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The Effect of *n*-Butanol Extract of Dewandaru Fruit (*Eugenia uniflora* L.) on the Leukocyte Count in Mice Exposed to Cigarette Smoke

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ABSTRACT

Dewandaru (*Eugenia uniflora* L.) is a medicinal plant that has been widely used in traditional medicine. Cigarettes are addictive substances that pose a significant threat to health and life sustainability. The principal toxic constituents of cigarettes are nicotine, tar, and carbon monoxide (CO). Long-term smoking can increase the total leukocyte count, particularly the number of polymorphonuclear neutrophils (PMN) in the peripheral blood circulation, due to free radical generation and inflammatory reactions. *Dewandaru* fruit (*Eugenia uniflora* L.) contains antioxidant and anti-inflammatory compounds that may inhibit leukocyte activation induced by oxidative stress associated with cigarette smoke exposure, thereby contributing to a reduction in the total leukocyte count. This study employed an experimental design with a *posttest*-only control group, using a sample of 30 male mice (*Mus musculus*) divided into three groups. Group I served as the negative control and received cigarette smoke exposure and distilled water. Groups II and III were designated as treatment groups and received 30 minutes of cigarette smoke exposure per day for 30 days, along with oral administration of *n*-butanol extract of *dewandaru* fruit at doses of 100 mg/kgBW and 200 mg/kgBW, respectively. On day 31, peripheral blood samples were collected from all subjects. The findings of this study indicate that administration of *n*-butanol extract of *dewandaru* fruit (*Eugenia uniflora* L.) resulted in a reduction in the total leukocyte count in male mice receiving the extract compared to the control group.

INTRODUCTION

Cigarettes are addictive substances that pose a significant threat to human survival in both developed and developing countries (Hasan, 2024; Hecht & Hatsukami, 2022; Sakthisankaran et al., 2024; Zeng et al., 2023). As a developing country, Indonesia has experienced a rapid increase in cigarette consumption from year to year, and currently ranks third globally in the number of smokers, after China and India (Achmad et al., 2026; Ahsan et al., 2022; Kishun et al., 2022; Prasetya et al., 2025). Based on data from the World Health Organization (WHO), cigarette consumption claims one life every 10 seconds, and the estimated number of smokers worldwide has reached 1.35 billion people (Minister of Health of the Republic of Indonesia, 2013). According to data from the Basic Health Research (*Riset Kesehatan Dasar*/Riskesdas) conducted in 2013, the mean prevalence of smokers in Indonesia was 29.3% (Devina et al., 2017).

Cigarette smoke contains approximately 4,000 chemical compounds, including carbon monoxide, carbon dioxide, phenol, ammonia, formaldehyde, pyrene, nitrosamines, nicotine, and tar, all of which are highly harmful to the human body. Cigarette smoke also comprises numerous oxidants and free radicals capable of damaging lipids, proteins, deoxyribonucleic

acid (DNA), carbohydrates, and various other biomolecules (Devina et al., 2017). Approximately 10^{17} oxidant molecules are present per cigarette. The most harmful constituent of cigarettes is nicotine, a rapidly acting toxic substance (Nowak & Pawliczak, 2022; Wang et al., 2023). Upon inhalation of cigarette smoke, an inflammatory response is triggered, as evidenced by increased production of pro-inflammatory mediators, which subsequently elevates the total leukocyte count in the peripheral blood (Dewi et al., 2018). Leukocytes, or white blood cells, serve as the primary cellular mediators of the body's defense against infection. The normal reference range for the leukocyte count is 4,000–10,000/mm^3 (Sylvia, 1995).

Leukocytes are blood cells that play a central role in the body's immune defense system (Akhand & Ahsan, 2023; Fırat, 2024; Harvanová et al., 2023; Zhu & Su, 2022). Individuals chronically exposed to cigarette smoke have been reported to exhibit a leukocyte count 20–25% higher than that of non-smokers (Dewi et al., 2018). Nicotine has been shown to induce leukocytosis through the stimulation of hormones such as epinephrine and cortisol (Ragil, 2016). Leukocytes serve as the body's principal defense agents against infection via the process of phagocytosis and play an essential role in the immune response to tissue injury (Sirih et al., 2017).

