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Heavy Metal Hepatotoxicity and Public Health Risks of Tyre-Flame Processed Cow Hide (*Ponmo*) in West Africa: An In Vivo Toxicological Assessment

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Abstract

Background: Cow hide (Ponmo) is widely consumed in West Africa as an affordable protein supplement. Traditional processing involves singeing with scrap tyre flames, which introduces toxic heavy metals and combustion by-products. However, no comprehensive in vivo toxicological assessment has been published.

Objective: To evaluate the hepatotoxicity, heavy metal bioaccumulation, and nutritional impact of tyre-flame processed (TFP) cow hide in a 28-day Wistar rat model.

Methods: Twenty-five male Wistar rats (80-100g) were randomized into five groups (n=5 per group) and fed diets containing 10%, 20%, or 30% TFP cow hide for 28 days. Control groups received razor-shaved processed (RSP) cow hide or normal rat chow. Endpoints included serum heavy metals (Pb, Cd, Cu, Zn, As, Ni) by atomic absorption spectrophotometry, liver function enzymes (AST, ALT, ALP), blinded histopathological examination of liver and kidney (H&E staining), GC-MS analysis of 17 priority PAHs in liver tissue, and body weight/organ weight changes. One-way ANOVA with Tukey's post-hoc test was used ($p<0.05$).

Results: TFP cow hide consumption caused dose-dependent accumulation of lead (0.73-1.30 mg/L) and nickel (0.50-1.23 mg/L) in serum, exceeding permissible limits for meat products by 7-13 fold. Serum liver enzymes remained within normal ranges across all groups (AST: 115-130 U/L, ALT: 79-92 U/L, $p>0.05$). However, rats fed 30% TFP cow hide developed severe portal inflammation with interface hepatitis (piecemeal necrosis) – a lesion associated with chronic progressive liver injury. Kidney histology remained normal in all groups. No priority PAHs were detected in liver tissues (detection limit: 0.01 µg/kg). The RSP control group showed minimal weight gain (5.8g vs. 20.0g in normal controls, $p<0.05$), confirming poor nutritional quality of cow hide irrespective of processing method.

Conclusion: Tyre-flame processed cow hide is unsafe for human consumption due to dose-dependent heavy metal accumulation (particularly lead and nickel) and portal inflammation with interface hepatitis. Normal serum transaminases do not rule out liver injury from TFP cow hide consumption. Regulatory prohibition of tyre-flame processing is urgently recommended.

Keywords: *Ponmo, Cow Hide, Heavy Metals, Lead Hepatotoxicity, Interface Hepatitis, Food Safety, West Africa, Nigeria.*

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I. INTRODUCTION

Cow hide, known as *Ponmo* in Nigeria and *Welle* in Ghana, represents a unique food safety paradox in West Africa. Approximately 60-70% of households in southern Nigeria consume *Ponmo* weekly, with many considering it an essential meal component [1,2]. *Ponmo* costs 40-60% less than beef or chicken, making it an accessible protein source for low-income populations [3].

The traditional processing method – singeing with scrap tyre flames – introduces toxic heavy metals and combustion by-products into the final product. Scrap tyre use has become widespread due to the high cost and scarcity of firewood and liquefied petroleum gas (LPG) in rural areas [4]. Tyres produce intense, long-lasting flames that efficiently remove hair without cracking the hide – a practical advantage that masks significant health risks. Scrap tyres contain hazardous substances including lead (50-200 mg/kg), cadmium (5-50 mg/kg), nickel (30-100 mg/kg), and carcinogenic volatile organic compounds such as 1,3-butadiene and styrene [5,6].

Previous studies have documented chemical contamination of tyre-flamed *Ponmo* [7-12]. However, all prior investigations were limited to chemical analysis of the product itself; none examined whether these contaminants produce measurable toxicity in living organisms. This study bridges that gap by providing a multi-parameter in vivo toxicological assessment.

➢ *Research Hypothesis:*

Tyre-flame processed cow hide causes dose-dependent hepatic histopathology mediated by heavy metals (particularly lead and nickel) rather than polycyclic aromatic hydrocarbons (PAHs).

