

## Acrylamide Toxicity in Processed Foods: A Growing Public Health Concern

P. W. Lende, P. S. Harinkhede, V. D. Marbate, V. T. Dhurvey,  
A. M. Nagwanshi, S. S. Shahare, A. Sharma

<sup>1</sup> Department of Zoology, RTM Nagpur University, Nagpur 440033, MS India

\*E-Mail: [swapnilshahare165@gmail.com](mailto:swapnilshahare165@gmail.com)

Received April 21, 2025

The rising demand for processed foods, driven by population growth and urbanization, has heightened concerns about food safety, particularly regarding acrylamide (ACR), a compound formed during high-temperature processing of carbohydrate-rich foods. Originally identified as an industrial neurotoxicant, ACR is now recognized for its carcinogenic, neurotoxic, genotoxic properties and reproductive toxicity, making its presence in fried, baked, and roasted foods, is a significant public health concern. This review explores acrylamide formation, dietary sources, and harmful effects on organs like liver, intestine and gonads, including its effects on oxidative stress, inflammation, and DNA damage, as revealed by experimental studies. Emphasizing its health risks, this work highlights the urgent need for innovative effective mitigation strategies to reduce ACR levels in processed foods, ensuring consumer safety without compromising the sensory and nutritional quality of processed foods.

*Key words:* processed food, food safety, acrylamide, high temperature, carcinogenic, neuro-toxic, genotoxic, reproductive toxicity, health risk, mitigation, nutritional quality

Food is the component required for physiological process such as proper growth, metabolic processes and survival. With increase in population and urbanization, the demand for variety of foods, including both nutritious as well as indulgent food is also increasing. The children's and modern-day people are more exposed to indulgent foods, which are often rich, flavourful, and appealing, commonly contain various types of additives to enhance their taste, texture, appearance, and shelf life

Processing toxicants are substances formed during food processing or preparation that can have adverse physiological effects on human health. Ingredients used in food formulations can undergo chemical reactions under processing conditions such as heat, irradiation, or fermentation. The reaction products formed can be beneficial or harmful, depending on the conditions and processes used (Lineback and Stadler, 2009).

ACR was first discovered in 1997 due to a tunnel leakage in Sweden, where tunnel workers developed signs of impaired nerve function (Spencer and Schaumburg, 1974; Exon, 2006). The presence of ACR haemoglobin adducts suggested the possibility that ACR might also be consumed through the diet (Reynold, 2002; Timilsena *et al.*, 2010). In 2002, Swedish researchers reported the presence of ACR in several heat-treated carbohydrate-rich foods at significantly higher levels than other well-known food carcinogens (Tareke *et al.*, 2002; Koszucka *et al.*, 2019).

It has been placed in Group 2A carcinogen according to International Agency for Research on Cancer (IARC) specifying ACR as probably carcinogenic to humans based on previous experiment, studies and limited evidence in humans (Stadler *et al.*, 2002; Tareke *et al.*, 2002; Larginho *et al.*, 2014, Capuano and Fogliano, 2011; Parzefall, 2008; Tan *et al.*, 2013).

ACR (2 - propenamide) is a colorless, odourless, non-volatile, unsaturated amide with a molecular weight of approximately 71.08 kDa and it is found to be soluble in water and other polar solvents (acetone, methanol, ethanol) but is nearly insoluble in nonpolar solvents like carbon tetrachloride. It has high boiling point of about

125 °C (25 mm Hg), and melting point is at 84.5 °C, (Erkekoğlu and Baydar, 2010; Timilsena *et al.*, 2009; Krishnakumar and Visvanathan, 2014; Zamani *et al.*, 2017).

## Effect on Liver

Histology enables the observation of tissue and any distinctive alterations the tissue might have experienced. This helps to determine adverse effects in multiple organs of the model organism (Larginho *et al.*, 2014). To evaluate how ACR influences biochemical responses and diagnose any alteration, serum concentrations of various analytes are measured. (Wolf, 2018; Larginho *et al.*, 2014). Liver plays a key role in many metabolic processes, and liver injury disrupts these functions.