The human body possesses endogenous enzymatic systems capable of neutralizing free radicals; however, when free radical load is excessive as occurs with prolonged cigarette smoke exposure exogenous antioxidants are required to supplement the body's defense mechanisms. Antioxidants are compounds that function to prevent, inhibit, and neutralize free radical reactions (Winarsi, 2007). Antioxidants are broadly classified into two categories: enzymatic and non-enzymatic. Enzymatic antioxidants are endogenously produced and constitute part of the primary intracellular defense system; these include superoxide dismutase (SOD), catalase, and glutathione peroxidase. Non-enzymatic antioxidants include vitamins A, E, C, and β-carotene, as well as other compounds such as bilirubin, albumin, and flavonoids. Non-enzymatic antioxidants are derived from exogenous dietary sources, including vitamins A, C, and E, β-carotene, selenium, lycopene, and flavonoids (Nadimin, 2018). Flavonoids are classified as non-enzymatic or chain-breaking antioxidants and have been demonstrated *in vitro* to exert potent biological effects (Winarsi, 2007).

The urgency of this research is underscored by Indonesia's status as the country with the third-largest smoking population globally, with smoking-related diseases contributing substantially to morbidity, mortality, and healthcare expenditure. The rising prevalence of smoking among adolescents and young adults further highlights the critical importance of identifying protective agents against cigarette smoke-induced oxidative stress. From a public health perspective, natural products capable of mitigating smoking-induced inflammation may offer affordable and accessible interventions for at-risk populations. The novel contributions of this research are: (1) utilization of the *n*-butanol fraction of *dewandaru* fruit, which was demonstrated by Santoso et al. (2020) to possess superior antioxidant capacity (IC_{50} = 8.893 ppm) relative to the ethyl acetate (IC_{50} = 15,203 ppm) and chloroform (IC_{50} = 75,873 ppm) fractions; (2) establishment of a dose-response relationship for leukocyte modulation at doses of 100 mg/kgBW and 200 mg/kgBW; (3) specifically targeting the leukocyte response to cigarette smoke exposure as a primary outcome measure; and (4) integration of phytochemical screening to identify the bioactive compounds responsible for the observed effects.

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The purpose of this study is to determine the effect of *n*-butanol extract of *dewandaru* fruit (*Eugenia uniflora* L.) on the leukocyte count in male mice (*Mus musculus*) exposed to cigarette smoke. The theoretical contribution of this study is to advance the understanding of how natural antioxidants modulate cigarette smoke-induced inflammatory responses. The practical contribution is to provide evidence supporting the potential use of *dewandaru* fruit extract as an adjunctive therapy for individuals exposed to cigarette smoke, whether as active smokers or through secondhand exposure. The objective is to measure and compare leukocyte counts across three experimental groups: cigarette smoke exposure only, cigarette smoke exposure with 100 mg/kgBW extract, and cigarette smoke exposure with 200 mg/kgBW extract. The anticipated benefits include: (1) establishing a scientific basis for the traditional use of *dewandaru* fruit in Balinese *Usadha* medicine; (2) providing preliminary data to support the potential development of standardised antioxidant supplements; and (3) contributing to the growing body of evidence on natural products for mitigating smoking-induced oxidative stress.

METHOD

This study used male mice (*Mus musculus*) as the animal model, with the fruit of *dewandaru* (*Eugenia uniflora* L.) as the plant material. *Dewandaru* fruit was obtained from the Mount Kawi area, Malang, East Java. Additional materials used in this study included ethanol, clove cigarettes, and aquadest.

Preparation of *n*-Butanol Extract of *Dewandaru* Fruit: *Dewandaru* fruit powder (*Eugenia uniflora* L.) was weighed to 300 grams and macerated using 1,000 ml of *n*-butanol solvent in a tightly sealed amber jar, protected from light. The *maserat* was stirred for 60 minutes using a glass stirring rod. On the second day, the *maserat* was stirred again for 60 minutes, then re-sealed and stored. On the third day, the *maserat* was filtered to yield filtrate-1. The marc was macerated twice further using the same type and volume of solvent following the same procedure, and maceration was continued until day seven. On the seventh day, the *maserat* was filtered again to obtain filtrate-3, which was then combined with filtrate-1 and filtrate-2. The combined filtrate was concentrated using a rotary evaporator until a thick extract was obtained.

