**Journal of Pioneering Medical Sciences**
Received: October 01, 2025 | Accepted: November 19, 2025 | Published: December 05, 2025
Volume 14, Issue 11, Pages 43-47

*DOI https://doi.org/10.47310/jpms2025141107*


# Genotoxic and Cytotoxic Effects of Quinoline Yellow in Albino Mice

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**Abstract** Quinoline Yellow (QY) is a synthetic dye frequently used in numerous products, such as foodstuffs and drinks, which raises concerns about its potential health hazards. This study aimed to assess the impact of QY on albino mice, with a specific focus on examining its effects on DNA and chromosomal integrity in bone marrow cells, as well as its influence on the proper functioning of the liver and kidney. It is an experimental *in vivo* study design. Six groups of Albino mice (each group containing 10 mice) were orally administered different dosages of QY and assessed using the Chromosomal Aberrations (CAs) assay and the Micronucleus Test (MNT) methods. To determine the influence of QY on liver and kidney function, serum markers, such as AST, ALT, urea and creatinine levels, were quantified. The most prevalent type of CA observed at all QY concentrations was sticky chromosomes. In each treatment group, a significant elevation ($p<0.05$) in AST, ALT, urea and creatinine levels was observed compared to the control group. Conclusively, the QY demonstrated a genotoxic impact and damage to bone marrow cell lines and had severely affected the normal functions of the liver and kidneys of the mice. Considering its usage, further research is crucial to ascertain the safety implications of QY in food and consumer products.

**Key Words** Quinoline, Chromosome Aberrations, Micronuclei Induction, DNA Damage

## INTRODUCTION

Numerous industries, including food, plastics, paper, cosmetics and pharmaceuticals, heavily rely on synthetic dyes. However, inadequate information is available about their potential to harm human health and the environment [1,2].

Synthetic dyes are artificial coloring agents used in foods and cosmetics, but their safety remains a topic of debate and is regulated differently across countries. Although health authorities have assessed its safety, research is still examining possible health concerns [3,4]. Additionally, if these colorants are metabolized or absorbed through the skin, they can have hazardous health consequences for people [1].

Quinoline yellow, commonly abbreviated as QY, is a yellow-green colorant (Figure 1) synthesized from the chinophthalon molecules. It finds extensive use in a wide range of products, including dietary supplements, health drinks, personal hygiene goods and medicine [1,5]. Nevertheless, artificial food colouring agents, particularly those containing aromatic azo compounds such as QY, have garnered attention due to the potential detrimental health consequences. Furthermore, a number of health ailments such as allergies, asthma, attention abnormalities and hyperactivity disorder have been associated with the continuously and excessive ingestion of QY [6,7]. In this direction, the Joint FAO/WHO Expert Committee on Food Additives (JECFA) has determined an acceptable limit of 10 mg/kg bw for QY ingestion [8].

Nevertheless, it has been found necessary to be aware of the acceptable limit of these dyes, specifically for children who are more sensitive than adults. As mentioned earlier, the accumulation of these colorants can result in potential health hazards [9].

There is limited data on the *in vivo* cytogenetic impact of QY stuffs. And still, *in vivo* cytogenetic investigations need to be conducted in conjunction with certain biochemical assays to confirm the QY activity and then assess safety. This study hypothesizes that QY significantly affects the functions of the liver and kidney in Albino mice. Therefore, this study aimed to evaluate the impact of QY on albino mice, with a specific focus on examining its effects on DNA and chromosomal integrity in bone marrow cells, as well as its influence on the proper functioning of the liver and kidney.

### Objectives

- **Primary:** To assess the genotoxic and cytotoxic effects of QY using CA and MN assays
- **Secondary:** To evaluate its effect on liver and kidney function markers

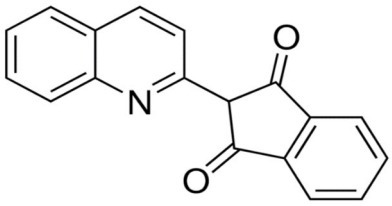


Figure 1: Quinoline Yellow (QY) Chemical Structure

## METHODS

### Study Design

It is an experimental *in vivo* study design conducted to assess the genotoxic and cytotoxic effects of QY using CA and MN assays.