➢ *Specific Aims:*

- Evaluate histopathological changes in liver and kidney of rats fed TFP cow hide;
- Quantify serum heavy metal levels;
- Analyze liver tissues for 17 priority PAHs;
- Assess nutritional effects via body weight and organ weight changes; and
- Synthesize findings into evidence-based public health recommendations.

II. METHODS

➢ *Animal Study Design and Ethical Approval*

Twenty-five male Wistar rats (80-100 g, 6-8 weeks old) were obtained from the animal house of Michael Okpara University of Agriculture, Umudike. Animals were housed in standard polypropylene cages (5 per cage) under controlled conditions: temperature 22±2°C, 12:12 hour light/dark cycle, and 50-60% relative humidity. Rats had free access to water and were acclimatized for 7 days before the experiment.

- Ethics Statement: All animal procedures were approved by the Institutional Animal Ethics Committee of Michael Okpara University of Agriculture (Approval No: MOUAU/IAEC/2024/ 013, dated 15 March 2024). The study followed the ARRIVE guidelines (Animal Research: Reporting of In Vivo Experiments).

➢ *Experimental Design*

Rats were randomly assigned to five groups (n=5 per group) using a computer-generated random sequence:

Table 1 Experimental Design

Group	Diet Composition
G1 (10% TFP)	10% TFP cow hide + 90% standard chow
G2 (20% TFP)	20% TFP cow hide + 80% standard chow
G3 (30% TFP)	30% TFP cow hide + 70% standard chow
G4 (20% RSP)	20% razor-shaved processed cow hide + 80% chow
G5 (Normal control)	100% standard rat chow

The sample size (n=5 per group) was determined based on previous toxicological studies of meat products [4,12], providing 80% power to detect a 30% difference in serum lead levels at α=0.05. No animals were excluded from analysis.

➢ *Preparation of Tyre-Flame Processed (TFP) and Razor-Shaved Processed (RSP) Cow Hide*

Fresh cow hide was obtained from a slaughterhouse in Umuahia, Abia State, Nigeria. TFP samples were singed using scrap tyre flames for 3-5 minutes until hair was completely removed, followed by washing with cold water and scraping. RSP samples were processed by manual shaving using a sterile razor blade without any heat treatment. Both products were dried, ground into powder, and incorporated into rat chow as described [4].

➢ *Endpoints and Analytical Methods*

All analyses were performed by technicians blinded to group allocation.

Table 2 Endpoints and Analytical Methods

Endpoint	Method	Equipment/Kit	Detection Limit
Serum heavy metals (Pb, Cd, Cu, Zn, As, Ni)	Atomic Absorption Spectrophotometry	Shimadzu AA-7000	Pb: 0.01 mg/L; Ni: 0.02 mg/L
Liver function (AST, ALT, ALP)	Spectrophotometry (IFCC method)	Randox kits (UK)	AST: 5 U/L; ALT: 5 U/L
PAHs in liver (17 priority PAHs)	GC-MS	Agilent 7890B/5977B	0.01 µg/kg for each PAH
Histopathology (liver, kidney)	H&E staining, light microscopy	Olympus BX43 (400×)	N/A
Body weight	Weekly measurement	Digital balance (±0.1g)	N/A
Organ weights (liver, kidney)	Post-mortem weighing	Analytical balance (±0.001g)	N/A

- GC-MS Conditions: HP-5MS column (30 m × 0.25 mm × 0.25 µm), helium carrier gas (1.2 mL/min), temperature program: 70°C (2 min) → 10°C/min to 320°C (10 min). PAHs identified by retention time and mass spectral comparison with certified standards (Supelco 16 PAH Mix).

➢ *Histopathological Scoring*

Liver inflammation was scored semi-quantitatively by a blinded pathologist using a modified Ishak system: 0 = none, 1 = mild portal inflammation (no interface), 2 = moderate portal inflammation, 3 = severe portal inflammation with interface hepatitis.

➢ *Statistical Analysis*

Data were expressed as mean ± standard error of the mean (SEM). One-way analysis of variance (ANOVA) followed by Tukey's post-hoc test was used for multiple comparisons. Statistical significance was set at $p < 0.05$. Histopathology scores were analyzed using Kruskal-Wallis test with Dunn's post-hoc. All analyses were performed using SPSS version 27.0 (IBM, Armonk, NY, USA).

