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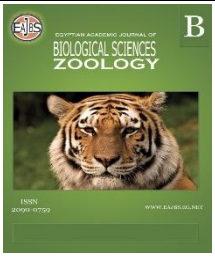

A New Trial to Induce Metabolic Disorders in Rats by Dietary Egg Yolk Supplementation with High-Fat Diet: A Comprehensive Study on BloodProfile and Selected Biochemical and Hormonal Parameters

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ABSTRACT

This study aimed to establish a novel model for inducing metabolic disorders in male albino rats through the incorporation of egg yolk supplements, in raw and cooked forms, alongside a high-fat diet (HFD). Over two experimental periods 6 and 12 weeks—the morphological, hematological, biochemical, and hormonal impacts of these dietary interventions were assessed. Experimental groups included rats fed HFD alone or in combination with either low or high doses of raw by gastric tube or boiled egg yolk by added to their diet, as well as a group receiving doses of yolk with a normal diet. Key parameters included body weight progression, liver and internal fat relative weight, complete blood count (CBC), glucose homeostasis markers (FBS, insulin, HbA1c, HOMA-IR), lipid profile (TGs, cholesterol fractions), liver function enzymes, thyroid hormones (TSH, T3, T4), and testosterone hormone. The results demonstrated that HFD and its combination with both low and high-dose raw yolk significantly aggravated obesity indices, insulin resistance and lipid abnormalities, in contrast, yolk supplementation under normal dietary conditions appeared to exert protective or neutral effects on most parameters. These findings suggest that raw egg yolk, particularly in the context of a high-fat diet, can potentiate metabolic disturbances, while its impact is modulated by the dietary background and method of preparation.This experimental model provides valuable insights for future investigations on diet-induced metabolic dysregulation.

INTRODUCTION

Metabolic disorders such as obesity, insulin resistance, dyslipidemia, and non-alcoholic fatty liver disease (NAFLD) represent major global health challenges due to their increasing prevalence and their association with cardiovascular complications and reduced quality of life. Their pathogenesis involves complex interactions among genetic, environmental, and dietary factors, with high-fat and high-sugar diets playing pivotal roles in disease onset and progression (Muscogiuri *et al.,* 2022).

Numerous earlier studies have explored the relationships between macronutrient intake

and metabolic health, yet findings remain inconsistent due to differences in population characteristics, dietary patterns, and study design (Hankinson *et al.,* 2013; Camhi *et al.,* 2015). An umbrella review encompassing studies up to mid-2024 highlighted conflicting associations between food groups such as whole grains, sugar-sweetened beverages, and processed meats and the risk of metabolic syndrome or type 2 diabetes (Amanda *et al.,* 2025). Moreover, a 2023 cross-sectional study in Brazil reported a strong association between ultra-processed food consumption and increased prevalence of metabolic syndrome in adult populations (Barbosa *et al.,* 2023).

To address these rising concerns, animal models particularly rodents have long been employed to study the mechanisms underlying metabolic diseases and to test dietary interventions. Rats are widely used due to their physiological resemblance to humans and their well-characterized metabolic responses. Various experimental protocols have been utilized to induce metabolic disorders in rats, such as feeding high-fat diets, fructose-enriched regimens, or high cholesterol that many disrupt insulin signaling pathways (Wang *et al.,* 2023).

Within this context, egg yolk has emerged as a controversial dietary component. It is a rich source of essential nutrients, including phospholipids, fat-soluble vitamins, choline, and high-quality protein. However, its high cholesterol and saturated fat content have led to concerns about its role in promoting metabolic dysfunction when consumed excessively or in combination with high-fat diets (Kovács *et al.,* 2019). Some studies highlight its antioxidative and neuroprotective properties, while others report that egg yolk supplementation may accelerate lipid accumulation, hepatic steatosis, insulin resistance, and hormonal disturbances (Zhou *et al.,* 2021; Nassar *et al.,* 2019).

Interestingly, the physiological impact of egg yolk may vary according to dose, method of preparation (raw vs. cooked), and background diet. For instance, raw egg yolk retains more active lipids and cholesterol that might amplify adverse effects, whereas cooking can alter its lipid profile and reduce bioavailability of certain compounds (Güzel-Seydim *et al.,* 2022).

Despite the extensive use of high-fat diet models, current experimental approaches that utilize egg yolk as a source of dietary cholesterol have shown considerable variability in disease onset and severity. This underscores the need to standardize protocols and explore novel dietary models. The present study therefore aimed to investigate the combined effects of raw and cooked egg yolk at different doses alongside HFD in male albino rats over two periods (6 and 12 weeks), evaluating a wide spectrum of morphological, hematological, biochemical, and hormonal parameters. This approach provides a new platform to better understand the metabolic consequences of egg yolk consumption under varied nutritional conditions. The future studies investigation pathophysiology of metabolic disorders and efficacy of therapeutic agent.

MATERIALS AND METHODS

Animals:

This experimental study was conducted on 72 adult male albino rats (140–160 g), housed under standard laboratory conditions at Al-Azhar University (boys branch) Cairo, Egypt. the animals were acclimated for two weeks before the experiment and maintained on a 12/12-hour light/dark ***cycle*** with free access to water and standard rat chow.

Experimental Design:

High Fat Diet (HFD)preparation.

Palm Stearn (solid part) from local markets (samn nabaty) was used as a source of fat (vegetable ghee)300g vegetable ghee were add to 230g protein and 470g carbohydrate to prepare the HFD 1KG.

Egg-Yolk Preparation.

Local chicken eggs were purchased from local market.
1-Raw eggs-yolk was carefully separated from white egg the egg yolk solution was given to rats as low or high dose (9g or 18g/kg respectively by gastric tube every day.
2-Boiled egg-yolk (Cooked egg-yolk) preparation Eggs were boiled for 15 min to become solid
Rats were randomly divided into six groups:
***1*-Control, Normal Diet (ND)**: Standard diet 1kg (50g fat, 230g protein, 720g carbohydrate)
2-High-fat diet (**HFD)1kg** (300g fat, 230g protein, 470g carbohydrate)
3-HFD + low dose egg-yolk (LD): 1kg (HFD as mentioned above with low dose raw egg yolk (9g/day) via gastric tube)
4-HFD +high dose egg-yolk HD:1kg (HFD as mentioned above with high dose raw egg yolk (18g/day via gastric tube)
5-HFD + Boiled Yolk: 1kg (HFD as mentioned above with 100 g boiled yolk ,180g protein, 250g vegetable ghee ,470g carbohydrate).
6-ND + HD: Normal diet plus high dose raw egg yolk (18g/day via gastric tube).

Measured Parameters:

Rats were sacrificed after two experimental periods: 6 weeks and 12 weeks, following overnight fasting. The following measurements were performed:
Morphological Parameters Body weight (weekly), BMI calculated from body and length (Novelli *et al.,* 2007)

- ***Relative liver weight*** to detect hepatic hypertrophy (El-Beshbishy *et al.,* 2012).
- ***Relative Internal lipid mass (%)*** calculated from epididymal, mesenteric, and perirenal fat (Buettner *et al.,* 2007)

Hematological Analysis:

- ***Hemoglobin* (Hb)** *and* **CBC** measured using Sysmex XN-Series analyzer (Greer *et al*., 2018)

Biochemical Analysis*:*

- ***Fasting glucose*** (Stein, 1987; Tietz, 1995).
- ***Insulin hormone*** measured via ECLIA method (Sapin *et al.,* 2001).
- ***HbA1c*** as a long-term glucose marker (Lenters-Westra & Slingerland, 2018).
- ***HOMA-IR*** calculated for insulin resistance (Matthews *et al.,* 1985).
- ***Lipid profile***: Total cholesterol, triglycerides, HDL, LDL, and VLDL (Roeschlau *et al., 1974***;** Gordon *et al.,* 1977).

Liver Function Tests:

- ***ALT, AST, ALP*** enzymatic activity (Reitman & Frankel, 1957).

Hormonal Profile:

- **TSH, Free T3, Free T4** measured via ECLIA method (Wheeler & Lazarus, 1994).
- **Testosterone** level (Roche Diagnostics, n.d.).

Statistical Analysis:

Data were analyzed using ***one-way ANOVA*** followed by Significance was set at $P < 0.05$, and results were reported as ***mean* ± *SE*** using R software (v4.4.3)..

RESULTS

A comprehensive experimental design was adopted to investigate the progressive metabolic impact of egg yolk supplementation in rats fed a high-fat diet (HFD). To closely mimic the real-time development of metabolic disorders in response to chronic dietary exposure, the experimental period was strategically divided into two time points***:***
Short-term (6 weeks) to assess early physiological and biochemical responses.
Long-term (12 weeks) to evaluate cumulative and potentially irreversible metabolic damage. This dual-phase approach allows for the evaluation of both acute and chronic effects of raw and cooked egg yolk consumption in various dietary contexts. It also provides insight into the

time-dependent progression of key metabolic dysfunctions such as insulin resistance, dyslipidemia, inflammation, hormonal imbalances, and organ-specific pathology.