### Animals

A total of 60 mice were distributed randomly (Randomization was done using a random number generator to assign mice to groups) into six groups of mice were orally given aqueous QY dosages of 0.01, 0.05, 0.1 and 0.2 g/kg b.wt. Daily for 45 days.

Mice were divided (n = 10 per group) as follows:

- Group I: Negative control (deionized water)
- Group II: Positive control (doxorubicin, 16 mg/kg b.wt.)
- Groups III–VI: QY-treated mice at doses of 0.01, 0.05, 0.1 and 0.2 g/kg bw

Albino mice, aged between 11 and 13 weeks old, weighing approximately 22 to 26 grams, were used in this study. The mice were housed in laboratory conditions with a temperature of 22°C. The humidity was maintained at 36.3±3.3% and the dark cycle followed a schedule of 12 hours of illumination followed by 12 hours of darkness. The mice were given unlimited accessibility to nutritious feed and clean drinking water throughout the study period.

### Treatment

All the chemical substances employed, including Quinoline Yellow (QY), were acquired from Sigma Chemical Co., based in St. Louis, MO, USA.

Doxorubicin was administered as the positive control at a dosage of 16 mg/kg body weight. Negative control involved the use of deionized water.

### Biochemical Analysis

Blood samples were collected from the vena cava and analyzed for biochemical parameters. Subsequently, the blood was centrifuged for 20 minutes at 3500 rpm.

Then, we collected and stored the resulting serum at -20°C until it was needed. Automated Chemical Analyzer 7070 (Hitachi, Japan) was used to measure the concentrations of ALT, AST, urea and creatinine in the blood.

### Cytogenic Analysis

### Chromosomal Aberrations (CA) Assay

The bone marrow cells were examined using a previously described method of Preston *et al.* [10] with suitable modifications. The mice were given an injection of colchicine (10 mg/kg b.wt.) two hours before, into their abdomen. Twenty-four hours after the last treatments, the mice were euthanized by cervical dislocation. The femur was collected from a warm KCl solution (0.075 M at 37°C) after all tissues were removed, following a 25-minute pre-incubation at 37°C.

The harvested bone marrow was subsequently centrifuged for 10 minutes at 2000 rpm and then fixed with a mixture of acid and methanol (1:3 volume ratio). This process involved centrifugation and fixation, which were repeated twice with a 10-minute interval. The cells were fixed in a solution and then placed on cooled glass slides, dried and stained with a 5% dilution of Giemsa solution. Two hundred metaphase cells were analysed in each group.

### Micronucleus (MN) Assay

According to Schmid's [11] methodology, the femoral bones were dissected to extract the bone marrow cells. Then, we centrifuged the cells at 2000 xg for 10 minutes. After removing the supernatant, we stained the bone marrow cell smears using the May Grunwald Giemsa solution, following the technique outlined in the study by Shahrim *et al.* [12]. For each treatment, a minimum of 400 cells were examined on encoded slides to assess the presence of micronuclei.

### Data Analysis

The SPSS program, version 27.0 and Microsoft Excel 2010 were used for the statistical analysis of the study findings. The data were documented as the Mean±SD. One-way ANOVA was used to perform the analysis of variance, followed by Tukey's multiple comparison tests. The Shapiro-Wilk test was chosen as the normality test. Statistical significance was determined for the results (*$p<0.05$ Vs control).

## RESULTS

### CA Assay

Taking into account the literature reports, it is evident that cytogenetic tests, such as the CA or micronuclei assay, are frequently employed to evaluate the mutagenic potential of chemical substances or food additives. The primary objective of this study was to investigate the cytotoxic and genotoxic effects of QY on the bone marrow cells of albino mice, utilizing CA and MN assays as key indicators of toxicity.