## Effect of ACR on Histology of Liver

Acrylamide (ACR) exposure has been shown to induce dose-dependent hepatotoxicity across various aquatic and terrestrial species. Ibrahim *et al.*, (2019) reported progressive liver damage in *Clarias gariepinus* exposed to 20–80% of the 96-hour LC50 (133 mg/L), ranging from mild necrosis and vessel congestion to extensive necrosis and cytoplasmic destruction at higher doses. Similarly, Kilicle *et al.*, (2020) observed sinusoid vasodilatation, nuclear anomalies, and severe fibrosis in *Capoeta capoeta* following intraperitoneal ACR administration (10–30 mg/L). In *Carassius auratus*, Larginho *et al.*, (2013) found eosinophilic alterations, necrosis, and inflammatory responses at concentrations up to 150 mg/L. Supporting these findings in mammals, Faridi *et al.*, (2024) showed that rats treated with 2 mg/kg ACR over four weeks developed significant hepatocellular damage, including necrosis, vacuolar degeneration, and pyknotic nuclei

## Biochemical Effects of ACR on Liver

Acrylamide (ACR) exposure has been linked to oxidative stress and metabolic disruption in various models. Ibrahim *et al.* (2019) reported increased malondialdehyde (MDA) levels and reduced GSH, SOD, and TAC in *Clarias gariepinus*, indicating lipid peroxidation and impaired antioxidant defenses. Kim *et al.*, (2014) found that zebrafish exposed to 150–300 ppm ACR showed elevated GOT and GPT levels, along

with increased serum glucose and triglycerides, reflecting liver damage and metabolic disturbances. In rats, Yalçın *et al.* (2020) observed reduced TAS, increased TOS, and heightened caspase-3 activity, suggesting oxidative stress and hepatocyte apoptosis. Similarly, Mallepogu *et al.* (2023) reported that ACR-treated chick embryos exhibited elevated ALT, AST, and ALP levels with reduced glutathione, further confirming oxidative liver damage.

### Effects of ACR On Gene Expression in Liver

In a study conducted by Ibrahim *et al.*, (2019) where fish were exposed to ACR at various concentrations of the 96-hour LC50 value (133 mg/L) for 2 weeks. According to this study genotoxicity was evident through DNA fragmentation assays, which showed dose-dependent increases in DNA fragmentation in the liver, indicating significant DNA damage. Accordingly, a study by Sheikh *et al.* (2010) exposed fertilized chick embryos to single doses of acrylamide (0.1 mg, 0.2 mg, and 0.3 mg in 50 µL saline) showed a dose-dependent induction of GST isoforms, including theta (CL1), mu (CL2), and alpha (CL3) classes, demonstrating an adaptive upregulation of detoxification pathways in response to acrylamide exposure. In another work by Chen *et al.*, (2021), acrylamide was administered orally to mice at doses of 50–75 mg/kg/day for 30 days, this showed upregulation of cytochrome P450-related genes, cancer-related genes (e.g., EGFR and Rad51), and inflammatory markers, while anti-oncogenes such as Bcl2 and P21 were downregulated. Kandemir *et al.*, (2020) carried his research by administering acrylamide at doses of 50 and 100 mg/kg body weight for a duration of 14 days in rats. Acrylamide affected gene expression by upregulating pro-apoptotic markers, including P53, which suggests enhanced apoptosis in liver cells.

### Effects On Intestine

Histological, Biochemical and Neurochemical biomarkers provide valuable data on specific organ toxicity, particularly in organs like the intestine, which are directly involved in the processing of food and toxins. Studies using these biomarkers help quantify the exposure to pollutants and elucidate the mechanisms of toxicity, providing insights into how toxicants like

acrylamide affect the organism's physiological functions (Samanta *et al.*, 2016). The intestines play a key role in the metabolism of orally ingested toxic substances, making them a critical organ for evaluating the impact of acrylamide in organisms (Van Dyk *et al.*, (2009).

### Effect of ACR on Histology of Intestine

Acrylamide (ACR) exposure has been shown to cause significant gastrointestinal toxicity in both aquatic and mammalian models. Modi *et al.* (2022) reported that zebrafish exposed to 1.22 mM ACR for 21 days experienced early mortality and intestinal damage, including goblet cell hyperplasia and enterocyte exfoliation. In rats, Ige *et al.* (2022) observed increased intestinal transit time, reduced mucosal cell counts, and severe duodenal lesions such as villi sloughing, crypt disruption, and inflammation following 28-day ACR exposure (7.5 and 15 mg/kg). Similarly, Koledin *et al.*, (2016) found that subchronic ACR treatment (25–50 mg/kg/day) in juvenile rats disrupted mucin production, notably reducing neutral, acidic, and MUC2 mucins, leading to compromised mucosal integrity. Sayyad *et al.*, (2017) also reported damage to the duodenal lining, villi, and lingual papillae in rats exposed to ACR and fried potato chips, along with evidence of DNA damage in intestinal and lingual tissues.