Morphological Parameters:

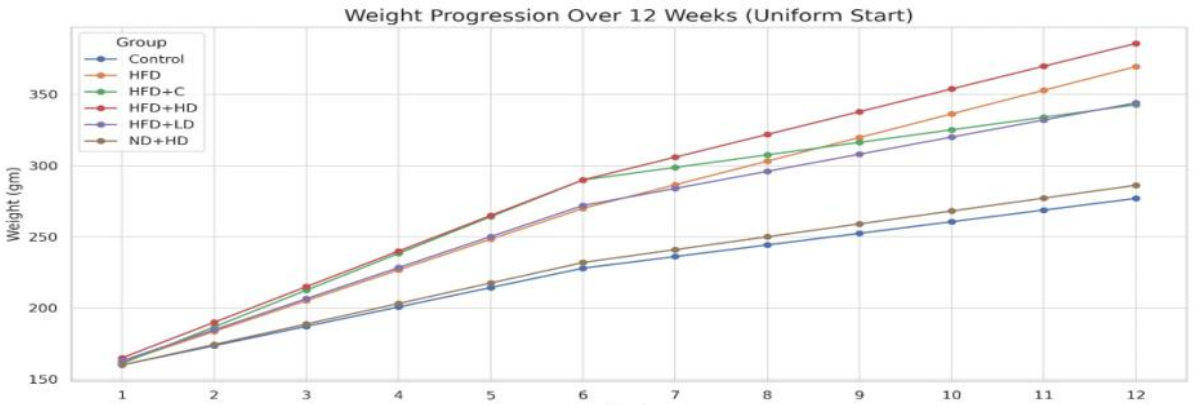
Weight Progression:

The Table (1) and Graph (1) displays the weekly progression of body weight (in grams) for six experimental groups over a 12-week period. Weight gain indicates differential responses depending on dietary treatment.After6 week All HFD-fed groups showed a significant increase in weight compared to control.HFD+HD and HFD+C groups had the highest body weights (~290 g), reflecting an enhanced obesogenic effect when egg yolk is combined with HFD.ND+HD group was only slightly higher than control, suggesting that egg yolk alone in a normal diet does not induce rapid weight gain. Weight differences began to emerge clearly by week 4 and became pronounced by week 6. HFD-fed groups rapidly diverged from the control due to increased caloric intake and fat storage. After12 week HFD and HFD+LD followed closely, with weights >344 g.ND+HD showed moderate increase (286.28 g), only 9 g above control, confirming that yolk in normal diet context has minimal impact on weight.Control group remained the lowest, reflecting expected baseline weight gain in standard-fed rats.

Table 1: The weekly progression of body weight (in grams) for six experimental groups over a 12-week period

weeks / group	1	2	3	4	5	6*	7	8	9	10	11	12**
Control	160	173.6	187.2	200.8	214.4	228	236.17	244.34	252.5	260.67	268.84	277.01
HFD	162	183.6	205.2	226.8	248.4	270	286.6	303.2	319.8	336.4	353	369.6
HFD+LD	163	184.8	206.6	228.4	250.2	272	284.03	296.05	308.08	320.11	332.14	344.16
HFD+HD	165	190	215	240	265	290	305.98	321.96	337.94	353.92	369.89	385.87
HFD+Boiled egg	161	186.8	212.6	238.4	264.2	290	298.8	307.6	316.4	325.21	334.01	342.81
ND+HD	160	174.4	188.8	203.2	217.6	232	241.05	250.09	259.14	268.18	277.23	286.28

(*)The end of 1st period, (**) The end of 2nd period.



Graph 1: the weekly progression of body weight (in grams) for six experimental groups over a 12-week period.

Body Weight (gm):

Based on the findings in Table (2), after 6 weeks, body weight in the HFD group (263.00 ± 7.96 g) was higher than the control (228.00 ± 5.71 g). The HFD+LD group also showed an increase (270.38 ± 6.30 g) compared with the control. A more pronounced elevation was observed in the HFD+HD (302.78 ± 8.25 g) and HFD+Boiled groups (299.50 ± 6.29 g). In contrast, the ND+HD group (231.00 ± 0.52 g) remained comparable to the control. After 12 weeks, body weight remained elevated in the HFD group (340.57 ± 15.39 g) relative to the control (265.52 ± 7.21 g). The HFD+LD group (324.58 ± 15.58 g) and HFD+Boiled group (328.93 ± 21.05 g) also demonstrated higher weights than the control, with the greatest increase

recorded in the HFD+HD group (358.13 ± 3.17 g). The ND+HD group (271.95 ± 4.96 g) did not show a meaningful difference from the control. Between the two periods, body weight in the control group rose from 228.00 ± 5.71 g after 6 weeks to 265.52 ± 7.21 g after 12 weeks. A more substantial increase was observed in HFD, which escalated from 263.00 ± 7.96 g to 340.57 ± 15.39 g, and in HFD+LD, which rose from 270.38 ± 6.30 g to 324.58 ± 15.58 g. The HFD+HD group also exhibited a robust gain from 302.78 ± 8.25 g to 358.13 ± 3.17 g. By contrast, the HFD+Boiled group showed only a modest rise from 299.50 ± 6.29 g to 328.93 ± 21.05 g, while ND+HD increased clearly from 231.00 ± 0.52 g to 271.95 ± 4.96 g.

Table 2*:* Body weight of rats in different experimental groups at 6- and 12-weeks values are expressed as Mean ± SE

Group	***6w (Mean±SE)***	***12w (Mean±SE)***
Control	228.00 ± 5.71	265.52 ± 7.21
HFD	263.00*** ± 7.96	340.57 ***± 15.39
HFD+LD	270.38 ***± 6.30	324.58 ***± 15.58
HFD+HD	302.78 ***± 8.25	358.13*** ± 3.17
HFD+Boiled	299.50*** ± 6.29	328.93*** ± 21.05
ND+HD	231.00 *± 0.52	271.95* ± 4.96

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Body Mass Index (BMI/gm/cm²):

As recorded in Table (3) After 6 weeks, BMI in the HFD group (0.51 ± 0.02) was higher than the control (0.46 ± 0.01), and a similar increase was recorded in the HFD+LD group (0.51 ± 0.02). More pronounced elevations were noted in HFD+HD (0.54 ± 0.02) and HFD+Boiled (0.55 ± 0.02), whereas the ND+HD group (0.44 ± 0.00) remained close to the control. After 12 weeks, BMI continued to be elevated in HFD (0.65 ± 0.03) relative to control (0.50 ± 0.01). The HFD+LD (0.58 ± 0.03) and HFD+Boiled (0.59 ± 0.03) groups also showed higher BMI values, with the highest level observed in HFD+HD (0.66 ± 0.01). The ND+HD group (0.51 ± 0.01) showed no clear deviation from the control. Between the two periods, BMI in the control group increased slightly from 0.46 ± 0.01 after 6 weeks to 0.50 ± 0.01 after 12 weeks. In HFD, BMI rose from 0.51 ± 0.02 to 0.65 ± 0.03, while HFD+LD increased from 0.51 ± 0.02 to 0.58 ± 0.03. A marked increase was also recorded in HFD+HD, which rose from 0.54 ± 0.02 to 0.66 ± 0.01. In HFD+Boiled, the rise was modest from 0.55 ± 0.02 to 0.59 ± 0.03, whereas ND+HD increased from 0.44 ± 0.00 to 0.51 ± 0.01.

Table 3: BMI of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE.

Group	***6w (Mean±SE)***	***12w (Mean±SE)***
Control	0.46 ± 0.01	0.50 ± 0.01
HFD	0.51** ± 0.02	0.65** ± 0.03
HFD+LD	0.51** ± 0.02	0.58* ± 0.03
HFD+HD	0.54 ***± 0.02	0.66 ***± 0.01
HFD+Boiled	0.55 ***± 0.02	0.59 **± 0.03
ND+HD	0.44 ± 0.00	0.51 ± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Relative Liver Weight (RLW/%):

Table (4) provides data, after 6 weeks, relative liver weight (RLW) in the HFD group (3.52 ± 0.06%) was slightly higher than the control (3.41 ± 0.06%). The HFD+LD group

showed a more noticeable increase (3.85 ± 0.18%) compared with control. The HFD+HD group (3.50 ± 0.23%) and HFD+Boiled group (3.43 ± 0.13%) remained close to control, while ND+HD (4.04 ± 0.15%) exhibited the highest value among all groups. After 12 weeks, relative liver weight in the HFD group (3.50 ± 0.04%) stayed very similar to the control (3.43 ± 0.13%). The HFD+LD group decreased to (3.44 ± 0.18%), almost equal to control. Interestingly, the HFD+HD group showed a reduction (2.78 ± 0.05%), becoming lower than the control. In contrast, HFD+Boiled (3.67 ± 0.18%) and ND+HD (3.88 ± 0.07%) presented higher values than control. Between the two periods, relative liver weight in the control group remained stable, changing only from 3.41 ± 0.06% after 6 weeks to 3.43 ± 0.13% after 12 weeks. The HFD group also showed stability, moving from 3.52 ± 0.06% to 3.50 ± 0.04%. The HFD+LD group decreased from 3.85 ± 0.18% to 3.44 ± 0.18%, while the HFD+HD group declined more sharply from 3.50 ± 0.23% to 2.78 ± 0.05%. Conversely, HFD+Boiled rose slightly from 3.43 ± 0.13% to 3.67 ± 0.18%, and ND+HD showed a modest reduction from 4.04 ± 0.15% to 3.88 ± 0.07%, though it still remained higher than control at both time points.