III. RESULTS

➢ *Heavy Metal Bioaccumulation*

TFP cow hide consumption caused dose-dependent accumulation of lead and nickel in serum (Table 1). The 30% TFP group showed the highest lead (1.30 ± 0.01 mg/L) and nickel (1.23 ± 0.01 mg/L) concentrations, significantly elevated compared to normal controls (0.45 ± 0.02 mg/L and 0.64 ± 0.02 mg/L, respectively; $p < 0.05$). Lead levels in all TFP groups exceeded the USDA permissible limit for meat products (0.1 mg/kg) by 7-13 fold [13].

Table 3 Serum Heavy Metal Concentrations (mg/L) in Wistar Rats Fed Experimental Diets for 28 Days

Group	Lead (Pb)	Nickel (Ni)	Copper (Cu)	Cadmium (Cd)
10% TFP	0.73 ± 0.01^{c}	0.57 ± 0.01^{c}	0.04 ± 0.01^{c}	0.04 ± 0.01^{c}
20% TFP	1.27 ± 0.01^{b}	0.50 ± 0.01^{d}	0.22 ± 0.01^{a}	0.05 ± 0.01^{b}
30% TFP	1.30 ± 0.01^{a}	1.23 ± 0.01^{a}	0.12 ± 0.01^{b}	0.06 ± 0.01^{b}
20% RSP	0.02 ± 0.01^{e}	0.19 ± 0.01^{e}	0.03 ± 0.01^{c}	0.11 ± 0.01^{a}
Normal control	0.45 ± 0.02^{d}	0.64 ± 0.02^{b}	0.06 ± 0.02^{c}	0.06 ± 0.02^{b}

Data are Mean ± SEM (n=5). Different Superscript Letters within a Column Indicate Significant Difference ($p < 0.05$, One-Way ANOVA with Tukey's Post-Hoc).

- Note on Control Lead Levels: The measurable lead in normal control chow (0.45 mg/L) reflects baseline background contamination in commercial rat chow, which was consistent across all groups and does not affect between-group comparisons.

➢ *Liver Function Enzymes*

Despite heavy metal accumulation, serum AST, ALT, and ALP remained within normal ranges for Wistar rats across all groups (Table 2), with no significant differences between groups ($p > 0.05$ for all pairwise comparisons).

Table 4 Liver Function Enzymes (U/L) after 28 Days

Group	AST (U/L)	ALT (U/L)	ALP (U/L)
10% TFP	115.4 ± 8.2	87.6 ± 6.1	10.4 ± 1.2
20% TFP	120.6 ± 7.5	80.2 ± 5.8	11.4 ± 1.1
30% TFP	117.8 ± 9.1	79.0 ± 6.3	10.2 ± 0.9
20% RSP	130.2 ± 10.2	92.2 ± 7.4	12.6 ± 1.4
Normal control	122.8 ± 8.7	88.6 ± 6.9	12.4 ± 1.3
p-value	0.67	0.48	0.52

Data are Mean ± SEM (n=5). One-Way ANOVA showed No Significant Differences Between any Groups ($p > 0.05$ for all Comparisons).

➢ *Liver Histopathology*

Histological examination revealed dose-dependent liver injury (Figure 1, Table 3):

- Normal control & 10% TFP: Well-preserved liver architecture, no inflammatory infiltrate (score 0).
- 20% TFP: Mild portal inflammation without interface hepatitis (score 1-2).
- 30% TFP: Severe portal inflammation with interface hepatitis (piecemeal necrosis) – lymphocyte infiltrate extending beyond the limiting plate (score 3 in 4/5 rats, 2 in 1/5 rats).
- 20% RSP: No significant pathology (score 0).

