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Therapeutic Potential of *Syzygium cumini* and *Terminalia chebula* against Silica-mediated Toxicity

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Abstract

Background: Silica is one of the well-known occupational pollutants associated with increased risk for respiratory diseases like silicosis, chronic bronchitis, chronic obstructive pulmonary disease, tuberculosis, liver fibrosis and lung cancer. **Aim**: The study aimed to evaluate the therapeutic effectiveness of simultaneous supplementation of ethanolic extract of *Terminalia chebula* and *Syzygium cumini* against silica-induced toxicity. **Methods:** To observe the alteration of blood parameters and histopathological variables after silica exposure (at a dose of 40 mg/kg, i.p.) for 28 days, and also used therapeutic agents, ethanolic extract of *T. chebula* (250 mg/kg, p. o.) and *S. cumini* (1000 mg/kg, p. o.). **Results**: Alterations were observed in the blood parameters after silica intoxication. Experimental rats, after being intoxicated with silica for 28 days, experimental rats showed a significant elevation in all blood biochemical parameters, which returned to normal after administration of therapeutic agents. **Conclusion:** Our experiment's findings clearly demonstrate that therapy with the ethanolic extract of *T. chebula* worked better than the ethanolic extract of *S. cumini.*

Major Findings: The ethanolic extract of both therapeutic agents—*S. cumini* and *T. chebula* recovered the blood parameters and histological changes to a significant degree, with *T. chebula* being shown to be more successful. This is because both plant extracts have medicinal qualities such as antioxidant, hepatoprotective, and anti-diabetic properties.

Keywords: Blood Parameters, Chronic Bronchitis, Occupational Exposure, Pulmonary Disease Therapeutic Agents

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1. Introduction

The most abundant mineral on Earth is silica, which is mostly made of silicon dioxide. Since prolonged exposure to respirable crystalline silica (RCS) can cause silicosis, chronic obstructive pulmonary disease (COPD), lung cancer, kidney diseases, and autoimmune diseases like rheumatoid arthritis and scleroderma, it is the latter of its two forms—amorphous and crystalline—that most clinicians and occupational health workers are concerned about[1]. Furthermore, exposure to RCS may worsen the onset of pulmonary tuberculosis[2]. Indeed, engineered silica, which contains up to 97% crystalline silica, has become a significant health concern for regulators, policy makers, and doctors[3]. The most common industries where silica exposure occurs are mining, construction, ceramic manufacturing, sand casting foundries, agate industry, stone cutting and polishing, road construction, sandblasting, brickmaking, fracking, and

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industries related to new technologies like solar panel manufacturing[4,5]. The fact that there are more than 20,000 new instances of silicosis each year suggests that the illness is still relevant even though it is one of the earliest lung diseases to be identified[6].

Silicosis was considered the most serious occupational hazard during the 1930s and was the focus of major federal, state and professional attention during this time[7]. The distribution of carbon in biological materials is comparable to that of silicon in the natural world. About 28% of the Earth's crust is made up of silicon[8]. Because silicon is so reactive, it either reacts with oxygen alone to produce free silica (SiO_2) or with oxygen and other elements to form silicates, such as asbestos[9,10,11]. Exposure to silica, however, caused severe testicular damage. The vascularisation had increased considerably in the seminiferous tubules[12].

Studies in silicotic pencil workers in central India demonstrated high mortality rates; the mean age of death was 35 years, and the mean duration of exposure was 12 years[13]. Silica exposure has also been associated with other lung diseases, such as tuberculosis[14,15] and lung cancer. Uterus histoarchitecture undergoes a significant change in its cellular organisation, with uterine lumen, endometrial cell height and uterine glands getting reduced after subchronic exposure to silica for 28 days at a dose of 40 mg/kg body weight[16].