Dosage Calculation: Dosage calculation for Group I (based on a mean body weight of 20 g per mouse): 100 mg/kgBW × 0.020 kg = 2 mg of *n*-butanol extract of *dewandaru* fruit per mouse per day. Total extract required for one group (consisting of 10 mice) over 30 days: 2 mg × 10 × 30 = 600 mg of *n*-butanol extract of *dewandaru* fruit. (Note: dosage was adjusted proportionally to the individual body weight of each mouse.) Dosage calculation for Group II (based on a mean body weight of 20 g per mouse): 200 mg/kgBW × 0.020 kg = 4 mg of *n*-butanol extract of *dewandaru* fruit per mouse per day. Total extract required for one group (consisting of 10 mice) over 30 days: 4 mg × 10 × 30 = 1,200 mg of *n*-butanol extract of *dewandaru* fruit. Total *n*-butanol extract of *dewandaru* fruit required for both treatment groups: 600 + 1,200 = 1,800 mg. (Note: dosage was adjusted proportionally to the individual body weight of each mouse.)

Testing Procedure: The mice underwent a 7-day acclimatization period prior to the commencement of the study. A total of 30 mice were randomly allocated into three groups of 10 mice each, comprising one negative control group and two treatment groups receiving *n*-butanol extract of *dewandaru* fruit at doses of 100 mg/kgBW and 200 mg/kgBW, respectively.

Beginning on day 8, all groups including the control group were subjected to cigarette smoke exposure for 30 minutes daily for 30 consecutive days. Concurrently, the treatment groups received oral administration of *n*-butanol extract of *dewandaru* fruit via orogastric *sonde* (gavage) at their respective doses, administered daily for 30 days following each cigarette smoke exposure session. On day 31, blood samples were collected from all subjects via the orbital sinus and subsequently analyzed using a hemocytometer and peripheral blood smear to determine the total leukocyte count.

Phytochemical Screening: The phytochemical test solution was prepared by dissolving 500 mg of *dewandaru* fruit extract in 50 ml of *n*-butanol solvent. Alkaloid test: 2 ml of the test solution was evaporated in a porcelain evaporating dish until a residue was obtained. The residue was then dissolved in 5 ml of 2N HCl. The resulting solution was divided into two test tubes, with the first tube serving as a blank. Three drops of Dragendorff reagent were added to the second tube; the formation of an orange precipitate indicated a positive result for alkaloids (Santoso et al., 2018). Flavonoid test: 1 ml of the test solution was combined with 0.5 g of magnesium powder and 3 drops of concentrated HCl. The formation of an orange-to-red color indicated the presence of flavones, red-to-crimson indicated flavanols, and pale red-to-purplish-red indicated flavanones (Santoso et al., 2018). Saponin test: 10 ml of the test solution was placed in a vertical test tube and shaken vigorously for 10 seconds, then allowed to stand for 10 seconds. The formation of stable persistent foam indicated the presence of saponins. Stability was confirmed by the addition of 1 drop of 2N HCl, which did not cause foam dissipation (Santoso et al., 2018). Tannin test: 2 ml of the test solution was treated with a few drops of 10% $FeCl_3$ solution; the formation of a dark blue or greenish-black color indicated the presence of tannins and polyphenols (Santoso et al., 2018). Steroid/triterpenoid test: 2 ml of the test solution was combined with 1 ml of chloroform, 1 ml of acetic anhydride, and 4 ml of concentrated H_2SO_4 (Liebermann–Burchard reaction). The formation of a brownish or violet ring at the solvent interface indicated a positive result for triterpenoids, while a greenish-blue ring indicated a positive result for steroids (Santoso et al., 2018).

Data Analysis: Data obtained from the study were statistically analyzed using SPSS version 20.0. Prior to inferential analysis, a normality test was performed using the Kolmogorov–Smirnov test and a homogeneity test using the Shapiro–Wilk test. Data were considered normally distributed if $p > 0.05$, and homogeneous if $p > 0.05$. If the data satisfied both assumptions of normality and homogeneity, parametric analysis was conducted using a one-way analysis of variance (one-way ANOVA). If the data did not meet the assumption of normal distribution, non-parametric analysis was performed using the Kruskal–Wallis test.