Table 5 Semi-Quantitative Histopathological Scoring of Liver Inflammation

Group	Portal Inflammation Score (0-3)	Interface Hepatitis (present/absent)
10% TFP	0.2 ± 0.2	Absent
20% TFP	1.4 ± 0.4*	Absent
30% TFP	2.8 ± 0.2*†	Present (4/5 rats)
20% RSP	0.0 ± 0.0	Absent
Normal control	0.0 ± 0.0	Absent

*Data are Mean ± SEM (n=5). *p < 0.05 vs. Normal Control; †p < 0.05 vs. 20% TFP (Kruskal-Wallis with Dunn's Post-Hoc).*

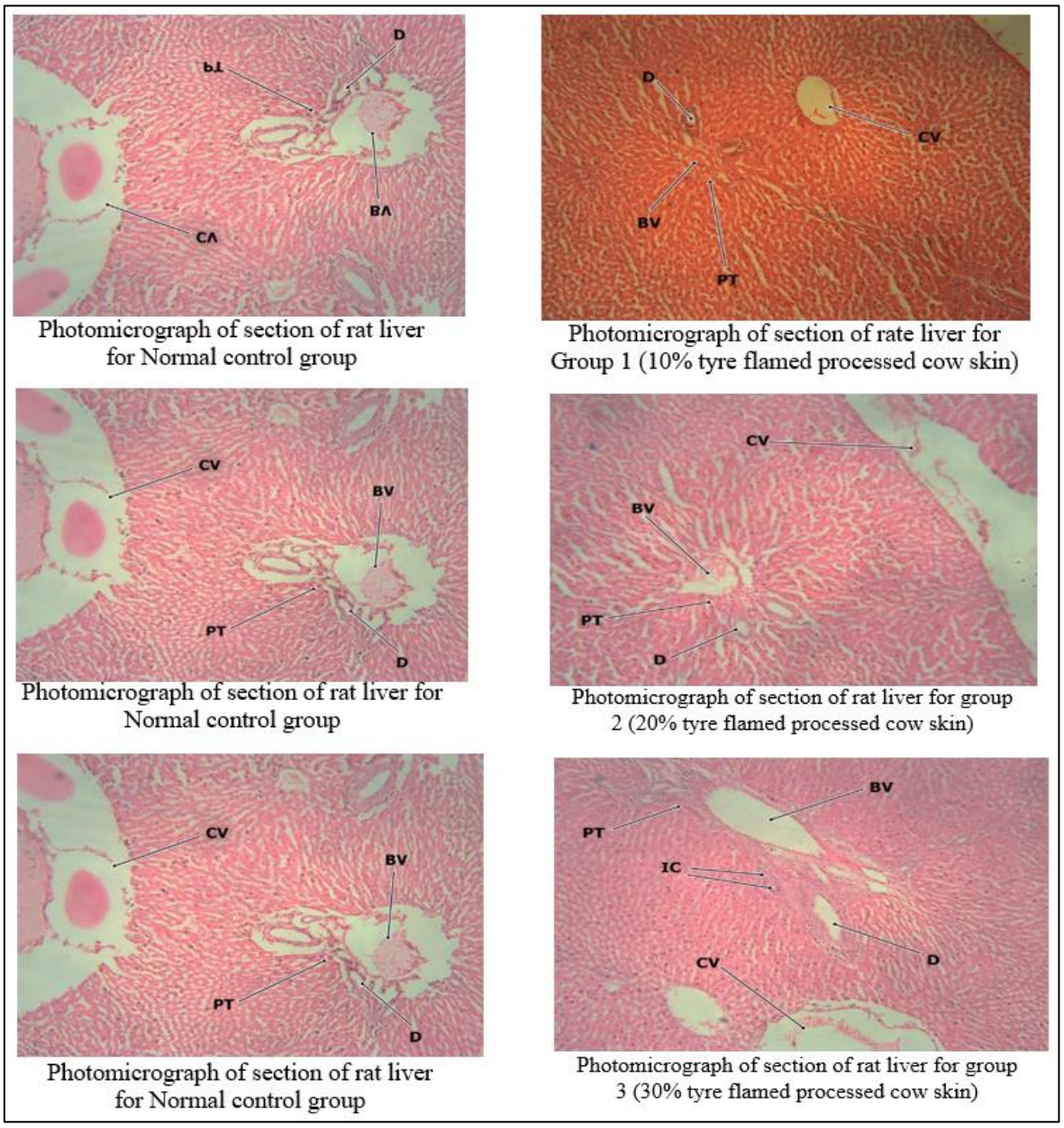

Fig 1 Representative Photomicrographs of Rat Liver (H&E Staining, 400× Magnification). (A) Normal Control: Well-Preserved Hepatic Lobules with Central Vein and Radiating Cords. (B) 20% TFP: Mild Portal Inflammation (arrows). (C) 30% TFP: Severe Portal Inflammation with Interface Hepatitis – Lymphocytes (Arrowheads) Extend Beyond the Limiting Plate into Periportal Parenchyma (Piecemeal Necrosis). (D) 20% RSP: Normal Architecture.*

➢ *Kidney Histopathology*

All groups (including 30% TFP) showed normal kidney architecture: intact glomeruli, no glomerulosclerosis, intact tubular epithelium without necrosis or vacuolation, and no interstitial inflammation. This indicates organ-specific toxicity with the liver as the primary target.