Terminalia chebula Retz is a flowering evergreen tree with a brown, roundish crown and widely dispersed branches that can grow up to 30 meters in height. It belongs to the *Combretaceae* family. The elliptical, oblong, acute-tipped leaves have cordate bases, complete margins, glabrous upper surfaces, and golden pubescence beneath. The blooms are monoecious, terminal spikes or short panicles, dull white to yellow, and have a strong, disagreeable smell. Because of its exceptional curative properties, *T. chebula* is referred to as the "King of Medicine" in Tibet and consistently appears at the top of the list in Ayurvedic Materia *Medica*[17]. It is said to be beneficial for respiratory disorders and has antifibrotic qualities (Ayurvedic Medicinal Library). Pioglitazone 2.7 mg/kg served as a positive control, and three oral dosages of *T. chebula* fruit extract — 50, 100, and 200 mg/kg — were given[18]. For six weeks during the clinical trial, 15 patients received 2 g of *T. chebula* powder mixed with lukewarm water. Serum TG, total lipids, low-density lipids, and very low-density lipids were all significantly reduced, but high-density lipids were significantly elevated, according to the patient assessment. *T. chebula* thus has a cardiotonic impact as well[19]. Hepatoprotective[20,21], antimicrobial, antioxidant[22], adaptogenic, anti-inflammatory[23] and immunomodulatory[24] are some of its reported properties. According to Naik *et al.*, tannic acid is the main component in *T. chebula* fruit[25]. *Salmonella typhi* is well inhibited by *T. chebula* extracts[26].

Local names for *Syzygium cumini* (L.) Skeels tree, a member of the *Myrtaceae* family, is *Jamun* and *Jambul* in Asia and *Jamelao* and *Jambolao* in Brazil. The tree is well-known for its medicinal qualities and is widely distributed throughout Asia and the Americas Sections[27,28]. As one moves from Malaysia to northeastern Australia, the level of diversity increases. When many people consider popular species to cultivate, *S. cumini* comes to mind. This tree is found in areas of Nepal, Sri Lanka, and Indonesia, in addition to the Indian subcontinent. Long ago, this tree was introduced to Malaysia, where it became native[29] Pharmacological research has demonstrated the hypoglycemic, antibacterial, and anti-HIV properties of the constituents of *S. cumini*, a well-known traditional medicinal herb. Plant parts such as bark, leaves, fruit, and seed have been used in many traditional medical systems. Leucorrhoea, stomachaches, fever, dermatitis, constipation, decrease of radiation-induced DNA damage, prevention of blood discharges in the faeces, and other conditions can all be treated with the leaves. The eleventh Traditional Indian medicinal practices, such as *Siddha, Ayurveda,* and *Unani,* make use of *S. cumini* fruits for a variety of purposes, including those of an astringent, antiscorbutic, diuretic, antidiabetic, spleen enlargement[30,31]; chronic diarrhoea treatment, and stomachic[32]. The leaves of *S. cumini* include a variety of alkaloids, terpenoids, lignins, and other phytochemical components in addition to phenolics such as myricetin, quercetin, and betulinic acid[27,33,34].

Syzygium cumini (*Myrtaceae* family). Long-lasting brown hues in a variety of tones are produced by *S. cumini* bark, depending on the mordant and extract strength. Fishing and tanning leather are two common uses for the bark, which has an 8–19% tannin content. Asthma and bronchitis are treated with bark decoctions. Additionally, they can be used as mouthwash or gargled

to treat mouth ulcers, spongy gums, and stomatitis. Bark ashes are either applied to localised inflammations or mixed with water and used for burns[35]. Indian Black Berry, Black Plum, and *Jamun* are common names for *S. cumini*. Anthocyanins, cyanidin, petunidin, malvidin glucoside, gallic acid, vitamin C, and tannins are all present in *S. cumini* fruits. Although *S. cumini* seeds have been shown to have antihyperglycemic qualities, there are few reports of the fruit pulp's antihyperglycemic qualities[36]. An ethanolic extract of *S. cumini* bark showed anti-inflammatory qualities in animal models. The extract did not show any toxicity in mice up to a dose of 10.125 g/kg, p.o. Significant anti-inflammatory effects were seen in rats evaluated for formaldehyde-induced paw oedema (sub-acute), cotton pellet granuloma (chronic), carrageenin (acute), and kaolin carrageenin (sub-acute). The extract did not result in any stomach sores in rats used for both acute and long-term ulcerogenic testing. Since ancient times, medicinal plants have been utilised to heal ailments and are a significant source of both natural and manufactured drugs[35,37].

