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Effectiveness of Okra (*Abelmoschus esculentus* (L.) Moench) as an Ethnomedicine Based Anticancer Agent through Teratogenic Risk Testing

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ABSTRACT

Okra (*Abelmoschus esculentus* L.) contains lectin compounds known for their potential anticancer properties. However, limited research has examined its possible teratogenic effects—adverse impacts that may cause fetal abnormalities. The urgency of this study lies in assessing the safety of okra extract as a natural anticancer candidate. This research aimed to evaluate the teratogenic effects of okra fruit extract using an *in vivo* model as a preliminary step toward developing safe ethnomedicine-based anticancer agents. The study employed a Completely Randomized Design (CRD) with one control and three treatment groups. Female mice (*Mus musculus* L., DDW strain) received 2%, 4%, and 6% ethanol extracts of okra orally at a dose of 0.01 mL/g body weight daily from gestation days 0–10. The control group received no extract. Observations included maternal and fetal body weights, number of fetuses, liver morphology, and congenital malformations such as cleft palate and hydrocephalus. Data were analyzed using ANOVA ($p < 0.05$) followed by Bonferroni Post Hoc tests in SPSS version 23. The results showed that okra extract significantly affected maternal weight, fetal number, and fetal weight, as well as liver morphology and cleft palate incidence. These findings indicate that although okra exhibits anticancer potential, its teratogenic risks must be carefully evaluated.

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Introduction

Cancer is a disease in which body cells grow uncontrollably and have the potential to invade and spread to other parts of the body. This condition has become a global health problem that continues to increase significantly. In 2018, an estimated 18.1 million new cancer cases and 9.6 million cancer-related deaths were reported worldwide (Bray et al., 2018). Despite advances in conventional treatments such as surgery, chemotherapy, radiation therapy, and immunotherapy, significant challenges remain in achieving long-term survival among cancer patients. Cancer continues to be a leading cause of death and poses a serious threat to global life expectancy.

In recent decades, complementary and alternative therapies have gained

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increasing attention as potential strategies for cancer management. The development of new anticancer agents is crucial due to the growing number of cancer cases exhibiting resistance to existing therapies (Elkhalifa et al., 2021). One promising alternative approach is the use of ethnomedicine. Okra (*Abelmoschus esculentus* (L.) Moench) is a plant that has been widely used in traditional medicine (Elkhalifa et al., 2021). Belonging to the Malvaceae family, okra possesses various pharmacological properties, including antioxidant, antibacterial, antifungal, and anticancer activities (Sami et al., 2019). The active compounds in okra, particularly those from the polyphenol group, have attracted attention for their anticancer potential (Ying et al., 2014; Esan et al., 2018).

Previous studies have demonstrated that okra exhibits angiogenic effects, influences body weight in mice, and significantly affects *p53* gene expression in murine models of breast cancer (Putri, 2020; Putri et al., 2019; Nasution et al., 2019). *In vitro* studies using human breast cancer cells have also shown that lectin proteins isolated from okra can inhibit cell proliferation by up to 63% (Monte et al., 2014).

Figure 1. Okra fruit (*Abelmoschus esculentus* (L.) Moench)

Although numerous studies have supported the development of okra as an anticancer therapy, none have specifically examined its teratogenic potential. In the pharmaceutical context, evaluating potential embryotoxicity is essential. During the early stages of drug development, a new compound must undergo acute, chronic, and developmental toxicity testing. Preclinical evaluation of a drug's long-term safety involves a thorough assessment of its potential cytotoxic, mutagenic, embryotoxic, and teratogenic effects (Schumann, 2010).

Various animal-based testing systems have been developed to screen for potential teratogenic activity. These tests are typically conducted on pregnant laboratory animals—such as mice, rabbits, and primates—by administering the test compound regularly during the period of fetal organogenesis. The fetuses are subsequently examined for the presence of abnormalities or defects (Kochhar, 2010; Fantel, 1982).

