,2,3-triol

5-[5-(phenylsulfanyl)-1,3,4-oxadiazol-2-yl]benzene-1,2,3-triol

Figure 2 Step-by-step procedure for the synthesis of 5-[5-9(phenylsulfonyl)-1,3,4-oxadiazol-2-yl] benzene-1,2,3-triol.

Table 3 Physiochemical data for synthesized compound mentioned in step iva-ivj

Compound	% yield	Rf value	Mol. formula	Mol. weight g/mol
iva	81.64	0.63	$C_{15}H_{12}N_2O_4S$	316.33
ivb	73.27	0.82	$C_{15}H_{12}N_2O_5S$	332.33
ivc	77.33	0.74	$C_{14}H_{10}N_2O_5S$	318.30
ivd	61.01	0.79	$C_{14}H_{19}N_3O_6S$	347.30
ive	69.82	0.81	$C_{16}H_{14}N_2O_4S$	330.35
ivf	78.62	0.58	$C_9H_8N_2O_4S$	240.23
ivg	76.23	0.68	$C_{10}H_{10}N_2O_4S$	254.26
ivh	65.73	0.66	$C_{11}H_{12}N_2O_4S$	268.28
ivi	71.98	0.85	$C_{14}H_{11}N_3O_4S$	317.31
ivj	91.82	0.71	$C_{14}H_{10}N_2O_4S$	302.30

3 PHARMACOLOGICAL SCREENING:

Inflammation is defined classically as a protective reaction by the body, in response to some physical or chemical injury. Inflammation is a protective reaction by the body. It is a part of the host defense tissue factors or mechanisms that are involved in inflammatory reactions such as the release of histamine, prostaglandins, and bradykinin. There are many

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diseases that are produced due to inflammation such as arthritis, fever, etc. Now, many medicines are also available for the treatment of inflammation. Recently many novel non-steroidal anti-inflammatory drugs have been available on the market. In rats the inflammatory reaction is produced in the form of paw edema with the help of some chemicals, some chemicals which are used are carrageenan, bradykinin, histamine, formalin, 5-hydroxytryptamine, and mustard or egg white. When this type of compound is injected into the plantar tissue of the right hind footpad of the rats, paw edema is produced within a few minutes of the injection. Carrageenan-induced paw edema is the most used method in experimental pharmacology for anti-inflammatory activity. Carrageenan is a sulfated polysaccharide, obtained from seaweed (Rhodophyceae). It produces edema by releasing histamine[46], 5-HT, bradykinin, and prostaglandins into the body.[26]

The anti-inflammatory activity of synthesized indole derivatives was carried out using the carrageenan-induced rat hind paw edema method.

3.1 Carrageenan-Induced Paw Edema Model in Albino Wistar Rats

Method:

Table 4 Specification for Invivo activity.

Parameter	Details
Experimental Animals	Albino Wistar male rats (200-300 g)
Source	Zydus Research Centre, Ahmedabad
Housing Conditions	Group of six in a clean plastic cage with a metal frame lid
Environmental Conditions	Temperature: 22°C (±3°C); Relative Humidity: 30%-70% (ideal: 50%-60%); Light Cycle: 12 hours light, 12 hours dark
Acclimatization	2 days before the experiment
Fasting Before Experiment	16 hours (free access to water)
Diet	Standard pellet diet
Bedding	Clean paddy husk (changed every alternate day)
Ethical Compliance	As per CPCSEA guidelines
Acute Toxicity Study	Conducted before anti-inflammatory evaluation
Method Used	Up and Down Procedure (OECD Guideline No. 425)
Test Compound	Indole derivatives suspended in Tween-80
Dose Administered	Up to 10 mg/kg (n=3 rats)
Fasting Before Toxicity Study	24 hours
Observation Period	First 30 minutes intensively, then periodically for 24 hours
Signs Monitored	Toxicity symptoms

The carrageenan-induced paw edema model was used to evaluate the anti-inflammatory activity of the test compound. This method involves inducing acute inflammation in the hind paw of rats by injecting carrageenan, a known inflammatory agent.

The experiment utilized Albino Wistar rats, with six rats assigned to each group. Inflammation was induced by injecting a carrageenan solution (1% w/v in saline) into the subplantar region of the rats' paws (0.1 ml per paw). A mercury displacement plethysmometer was used to measure paw edema. The standard anti-inflammatory drug, Indomethacin (3 mg/kg), was prepared as an aqueous suspension using Tween-80. The test compounds were also suspended in 1% Tween-80 and administered intraperitoneally, following the same procedure as the standard drug. The necessary apparatus included syringes (1 ml and 2 ml) and sample tubes for suspension preparation. Before the experiment, animals were weighed and numbered. This model serves to evaluate the anti-inflammatory potential of the test compound by assessing its ability to reduce paw edema in comparison to the standard drug, Indomethacin.