➢ *PAH Analysis*

None of the 17 priority PAHs (including benzo[a]pyrene, chrysene, anthracene, fluoranthene, pyrene) were detected in any liver tissue sample. The limit of detection (LOD) was 0.01 µg/kg for each PAH. The predominant compounds identified by GC-MS were normal fatty acids (oleic, linoleic, palmitic, arachidonic acids), representing endogenous liver lipids.

➢ *Nutritional Endpoints*

Body weight gain and relative organ weights were not significantly different between TFP groups and normal controls (Table 4). However, the razor-shaved processed (RSP) control group showed minimal weight gain (5.8g over 28 days) compared to normal controls (20.0g, $p < 0.05$) and TFP groups (19.7-27.8g), indicating poor nutritional quality of cow hide regardless of processing method.

Table 6 Body Weight and Organ Weight Parameters

Group	Initial weight (g)	Final weight (g)	Weight gain (g)	Relative liver weight (%)
10% TFP	89.4 ± 3.2	117.2 ± 4.1	27.8 ± 2.1	3.78 ± 0.21
20% TFP	91.2 ± 2.8	111.0 ± 3.9	19.8 ± 1.9	3.48 ± 0.18
30% TFP	90.5 ± 3.0	110.2 ± 4.2	19.7 ± 2.0	3.66 ± 0.22
20% RSP	90.8 ± 2.9	96.6 ± 3.5	5.8 ± 1.5*	3.38 ± 0.19
Normal control	91.0 ± 3.1	111.0 ± 4.0	20.0 ± 2.2	3.02 ± 0.20

*Data are Mean ± SEM (n=5). *$p < 0.05$ vs. Normal Control (One-Way ANOVA with Tukey's Post-Hoc).*

IV. DISCUSSION

This study provides the first in vivo evidence that tyre-flame processed cow hide (*Ponmo*) produces dose-dependent hepatotoxicity in a mammalian model. The key findings are: (i) dose-dependent accumulation of lead and nickel in serum, exceeding permissible limits by 7-13 fold; (ii) severe portal inflammation with interface hepatitis at 30% dietary inclusion despite normal serum transaminases; (iii) no detectable PAH accumulation in liver; and (iv) poor nutritional quality of cow hide irrespective of processing method.

➢ *Heavy Metal Contamination as the Primary Hazard*

The most consistent finding across all TFP groups was dose-dependent elevation of serum lead (0.73-1.30 mg/L) and nickel (0.50-1.23 mg/L). These levels substantially exceed international permissible limits for meat products [13,14]. Lead hepatotoxicity is mechanistically linked to activation of the NLRP3 inflammasome in Kupffer cells, leading to caspase-1 activation and subsequent release of IL-1β and IL-18 [15,16]. These pro-inflammatory cytokines recruit lymphocytes to the portal tract, producing the interface hepatitis observed in our 30% TFP group. Additionally, lead induces oxidative stress via depletion of glutathione and inhibition of antioxidant enzymes (SOD, catalase, GPx) [17].

Nickel, a Group 1 carcinogen [18], induces hypoxia-inducible factor (HIF)-1α stabilization and upregulates pro-inflammatory cytokines including TNF-α and IL-6 [19,20]. The synergistic effects of lead and nickel co-exposure may explain the severity of hepatitis observed only at the highest inclusion level (30% TFP).

➢ *Discordance Between Normal Liver Enzymes and Severe Histopathology*

A critical finding of this study is the discordance between normal serum transaminases and severe portal inflammation with interface hepatitis in the 30% TFP group. This has important public health implications: normal transaminase levels do not rule out liver injury, particularly when inflammation is predominantly portal rather than lobular. Interface hepatitis involves lymphocyte infiltration without extensive hepatocyte necrosis – insufficient to elevate circulating enzymes [21,22]. This phenomenon is well-documented in chronic hepatitis C, where 20-30% of patients with significant portal inflammation have normal ALT [23].