Table 4: RLW of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6w (Mean±SE)***	***12w (Mean±SE)***
Control	3.41 ± 0.06	3.43 ± 0.13
HFD	3.52 ± 0.06	3.50* ± 0.04
HFD+LD	3.85 ± 0.18	3.44 *± 0.18
HFD+HD	3.50 ± 0.23	2.78* ± 0.05
HFD+Boiled	3.43 ± 0.13	3.67* ± 0.18
ND+HD	4.04 ± 0.15	3.88* ± 0.07

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Relative Internal Lipids Weight (RILW/%):

Table (5) illustrates, after 6 weeks, relative internal lipids weight (RILW) in the HFD group (4.48 ± 0.50%) was higher than the control (3.56 ± 0.23%). The HFD+LD group showed a more evident increase (5.31 ± 0.55%) compared with control, and the HFD+HD group recorded the highest level (5.71 ± 0.35%). The HFD+Boiled group (4.57 ± 0.15%) was moderately higher than control, while the ND+HD group (3.88 ± 0.15%) remained close to the control value. After 12 weeks, relative internal lipids weight in the HFD group decreased to (3.28 ± 0.12%) and became lower than the control (3.54 ± 0.18%). The HFD+LD group remained elevated (4.89 ± 0.43%) compared with control, and the HFD+HD group also sustained a higher value (5.48 ± 0.25%). Interestingly, the HFD+Boiled group increased further to (4.90 ± 0.30%), while ND+HD (3.75 ± 0.05%) stayed close to control. between the two periods, relative internal lipids weight in the control group showed no change, remaining almost constant at 3.56 ± 0.23% after 6 weeks and 3.54 ± 0.18% after 12 weeks. In the HFD group, RILW decreased from 4.48 ± 0.50% to 3.28 ± 0.12%. The HFD+LD group also declined slightly from 5.31 ± 0.55% to 4.89 ± 0.43%, whereas the HFD+HD group showed a mild reduction from 5.71 ± 0.35% to 5.48 ± 0.25%. By contrast, HFD+Boiled increased from 4.57 ± 0.15% to 4.90 ± 0.30%, while ND+HD showed a slight decrease from 3.88 ± 0.15% to 3.75 ± 0.05%.

Table 5: RILW of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6w (Mean±SE)*	*12w (Mean±SE)*
Control	3.56 ± 0.23	3.54 ± 0.18
HFD	4.48** ± 0.50	3.28** ± 0.12
HFD + LD egg yolk	5.31**± 0.55	4.89*** ± 0.43
HFD + HD egg yolk	5.71** ± 0.35	5.48*** ± 0.25
HFD + Boiled egg yolk	4.57** ± 0.15	4.90** ± 0.30
ND + HD egg yolk	3.88* ± 0.15	3.75 **± 0.05

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Hematological Parameters:

Hemoglobin Hb (g%):

As recorded in Table (6), after 6 weeks, hemoglobin concentration in the HFD group (13.00 ± 0.04 g%) was lower than the control (14.55 ± 0.20 g%). The HFD+LD group also showed a reduction (12.72 ± 0.21 g%), with an even greater decrease in the HFD+HD group (11.55 ± 0.36 g%). In contrast, the HFD+Boiled group (14.10 ± 0.12 g%) remained close to the control, while the ND+HD group (15.28 ± 0.18 g%) displayed a higher hemoglobin value than the control. After 12 weeks, hemoglobin concentration in the HFD group (12.12 ± 0.24 g%) declined further compared with the control (14.89 ± 0.07 g%). The HFD+LD group (12.56 ± 0.19 g%) and the HFD+HD group (12.50 ± 0.09 g%) also remained below control. The HFD+Boiled group (14.16 ± 0.12 g%) was similar to control, while ND+HD (15.34 ± 0.01 g%) sustained higher levels than control. Between the two periods, hemoglobin concentration in the control group increased slightly from 14.55 ± 0.20 g% after 6 weeks to 14.89 ± 0.07 g% after 12 weeks. In the HFD group, hemoglobin decreased from 13.00 ± 0.04 g% to 12.12 ± 0.24 g%, and in the HFD+LD group it declined modestly from 12.72 ± 0.21 g% to 12.56 ± 0.19 g%. The HFD+HD group showed an improvement from 11.55 ± 0.36 g% to 12.50 ± 0.09 g%, yet values remained lower than control. The HFD+Boiled group was stable, rising only slightly from 14.10 ± 0.12 g% to 14.16 ± 0.12 g%, while ND+HD showed a small increase from 15.28 ± 0.18 g% to 15.34 ± 0.01 g%, maintaining the highest values among the groups.

Table 6: HB of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE.

Group	*6w (Mean±SE)*	*12w (Mean±SE)*
Control	14.55 ± 0.20	14.89 ± 0.07
HFD	13.00*** ± 0.04	12.12*** ± 0.24
HFD+LD	12.72*** ± 0.21	12.56 ***± 0.19
HFD+HD	11.55** ± 0.36	12.50 ***± 0.09
HFD+Boiled	14.10* ± 0.12	14.16 *± 0.12
ND+HD	15.28** ± 0.18	15.34 *± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Red Blood Cells (RBCs/cells×10^6/µL):

It is evident from Table (7) that after 6 weeks, RBC count in the HFD group (4.59 ± 0.01 ×10^6/µL) was slightly lower than the control (4.82 ± 0.12 ×10^6/µL). The HFD+LD group also showed a mild reduction (4.48 ± 0.03 ×10^6/µL), with a more pronounced decrease in the HFD+HD group (4.35 ± 0.09 ×10^6/µL). The HFD+Boiled group (4.50 ± 0.02 ×10^6/µL) was close to HFD+LD and remained lower than control. By contrast, ND+HD (4.95 ± 0.06 ×10^6/µL) exceeded the control value.After 12 weeks, RBC count in the HFD group (4.43 ±

0.03 ×10⁶/µL) remained below the control (5.09 ± 0.04 ×10⁶/µL). The HFD+LD group (4.47 ± 0.02 ×10⁶/µL) also stayed lower than control, while the HFD+HD group (4.54 ± 0.03 ×10⁶/µL) showed a slight improvement compared with its 6-week level but remained reduced relative to control. The HFD+Boiled group (4.55 ± 0.01 ×10⁶/µL) showed stability close to HFD+HD. ND+HD (5.30 ± 0.01 ×10⁶/µL) presented a higher RBC count than control.Between the two periods, RBC count in the control group rose from 4.82 ± 0.12 ×10⁶/µL after 6 weeks to 5.09 ± 0.04 ×10⁶/µL after 12 weeks. In the HFD group, RBCs decreased slightly from 4.59 ± 0.01 ×10⁶/µL to 4.43 ± 0.03 ×10⁶/µL. The HFD+LD group was nearly unchanged, moving from 4.48 ± 0.03 ×10⁶/µL to 4.47 ± 0.02 ×10⁶/µL, while the HFD+HD group increased from 4.35 ± 0.09 ×10⁶/µL to 4.54 ± 0.03 ×10⁶/µL. The HFD+Boiled group also showed a modest rise from 4.50 ± 0.02 ×10⁶/µL to 4.55 ± 0.01 ×10⁶/µL. Finally, ND+HD increased more clearly from 4.95 ± 0.06 ×10⁶/µL to 5.30 ± 0.01 ×10⁶/µL, maintaining the highest levels among all groups.

Table 7: RBCs of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	4.82 ± 0.12	5.09 ± 0.04
HFD	4.59* ± 0.01	4.43*** ± 0.03
HFD + LD yolk	4.48*± 0.03	4.47 ***± 0.02
HFD + HD yolk	4.35** ± 0.09	4.54 ***± 0.03
HFD + boiled yolk	4.50* ± 0.02	4.55 ***± 0.01
ND + HD yolk	4.95* ± 0.06	5.30*± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Packed Cell Volume (PCV/%):

Based on the findings in Table (8), after 6 weeks, PCV in the HFD group (39.52 ± 0.17%) was lower than the control (43.36 ± 0.68%). The HFD+LD group also showed a reduction (38.08 ± 0.71%), with the lowest value observed in the HFD+HD group (35.00 ± 0.94%). The HFD+Boiled group (42.32 ± 0.35%) was similar to control, while ND+HD (46.27 ± 0.47%) presented higher values than the control.After 12 weeks, PCV in the HFD group (36.62 ± 0.69%) decreased further compared with the control (44.97 ± 0.13%). The HFD+LD group (37.82 ± 0.58%) remained lower than control, as did the HFD+HD group (37.38 ± 0.30%). The HFD+Boiled group (42.56 ± 0.31%) was close to the control, whereas ND+HD (46.06 ± 0.09%) remained higher than control. Between the two periods, PCV in the control group increased slightly from 43.36 ± 0.68% after 6 weeks to 44.97 ± 0.13% after 12 weeks. The HFD group declined from 39.52 ± 0.17% to 36.62 ± 0.69%, and the HFD+LD group also showed a mild decrease from 38.08 ± 0.71% to 37.82 ± 0.58%. The HFD+HD group rose slightly from 35.00 ± 0.94% to 37.38 ± 0.30% but remained below control values. The HFD+Boiled group showed stability with only a small increase from 42.32 ± 0.35% to 42.56 ± 0.31%. ND+HD maintained the highest levels, moving from 46.27 ± 0.47% to 46.06 ± 0.09%, remaining consistently above control.