### Biochemical Effects of ACR on Intestine

Acrylamide (ACR) exposure has been shown to induce significant neurochemical and inflammatory changes in the gastrointestinal tract. Palus *et al.*, (2019) demonstrated that juvenile pigs exposed to ACR at 0.5 and 5 µg/kg/day for 28 days exhibited increased immunoreactivity to SP, CGRP, GAL, and VACHT in ileal and duodenal enteric neurons, with reduced nNOS in the ileum, suggesting altered ENS function. A follow-up study by Palus *et al.*, (2020) further showed that ACR exposure increased VIP-like immunoreactive neurons and altered their co-localization with neuroactive substances such as nNOS, SP, and CART, highlighting disruptions in intestinal neuronal signaling. Complementing these findings, Amirshahrokhi (2021) reported that rats treated with higher ACR doses (20–30 mg/kg/day) showed elevated colonic inflammation, oxidative stress markers, pro-inflammatory cytokines,

and NF- $\kappa$ B expression, along with reduced antioxidant defenses, indicating severe gastrointestinal and immunological disturbances.

## Effects On Gonad –

### Effect of ACR on Histology of Gonad

#### Male Gonad

Acrylamide exposure induces significant structural damage to testicular tissues. Studies in rodents show reduced testicular weight, degeneration of seminiferous tubules, and vacuolation in Sertoli cells (Mazen and Elnegris, 2013; Yilmaz *et al.*, 2017). Leydig cell hyperplasia and interstitial edema are frequently observed, accompanied by disrupted spermatogenesis and germ cell apoptosis (Wang *et al.*, 2010; Kucukler *et al.*, 2020). Histopathological findings reveal the severity of acrylamide's effects on the structural and functional integrity of testicular tissues.

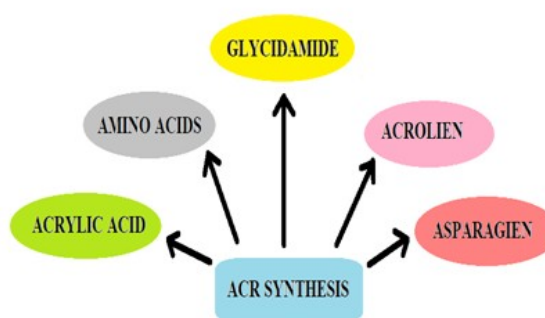
#### Female Gonad

Acrylamide exposure reduces follicular reserves and induces atresia in ovarian follicles. Pregnant guinea pigs exposed to acrylamide showed heightened follicular apoptosis, affecting ovarian function in offspring (Hulas-Stasiak *et al.*, 2013). Studies highlight the vulnerability of the ovarian reserve to acrylamide, particularly during critical developmental windows. Acrylamide crosses the placental barrier, disrupting fetal ovarian and testicular

development. This includes decreased follicle numbers, impaired Leydig cell function, and epigenetic alterations that may affect future generations (Aldawood *et al.*, 2022). Acrylamide's transgenerational impact underscores the need for protective measures, particularly for pregnant populations.

### Biochemical Effects of ACR on Gonad

Acrylamide induces oxidative stress by increasing reactive oxygen species (ROS) and lipid peroxidation while depleting antioxidant defenses like superoxide dismutase (SOD) and glutathione peroxidase (GPx) (Khalil *et al.*, 2014; Zhang *et al.*, 2010). Elevated malondialdehyde (MDA) levels in testes and ovaries indicate heightened lipid peroxidation, a marker of cellular damage (Lebda *et al.*, 2014). This imbalance between ROS production and antioxidant defenses plays a central role in acrylamide-induced gonadotoxicity. Acrylamide exposure alters key reproductive hormones, including testosterone, estradiol, luteinizing hormone (LH), and follicle-stimulating hormone (FSH). Decreased testosterone and increased LH levels in males disrupt spermatogenesis, while altered estradiol levels in females impair follicular development (Ma *et al.*, 2011; Wei *et al.*, 2014).