Teratogenicity testing is crucial in this research because it enables the evaluation of new substances or compounds for their potential effects on embryonic and fetal development. This testing helps identify possible adverse impacts on fetal health resulting from exposure to specific substances. The results of teratogenicity testing provide important insights for decision-making in drug development, health policy formulation, and the

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protection of human health and the environment. Overall, teratogenicity testing plays a vital role in ensuring the safety and efficacy of substances used across various sectors, including the pharmaceutical, food, and chemical industries—thereby underscoring the importance of this study.

Materials and Methods

Time and Place of Research

This research was conducted from May 2025 to November 2025 at the Central Laboratory of STIKes Widya Husada Medan and the Experimental Animal Care Facility of STIKes Widya Husada Medan.

Research Design

This study employed a Completely Randomized Design (CRD) consisting of three treatment groups with different extract concentrations and one control group. Both the control and treatment groups included six replications, resulting in a total of 24 mice.

The number of replications was determined using the Federer formula:

$$(t-1)\,(n-1) \geq 15$$

Where:
t = number of treatments
n = number of replications

Preparation of Test Materials

Okra fruit was obtained from the medicinal plant cultivation field of STIKes Widya Husada Medan. After collection, 10 kg of okra fruit was washed thoroughly and air-dried at room temperature until it reached the general moisture content requirement for simple herbs. The dried material was then ground into a fine powder and sieved using a B30 mesh sieve.

The ethanol extract of okra fruit was prepared using the maceration method. The powdered okra fruit was placed in a brown glass container and soaked in ethanol until completely submerged. The mixture was stirred and left to stand overnight. The filtrate was then collected, while the residue was re-soaked in ethanol until a clear filtrate was obtained. The combined filtrates were concentrated using a rotary evaporator to produce a thick extract.

Preparation of Test Animals

The experimental animals used were 24 healthy, fertile female mice (*Mus musculus* L.) of the DDW strain, aged 8–11 weeks and weighing 25–30 g. The mice were housed in clean cages and provided with food and water *ad libitum*. Handling of the experimental animals complied with the institutional animal ethics guidelines.

Treatment

The test material was administered to pregnant female mice using a gavage needle at a dose of 0.01 mL/g body weight per day. For the control group, pregnant mice were maintained until the 18th day of gestation without receiving any treatment. Meanwhile, mice in the treatment groups were orally administered ethanol extracts of okra at concentrations of 2%, 4%, and 6% through force-feeding once daily from day 0 to day 10 of gestation.

Maternal body weight was measured daily using a digital scale. Fetal condition was observed after preservation, beginning with washing in physiological solution followed by fixation in Bouin's solution.

The treatments were as follows:

a. Negative control: no treatment
b. Treatment I: administration of 2% ethanol extract (0.01 mL/g body weight/day)
c. Treatment II: administration of 4% ethanol extract (0.01 mL/g body weight/day)
d. Treatment III: administration of 6% ethanol extract (0.01 mL/g body weight/day)

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Research Parameters

The parameters observed in this study included:

a. Maternal body weight
b. Fetal weight
c. Number of fetuses
d. Liver morphology
e. Cleft palate condition
f. Hydrocephalus

Statistical Analysis

Data obtained from each observation parameter were recorded and tabulated. Quantitative data (dependent variables) were analyzed for their significance relative to the treatment groups (independent variables) using SPSS version 23.

The analysis sequence began with normality and homogeneity tests. If the results showed $p < 0.05$, data transformation was performed, followed by non-parametric testing. Differences between two treatments were analyzed using the Mann–Whitney test. If the normality and homogeneity tests showed $p > 0.05$, data were analyzed using one-way ANOVA. When significant differences were found ($p < 0.05$), the analysis was continued with a Bonferroni Post Hoc test at a 5% significance level.