Table 8: PCV of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6w (Mean±SE)*	*12w (Mean±SE)*
Control	43.36 ± 0.68	44.97 ± 0.13
HFD	39.52*** ± 0.17	36.62 ± 0.69
HFD+LD	38.08*** ± 0.71	37.82 ± 0.58
HFD+HD	35.00*** ± 0.94	37.38 ± 0.30
HFD+Boiled	42.32*± 0.35	42.56 ± 0.31
ND+HD	46.27** ± 0.47	46.06 ± 0.09

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Total Leucocyte Count (TLC/cells×10³/µL):

It is evident from Table (9) that after 6 weeks, total leukocyte counts in the HFD group (13.03 ± 0.14 ×10³/µL) was elevated compared with the control (10.82 ± 0.05 ×10³/µL). The HFD+LD group (12.06 ± 0.13 ×10³/µL) and HFD+HD group (11.97 ± 0.56 ×10³/µL) also showed higher values than control. By contrast, the HFD+Boiled group (10.00 ± 0.12 ×10³/µL) presented slightly lower levels than control, while ND+HD (11.00 ± 0.05 ×10³/µL) was close to the control group.After 12 weeks, total leukocyte count in the HFD group (13.80 ± 0.62 ×10³/µL) remained higher than the control (10.44 ± 0.06 ×10³/µL). The HFD+LD group (11.49 ± 0.19 ×10³/µL) was still above control, and HFD+HD demonstrated a marked elevation (27.91 ± 13.70 ×10³/µL). The HFD+Boiled group (11.92 ± 1.35 ×10³/µL) showed a moderate increase compared with control, while ND+HD (10.88 ± 1.31 ×10³/µL) was nearly equal to control.Between the two periods, total leukocyte count in the control group decreased slightly from 10.82 ± 0.05 ×10³/µL after 6 weeks to 10.44 ± 0.06 ×10³/µL after 12 weeks. In the HFD group, TLC increased from 13.03 ± 0.14 ×10³/µL to 13.80 ± 0.62 ×10³/µL. The HFD+LD group declined from 12.06 ± 0.13 ×10³/µL to 11.49 ± 0.19 ×10³/µL, while the HFD+HD group rose dramatically from 11.97 ± 0.56 ×10³/µL to 27.91 ± 13.70 ×10³/µL. The HFD+Boiled group increased from 10.00 ± 0.12 ×10³/µL to 11.92 ± 1.35 ×10³/µL, whereas ND+HD remained nearly stable, shifting slightly from 11.00 ± 0.05 ×10³/µL to 10.88 ± 1.31 ×10³/µL.

Table 9: TLC *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	10.82 ± 0.05	10.44 ± 0.06
HFD	13.03*** ± 0.14	13.80*** ± 0.62
HFD + LD yolk	12.06***± 0.13	11.49 ***± 0.19
HFD + HD yolk	11.97** ± 0.56	27.91*** ± 13.70
HFD + boiled yolk	10.00* ± 0.12	11.92** ± 1.35
ND + HD yolk	11.00** ± 0.05	10.88* ± 1.31

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Fasting Blood Sugar (FBS/mg%):

Table (10) provides data, after 6 weeks, fasting blood sugar in the HFD group (106.25 ± 1.37 mg%) was higher than the control (86.12 ± 1.10 mg%). The HFD+LD group (102.62 ± 3.20 mg%) and HFD+HD group (96.83 ± 2.63 mg%) were also elevated compared with control. The HFD+Boiled group (93.20 ± 2.69 mg%) was moderately higher than control, while ND+HD (89.56 ± 2.46 mg%) remained close to control values.After 12 weeks, fasting blood sugar in the HFD group (97.00 ± 1.59 mg%) was still above the control (93.12 ± 1.11 mg%) but with a smaller difference compared to the 6-week level. The HFD+LD group (102.40

± 2.55 mg%) stayed elevated relative to control, and HFD+HD (105.20 ± 4.74 mg%) showed a further rise compared with its 6-week level. The HFD+Boiled group (92.60 ± 1.47 mg%) and ND+HD group (92.79 ± 0.29 mg%) were very close to the control value. Between the two periods, fasting blood sugar in the control group increased from 86.12 ± 1.10 mg% after 6 weeks to 93.12 ± 1.11 mg% after 12 weeks. In the HFD group, FBS decreased from 106.25 ± 1.37 mg% to 97.00 ± 1.59 mg%, while HFD+LD remained almost stable, moving from 102.62 ± 3.20 mg% to 102.40 ± 2.55 mg%. The HFD+HD group increased from 96.83 ± 2.63 mg% to 105.20 ± 4.74 mg%, while the HFD+Boiled group declined slightly from 93.20 ± 2.69 mg% to 92.60 ± 1.47 mg%. ND+HD also showed a small reduction from 89.56 ± 2.46 mg% to 92.79 ± 0.29 mg%, maintaining values close to control at both time points.

Table 10: FBS *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6 W (Mean±SE)***	***12 W(Mean±SE)***
Control	86.12± 1.10	93.12± 1.11
HFD	106.25***± 1.37	97.00*± 1.59
HFD + LD yolk	102.62***± 3.20	102.40*± 2.55
HFD + HD yolk	96.83**± 2.63	105.20**± 4.74
HFD + boiled yolk	93.20**± 2.69	92.60*± 1.47
ND + HD yolk	89.56*± 2.46	92.79*± 0.29

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Insulin (mIU/ml):

Table (11) illustrates After 6 weeks, insulin level in the HFD group (3.47 ± 0.07 mIU/ml) was higher than the control (1.86 ± 0.05 mIU/ml). The HFD+LD group (2.91 ± 0.11 mIU/ml) and HFD+HD group (3.86 ± 0.13 mIU/ml) also showed marked increases compared with control. The HFD+Boiled group (3.52 ± 0.12 mIU/ml) was similarly elevated, while ND+HD (2.02 ± 0.03 mIU/ml) remained close to control. After 12 weeks, insulin level in the HFD group (3.85 ± 0.09 mIU/ml) remained elevated compared with the control (3.46 ± 0.14 mIU/ml). The HFD+LD group (4.02 ± 0.15 mIU/ml) and HFD+HD group (3.88 ± 0.17 mIU/ml) continued to show higher levels, while the HFD+Boiled group (3.68 ± 0.12 mIU/ml) was slightly above control. In contrast, ND+HD (2.94 ± 0.06 mIU/ml) was lower than control at this time point. Between the two periods, insulin level in the control group increased from 1.86 ± 0.05 mIU/ml after 6 weeks to 3.46 ± 0.14 mIU/ml after 12 weeks. The HFD group also rose from 3.47 ± 0.07 mIU/ml to 3.85 ± 0.09 mIU/ml, while HFD+LD increased from 2.91 ± 0.11 mIU/ml to 4.02 ± 0.15 mIU/ml. The HFD+HD group remained almost stable, moving slightly from 3.86 ± 0.13 mIU/ml to 3.88 ± 0.17 mIU/ml, and the HFD+Boiled group showed a modest decline from 3.52 ± 0.12 mIU/ml to 3.68 ± 0.12 mIU/ml. ND+HD, however, increased more clearly from 2.02 ± 0.03 mIU/ml to 2.94 ± 0.06 mIU/ml, but still stayed lower than most HFD-fed groups.

Table 11: INSULIN O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE*

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	1.86± 0.05	3.46± 0.14
HFD	3.47***± 0.07	3.85**± 0.09
HFD + LD yolk	2.91**± 0.11	4.02**± 0.15
HFD + HD yolk	3.86***± 0.13	3.88*± 0.17
HFD + boiled yolk	3.52***± 0.12	3.68*± 0.12
ND + HD yolk	2.02*± 0.03	2.94*± 0.06

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Hemoglobin A1C (HbA1c /%):

As recorded in Table (12), After 6 weeks, HbA1c percentage in the HFD group (4.58 ± 0.05%) was higher than the control (3.33 ± 0.09%). The HFD+LD group (4.56 ± 0.09%) and HFD+HD group (4.38 ± 0.09%) also recorded elevated values compared with control. The HFD+Boiled group (3.66 ± 0.06%) remained close to control, while ND+HD (4.06 ± 0.10%) was moderately higher than the control. After 12 weeks, HbA1c percentage in the HFD group (4.52 ± 0.10%) stayed above the control (3.34 ± 0.05%). The HFD+LD group (4.62 ± 0.10%) and HFD+HD group (4.44 ± 0.08%) maintained higher values, while HFD+Boiled (3.66 ± 0.07%) remained very close to control. ND+HD (3.76 ± 0.01%) showed a slight decrease compared with its 6-week level but still above the control. Between the two periods, HbA1c percentage in the control group remained stable, moving only from 3.33 ± 0.09% after 6 weeks to 3.34 ± 0.05% after 12 weeks. In the HFD group, HbA1c decreased slightly from 4.58 ± 0.05% to 4.52 ± 0.10%, and in HFD+LD it rose slightly from 4.56 ± 0.09% to 4.62 ± 0.10%. The HFD+HD group increased modestly from 4.38 ± 0.09% to 4.44 ± 0.08%, while HFD+Boiled remained unchanged at 3.66 ± 0.06–0.07% across both periods. ND+HD decreased from 4.06 ± 0.10% to 3.76 ± 0.01%, showing an opposite trend compared with the HFD-fed groups.