**Figure 1.** Structure of Acrylamide. **Figure 2.** Probable ways of ACR formation

**Table 1.-** Effect of Acrylamide on histology of liver

Model Organism	Dosage	Duration	Inference	References
<i>Clarias gariepinus</i>	20%, 40%, 60%, and 80%	2 weeks	vessel congestion, necrobiotic changes, diffuse necrosis, cytoplasmic destruction, nuclear damage, severe liver damage	Ibrahim et al., (2020)
<i>Capoeta capoeta</i>	10mg/L, 20mg/L, 30mg/L	4 days	sinusoid and central vein vasodilatation, melanomacrophages, necrosis, nuclear anomalies, pyknotic, karyorrhectic, and karyolytic nuclei, fibrosis, blood leakage into the liver parenchyma	Kilicle et al., (2020)
<i>Carassium auratus</i>	50 - 200 mg l <sup>-1</sup>	96h	eosinophilic alterations, focal necrosis, nuclear pleomorphisms (pyknosis), diffuse eosinophilic hepatocellular alterations, lipidic and eosinophilic degeneration, inflammatory responses, reduced pancreatic intrusions into the hepatic parenchyma	Larguinho et al., (2013),
<i>Rattus norvegicus</i>	2mg/kg	4 weeks	necrosis, vacuolar degeneration, granular cytoplasmic changes, pyknotic nuclei in liver tissue	Faridi et al., (2024)

**Table 2.** Biochemical effect of Acrylamide on liver

Model Organism	Dosage	Duration	Inference	References
<i>Clarias gariepinus</i>	20%, 40%, 60%, 80%	2 weeks	increased malondialdehyde (MDA) levels, decreased levels of reduced glutathione (GSH), superoxide dismutase (SOD), and total antioxidant capacity (TAC)	Ibrahim et al., (2019)
<i>Danio rerio</i>	300 ppm	26 hours	increases in serum glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) levels, acute elevations in serum glucose and triglyceride levels	Kim et al., (2015)
<i>Rattus norvegicus</i>	20mg/kg	30 days.	reduced the total antioxidant status (TAS) and increased the total oxidant status (TOS) in serum, immunohistochemically, ACR significantly increased caspase-3 immunoreactivity.	Yalçin et al., (2020)
fertilized eggs from the Babcock strain	0.5 mg and 1.0 mg	11 days	elevated levels of alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP), alongside decreased glutathione levels, which denote oxidative stress.	Mallepogu et al., (2023)

**Table 3.** Effect of Acrylamide on Gene Expression in Liver

Model Organism	Dosage	Duration	Inference	References
<i>Clarias gariepinus</i>	20%, 40%, 60%, 80%	2 weeks	increases in DNA fragmentation in the liver, indicating significant DNA damage	Ibrahim et al., (2019)
Chick embryo	0.1, 0.2, and 0.3 mg	72 hours	dose-dependent induction of GST isoforms (theta (CL1), mu (CL2), and alpha (CL3) classes),	Sheikh et al., (2010)
<i>Rattus norvegicus</i>	25- 75 mg/kg	30 days	upregulation of cytochrome P450-related genes, cancer-related genes (e.g., EGFR and Rad51), and inflammatory markers, while anti-oncogenes such as Bcl2 and P21 were downregulated	Chen et al., (2021)
<i>Rattus norvegicus</i>	50 and 100 mg/kg body weight	14 days	upregulating pro-apoptotic markers, including P53, which suggests enhanced apoptosis in liver cells	Kandemir et al., (2020)

**Table 4.** Effect of ACR on histology of Intestine.

Model Organism	Doses	Duration	Inference	Reference
Zebrafish	0.152, 0.305, 0.610 and 1.22 mM	21 days	widening of intracellular gaps, protein fluid accumulation, ovarian depletion of yolk granules in mature oocytes, disruption of intestinal prismatic structure, leading to goblet cell hyperplasia and enterocyte exfoliation.	Modi <i>et al.</i> , (2022)
Wistar Rat	0.2ml/ animal/day, 7.5mg/kg BD, 15mg/kg BD	28 days	Acrylamide reduced mucosal and intestinal cell counts in the duodenum, jejunum, and ileum. Duodenal samples showed severe coagulative necrosis, villus sloughing, necrotic debris, crypt disruption, moderate cell infiltration, and vascular congestion.	Ige <i>et al.</i> , (2022)
Rat	0,25, 50 mg/kg/day	five days per week for 21 days	Acrylamide reduced mucin production and secretion, with a notable decrease in goblet cells in the upper crypt. Neutral mucins and MUC2 mucins declined significantly, while acidic mucins showed a dose-dependent decrease. Sulfomucins were absent in the lower crypt and significantly reduced alongside sialomucins in the upper crypt.	Koledin <i>et al.</i> , (2016)
Neonatal rats	30,50 mg/kg body weight/day	21 days	Acrylamide caused mucosal injury, cell sloughing, glandular disarrangement, reduced mucosal thickness, and decreased PAS-positive reactions. Microscopic analysis revealed damage to parietal, chief, mucous, and enteroendocrine cells. Quercetin co-treatment mitigated most of these effects.	Sayyad <i>et al.</i> , (2017)