Various types of CAs were detected, including polyploidy, RCF, stickiness, fragmentation and gaps (Figure 2). The mean proportions of CAs were documented as 11.5, 14.25, 16.25 and 17% for QY dosages of 0.02, 0.05, 0.1 and 0.2 g/kg b.wt., respectively (Figure 3). QY demonstrated an increase in CA frequency that was concentration dependent.

### MN Assay

The QY treatment demonstrated an elevation in micronuclei frequency. Specifically, at QY doses of 0.02, 0.05, 0.1 and 0.2 g/kg body weight (b.wt.), the percentage of micronucleus frequencies was observed to be 6, 11.8, 12 and 18%, respectively. From the data obtained, it is clear that the concentration of micronuclei formation is directly related to QY concentration (Figure 4).

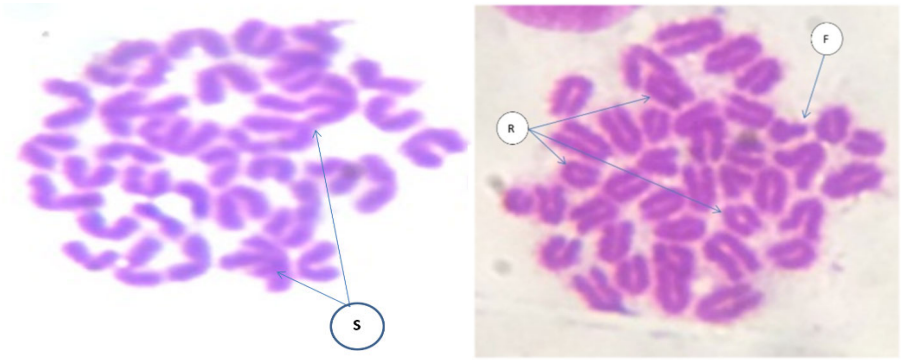


Figure 2: The Occurrence of CAs in Bone Marrow Cells Caused by Exposure to QY (Abnormalities Induced by QY in Mouse Bone Marrow. (S) Sticky Chromosomes, (F) Chromosomal Fragments, (R) Ranges)