Table 12: HBA1C *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	3.33± 0.09	3.34± 0.05
HFD	4.58***± 0.05	4.52***± 0.10
HFD + LD yolk	4.56***± 0.09	4.62***± 0.10
HFD + HD yolk	4.38**± 0.09	4.44***± 0.08
HFD + boiled yolk	3.66*± 0.06	3.66* ± 0.07
ND + HD yolk	4.06**± 0.10	3.76 **± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Homeostatic Model Assessment for Insulin Resistance (HomaIR):

Table (13) provides data, after 6 weeks, HOMA-IR in the HFD group (0.91 ± 0.01) was higher than the control (0.40 ± 0.01). The HFD+LD group (0.73 ± 0.01) and HFD+HD group (0.93 ± 0.05) also showed increased values compared with control. The HFD+Boiled group (0.81 ± 0.02) was moderately elevated, while ND+HD (0.45 ± 0.02) remained close to control. After 12 weeks, HOMA-IR in the HFD group (0.92 ± 0.01) stayed above the control (0.79 ± 0.03). The HFD+LD group (1.01 ± 0.04) and HFD+HD group (1.01 ± 0.07) recorded higher values than control, while the HFD+Boiled group (0.84 ± 0.03) remained close to

control. ND+HD (0.67 ± 0.01) increased compared with its 6-week value but still stayed lower than the HFD-fed groups.Between the two periods, HOMA-IR in the control group increased from 0.40 ± 0.01 after 6 weeks to 0.79 ± 0.03 after 12 weeks. The HFD group remained relatively stable, rising only slightly from 0.91 ± 0.01 to 0.92 ± 0.01. The HFD+LD group increased from 0.73 ± 0.01 to 1.01 ± 0.04, while the HFD+HD group also rose modestly from 0.93 ± 0.05 to 1.01 ± 0.07. The HFD+Boiled group showed little change, moving from 0.81 ± 0.02 to 0.84 ± 0.03. ND+HD, however, increased from 0.45 ± 0.02 to 0.67 ± 0.01, indicating a noticeable elevation compared with its baseline period.

Table 13: HOMIR *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	0.40 ± 0.01	0.79± 0.03
HFD	0.91**± 0.01	0.92** ± 0.01
HFD + LD yolk	0.73**± 0.01	1.01***± 0.04
HFD + HD yolk	0.93**± 0.05	1.01***± 0.07
HFD + boiled yolk	0.81**± 0.02	0.84**± 0.03
ND + HD yolk	0.45*± 0.02	0.67 *± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Lipid Profile Parameters:

Triglycerides (TGs/mg%): It is evident from Table (14), after 6 weeks, triglyceride levels in the HFD group (136.25 ± 0.46 mg%) were markedly higher than the control (78.62 ± 2.43 mg%). The HFD+LD group (126.33 ± 2.85 mg%) and HFD+HD group (137.83 ± 5.89 mg%) also showed strong elevations compared with control. The HFD+Boiled group (106.60 ± 4.18 mg%) was moderately higher, while ND+HD (101.81 ± 0.82 mg%) also exceeded the control but to a lesser degree. After 12 weeks, triglyceride levels in the HFD group (140.67 ± 0.76 mg%) remained elevated compared with the control (84.50 ± 1.88 mg%). The HFD+LD group (138.80 ± 1.28 mg%) and HFD+HD group (137.60 ± 3.58 mg%) continued to show high values, while the HFD+Boiled group (103.20 ± 2.75 mg%) was lower than the other HFD-fed groups but still above control. The ND+HD group (112.54 ± 2.04 mg%) also showed a clear increase compared with control. Between the two periods, triglyceride levels in the control group rose slightly from 78.62 ± 2.43 mg% after 6 weeks to 84.50 ± 1.88 mg% after 12 weeks. The HFD group increased from 136.25 ± 0.46 mg% to 140.67 ± 0.76 mg%, and HFD+LD rose from 126.33 ± 2.85 mg% to 138.80 ± 1.28 mg%. The HFD+HD group remained stable, moving from 137.83 ± 5.89 mg% to 137.60 ± 3.58 mg%. By contrast, the HFD+Boiled group decreased slightly from 106.60 ± 4.18 mg% to 103.20 ± 2.75 mg%, while ND+HD increased from 101.81 ± 0.82 mg% to 112.54 ± 2.04 mg%.

Table **14:** TGs *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE.

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	78.62± 2.43	84.50± 1.88
HFD	136.25***± 0.46	140.67***± 0.76
HFD + LD yolk	126.33***± 2.85	138.8 ***± 1.28
HFD + HD yolk	137.83***± 5.89	137.6***± 3.58
HFD + boiled yolk	106.60**± 4.18	103.20**± 2.75
ND + HD yolk	101.81** ± 0.82	112.54*** ± 2.04

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period)

Total Cholsterol (mg%):

As recorded in Table (15), after 6 weeks, total cholesterol in the HFD group (112.00 ± 1.46 mg%) was slightly lower than the control (118.00 ± 0.97 mg%). The HFD+LD group (115.25 ± 2.14 mg%) and HFD+HD group (118.33 ± 3.14 mg%) were close to control, while the HFD+Boiled group (111.40 ± 3.16 mg%) was marginally reduced. ND+HD (104.00 ± 2.62 mg%) showed the lowest cholesterol level among the groups.After 12 weeks, total cholesterol in the HFD group (117.83 ± 2.12 mg%) became similar to the control (111.25 ± 0.81 mg%). The HFD+LD group (115.20 ± 2.57 mg%) and HFD+HD group (116.00 ± 3.59 mg%) also remained close to control values. The HFD+Boiled group increased to (118.60 ± 3.06 mg%), slightly higher than control, while ND+HD (117.98 ± 0.73 mg%) also showed a modest increase compared with its 6-week level.Between the two periods, total cholesterol in the control group decreased slightly from 118.00 ± 0.97 mg% after 6 weeks to 111.25 ± 0.81 mg% after 12 weeks. The HFD group rose from 112.00 ± 1.46 mg% to 117.83 ± 2.12 mg%, while HFD+LD remained stable with only a minor change from 115.25 ± 2.14 mg% to 115.20 ± 2.57 mg%. The HFD+HD group declined slightly from 118.33 ± 3.14 mg% to 116.00 ± 3.59 mg%. The HFD+Boiled group increased from 111.40 ± 3.16 mg% to 118.60 ± 3.06 mg%, and ND+HD rose from 104.00 ± 2.62 mg% to 117.98 ± 0.73 mg%, reflecting a clear upward shift.

Table 15: T.CHOL *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	118.00 ± 0.97	111.25 ± 0.81
HFD	112.00* ± 1.46	117.83* ± 2.12
HFD + LD yolk	115.25* ± 2.14	115.20* ± 2.57
HFD + HD yolk	118.33 ± 3.14	116.00* ± 3.59
HFD + boiled yolk	111.40* ± 3.16	118.60 *± 3.06
ND + HD yolk	104.00*± 2.62	117.98* ± 0.73

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period)

High-Density Lipoprotein Cholesterol (HDL-C/mg%):

Based on the findings in Table (16), after 6 weeks, HDL-C in the HFD group (33.50 ± 0.18 mg%) was lower than the control (37.25 ± 0.63 mg%). The HFD+LD group (36.96 ± 0.36 mg%) remained close to control, while the HFD+HD group (30.00 ± 1.10 mg%) showed the lowest value. The HFD+Boiled group (36.40 ± 1.08 mg%) was comparable to control, and ND+HD (38.56 ± 0.16 mg%) showed the highest HDL-C among all groups. After 12 weeks, HDL-C in the HFD group (34.17 ± 0.28 mg%) was still slightly below the control (34.96 ± 0.29 mg%). The HFD+LD group (33.20 ± 0.40 mg%) decreased compared with its earlier level and was lower than control, while HFD+HD (31.00 ± 1.13 mg%) remained reduced. The HFD+Boiled group (35.60 ± 0.80 mg%) was slightly above control, and ND+HD (36.40 ± 0.15 mg%) continued to present the highest values relative to control.Between the two periods, HDL-C in the control group decreased from 37.25 ± 0.63 mg% after 6 weeks to 34.96 ± 0.29 mg% after 12 weeks. The HFD group increased slightly from 33.50 ± 0.18 mg% to 34.17 ± 0.28 mg%, while HFD+LD declined from 36.96 ± 0.36 mg% to 33.20 ± 0.40 mg%. The HFD+HD group rose modestly from 30.00 ± 1.10 mg% to 31.00 ± 1.13 mg%, while the HFD+Boiled group also increased from 36.40 ± 1.08 mg% to 35.60 ± 0.80 mg%, remaining relatively stable. ND+HD decreased slightly from 38.56 ± 0.16 mg% to 36.40 ± 0.15 mg% but still maintained higher levels than most groups.