**Table 5.** Biochemical effect of ACR on Intestine.

Model Organism	Doses	Duration	Inference
female Danish Landrace pigs	0.5 ug/kg bw, 5 ug/kg bw	28 days	increase in substance P (SP), calcitonin generelated peptide (CGRP), galanin (GAL) and vesicular acetylcholine transporter (VACHT)-like immunoreactive (LI) neurons as well as a decrease in neuronal nitric oxide synthase (nNOS) -like immunoreactivity in all types of ileum intramural plexuses.
	0.5 -5 µg/kg body weight/day	4 weeks	Acrylamide caused increased immunoreactivity to neuroactive substances (e.g., substance P, CGRP, galanin, nNOS, and VACHT) within the duodenal ENS, indicating neurochemical changes. Increased oxidative stress and neurotoxic effects were suggested, impacting the enteric nervous system, as well as potential systemic effects such as neurotoxicity and carcinogenicity
Mouse (NMRI strain)	20 and 30 mg/kg/day.	21 days	Acrylamide exposure worsened colitis in mice, increasing disease activity index (DAI), body weight loss, mortality, and oxidative stress markers (MDA, carbonyl proteins). It also elevated proinflammatory cytokines (TNF-α, IL-1β, IL-6) and caused greater macroscopic and histological damage.
Pig (Danish landrace gilts)	Low dose (0.5 µg/kg body weight/day) and high dose (5 µg/kg body weight/day)	28 days	Acrylamide exposure led to significant changes in the population of vasoactive intestinal peptide (VIP)-like immunoreactive neurons in the small intestine's enteric nervous system. Increased VIP immunoreactivity was associated with signs of local inflammation, oxidative stress, and potential neuronal plasticity as an adaptation to acrylamide exposure.

**Table 6.** Histological Effects of Acrylamide on Gonads

Model Organism	Dosage	Duration	Inference	References
Albino rat	15 mg/kg	8 Weeks	Shrunken tubules, vacuolations, sloughed spermatogenic cells into the lumen	Mazen and Elnegris (2013)
Sprague Dawley	5 mg/kg/day and 10 mg/kg/day	8 Weeks	germ cell depletion, leydig cell hyperplasia, vacuolation in seminiferous tubules	Wang <i>et al.</i> , (2010)
Mice	1mM and 10mM	24 Hours	Oxidative stress, apoptosis in Leydig and Sertoli cell.	Yilmaz <i>et al.</i> , (2017)
Guinea pigs	3 mg/kg/day	32 Days	Reduction in the number of healthy ovarian follicles, increase in atretic follicles.	Hulas-Stasiak <i>et al.</i> , (2013)
Wistar Albino	2.5, 10 or 50 mg/kg	14 Days	Reduction in ovaries weight, apoptosis increased in granulosa cells.	Aldawood <i>et al.</i> , (2020)

**Table 7.** Biochemical effects of Acrylamide on Gonads

Model Organism	Dosage	Duration	Inference	References
Albino rat	5, 10 and 15 mg/kg bw	4 or 12 Weeks	Imbalance in the oxidant /antioxidant status, impairment in the testicular function and perturbations in the immune response genes in rats.	Khalil <i>et al.</i> , (2014)
Mice	0.05% (w/v)	21 Days	Elevation in malondialdehyde level (MDA), reduction in the level of reduced glutathione (GSH) and the glutathione-S-transferase (GST), glutathione peroxidase (GPX) and glutathione reductase (GR), reduced serum total testosterone and progesterone but increased estradiol (E2) levels.	Lebda <i>et al.</i> , (2014)
Sprague Dawley	5, 15, and 30 mg/kg per day	4 Weeks	Reduced testis and seminal vesicle weights, altered hormone levels (increased FSH and testosterone, decreased LH), and led to histological damage in the reproductive organs, such as epithelial cell degeneration in the epididymis.	Ma <i>et al.</i> , (2011)
Mice	20 mg/kg and 40 mg/kg per day	30 Days	Potential disruption of ovarian function and decrease in the number of corpora lutea	Wei <i>et al.</i> , (2014)