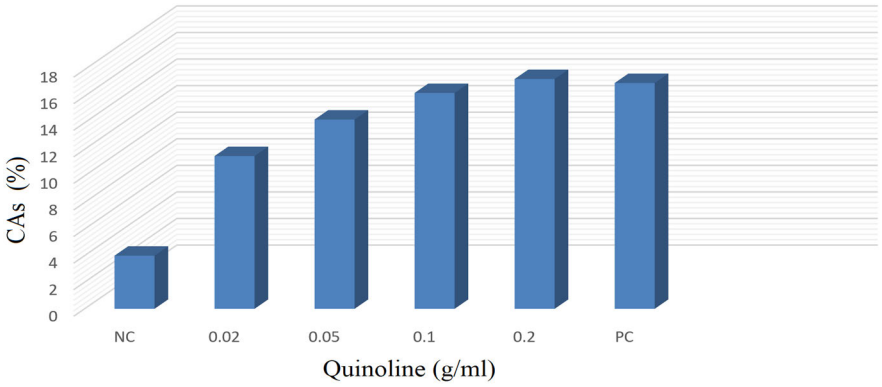


Figure 3: The Impact of Quinoline on the Occurrence of Chromosomal Aberrations

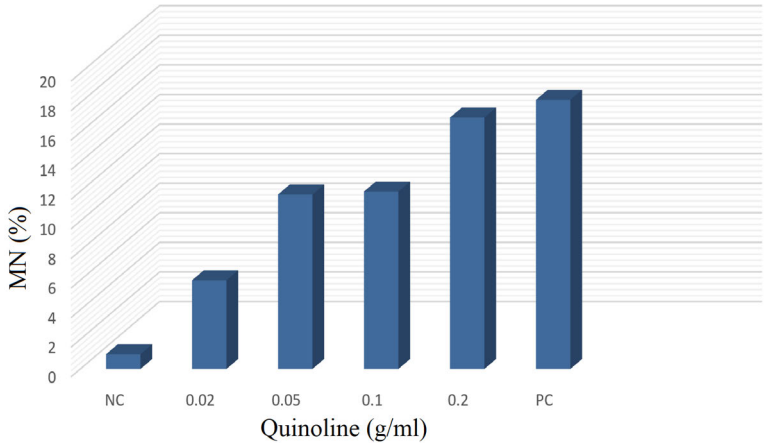


Figure 4: The Micronucleus Frequency in Bone Marrow Cells following QY Treatment

**Biochemical Parameters**

The impact of QY on liver and kidney functions in mice was reported. In every treatment group, a significant increase in AST and ALT levels was observed compared to the control group ($p<0.05$). As well, AST, ALT and urea levels at a dose of 0.2 g/mL of QY were significantly different (higher than controls) ($p<0.01$). A progressive dose-dependent alteration in renal and hepatic biochemical markers was observed following QY administration. At the lower doses of 0.02 and 0.05 g/mL, creatinine, blood urea, AST and ALT levels remained comparable to the negative control, suggesting no noticeable renal or hepatic toxicity. However, beginning at 0.1 g/mL, early signs of organ stress emerged, as evidenced by increases in creatinine (1.16 g/dL) and blood urea (39.70 g/dL), along with significant elevations in AST and ALT levels, indicating initial hepatocellular injury. Marked toxicity became evident at 0.2 g/mL, where creatinine and urea rose sharply to 1.40 g/dL and 56.10 g/dL, respectively, accompanied by substantial increases in AST (36.90 U/L) and ALT (42.00 U/L). As expected, the positive control group showed the highest elevations across all parameters, confirming severe hepatorenal dysfunction.

Table 1: The Serum Biochemical Parameters (Mean ± S.D) Reported by Effect of QY

| Creatinine (g/dL) | Blood Urea (g/dL) | AST (U/L) | ALT (U/L) | QY dose (g/mL) |
|---|---|---|---|---|
| 0.94±0.15 | 35.58±0.68 | 21.40±0.96 | 25.86±0.61 | Negative control |
| 0.90±0.16 | 28.60±0.96 | 20.0±0.79 | 25.84±0.59 | 0.02 |
| 0.80±0.16 | 30.78±0.56 | 20.60±0.96 | 25.42±0.68 | 0.05 |
| 1.16±0.11 | 39.70±1.20 | 23.0±0.79* | 29.50±0.79* | 0.1 |
| 1.40±0.16* | 56.10±2.30** | 36.90±1.29** | 42.0±0.79** | 0.2 |
| 3.06±0.11 | 133.0 | 55.0±3.61 | 81.0±0.79 | Positive control |

ALT Alanine aminotransferase, AST Aspartate aminotransferase. Level of significance *p<0.05, **p<0.01. All values are expressed as mean ± SD

Overall, the data demonstrate that QY is well tolerated at low doses, while doses at or above 0.1 g/mL trigger measurable toxicity, with 0.2 g/mL producing significant adverse biochemical effects (Table 1).

## DISCUSSION

The QY is a colorant dye that has been extensively used in dietary supplements, medications, personal care products, makeup, etc. Additionally, the QY possesses industrial applications as well. In the present investigation, CAs and MNT tests were applied to evaluate the effects of QY on the genetic material of mice. Additionally, the impact of QY on liver and kidney function was also investigated in the current study. Furthermore, these CAs and MNT tests were conducted to scan the DNA impairment and classify the variations observed in the treated cells. These assays aid in the investigation of changes that are typically the result of exposure to mutagens or carcinogens [13,14]. Our results demonstrated the dose-dependent increase in CAs and micronuclei induction after treatment with QY of bone marrow culture.

In a recent investigation, Chequer *et al.* [1] have also demonstrated the ill effects of QY in HepG2 cells and concluded that it has the capacity to damage and cause permanent mutations in DNA, therefore causing instability. Additionally, using the comet assay and CBMN-Cyt techniques, they confirmed that QY is genotoxic. Moreover, further insights obtained from Fluorescence In Situ Hybridization (FISH) assays have unveiled that QY has the potential to cause DNA damage through both aneugenic and clastogenic mechanisms.