RESULT AND DISCUSSION

Observation Results of Research Subjects: This study was conducted to determine the effect of giving n-Butanol extract of *dewandaru* fruit on the number of leukocytes in male mice exposed to cigarette smoke. A total of 30 male white mice *(Mus muscullus)* exposed to cigarette smoke were randomly divided into three treatment groups. The first group (K) was a control group that during treatment was given exposure to cigarette smoke and water, the second group (P1) was a treatment group that during treatment was given exposure to cigarette smoke and given n-Butanol extract of *dewandaru* fruit at a dose of 100 mg/KgBB, and the third group

(P2) was a treatment group that during treatment was given exposure to cigarette smoke and n-Butanol extract of *dewandaru* fruit at a dose of 200 mg/KgBB.

Normality Test of Leukocyte Count

Table 1. Results of the Normality Test of Mouse Leukocyte Number Data

Groups	***Shapiro-Wilk***		
	Statistic	*Df*	*Sig.*
Controls	.942	10	.573
Group 1	.898	10	.208
Group 2	.923	10	.383

The number of leukocytes in each group (Control), (P1), (P2) was tested for distribution using the *Shapiro-Wilk* the significance of the results of data analysis was guided by a confidence level of 95% ($\alpha = 0.5$). Test results *Shapiro-Wilk* The number of leukocytes in mice (Control) showed that $p = 0.573$, (P1) showed a value of $p = 0.208$, and for (P2) showed a value of $p = 0.383$, the result was the average value of (p) of each group and it can be concluded that the data was distributed normally because the p value > 0.05.

Data Homogeneity (Variance) Test

Table 2. Results of the Mouse Leukocyte Number Data Homogeneity Tes**t**

Levene Statistic	*df1*	*df2*	*Say.*
.180	2	27	.836

The number of leukocytes in each group (K), (P1), (P2) was carried out a homogeneity test, using the test *Levene's statistic*. To find out whether two or more data groups have the same variant or not. If the variance test yields a p> value of 0.05, then the variance of the tested data is the same. The results obtained from the three groups showed that the value of $p = 0.836$ means that the data distribution has a homogeneous distribution, indicated by a p value > 0.05, so that it can be continued with the Test *One Way Anova*.

One Way Anova Analysis: The average number of male mouse leukocytes given exposure to cigarette smoke was 3.1±(0.4) in the control group, the average number of leukocytes was 3.1(0.4), the (P1) group had an average number of leukocytes of 2.8±(0.3), while in the (P2) group, the average number of leukocytes was 2.5±(0.3).

Analysis results *One Way Anova*, showed that the administration of n-butanol extract of *dewandaru* fruit between the P1 group with a dose of 100 mg/KgBB and the P2 group with a dose of 200 mg/KgBB compared to the control group had an average number of mouse leukocytes. The results are presented in table 3.

Table 3. Average Mouse Leukocyte Count

Groups	Average (SD) 103µl	Value p
K	3,1 (0,4)	
P1	2,8 (0,3)	0,005
P2	2,5 (0,3)	

Description:

K : exposed to cigarette smoke without extract (only *aquadest*)

P1 : exposed to cigarette smoke given a suspension of n-butanol extract 100 mg/KgBB

P2 : exposed to cigarette smoke given n-butanol extract 200 mg/KgBB

Test *One Way Anova* It shows that all data in each group have significant differences shown by a p value of < 0.05. This means that there is a significant difference in the number of mouse leukocytes between the three treatment groups. To find out which groups have differences, a test is carried out *Post Hoc* LSD.

Post Hoc *Test*

Test *Post Hoc Test* showed there was a significant mean difference between the control group and the treatment group. How far the differences between the different treatment groups are presented in the table

Table 4. Data Interpretation

Groups		**P value (sig)**	**Remarks**
Control	P1	0,153	There is no meaningful difference
	P2	0,001	There is a significant difference
P1	Controls	0,153	There is no meaningful difference
	P2	0,045	There is a significant difference
P2	Controls	0,001	There is a significant difference
	P1	0,045	There is a significant difference

Description:

K : exposed to cigarette smoke without extract (*aquadest* only)

P1 : exposed to cigarette smoke given a suspension of n-butanol extract 100 mg/KgBB

P2 : exposed to cigarette smoke given n-butanol extract 200 mg/KgBB

Based on the results of *the Post Hoc* analysis, it can be explained as follows:

1. There was no significant difference between the control group and the group administered the extract dose of 100 mg/KgBB (P1) with a value of p = 0.153
2. There was a significant difference between the control group and the group given a dose of 200 mg/KgBB (P2) with a value of p = 0.001
3. There was a significant difference between the group administering a dose of 100 mg/KgBB (P1) and the group administering a dose of 200 mg/KgBB (P2) with a value of p = 0.045

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Average Graph of Mouse Leukocyte Count

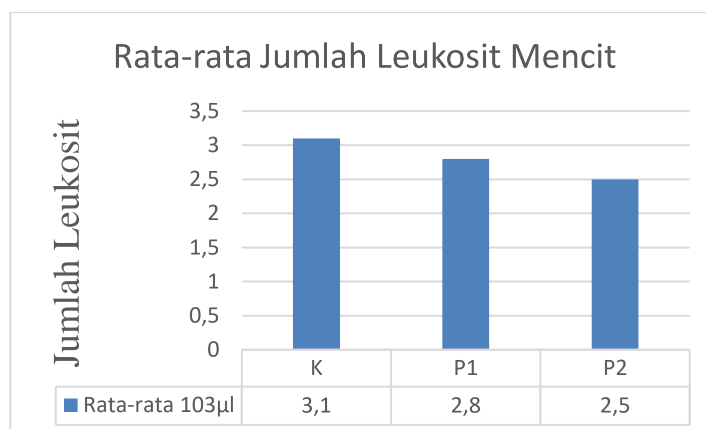

Figure 1. Graph of Average Leukocyte Count Between Treatment Groups

In the control group with the administration of cigarette smoke and water, the average number of leukocytes in the control group was 3.1 x $10^{3µl,}$ the number was lower than the normal number of leukocytes in mice, which was 0.4 – 1 x 10^3/µl (Sylvia, 1995). This is because the nicotine content is found in cigarette smoke. Nicotine can cause *leocytosis* with an increase in hormones such as *ephinephrine* and cortisol (Ragil, 2016). Nicotine induces *ketocolamin* and steroid hormones from the adrenal glands. This can trigger an increase in the levels of a number of endogenous hormones such as *ephinephrine* and cortisol, which in turn increases the number of leukocytes. Smoke will irritate breathing which also contributes to an increase in the number of leukocytes. This is in accordance with the theory that a person who is exposed to cigarette smoke for a long period of time continuously has a 20-25% higher number of leukocytes than a person who does not (Dewi, et al., 2018).

In the second group (P1) that was given a cigarette smoke treatment and an extract with a dose of 100 mg/kgBB, the average number of leukocytes of the P1 group was lower when compared to the control group of 2.8 x 10^3/µl

In the third group (P2) that was given a cigarette smoke treatment and given an extract with a dose of 200 mg/kgBB, the average number of leukocytes in the P2 group was also lower when compared to the control group, which was 2.5 x 10^3/µl.

The decrease in the number of leukocytes in the P1 and P2 groups is likely due to the presence of antioxidants contained in the extract of n-butanol of *Dewandaru* fruit. Antioxidants are compounds that are able to neutralize or reduce the negative impact of free radicals in the body (Winarsi, 2007), one of which is free radicals contained in cigarette smoke. The antioxidant capacity of *dewandaru* fruit with n-butanol solvent in vitro has the highest antioxidant capacity (Santoso, 2018). Based on the results of phytochemical screening, n-butanol extract of *dewandaru* fruit also contains flavonoids. Flavonoids are essential components of natural antioxidants (Jakub, T and Karel, S., 2016). Flavonoid compounds are non-enzymatic antioxidants or chain-breaking antioxidants. In vitro flavonoids have been shown to have very strong biological effects. As antioxidants, flavonoids can inhibit the clot of blood vessels and can dilate (relax) blood vessels. (Winarsi, 2007). So that oxygen transport in the body becomes smoother.

CONCLUSION

Based on the administration of N-Butanol extract, *dewandaru* fruit (*Eugenia uniflora L.*) had an effect on reducing the number of leukocytes at a dose of 100 mg/KgBb and a dose of 200 mg/KgBb in mice exposed to cigarette smoke. Cigarette smoke has an effect on the total number of leukocytes due to free radicals. The antioxidants contained in the fruit of the *dewandaru* (*Eugenia uniflora L.)* can react and are able to neutralize free radicals, so that by giving the extract of the *dewandaru* fruit (*Eugenia uniflora L.)* can reduce the total number of leukocytes.

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