**Table 8.** Molecular Effects of Acrylamide on Gonads

Model Organism	Dosage	Duration	Inference	References
Mice	1mM and 10mM	24 Hours	upregulated pro-apoptotic genes like Bax and Caspase-3, downregulated anti-apoptotic genes such as Bcl-2 in leydig and sertoli cells	Yilmaz <i>et al.</i> , (2017)
Mice	ACR- 0, 100, 500 and 1000uM GLY 25, or 250 µM	7 Days	Elevated expression of p53, a tumor suppressor gene, suggesting DNA damage.	Aras <i>et al.</i> , (2017)
F344	0.0, 0.5, 1.5, 3.0, 6.0 or 12 mg/kg	31 Days	Reduced expression of the StAR, altered expression of FSHR and LHR affects germ cell function	Recio <i>et al.</i> , (2017)
Mice	0.001, 0.01, 0.1, 1 and 10 µg/ml	1 Year	Downregulation of DNA repair genes increases susceptibility to mutations and chromosomal aberrations	Nixon <i>et al.</i> , (2012)

### Effects of ACR on Gene Expression in Gonad

Acrylamide upregulates pro-apoptotic genes like Bax and Caspase-3 while downregulating anti-apoptotic genes such as Bcl-2 in Leydig and Sertoli cells (Yilmaz *et al.*, 2017). Elevated expression of p53, a tumor suppressor gene, suggests a cellular response to DNA damage (Aras *et al.*, 2017). These genetic alterations initiate apoptosis and exacerbate gonadal dysfunction. Acrylamide disrupts genes involved in steroid hormone biosynthesis and spermatogenesis. Reduced expression of the StAR (steroidogenic acute regulatory) protein impacts testosterone production, while altered expression of FSHR and LHR affects germ cell function (Recio *et al.*, 2017; Ma *et al.*, 2011). Such disruptions in gene expression interfere with the production of critical reproductive hormones and gametes. Glycidamide, acrylamide's active metabolite, forms DNA adducts that interfere with replication and repair. Downregulation of DNA repair genes increases susceptibility to mutations and chromosomal aberrations (Nixon *et al.*, 2012; Katen and Roman, 2015).

### SUMMARY AND CONCLUSION

Acrylamide (ACR), a chemical formed during high-temperature cooking of carbohydrate-rich foods, is increasingly recognized as a public health concern due to its carcinogenic, neurotoxic, genotoxic, and reproductive effects. Experimental studies have shown that ACR exposure causes oxidative stress, inflammation, DNA damage, and hormonal imbalances, affecting key organs like the liver, intestine, and gonads in both animals and humans.

Given its widespread presence in processed foods and its toxic effects on multiple organ systems, reducing acrylamide exposure is essential. This can be achieved through improved food processing techniques, regulatory policies, and public education, ensuring food safety while maintaining nutritional and sensory quality.

### CONFLICTS OF INTEREST

The authors declare that they have no potential conflicts of interest.