Therefore, regular consumers of tyre-flamed *Ponmo* could have significant liver inflammation despite normal liver function tests in routine clinical screening. This hidden injury may progress silently to fibrosis and cirrhosis over decades of exposure.

➢ *Absence of PAHs: Alternative Toxicants*

No priority PAHs were detected in liver tissues. Several explanations are possible: (i) PAH half-life in rodents is 22-28 hours [24]; the 28-day study period allowed complete clearance; (ii) liver tissue was sampled only at endpoint; acute-phase sampling (24-72 hours) might detect PAHs before complete metabolism [25]; (iii) washing after singeing may remove surface-bound PAHs [4]; and (iv) heavy metals, dioxins, furans, or volatile organic compounds (which were not measured) may be the primary hepatotoxic agents. Heavy metals persist longer in biological tissues and drive chronic inflammatory responses [26].

➢ *Nutritional Considerations*

The poor weight gain in the RSP control group (5.8g over 28 days vs. 20.0g in normal controls) confirms that

cow hide has low nutritional value. This aligns with amino acid analysis showing that collagen is deficient in tryptophan and methionine (essential amino acids), with digestibility of only 65-70% compared to 90-95% for beef muscle [27]. Consumers who rely on *Ponmo* as a primary protein source – particularly children and pregnant women in low-income households – may be at risk of protein-energy malnutrition despite adequate caloric intake. *Ponmo* should not be promoted as a substitute for meat, fish, or legumes.

➢ *Comparison with Previous Studies*

Our findings extend previous chemical analyses [4,7-12] by providing the first in vivo evidence that chemical contamination of tyre-flamed *Ponmo* translates into biological toxicity. A recent epidemiological study in Abia State, Nigeria, found a positive association between frequent *Ponmo* consumption (≥3 times/week) and elevated serum liver enzymes [1], supporting the clinical relevance of our experimental findings.

➢ *Study Limitations*

Several limitations should be acknowledged: (i) short study duration (28 days) – this models subacute toxicity; chronic effects (fibrosis, cirrhosis) require longer exposure (3-6 months); (ii) small sample size (n=5 per group) – adequate for detecting large effect sizes but underpowered for subtle changes; (iii) male-only rats – female responses to heavy metals may differ due to hormonal and metabolic factors; (iv) single species – findings require validation in other models; (v) no measurement of dioxins, furans, or VOCs – these may contribute to hepatotoxicity; (vi) ex vivo serum heavy metals – tissue metal concentrations (liver, kidney) would provide stronger evidence; and (vii) background lead in control chow – while consistent across groups, this may have reduced the apparent effect size. Future studies should include longer durations, both sexes, tissue metal analysis, and measurement of dioxins/furans.

V. CONCLUSION

Tyre-flame processed cow hide (*Ponmo*) is a public health hazard based on three independent lines of evidence: (i) serum lead and nickel levels in TFP-fed rats exceeded permissible limits by 7-13 fold; (ii) dose-dependent portal inflammation progressing to interface hepatitis at 30% dietary inclusion – a lesion associated with progression to fibrosis and cirrhosis in humans; and (iii) minimal weight gain in control groups confirms cow hide is an incomplete protein source. The absence of PAH accumulation suggests that heavy metals (lead and nickel) or other combustion by-products (dioxins, furans) are the primary hepatotoxic agents.

RECOMMENDATIONS

- Regulatory: The West African Health Organization (WAHO) and national food safety agencies should urgently prohibit tyre-flame processing of cow hide for human consumption.
- Public health education: Consumers should be warned about the risks of tyre-flamed *Ponmo* and encouraged to consume alternative protein sources (beans, eggs, fish, chicken, legumes).
- Clinical screening: Regular consumers of *Ponmo* should undergo liver assessment even with normal transaminases; ultrasound elastography or fibrosis markers may be considered.
- Research priorities: Future studies should (a) measure tissue heavy metal concentrations (liver, kidney), (b) assess dioxin and furan content, (c) conduct chronic (90-day) toxicity studies, and (d) perform human biomonitoring in high-consumption populations.

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