2. Materials and Methods

Albino rats (female) weighing 150 ±10 gmwere used for the investigation in their conventional housing, which included a temperature of 25± 20°C, a relative humidity of 60–70%, and a 12-hour photoperiod. They were given unlimited access to food and water. Animals were maintained as per the Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). Approval of the Institutional Animal Ethics Committee, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana (IAEC/2018/1216-1250). SiO_2 was created as a toxicant dosage in regular saline. Distilled water was used to dissolve *T. chebula* and *S. cumini*.

Animals were split up into four groups of five animals each (Total 20 animals were used).

Group 1 (Control) was given normal saline intraperitoneally (4 ml/kg) and represented as a standard control. Group 2 (Experimental) received SiO_2 (40 mg/kg, *ip*) daily for 28 consecutive days and served as the experimental group. Group 3 (Treated with *T. chebula*) received the toxicant as in Group 2 and was treated with ethanolic extract of *T. chebula* per se (250 mg/kg, p. o.) for 5 consecutive days. Group 4 (Treated with *S. cumini*) was treated with an ethanolic extract of *S. cumini* (1000 mg/kg, p. o.) for five days in a row after receiving the same toxicant as Group 2.

2.1 Collection of Blood and Preparation of Serum

Animal blood was drawn by puncturing the retroorbital sinus, centrifuged, and the serum was refrigerated for 2-3 days and analysed for 2 times.

2.2 Necropsy of Animals

Animals were given ether anaesthesia and had their abdomens cut open 24 hours later. The vital organs (Liver, kidney, and Lung) were removed right away. They were immediately fixed in Bouin's fixative for histology. Following their blotting and removal of unnecessary materials, they were weighed using a digital monopan electronic balance and subjected to histology.

Blood parameters were estimated by the following methods: Transaminases by[38], Blood sugar by[39], Proteins by[40] and Alkaline phosphatase by[41]. Kit method (CHOD-PAP method, No. 1117678.0001) from Merck was utilised to estimate the levels of Cholesterol, urea, and Creatinine. Every result was given as a mean ± SE. The Student's t-test and P-values were used to compare two independent groups.

3. Results

Table 1 depicts that silica led to a clear toxic response by significantly elevating the activity of the serum transaminases AST and ALT levels significantly. The therapy with the therapeutic agents prevented the silica-induced toxicity. *T. chebula* was found to be very effective in reducing the elevated levels, while the treatment with *S. cumini* was ineffective in recouping the values when compared to controls.

Table 1 showed a significant elevation in the serum alkaline phosphatase activity in silica-treated animals when compared to control groups. Both therapeutic agents, i.e., *S. cumini* and *T. chebula,* recouped the values to a considerable extent, but *S. cumini* was found to be more effective. Control rats did not register any significant change during the entire experimental regimen.

Significant elevation in the serum protein content in silica-treated animals was seen when compared to

control groups (Table 1). Therapy with *T. chebula* was more effective in the recoupment of serum protein content than *S. cumini*. Control rats did not register any significant change during the entire experimental regimen.

Table 1 demonstrates the efficacy of therapeutic agents on the blood sugar level in silica-intoxicated rats. The mean blood sugar level of the various groups of rats receiving 40 mg/kg silica as silicon dioxide-exposed rats was statistically higher than that of the level of basal control animals. Both therapeutic agents, i.e., ethanolic extract of *T. chebula* and *S. cumini,* recouped the values to normal control, but *S. cumini* was found to be more effective than the other therapeutic agent.