Results and Discussion

Table 1. Effects of Okra (Abelmoschus esculentus L.) Fruit Extract

Variable	Control	P1	P2	P3	p-value
Maternal Body Weight (g)	27.50 ± 1.05^{a}	27.50 ± 1.05^{a}	28.50 ± 1.05^{ab}	29.50 ± 1.05^{b}	0.010
Number of Fetuses	7.83 ± 0.75^{a}	6.83 ± 0.75^{ab}	6.17 ± 0.75^{b}	6.17 ± 1.17^{b}	0.011
Fetal Body Weight (g)	12.83 ± 0.75^{a}	11.83 ± 0.75^{ab}	7.83 ± 5.31^{b}	11.17 ± 1.83^{ab}	0.036
Cleft Palate	0.00 ± 0.00^{a}	0.00 ± 0.00^{a}	0.50 ± 0.55^{ab}	0.67 ± 0.52^{b}	0.010
Hydrocephalus	0.00 ± 0.00	0.17 ± 0.41	0.33 ± 0.52	0.50 ± 0.55	0.241

The results of this study showed significant differences in several reproductive variables and fetal development parameters following the administration of okra fruit extract. In the control group, maternal body weight was recorded at 27.50 ± 1.05 g, with an average fetal count of 7.83 ± 0.75 and a fetal weight of 12.83 ± 0.75 g. No congenital abnormalities, such as cleft palate or hydrocephalus, were observed, making this group the baseline reference for normal conditions.

In the first treatment group (P1), maternal body weight did not differ significantly from the control (27.50 ± 1.05 g). The number of fetuses decreased slightly to 6.83 ± 0.75, and fetal weight declined modestly to 11.83 ± 0.75 g. No cases of cleft palate were observed in this group, although instances of hydrocephalus (0.17 ± 0.41) began to appear but were statistically insignificant. These findings suggest that low-dose okra extract administration remained relatively safe.

In contrast, toxic effects began to emerge in the second treatment group (P2). Maternal body weight increased to 28.50 ± 1.05 g, but the number of fetuses decreased significantly to 6.17 ± 0.75, and fetal weight

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dropped sharply to 7.83 ± 5.31 g. Moreover, cases of cleft palate (0.50 ± 0.55) and hydrocephalus (0.33 ± 0.52) were recorded, although the latter remained statistically insignificant. These results indicate that moderate doses of okra extract negatively affected fetal growth and development.

In the third treatment group (P3), maternal body weight increased significantly to 29.50 ± 1.05 g, while the number of fetuses remained low at 6.17 ± 1.17. Fetal weight increased again to 11.17 ± 1.83 g; however, the incidence of cleft palate rose to 0.67 ± 0.52, showing a significant difference compared with the control group. Hydrocephalus cases also increased to 0.50 ± 0.55, though not significantly. These findings indicate that while higher doses of okra extract increased maternal and fetal weight, they also elevated the teratogenic risk, particularly in the form of cleft palate.

Overall, this study demonstrated that okra fruit extract influenced maternal body weight, fetal count, and fetal weight, and posed a teratogenic risk manifested as cleft palate. These effects were evident at moderate to high doses, while lower doses appeared relatively safe. Therefore, although okra shows promise as an anticancer agent, its use requires caution due to its potential negative effects on reproduction and fetal development.

Previous studies have similarly reported reproductive effects of *Abelmoschus esculentus* extracts. Ogunwole et al. (2022) found that methanolic extracts of okra fruit significantly disrupted ovarian and uterine cytology in Wistar rats, suggesting potential reproductive risks at certain doses. Bello et al. (2009) also observed reductions in testicular weight, hormonal imbalances, and inflammation of female reproductive organs following okra extract administration, indicating both short- and long-term reproductive toxicity. Supporting these observations, an embryotoxicity study in zebrafish by Veshalini et al. (2022) revealed that okra extract induced developmental abnormalities such as scoliosis and pericardial edema, consistent with the cleft palate and possible hydrocephalus findings in the present study. Conversely, Ajewole et al. (2024) reported that administration of dried okra fruit and leaves increased progesterone levels in female goats, suggesting that okra may modulate female hormonal parameters. Taken together, the literature indicates that while okra possesses hormonal and therapeutic potential, high doses or specific extract types may exert adverse effects on reproduction and fetal development—consistent with the patterns observed in the P2 and P3 treatments in this study.