Similarly, in an earlier investigation, Macioszek and Kononowicz [15] examined the genotoxic effects of QY on Human lymphocytes and Vicia faba root tips using micronucleus and Comet assays. They found that QY exhibited genotoxic effects. The outcomes of this study are in line with earlier investigations, thus clearly indicating that some synthetic compounds used as supplements exhibit genotoxic behavior [16,17]. The presence of Robertsonian Centric Fusion (RCF), chromatid gaps and chromosomes sticking together, etc., undoubtedly suggests that QY can disrupt the regular morphology and function of chromosomes. Conclusively, these genetic changes result in genomic instability as well as reorganization [17,18]. Herein, the sticking of chromosomes was the predominant type of CAs that have been visualized, thus once again highlighting the genotoxicity of QY. Likewise, the excessive presence of micronuclei at high concentrations of QY in bone marrow cells again suggests that QY has the ability to stimulate DNA damage and chromosomal instability [19].

On the other hand, numerous studies have investigated the effects of QY on the liver and kidney functions in mice.

The safety of QY were readily and powerfully illustrated in this study and dose-dependent effect in enhancing liver and kidney function were also reported, similar studies were reporting the same findings [20-23].

Among all experimental groups compared to the control group, the present findings indicated an increase in AST and ALT levels. Elevated levels of AST and ALT suggest liver damage or stress. Moreover, increased urea and creatinine levels with higher doses of QY indicate kidney dysfunction. The results of this study align with previous studies that have reported associations between the consumption of food additives and elevated levels of liver and kidney enzymes. For instance, Amin *et al.* [20] observed an elevation in several biochemical parameters, including blood levels of ALP, total protein, creatinine, AST, ALT, albumin and urea in the blood of rats following treatment with tartrazine [22-23]. Similarly, Sunset yellow was found to induce an increase in liver and kidney enzymes functions in male Sprague-Dawley rats [17].

These results imply that QY could have effects on both liver and kidney functions in mice. These biochemical markers indicate the functions of the liver and kidneys, which play a crucial role in detoxification and elimination of substances from the body. Several studies have observed changes, raising concerns about the safety of prolonged exposure to QY [21]. Our results are in close agreement with the previous investigations. These findings have significant implications for food safety and public health. Because QY is found in both food products and consumer products, the observed genotoxic and cytotoxic effects have raised potential health risks associated with its consumption.

## CONCLUSION

Quinoline Yellow (QY) demonstrated clear genotoxic and cytotoxic effects in albino mice. The observed dose-dependent increase in chromosomal aberrations and micronuclei formation confirms its ability to induce DNA damage and genomic instability. Consistent with previous studies, these findings suggest that QY may act through both clastogenic and aneugenic mechanisms. Additionally, elevated liver and kidney biochemical markers indicate that QY exposure can impair hepatic and renal functions. Together, these results highlight potential health risks associated with prolonged or high-dose exposure to QY and underscore the need for cautious use and further toxicological evaluation.

This study has few limitations, firstly, performing the study in *in vivo* models only and neglecting the *in vitro* studies (on cell lines), as well, studying other biochemical markers and antioxidant enzymes as well as the *in vitro* antioxidant investigation, short exposure duration, or lack of mechanistic assays are lacking in this study.

This study provides valuable data for the genotoxic and cytotoxic impacts of QY. Therefore, this study provides valuable insights into the genotoxic and cytotoxic effects of QY. Additional investigations are essential to assess the safety of synthetic food additives containing aromatic azo compounds.

Future studies exploring the genotoxic and cytotoxic effects of Quinoline Yellow (QY) in albino mice should aim to deepen mechanistic understanding, refine dose relationships and enhance translational relevance. While current findings highlight dose-dependent toxicity, further work is required to determine more precise safety thresholds. Expanding the dosing range, particularly around the lower concentrations where toxicity begins to appear, would allow clearer identification of NOAEL and LOAEL values and provide insight into potential reversibility through recovery-period assessments. Longitudinal designs, including acute, sub-acute and longer-term exposures, would also be valuable to clarify the onset, persistence and progression of genotoxic

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