On sub-chronic exposure to silica, the cholesterol level was significantly raised (Table 1). The administration of *S. cumini* was more effective in recouping the silica-induced rise when compared to *T. chebula*. Table 1 showed a significant elevation in the level of urea in silica-treated animals when compared to control groups. Both therapeutic agents, i.e. *S. cumini* and *T. chebula,* recouped the values to a considerable extent, but *T. chebula* was found to be more effective. Table 1 depicts a considerable elevation in the level of creatinine on silica administration, at a dose of 40 mg/kg for 28 days. *S.cumini* and *T. chebul* were effective in the recoupment of creatinine concentration when compared to silica-exposed groups.

3.1 Blood Parameters

3.2 Photograph

Photomicrograph of the T. S. of the liver of an albino rat of the control group. Shows the normal cellular organisation (Figure 1). Silica exposure caused severe degeneration in hepatocytes along with the accumulation of fluid in the sinus. The number of Kupffer cells was decreased (Figure 2). Treatment with *S. cumini* showed slight improvement in hepatocytes, fewer degenerative changes and recoupment in the chord arrangement when compared to the silica per se exposed group(Figure 3). Treatment with *T. chebula*showed comparatively better-formed hepatocytes. Maintained chord arrangement with the normal size of Kupffer cells was observed, but mild vacuolation was noted in some hepatocytes (Figure 4).

Photomicrograph of the T. S. of the kidney of an albino rat of the control group showing normal features (Figure 5). Silica exposure caused severe

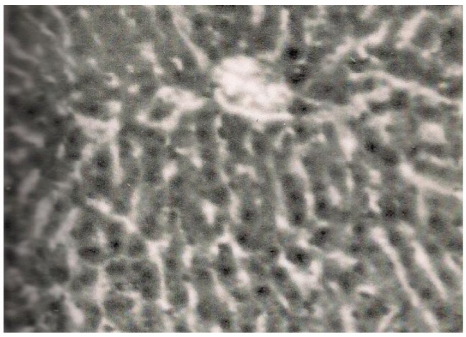


Figure 1. T. S. of the liver of an albino rat of the control group showing normal features (X 140).

Table 1. Blood parameters of control, experimental and treated groups

Sr. No.	Parameter	Control Mean+SE	SiO_2 Mean+SE	*Syzygium cumini* Mean+SE	*Terminalia chebula* Mean+SE
1	AST(IU/L)	68.0± 3.12	116 ± 5.55	104+7.24	98.4+8.85
2	ALT(IU/L)	43.2± 2.58	82.0 ± 4.14	79+4.22	70.4+1.13
3	ALP (mg Pi/100ml/h)	206±15.8	370± 30.1	267.32+17.7	269.95+1.45
4	S-Protein (mg/100ml)	37.3 ± 2.64	48.2 ± 2.78	47.95+2.57	45.6+0.77
5	Blood Sugar (mg/100ml)	108.0 ± 6.70	136.0 ± 7.60	122.32+6.85	124.9+0.88
6	Cholesterol (mg/dl)	54.3 ± 2.73	94.0 ± 4.99	67.0+3.85	68.6+0.66
7	Urea (mg/dl)	26+0.31	47.22+0.86	38.89+0.44	34.11+0.75
8	Creatinine (mg/dl)	0.35+0.01	1.72+0.03	1.07+0.05	1.13+0.08

ANOVA at 5 % level: * - Significant; ns - Not significant; P value SiO_2 *vs* control at a<0.05, b<0.01; P value Drugs *vs* SiO_2 at c<0.05, d<0.01.



Figure 2. Administration of Silicon dioxide, showing severe degeneration in hepatocytes along with accumulation of fluid in the sinus and disrupted portal triad (X 140).

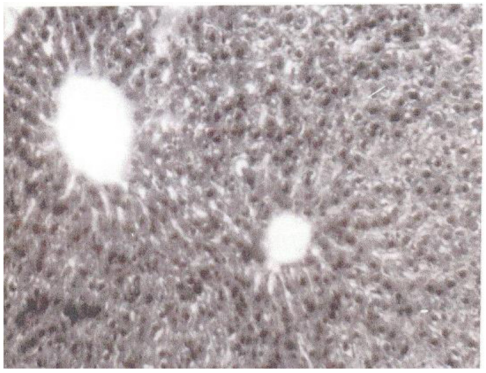


Figure 3. Treatment with *Syzygium cumini.*
Note: comparatively betterformed hepatocytes (c. f. 2) but mild degeneration persists (X 140)..