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3.2 Experimental design:

Weigh the animals and number them. Mark the animals with picric acid for individual animal identification. Divide rats into 5 groups of 6 rats each. Note the initial paw volume of each rat by dipping just beyond the tibio-tarsal junction by mercury displacement method, so that every time the paw is dipped in the mercury column up to the fixed mark to ensure constant paw volume. The animals were deprived of food overnight (allowed free access to water) and synthetic compounds were administered once 30 minutes before the injection of carrageenan. A dose-volume not exceeding 0.5ml/100gm intraperitoneal was administered.

Group I: -The solvent control received normal saline.

Group II: -Positive control received Indomethacin (3 mg/kg).

Group III: -Received indole derivative-4.7 at a dose of 3 mg/kg suspended in 1%w/v tween-80

Group IV: -Received indole derivative-4.8 at a dose of 3 mg/kg suspended in 1%w/v. tween-80

Group V: -Received indole derivative-4.9 at a dose of 3 mg/kg suspended in 1%w/v tween-80.

Group VI: -Received indole derivative-4.10 at a dose of 3 mg/kg suspended in 1%w/v tween-80.

After 30 minutes of test compound administration, 0.1 ml of 1%w/v of carrageenan in normal saline was injected into the subplantar region of the left hind paw of the rat. Immediately after the carrageenan injection, the volume of its displacement was measured using a plethysmometer. [27,28,29]

The reading was recorded at 0, ½, 1, 2, 3 hrs.

The % inhibition of edema was calculated at the end of 3 hrs. by using the formula.[30]

Percent (%) inhibition = 1 – Vt/Vc X 100,

Where Vt: - edema volume in test group,

Vc: -edema volume in control group

Results were expressed as mean ± standard deviation.

RESULT & DISCUSSION:

Screening of Anti-inflammatory activity

Table 5 Screening of Anti-inflammatory activity in Albino Wistar rat.

Compound code	Inhibition of inflammation in cm					% Inhibition			
	0 hr.	1 hr.	2 hr.	3 hr.	4 hr.	1 hr.	2 hr.	3 hr.	4 hr.
Control	0.36±0.02	0.33±0.02	0.31±0.02	0.30±0.02	0.29±0.02	-	-	-	
Standard (Indomethacin)	0.33±0.02	0.30±0.02	0.26±0.02	0.23±0.02	0.20±0.18	09.09	16.13	41.33	42.45
Comp-Iva	0.33±0.02	0.28±0.02	0.26±0.02	0.22±0.02	0.18±0.02	15.15	16.12	26.66	37.93
Comp-Ivb	0.31±0.02	0.27±0.02	0.17±0.02	0.14±0.09	0.11±0.009	18.18	16.12	53.33	62.06
Comp-Ivc	0.34±0.07	0.30±0.02	0.21±0.008	0.17±0.08	0.21±0.01	09.09	32.19	43.33	27.58
Comp-Ivd	0.32±0.02	0.31±0.1	0.21±0.02	0.14±0.02	0.10±0.01	12.12	19.09	53.33	65.51
Comp-Ive	0.32±0.02	0.32±0.02	0.25±0.01	0.15±0.007	0.13±0.02	13.13	20.13	27.77	37.58
Comp-Ivf	0.31±0.02	0.31±0.02	0.21±0.02	0.21±0.01	09.09	12.12	18.18	26.66	38.98
Comp-Ivg	0.31±0.02	0.27±0.02	0.17±0.02	0.14±0.02	0.10±0.01	12.12	19.09	49.11	51.12
Comp-Ivh	0.32±0.02	0.32±0.02	0.25±0.01	0.17±0.08	0.21±0.01	09.09	32.19	43.33	47.12
Comp-Ivi	0.31±0.02	0.27±0.02	0.17±0.02	0.14±0.02	0.10±0.01	12.12	18.18	26.66	38.54
Comp-Ivj	0.33±0.02	0.28±0.02	0.26±0.02	0.22±0.02	0.21±0.01	09.09	32.19	42.84	46.55

No. of animals used in each Group (n) = 6, Values are expressed as Mean ± SEM

Dose of test compound = 3 mg/kg, Dose of Indomethacin = 3 mg/kg

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- The pharmacological screening of the synthesized compounds showed anti-inflammatory activity ranging from 27.58 to 65.51 % inhibition of rat paw edema volume after 3 hours, whereas the standard drug Indomethacin showed 42.45 % inhibition of rat paw edema volume after 3 hours.
- The compounds –Ivb, Ivd, Ivg, and Ivj were found to be nearly more potent than indomethacin which is used as a standard drug. Compounds Iva, Ivc, Ive, Ivf, and Ivi have shown less activity than indomethacin.

CONCLUSION:

The present work, which has been undertaken is bonafide, and novel for the synthesis of oxadiazole derivatives. In this view, we have made an attempt to review the literature on substituted oxadiazole for its medicinal significance with the help of chemical abstracts, journals, and internet sites.

The compounds –Ivb, Ivd, Ivg, and Ivj were found to be nearly more potent than indomethacin which is used as a standard drug. Compounds Iva, Ivc, Ive, Ivf, and Ivi has shown less activity than indomethacin.

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