Table 16: HDL-C *o*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	37.25 ± 0.63	34.96± 0.29
HFD	33.50***± 0.18	34.17 ± 0.28
HFD + LD yolk	36.96*± 0.36	33.20± 0.40
HFD + HD yolk	30.00*** ± 1.10	31.00± 1.13
HFD + boiled yolk	36.40*± 1.08	35.60*± 0.80
ND + HD yolk	38.56 **± 0.16	36.40 *± 0.15

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Low-Density Lipoprotein Cholesterol (LDL-C/ mg%):

Table (17) illustrates after 6 weeks, LDL-C in the HFD group (51.25 ± 1.19 mg%) was lower than the control (65.02 ± 1.91 mg%). The HFD+LD group (53.02 ± 1.92 mg%) was also reduced compared with control. In contrast, the HFD+HD group (60.77 ± 2.24 mg%) was closer to control, while the HFD+Boiled group (53.68 ± 3.57 mg%) was moderately lower. ND+HD (45.08 ± 2.95 mg%) showed the lowest LDL-C among the groups.After 12 weeks, LDL-C in the HFD group (55.53 ± 1.82 mg%) remained lower than the control (59.39 ± 1.06 mg%). The HFD+LD group (54.24 ± 2.29 mg%) was also close to control but slightly reduced. The HFD+HD group (57.48 ± 3.28 mg%) was nearly comparable to control, while the HFD+Boiled group (62.36 ± 3.53 mg%) was slightly higher than control. ND+HD (59.08 ± 0.17 mg%) increased relative to its 6-week level, becoming similar to control.Between the two periods, LDL-C in the control group decreased from 65.02 ± 1.91 mg% after 6 weeks to 59.39 ± 1.06 mg% after 12 weeks. The HFD group rose slightly from 51.25 ± 1.19 mg% to 55.53 ± 1.82 mg%, while HFD+LD remained stable, moving from 53.02 ± 1.92 mg% to 54.24 ± 2.29 mg%. The HFD+HD group decreased from 60.77 ± 2.24 mg% to 57.48 ± 3.28 mg%, whereas HFD+Boiled increased from 53.68 ± 3.57 mg% to 62.36 ± 3.53 mg%. ND+HD also showed a marked rise from 45.08 ± 2.95 mg% to 59.08 ± 0.17 mg%, bringing it closer to the control value at 12 weeks.

Table 17: LDL-C *o*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12W(Mean±SE)*
Control	65.02± 1.91	59.39 ± 1.06
HFD	51.25 ***± 1.19	55.53 ± 1.82
HFD + LD yolk	53.02***± 1.92	54.24 ± 2.29
HFD + HD yolk	60.77**± 2.24	57.48* ± 3.28
HFD + boiled yolk	53.68** ± 3.57	62.36* ± 3.53
ND + HD yolk	45.08* ± 2.95	59.08 ± 0.17

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Very Low-Density Lipoprotein Cholesterol (VLDL-C/ mg%)

Table (18) provides data, after 6 weeks, VLDL-C in the HFD group (27.25 ± 0.09 mg%) was higher than the control (15.73 ± 0.49 mg%). The HFD+LD group (25.27 ± 0.57 mg%) and HFD+HD group (27.57 ± 1.18 mg%) also showed pronounced increases compared with control. The HFD+Boiled group (21.32 ± 0.84 mg%) was moderately higher than control, while ND+HD (20.36 ± 0.16 mg%) also exceeded the control value.After 12 weeks, VLDL-C in the HFD group (28.13 ± 0.15 mg%) remained elevated compared with the control (16.90 ± 0.38 mg%). The HFD+LD group (27.76 ± 0.26 mg%) and HFD+HD group (27.52 ± 0.72 mg%)

were both sustained at high levels. The HFD+Boiled group (20.64 ± 0.55 mg%) was lower than other HFD-fed groups but still higher than control, while ND+HD (22.51 ± 0.41 mg%) also showed an increase compared with control. Between the two periods, VLDL-C in the control group rose slightly from 15.73 ± 0.49 mg% after 6 weeks to 16.90 ± 0.38 mg% after 12 weeks. The HFD group increased modestly from 27.25 ± 0.09 mg% to 28.13 ± 0.15 mg%, while HFD+LD also rose from 25.27 ± 0.57 mg% to 27.76 ± 0.26 mg%. The HFD+HD group remained nearly stable, changing from 27.57 ± 1.18 mg% to 27.52 ± 0.72 mg%. The HFD+Boiled group declined slightly from 21.32 ± 0.84 mg% to 20.64 ± 0.55 mg%, while ND+HD increased from 20.36 ± 0.16 mg% to 22.51 ± 0.41 mg%.

Table 18: VLDL-C *o*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE.

Group	***6W(Mean±SE)***	***12W(Mean±SE)***
Control	15.73 ± 0.49	16.90 ± 0.38
HFD	27.25*** ± 0.09	28.13*** ± 0.15
HFD + LD yolk	25.27***± 0.57	27.76***± 0.26
HFD + HD yolk	27.57***± 1.18	27.52***± 0.72
HFD + boiled yolk	21.32***± 0.84	20.64***± 0.55
ND + HD yolk	20.36**± 0.16	22.51***± 0.41

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Liver Enzymes:

Aspartate Aminotransferase (AST/ U/L): Based on the findings in Table (19), after 6 weeks, AST activity in the HFD group (141.50 ± 0.91 U/L) was elevated compared with the control (92.62 ± 2.73 U/L). The HFD+LD group (174.25 ± 6.41 U/L) and HFD+HD group (139.50 ± 6.49 U/L) also showed strong increases. The HFD+Boiled group (128.80 ± 2.91 U/L) was moderately higher than control, while ND+HD (140.06 ± 1.15 U/L) also exhibited a marked elevation. After 12 weeks, AST activity in the HFD group (143.33 ± 1.80 U/L) remained higher than the control (92.31 ± 1.15 U/L). The HFD+LD group (152.40 ± 2.43 U/L) and HFD+HD group (147.00 ± 2.13 U/L) both stayed elevated, while the HFD+Boiled group (125.80 ± 2.60 U/L) was moderately above control. ND+HD (132.83 ± 1.17 U/L) also remained higher than control. Between the two periods, AST activity in the control group stayed almost constant (92.62 ± 2.73 U/L at 6 weeks and 92.31 ± 1.15 U/L at 12 weeks). The HFD group increased slightly from 141.50 ± 0.91 U/L to 143.33 ± 1.80 U/L, while HFD+LD declined from 174.25 ± 6.41 U/L to 152.40 ± 2.43 U/L. The HFD+HD group rose slightly from 139.50 ± 6.49 U/L to 147.00 ± 2.13 U/L, and the HFD+Boiled group decreased modestly from 128.80 ± 2.91 U/L to 125.80 ± 2.60 U/L. ND+HD also declined from 140.06 ± 1.15 U/L to 132.83 ± 1.17 U/L, though still higher than control.

Table 19: AST *o*f rats in different experimental groups at 6- and 12-weeks. values are expressed as mean ± SE

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	92.62 ± 2.73	92.31± 1.15
HFD	141.50*** ± 0.91	143.33***± 1.80
HFD + LD yolk	174.25***± 6.41	152.40***± 2.43
HFD + HD yolk	139.5***± 6.49	147.00*** ± 2.13
HFD + boiled yolk	128.80***± 2.91	125.80***± 2.60
ND + HD yolk	140.06 ***± 1.15	132.83***± 1.17

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Alanine Aminotransferase (ALT/ U/L):

Table (20) provides data,after 6 weeks, ALT activity in the HFD group (62.50 ± 1.22 U/L) was elevated compared with the control (34.12 ± 1.37 U/L). The HFD+LD group (58.35 ± 1.25 U/L) and HFD+HD group (59.17 ± 2.95 U/L) also showed high levels. The HFD+Boiled group (48.40 ± 1.38 U/L) was moderately above control, while ND+HD (36.31 ± 0.49 U/L) remained close to control.After 12 weeks, ALT activity in the HFD group (61.83 ± 2.79 U/L) was still higher than the control (35.08 ± 0.58 U/L). The HFD+LD group (51.60 ± 1.33 U/L) and HFD+HD group (59.60 ± 3.06 U/L) also maintained higher values, while the HFD+Boiled group (47.40 ± 2.46 U/L) was moderately above control. ND+HD (41.23 ± 1.02 U/L) remained close to control.Between the two periods, ALT activity in the control group was stable, moving slightly from 34.12 ± 1.37 U/L to 35.08 ± 0.58 U/L. The HFD group decreased slightly from 62.50 ± 1.22 U/L to 61.83 ± 2.79 U/L, while HFD+LD declined more noticeably from 58.35 ± 1.25 U/L to 51.60 ± 1.33 U/L. The HFD+HD group increased slightly from 59.17 ± 2.95 U/L to 59.60 ± 3.06 U/L, while HFD+Boiled decreased from 48.40 ± 1.38 U/L to 47.40 ± 2.46 U/L. ND+HD rose modestly from 36.31 ± 0.49 U/L to 41.23 ± 1.02 U/L.