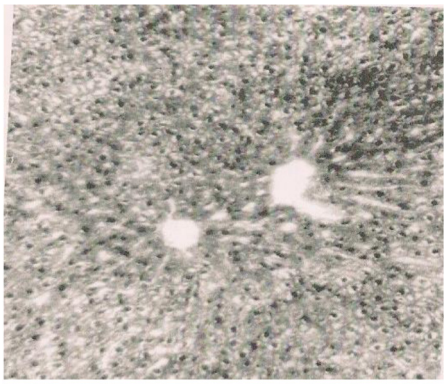


Figure 4. Treatment with *Terminalia chebula* maintainedchord arrangement with normal kupffer cells(X 140).

damage in the uriniferous tubules and dilatation in glomerular arteries (Figure 6). Treatment with *S. Cumini* formed better-formed cortical tubules and compact glomeruli (Figure 7). Treatment with

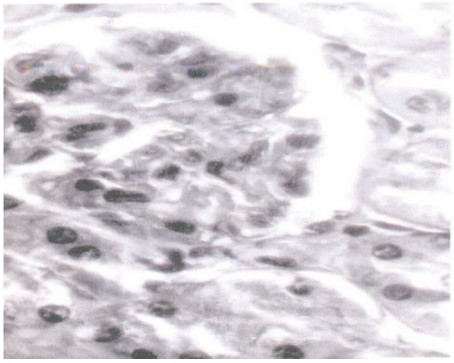


Figure 5. T. S. of Kidney of albino rat of control group showing normal features (X 400).



Figure 6. Administration of Silicon dioxide showing damage in uriniferoustubules (X 140) and dilatation in glomerular arteries.

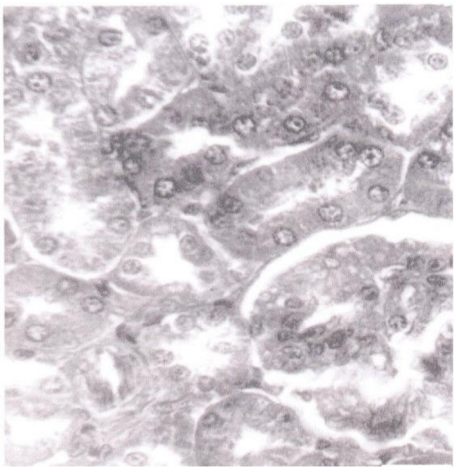


Figure 7. Treatment with *Syzygium cumini* showing better formed cortical tubules andcompact glomeruli (X 400).

T. chebulai improved the kidney architecture to a great extent, with fairly normal renal tubules and Bowman's capsule (Figure 8).

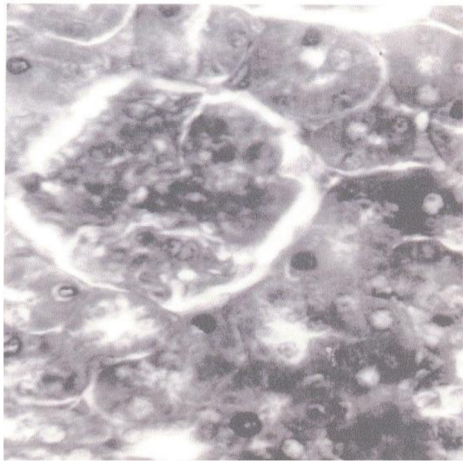


Figure 8. Treatment *Terminalia chebula* showing fairly normal renaltubules and Bowman's capsule (X 140).