Table 2. Effects of Okra (Abelmoschus esculentus L.) Fruit Extract on Liver Morphology and Liver Weight in Mice (Mean ± SD)

Group	Liver Morphology (Mean ± SD)	Liver Weight (g, Mean ± SD)
Control	1.33 ± 0.52[a]	1.25 ± 0.07[a]
P1 (2%)	1.17 ± 0.41[a]	1.28 ± 0.05[a]
P2 (4%)	2.17 ± 0.41[b]	1.40 ± 0.07[b]
P3 (6%)	2.50 ± 0.55[b]	1.55 ± 0.07[c]

Based on the results of the descriptive analysis, there were significant differences in liver morphology and liver weight among the treatment groups. The average liver morphology score in the control group was 1.33 ± 0.52, in group P1 it was 1.16 ± 0.41, in group P2 it was 2.17 ± 0.41, and in group P3 it was 2.50 ± 0.55. These results indicate a progressive change in liver morphology from normal to slightly pale and pale in the treatment groups, particularly in P2 and P3. The ANOVA test showed significant differences among the groups ($p < 0.001$), and the Bonferroni post hoc test confirmed

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that the control and P1 groups differed significantly from P2 and P3, whereas there was no significant difference between the control and P1.

Regarding liver weight, the mean values were 1.25 ± 0.07 g for the control group, 1.28 ± 0.05 g for P1, 1.40 ± 0.07 g for P2, and 1.55 ± 0.07 g for P3. ANOVA analysis revealed significant differences among the groups ($p < 0.001$). The Bonferroni post hoc test indicated that the control and P1 groups did not differ significantly, but both were significantly different from P2 and P3. Additionally, a significant difference was observed between P2 and P3, indicating a dose-dependent increase in liver weight with higher treatment concentrations.

Overall, these data demonstrate that okra extract treatment significantly affected the liver morphology and liver weight of mice. Groups P2 and P3 tended to exhibit pathological morphological changes (from slightly pale to pale) and increased liver weight compared with the control group, which may reflect a biological response to the treatment agent.

Several previous studies have reported that extracts or active compounds from *Abelmoschus esculentus* (okra) can influence liver structure and weight through both hepatoprotective and toxicological mechanisms. Wahyuningsih et al. (2020) found that okra fruit methanolic extract (OPME) exerted a hepatoprotective effect in mice with sodium nitrite–induced hepatotoxicity, improving liver histology by reducing tissue damage and restoring biochemical parameters. Similarly, Belkhodja et al. (2025) reported that polyphenolic fractions of okra exhibited significant hepatoprotective activity in a carbon tetrachloride–induced liver injury model by improving the hepato-somatic index and restoring liver function markers. Another study by Kwok et al. (2025) summarized several *in vivo* experiments showing that okra can increase antioxidant enzymes (catalase, SOD, and GSH), reduce liver injury markers such as AST, ALT, ALP, and GGT, and mitigate steatosis and inflammation in hepatic tissue.

These findings support the present study's observation that administration of okra extract at moderate to high doses induces morphological changes toward paleness and increases liver weight. Such morphological alterations may reflect an adaptive or pathological hepatic response—such as lipidosis, edema, or cellular hypertrophy—resulting from oxidative or metabolic stress induced by the treatment. Although okra is generally recognized for its hepatoprotective properties against chemical-induced liver injury, the results of this study suggest that at certain concentrations, it may exert the opposite effect, leading to structural deterioration and increased liver weight, which could represent early signs of hepatocellular hypertrophy or substance accumulation within liver cells.

Conclusions

The administration of okra fruit extract to mice significantly affected reproductive parameters, fetal development, and liver function. Low doses were relatively safe and did not cause significant changes, whereas medium to high doses resulted in a reduction in fetal number and weight, the appearance of teratogenic risks such as cleft palate, and morphological alterations along with increased liver weight that may indicate pathological conditions. Therefore, although okra possesses potential biological benefits, its use at high doses requires caution, as it may induce reproductive and hepatic toxicity.

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