Table 20: ALT *o*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	***6 W(Mean±SE)***	***12 W(Mean±SE)***
Control	34.12± 1.37	35.08± 0.58
HFD	50.50***± 0.18	56.67*** ± 1.84
HFD + LD yolk	58.35***± 1.25	51.60 ***± 1.33
HFD + HD yolk	59.17***± 2.95	59.60***± 3.06
HFD + boiled yolk	48.40***± 1.38	47.40***± 2.46
ND + HD yolk	36.31 *± 0.49	41.23**± 1.02

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period),

Alkaline Phosphatase (ALP/ U/L**):**

It is evident from Table (21), after 6 weeks, ALP activity in the HFD group (116.25 ± 3.01 U/L) was higher than the control (62.50 ± 1.22 U/L). The HFD+LD group (122.00 ± 8.54 U/L) and HFD+HD group (136.00 ± 10.17 U/L) showed further increases. The HFD+Boiled group (106.40 ± 4.17 U/L) was also elevated, while ND+HD (81.94 ± 1.15 U/L) was moderately higher than control. After 12 weeks, ALP activity in the HFD group (134.50 ± 3.50 U/L) remained elevated compared with the control (61.83 ± 2.79 U/L). The HFD+LD group (102.20 ± 5.36 U/L) and HFD+HD group (120.60 ± 4.66 U/L) were also above control, while the HFD+Boiled group (97.20 ± 3.41 U/L) was moderately elevated. ND+HD (79.62 ± 0.88 U/L) was closer to control than the HFD-fed groups. Between the two periods, ALP activity in the control group remained almost unchanged (62.50 ± 1.22 U/L at 6 weeks and 61.83 ± 2.79 U/L at 12 weeks). The HFD group increased from 116.25 ± 3.01 U/L to 134.50 ± 3.50 U/L, while HFD+LD declined from 122.00 ± 8.54 U/L to 102.20 ± 5.36 U/L. The HFD+HD group decreased slightly from 136.00 ± 10.17 U/L to 120.60 ± 4.66 U/L, and HFD+Boiled declined from 106.40 ± 4.17 U/L to 97.20 ± 3.41 U/L. ND+HD decreased slightly from 81.94 ± 1.15 U/L to 79.62 ± 0.88 U/L.

Table 21: ALP Of rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6W(Mean±SE)*	*12 W(Mean±SE)*
Control	62.50± 1.22	61.83 ± 2.79
HFD	116.25***± 3.01	134.50***± 3.50
HFD + LD yolk	122.00***± 8.54	102.20***± 5.36
HFD + HD yolk	136.00***± 10.17	120.60***± 4.66
HFD + boiled yolk	106.40***± 4.17	97.20 ***± 3.41
ND + HD yolk	81.94**± 1.15	79.62***± 0.88

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period)

Thyroid Function:

Thyroid Stimulation Hormone (TSH/IU/ml):

Table (22) illustrates, after 6 weeks, TSH was 0.25 ± 0.01 in control. TSH rose in HFD (0.36 ± 0.00), HFD+LD yolk (0.29 ± 0.02), HFD+HD yolk (0.43 ± 0.04), HFD+boiled yolk (0.32 ± 0.01), and ND+HD yolk (0.40 ± 0.01) relative to control. After 12 weeks, TSH was 0.37 ± 0.01 in control. TSH remained higher in HFD (0.49 ± 0.04), HFD+LD yolk (0.41 ± 0.01), and ND+HD yolk (0.39 ± 0.01); TSH in HFD+HD yolk (0.38 ± 0.02) was just above control, while HFD+boiled yolk (0.33 ± 0.01) was slightly below control. Between the two periods, TSH increased in control (0.25 → 0.37), HFD (0.36 → 0.49), and HFD+LD yolk (0.29 → 0.41); TSH decreased in HFD+HD yolk (0.43 → 0.38) and ND+HD yolk (0.40 → 0.39) and rose marginally in HFD+boiled yolk (0.32 → 0.33).

Table 22: TSH *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	0.25± 0.01	0.37 ± 0.01
HFD	0.36** ± 0.00	0.49**± 0.04
HFD + LD yolk	0.29**± 0.02	0.41** ± 0.01
HFD + HD yolk	0.43***± 0.04	0.38 **± 0.02
HFD + boiled yolk	0.32 **± 0.01	0.33 *± 0.01
ND + HD yolk	0.40***± 0.01	0.39 **± 0.01

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Triiodothyronine (T3/ng%):

Table (23) provides data, after 6 weeks, T3 was 45.5 ± 2.11 in control. T3 was higher in HFD (63.50 ± 0.55), HFD+LD yolk (56.25 ± 2.14), HFD+HD yolk (67.67 ± 3.68), HFD+boiled yolk (48.80 ± 1.14), and ND+HD yolk (55.81 ± 1.80) compared with control. After 12 weeks, T3 was 56.21 ± 1.49 in control. T3 stayed higher in HFD (67.50 ± 0.66), HFD+LD yolk (66.20 ± 1.19), HFD+HD yolk (65.6 ± 1.76), HFD+boiled yolk (59.20 ± 2.45), and ND+HD yolk (63.23 ± 1.02) than control. Between the two periods, T3 increased in control (45.5 → 56.21), HFD (63.50 → 67.50), HFD+LD yolk (56.25 → 66.20), HFD+boiled yolk (48.80 → 59.20), and ND+HD yolk (55.81 → 63.23), while T3 decreased slightly in HFD+HD yolk (67.67 → 65.6).

Table 23: T3 *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	45.5± 2.11	56.21± 1.49
HFD	63.50**± 0.55	67.50**± 0.66
HFD + LD yolk	56.25**± 2.14	66.20***± 1.19
HFD + HD yolk	67.67**± 3.68	65.6 **± 1.76
HFD + boiled yolk	48.80*± 1.14	59.20** ± 2.45
ND + HD yolk	55.81**± 1.80	63.23**± 1.02

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period).

Thyroxine (T4/ng%):

As recorded in Table (24), after 6 weeks, T4 was 1.45 ± 0.07 in control. T4 was higher in HFD (1.62 ± 0.01), HFD+LD yolk (1.64 ± 0.09), HFD+HD yolk (1.90 ± 0.14), and ND+HD yolk (1.53 ± 0.05), while T4 in HFD+boiled yolk (1.36 ± 0.04) was lower than control. After 12 weeks, T4 was 1.52 ± 0.10 in control. T4 remained higher in HFD (1.93 ± 0.09), HFD+LD yolk (1.60 ± 0.09), HFD+HD yolk (1.62 ± 0.07), and ND+HD yolk (1.56 ± 0.06), with T4 in HFD+boiled yolk (1.28 ± 0.03) still below control. Between the two periods, T4 increased in control (1.45 → 1.52), HFD (1.62 → 1.93), and ND+HD yolk (1.53 → 1.56), while T4 decreased in HFD+LD yolk (1.64 → 1.60), HFD+HD yolk (1.90 → 1.62), and HFD+boiled yolk (1.36 → 1.28).

Table24: T4 *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	1.45 ± 0.07	1.52 ± 0.10
HFD	1.62** ± 0.01	1.93**± 0.09
HFD + LD yolk	1.64** ± 0.09	1.60* ± 0.09
HFD + HD yolk	1.90*** ± 0.14	1.62* ± 0.07
HFD + boiled yolk	1.36 **± 0.04	1.28** ± 0.03
ND + HD yolk	1.53* ± 0.05	1.56**± 0.06

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period)

Reproductive Hormone:

Testosterone (ng/ml):

It is evident from Table (25), after 6 weeks, testosterone was 380.50 ± 11.90 in control. Testosterone was lower in HFD (328.75 ± 0.64), HFD+LD yolk (346.75 ± 6.41), HFD+HD yolk (301.67 ± 13.32), HFD+boiled yolk (334.80 ± 9.44), and ND+HD yolk (361.62 ± 3.60) relative to control. After 12 weeks, testosterone was 381.67 ± 4.61 in control. Testosterone remained lower in HFD (309.83 ± 8.43), HFD+LD yolk (349.60 ± 3.74), HFD+HD yolk (328.20 ± 2.18), HFD+boiled yolk (322.60 ± 3.74), and ND+HD yolk (341.94 ± 2.19) than control. Between the two periods, testosterone was stable in control (380.50 → 381.67), decreased in HFD (328.75 → 309.83), increased slightly in HFD+LD yolk (346.75 → 349.60) and HFD+HD yolk (301.67 → 328.20), decreased in HFD+boiled yolk (334.80 → 322.60), and decreased in ND+HD yolk (361.62 → 341.94).

Table 25: Testosterone *O*f rats in different experimental groups at 6- and 12-weeks. values are expressed as Mean ± SE

Group	*6 W(Mean±SE)*	*12 W(Mean±SE)*
Control	380.50 ± 11.90	381.67 ± 4.61
HFD	328.75***± 0.64	309.83*** ± 8.43
HFD + LD yolk	346.75 ***± 6.41	349.60***± 3.74
HFD + HD yolk	301.67***± 13.32	328.20***± 2.18
HFD + boiled yolk	334.80***± 9.44	322.60 ***± 3.74
ND + HD yolk	361.62** ± 3.60	341.94*** ± 2.19

(*) significant at ($p < 0.05$), (**) high significant at ($p < 0.01$), and (***) very high significant at ($p < 0.001$) Relative to control (same period.

DISCUSSION

The present study investigated the combined effects of a high-fat diet (HFD) based on a commonly marketed non-hydrogenated vegetable ghee and egg yolk supplementation on metabolic health indicators in male rats. The findings revealed significant metabolic, hematological, immunological, and hormonal alterations, particularly in groups receiving a high-fat diet enriched with high doses of egg yolk. These results provide important insights into the potential risks associated with the concurrent consumption of cholesterol-rich foods and dietary fats that are perceived as less harmful due to marketing claims, as well as the dose-dependent role of egg yolk in modulating metabolic health.