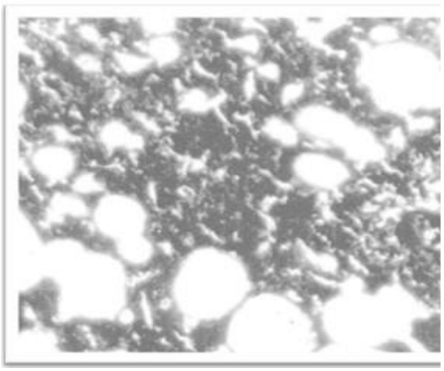
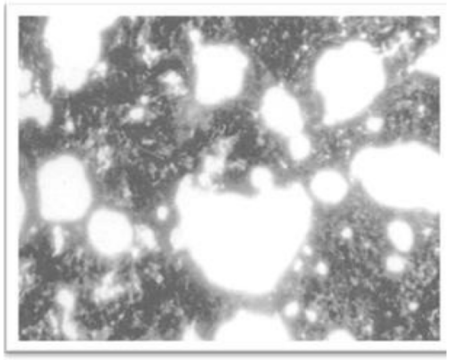


Figure 9. T. S. of Lung of albino rat of control group showing normal features (X 140).



Note: Ruptured alveolar septa and pulmonary artery showed fibrotic structure(X 140).

Figure 10. Administration of silicon dioxide.

Lungs of the control rat showed normal features (Figure 9). Administration of silicon dioxide ruptured the alveolar septa; the pulmonary artery showed a fibrotic structure (Figure 10). Treatment

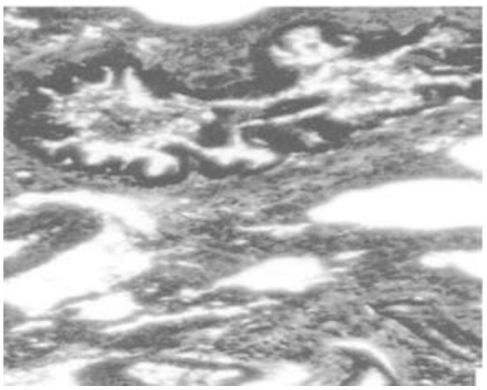


Figure 11. Treatment with *Syzygium cumini*. Note larger alveoli indicating border line emphysema (X400).

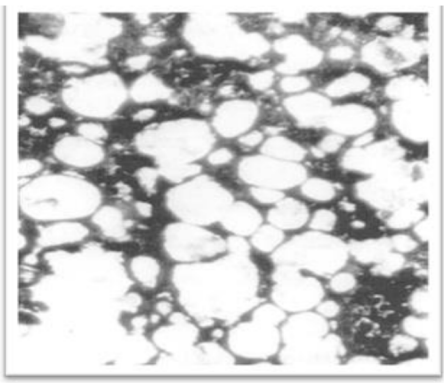


Figure 12. Treatment with *Terminalia chebula* showing moderate air spaces (X140).

with *S. cumin* showed larger alveoli, indicating borderline emphysema and degeneration in parenchymatous tissues (Figure 11). Treatment with *T. chebula* showed that respiratory bronchioles were generally lined with ciliated cuboidal epithelium. Alveolar ducts were long and branched repeatedly, and damaged alveoli of various shapes and sizes (Figure 12).

4. Discussion

Silicosis is a disease of the industrial period caused by the accumulation of silica particles in the lungs of humans and experimental animals. The IARC working group highlighted in its overall assessment that carcinogenicity was not found in every industrial setting examined and that this could depend on the crystalline silica's intrinsic properties or on outside variables influencing its biological activity or polymorph dispersion. Silicosis, an industrial-era

disease, is brought on by silica particles building up in the lungs of both humans and test animals.

In this study, rats were exposed to silicon dioxide subchronically for 28 days, after which they were treated with ethanolic extracts of *T. chebula* fruit and *S. cumini* bark. The study's findings showed that silica caused oxidative stress (AST, ALT and ALP), significant changes in blood parameters, and histological findings. The liver, kidney, and lung were among the organs where the protective effects of *S. cumini*[42] and *T. chebula* extract were observed[43].