### REFERENCES

- Aldawood, N., Jalouli, M., Alrezaki, A., Nahdi, S., Alamri, A., Alanazi, M., ... and Harrath, A. H. (2022). Fetal programming: In utero exposure to acrylamide leads to intergenerational disrupted ovarian function and accelerated ovarian aging. *Aging (Albany NY)*, 14(17), 6887.
- Aras, D., Cakar, Z., Ozkavukcu, S., Can, A., and Cinar, O. (2017). In Vivo acrylamide exposure may cause severe toxicity to mouse oocytes through its metabolite glycidamide. *PLoS One*, 12(2), e0172026.
- Begum Sheikh, R., and Kedam, T. (2010). Effect of acrylamide on chick embryonic liver glutathione S-transferases. *Mediterranean Journal of Nutrition and Metabolism*, 3, 31-38.
- El-Sayyad, H., SOAD, A., AFAF, A., Eman, A., and EHSAN, M. (2017). Abnormal soft palate, lingual mucosa and intestine of neonates maternally fed on diet containing fried potatoes chips or received acrylamide treatment. *Journal of Pharmacological Reports*, 2(3).
- Erkekoğlu, P., and Baydar, T. (2010). Toxicity of acrylamide and evaluation of its exposure in baby foods. *Nutrition research reviews*, 23(2), 323-333.
- Exon, J. H. (2006). A review of the toxicology of acrylamide. *Journal of Toxicology and Environmental Health, Part B*, 9(5), 397-412.
- Faridi, U., Zidan, N., Atteia, H., Al-Awthan, Y., Alalawy, A. I., Akhtar, M. Q., ... and Parveen, H. (2024). Anticancer and Hepatoprotective Role of Selenium Nanoparticles against Liver Carcinogen Acrylamide Induced Toxicity: In vitro and in vivo Studies. *Indian Journal of Pharmaceutical Education and Research*, 58(3), 830-836.
- Hułas-Stasiak, M., Dobrowolski, P., Tomaszewska, E., and Kostro, K. (2013). Maternal acrylamide treatment reduces ovarian follicle number in newborn guinea pig offspring. *Reproductive Toxicology*, 42, 125-131.
- Ibrahim, M. A., and Ibrahim, M. D. (2020). Acrylamide-induced hematotoxicity, oxidative stress, and DNA



- damage in liver, kidney, and brain of catfish (*Clarias gariepinus*). *Environmental toxicology*, 35(2), 300–308.
- Ige, A. O., Ayoola, O. I., Oladejo, E. O., Adele, B. O., Ola, O. O., and Adewoye, E. O. (2022). Gastrointestinal motility and Intestinal structure following oral exposure to acrylamide in Wistar rats. *Research journal of health sciences*, 10(1), 27-39.
- Jiang, G., Lei, A., Chen, Y., Yu, Q., Xie, J., Yang, Y., ... and Su, D. (2021). The protective effects of the *Ganoderma atrum* polysaccharide against acrylamide-induced inflammation and oxidative damage in rats. *Food and function*, 12(1), 397-407.
- Kandemir, F. M., Yıldırım, S., Kucukler, S., Caglayan, C., Darendelioğlu, E., and Dortbudak, M. B. (2020). Protective effects of morin against acrylamide-induced hepatotoxicity and nephrotoxicity: A multi-biomarker approach. *Food and chemical toxicology*, 138, 111190.
- Khalil, W. K. B., Ahmed, H. H., Hanan, F., Aly, H., and Eshak, M. (2014). Toxicological effects of acrylamide on testicular function and immune genes expression profile in rats. *J Pharm Sci Rev Res*, 24(1), 143-151.
- Kılıçlı, P. A., Koç, E., Doğan, A., Gül, S., Doğan, A. N. C., and Ersan, Y. (2020). Investigation of the effect of acrylamide on *Capoeta capoeta* (Guldenstead 1773) by histopathological, electrophoretic and biochemical methods. *Caucasian Journal of Science*, 7(2), 153-166.
- Kim, S. M., Baek, J. M., Lim, S. M., Kim, J. Y., Kim, J., Choi, I., and Cho, K. H. (2015). Modified lipoproteins by acrylamide showed more atherogenic properties and exposure of acrylamide induces acute hyperlipidemia and fatty liver changes in zebrafish. *Cardiovascular toxicology*, 15, 300-308.
- Koledin, I., Kovač, R., Rajković, V., and Matavulj, M. (2016). Subchronic exposure to acrylamide affects colon mucin secretion in juvenile wistar rats. *Archives of Biological Sciences*, 68(3), 641-649.
- Koszucka, A., and Nowak, A. (2019). Thermal processing food-related toxicants: A review. *Critical reviews in food science and nutrition*, 59(22), 3579-3596.
- Kotsiou, K., Tasioula-Margari, M., Capuano, E., and Fogliano, V. (2011). Effect of standard phenolic compounds and olive oil phenolic extracts on acrylamide formation in an emulsion system. *Food chemistry*, 124(1), 242-247.
- Krishnakumar, T., and Visvanathan, R. (2014). Acrylamide in food products: a review. *J Food Process Technol* 5: 344.
- Larguinho, M., Costa, P. M., Sousa, G., Costa, M. H., Diniz, M. S., and Baptista, P. V. (2014). Histopathological findings on *Carassius auratus* hepatopancreas upon exposure to acrylamide: correlation with genotoxicity and metabolic alterations. *Journal of Applied Toxicology*, 34(12), 1293-1302.
- Lebda, M., Gad, S., and Gaafar, H. (2014). Effects of lipoic acid on acrylamide induced testicular damage. *Materia socio-medica*, 26(3), 208.
- Lineback, D. R., and Stadler, R. H. (2009). Introduction to food process toxicants. *Process-induced food toxicants*, 3-21.
- Ma, Y., Shi, J., Zheng, M., Liu, J., Tian, S., He, X., ... and Zhu, J. (2011). Toxicological effects of acrylamide on the reproductive system of weaning male rats. *Toxicology and industrial health*, 27(7), 617-627.
- Mallepogu, V., Mallapu, R. E., Vadabingi, N., Pasala, C., Punuri, J. B., Amineni, U., ... and Kedam, T. R. (2023). Acrylamide toxicity inhibits chick embryo hepatic alpha, mu, and pi-glutathione S-transferases: molecular dynamic simulations, substrate specificity, and docking. *Toxicology and Environmental Health Sciences*, 15(3), 289-302.
- Mazen, N. F., and Elnegris, H. M. (2013). Role of coenzyme Q10 in testicular damage induced by acrylamide in weaned albino rats: a histological and immunohistochemical study. *Egyptian Journal of Histology*, 36(1), 164-174.
- Modi, C. M., Kachot, R. L., Patel, H. B., Patel, U. D., and Kariya, M. H. (2022). Histopathological evaluation of ovary and intestine of adult zebrafish exposed to