The study demonstrated that rats fed on HFD exhibited substantial weight gain and increased BMI compared to the control group, confirming the obesogenic nature of the high-fat diet. The most pronounced effects were observed in the HFD combined with high-dose egg yolk (HFD+HD) and boiled yolk (HFD+boiled yolk) groups, indicating a synergistic effect of dietary cholesterol and saturated fats in promoting adiposity. This trend was consistent with the enhanced visceral fat accumulation and increased liver weights recorded in these groups. Interestingly, the group receiving a normal diet supplemented with high-dose egg yolk (ND+HD) maintained body weight and BMI values comparable to the control group, suggesting that the detrimental effect of egg yolk is highly dependent on the background diet composition. These observations align with previous evidence demonstrating that while egg yolk alone does not necessarily induce obesity in normocaloric settings, its combination with high-fat diets accelerates fat deposition and hepatic lipid overload (Erami *et al.,* 2019; Blesso & Fernandez, 2018).

An additional critical point is the role of the fat source used in this study. The vegetable ghee, marketed as non-hydrogenated and perceived as a healthier alternative to partially hydrogenated oils, was expected to exert milder metabolic effects. However, the results demonstrated significant adverse impacts, including obesity, dyslipidemia, inflammation, and hepatic stress. This finding raises concerns about the actual fatty acid profile and oxidative stability of such products, as even non-hydrogenated fats can contain high levels of saturated fatty acids and oxidation by-products (Mozaffarian *et al.,* 2006; Choe & Min, 2007). These results underscore the need for stricter quality control and transparency in labeling fat products, as consumer perception of safety may not reflect biochemical reality.

Body Weight In the present study, rats fed a high-fat diet (HFD) exhibited a significant increase in body weight after both 6 and 12 weeks compared to the control group. This finding agrees with previous reports showing that HFD feeding induces progressive weight gain due to increased caloric intake and fat accumulation (Buettner *et al.,* 2007). The inclusion of raw egg yolk in HFD-fed groups further exaggerated body weight gain, which is likely attributed to the high lipid and cholesterol content of egg yolk enhancing adiposity. However, cooked yolk supplementation showed a relatively attenuated effect, suggesting that thermal processing

may alter lipid bioavailability and reduce the obesogenic impact. The mechanisms underlying these findings may involve hyperphagia, elevated lipogenesis, and impaired satiety signaling mediated by leptin resistance and hypothalamic inflammation induced by saturated fats.

Hematological Parameters The results revealed a significant decline in RBC count, Hb, and PCV in HFD-fed groups, indicating the onset of anemia-like features. This is consistent with previous studies reporting that obesity and dyslipidemia are associated with altered hematopoiesis and reduced erythrocyte survival due to oxidative stress and inflammation (Guzik & Korbut, 2003). Furthermore, WBC count was significantly elevated in HFD groups, reflecting systemic inflammation and activation of innate immunity. Egg yolk supplementation further intensified leukocytosis, possibly due to the pro-inflammatory role of oxidized lipids. These alterations suggest that chronic HFD feeding induces hematological disturbances that mimic metabolic syndrome–associated inflammation.

Glucose Homeostasis and Insulin Resistance HFD-fed rats developed hyperglycemia and elevated HOMA-IR, indicating insulin resistance. This agrees with the work of Samuel and Shulman (2012), who demonstrated that high-fat feeding impairs insulin signaling in skeletal muscle and liver, leading to glucose intolerance. Egg yolk supplementation worsened insulin resistance, particularly with high doses of raw yolk, likely because dietary cholesterol and saturated fat disrupt insulin receptor signaling and promote ectopic lipid accumulation in insulin-sensitive tissues. Interestingly, the ND+HD group also displayed increased glucose and HOMA-IR, emphasizing that excess yolk consumption even under normal diet can impair glucose metabolism. Mechanistically, these effects may be mediated through mitochondrial dysfunction, increased reactive oxygen species (ROS), and activation of inflammatory cascades (NF-κB, TNF-α).

Lipid Profile Serum cholesterol, triglycerides, and LDL-c were markedly elevated in HFD groups, while HDL-c decreased significantly, indicating dyslipidemia. These results align with prior studies confirming that HFD induces hyperlipidemia and atherogenic lipid patterns (Rhee *et al.,* 2011). Egg yolk, rich in cholesterol, further aggravated dyslipidemia, with raw yolk being more potent than cooked yolk. The ND+HD group also showed elevated lipid levels, highlighting the strong hypercholesterolemic effect of egg yolk even without HFD background. The underlying mechanism involves increased intestinal cholesterol absorption, reduced LDL receptor expression, and hepatic overproduction of VLDL. Heat denaturation during cooking may reduce phospholipid availability, partly explaining the milder effect of cooked yolk.

Liver Function HFD-fed rats demonstrated elevated ALT, AST, and ALP levels, reflecting hepatocellular damage and cholestasis. This concurs with histological reports of HFD-induced non-alcoholic fatty liver disease (NAFLD) (Postic & Girard, 2008). Egg yolk supplementation intensified liver enzyme elevations, consistent with cholesterol-driven steatosis and oxidative stress. Cooked yolk showed relatively less hepatic enzyme elevation, suggesting a partial protective effect against severe steatosis. The mechanism may involve lipid peroxidation, mitochondrial dysfunction, and ER stress leading to hepatocyte injury.

Thyroid Function TSH was significantly elevated, while T3 and T4 levels declined in HFD groups. These results are in line with findings that obesity and hyperlipidemia are linked to hypothyroidism-like features (Mullur *et al.,* 2014). Egg yolk supplementation further suppressed thyroid hormones, possibly due to increased lipid peroxidation impairing thyroid gland activity and altered hepatic deiodinase function. This suggests that high-fat and cholesterol feeding induces thyroid dysfunction, contributing to metabolic slowdown and weight gain.

Reproductive Hormone (Testosterone) HFD feeding resulted in a marked reduction in serum testosterone levels, consistent with previous reports linking obesity to hypogonadism (Kelly & Jones, 2013). Egg yolk supplementation, particularly at high doses, further suppressed testosterone, potentially through increased aromatization of androgens to estrogens

in adipose tissue and oxidative stress impairing Leydig cell function. These findings imply that chronic high-fat and cholesterol intake negatively affects male reproductive health.

Conclusion:

The present study demonstrated that high-fat diet (HFD) feeding in male rats induced a wide spectrum of metabolic disturbances, including obesity, anemia-like hematological changes, dyslipidemia, insulin resistance, hepatic dysfunction, renal impairment, thyroid dysregulation, and reduced testosterone levels. Supplementation with egg yolk, especially in raw and high doses, further aggravated most of these alterations, suggesting that excessive dietary cholesterol and saturated fats exacerbate metabolic stress. Interestingly, cooked egg yolk showed a relatively attenuated effect, indicating that thermal processing may reduce some of the harmful impacts of yolk lipids.Overall, these findings highlight the detrimental impact of excessive fat and cholesterol intake on metabolic health and endocrine balance. The data suggest that egg yolk, although nutritionally rich, should be consumed with caution, particularly in the context of high-fat diets, to avoid aggravation of obesity-related complications. Future studies are recommended to further elucidate the molecular mechanisms underlying yolk-induced metabolic disturbances and to determine safe consumption levels in different dietary backgrounds.

Implications:

The findings of this study carry important implications for both experimental research and public health nutrition. From a scientific perspective, the results emphasize the need to consider not only the type of dietary fat but also the source and preparation method when modeling metabolic disorders in animals. The observed differences between raw and cooked egg yolk highlight the significance of food processing in modulating metabolic outcomes, which may guide future nutritional experiments. Clinically and nutritionally, the results suggest that excessive egg yolk intake, particularly when combined with high-fat diets, poses substantial risks for metabolic health, including obesity, insulin resistance, dyslipidemia, hepatic and renal dysfunction, thyroid disturbance, and reproductive hormone imbalance. These outcomes underscore the importance of revisiting dietary guidelines concerning egg yolk consumption, especially in populations with high prevalence of obesity and metabolic syndrome. Furthermore, the study provides a basis for preventive strategies that integrate dietary modifications and lifestyle interventions to mitigate the adverse effects of excessive fat and cholesterol intake.

Declarations:

Ethical Approval: This study was conducted in accordance with ethical procedures and policies approved by Animal care and Use Committee of Faculty of Science Al-Azhar University, Cairo, Egypt. The study was approved by Ethics Board of Al-Azhar University.

Competing Interests: The authors declare that they have no competing interests.

Authors' Contributions: Rawda E. Alnfrawy: Designed the study, conducted the experimental work on animals, collected blood and tissue samples, performed biochemical and hematological analyses, carried out statistical evaluation and data interpretation, and drafted the initial manuscript.**Prof. Ahkam**: Supervised the experimental design, provided scientific guidance, and critically revised the manuscript for important intellectual content.**Dr. Salah**: Assisted in supervising the biochemical and physiological analyses, validated the data, and contributed to manuscript revision.All authors read and approved the final version of the manuscript.

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