Enzymes catalysed reactions occur within living cells, but leak out on exposure to certain toxicants into the bloodstream. Measurement of the extent and duration of these raised enzyme levels provides information on the identity of damaged cells and indicates the degree of injury and its response to corrective treatment. Serum AST and ALT are excellent markers of liver damage caused by exposure to toxic substances. AST is a mitochondrial enzyme that is found in the heart, liver, kidneys, and skeletal muscles. ALT is localised mainly in the cytoplasm, whereas AST activity is distributed fairly equally between cytoplasm and mitochondria. ALT is the cytosolic enzyme, which is more specific for the liver than AST.

In severe acute liver damage, serum transaminase levels parallel those of the organs, indicating that both the cellular and mitochondrial membranes have been damaged. Our study reveals a significant increase in serum transaminase activity on administration of silicon dioxide at a dose of 40 mg/ kg, conceptualising liver phagocytosis and necrosis. Increased serum enzymes, mainly transaminases, give an indication of the leakage of the enzymes out of the liver cell, not only with necrosis but also with changes in cell permeability. Since membrane integrity is linked with intracellular metabolic states, disturbance of the latter results in membrane lesions with concomitant increase in enzyme leakage, giving rise to hypoxia and membrane hyperpermeability. In our experiment, we found that, after subchronic exposure to silica, cell damaged and the level of transaminases (AST/ALT) was increased; these findings are substantiated by other authors after administration of toxic compounds[44-46]. Traditional Indian medicinal practices such as *Siddha, Ayurveda*, and *Unani* make use of *S. cumini* fruits for a variety of purposes, including those of an astringent, antiscorbutic, diuretic, antidiabetic, spleen enlargement[47,48], chronic diarrhoea treatment, and stomachic[49].

Alkaline phosphatase is abundant in the liver, which is referred to as the body's powerhouse. All bodily tissues contain phosphatase, which is crucial to the activity of inorganic pyrophosphatase. This enzyme's primary role is to supply energy for the cell wall's differential permeability and to facilitate active transport across the capillary wall. The kidney's brush border and the intestinal mucosa are the sources of alkaline phosphatase. They mediate membrane trafficking and participate in transphosphorylation processes. It has been proposed that the intestinal mucosa and the kidney's proximal tubules have high levels of alkaline phosphatase activity and are crucial for the reabsorption of glucose from renal tubules. Alkaline phosphatase is involved in the hydrolysis of phosphohexoses[50]. In our study, sub-chronic exposure to silica resulted in a notable increase in alkaline phosphatase activity; however, this elevation was diminished and found to be closer to normal when the subjects were treated with *S. cumini* ethanolic extract. When animals were given *T. chebula*, this level was likewise significantly restored.

Blood sugar levels also significantly increased following subchronic silica exposure. The dysfunctional and dystrophic alterations in the liver following the ingestion of hazardous substances could be the cause of this increase in blood sugar. This high level was considerably reduced when these silicotic rats were treated with *S. cumini*. Additionally, *T. chebula* had a protective effect. In the current investigation, animals treated with silica for 28 days showed notable alterations in their serum protein and cholesterol levels. Recovery was also shown when the same silicotic rats were given an ethanolic extract of *S. cumini* and *T. chabula*.

5. Conclusion

In conclusion, the results of our experiment clearly demonstrate that the ethanolic extract of both therapeutic agents—*S. cumini* and *T. chebula*re covered the blood parameters and histological changes to a significant degree, with *T. chebula* being shown to be more successful. This is because both plant extracts have medicinal qualities such as antioxidant, hepatoprotective, antibacterial, anti-diabetic, and

anti-hyperglycemic. *T. chebula* is essential for the detoxification of dangerous substances and the microsomal oxygenation of various xenobiotics because of its strong antioxidant properties, which directly interact with oxidising radicals to protect cells from reactive oxygen species.

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Author Contribution

Shruti Saxena: Conceptualisation, data curation, investigation; Priyanka Rana: Formal analysis, investigation; Kanchan Chatterjee: Investigation, resources; Rupinder Kaur: Supervision, writing; Diksha Nautiyal: Investigation, software handling. All authors have read and agreed to the published version of the manuscript.