- acrylamide. *Journal Of Veterinary Pharmacology And Toxicology*, 21(1), 32-34.
- Nixon, B. J., Stanger, S. J., Nixon, B., and Roman, S. D. (2012). Chronic exposure to acrylamide induces DNA damage in male germ cells of mice. *Toxicological Sciences*, 129(1), 135-145.
- Palus, K., and Calka, J. (2019). Influence of acrylamide administration on the neurochemical characteristics of enteric nervous system (ENS) neurons in the porcine duodenum. *International Journal of Molecular Sciences*, 21(1), 15.
- Palus, K., Bulc, M., and Calka, J. (2020). Effect of acrylamide supplementation on the population of vasoactive intestinal peptide (VIP)-like immunoreactive neurons in the porcine small intestine. *International Journal of Molecular Sciences*, 21(24), 9691.
- Palus, K., Obremski, K., Bulc, M., and Calka, J. (2019). The impact of low and high doses of acrylamide on the intramural neurons of the porcine ileum. *Food and Chemical Toxicology*, 132, 110673.
- Park, J. S., Samanta, P., Lee, S., Lee, J., Cho, J. W., Chun, H. S., ... and Kim, W. K. (2021). Developmental and neurotoxicity of acrylamide to zebrafish. *International Journal of Molecular Sciences*, 22(7), 3518.
- Parzefall, W. (2008). Minireview on the toxicity of dietary acrylamide. *Food and Chemical Toxicology*, 46(4), 1360-1364.
- Recio, L., Friedman, M., Marroni, D., Maynor, T., and Chepelev, N. L. (2017). Impact of acrylamide on calcium signaling and cytoskeletal filaments in testes from F344 rat. *International journal of toxicology*, 36(2), 124-132.
- Reynolds, T. (2002). Acrylamide and cancer: tunnel leak in Sweden prompted studies. *Journal of the National Cancer Institute*, 94(12), 876-878.
- Spencer, P. S., and Schaumburg, H. H. (1974). A review of acrylamide neurotoxicity part I. Properties, uses and human exposure. *Canadian Journal of Neurological Sciences*, 1(2), 143-150.
- Stadler, R. H., Blank, I., Varga, N., Robert, F., Hau, J., Guy, P. A., ... and Riediker, S. (2002). Acrylamide from Maillard reaction products. *Nature*, 419(6906), 449-450.
- Tan, D., Li, L., Wang, S., Wei, B., Zhang, X., Sun, B., and Ji, S. (2013). The cytogenetic effects of acrylamide on *Carassius auratus* peripherial blood cells. *Food and chemical toxicology*, 62, 318-322.
- Tareke, E., Rydberg, P., Karlsson, P., Eriksson, S., and Törnqvist, M. (2002). Analysis of acrylamide, a carcinogen formed in heated foodstuffs. *Journal of agricultural and food chemistry*, 50(17), 4998-5006.
- Timilsena, Y. P., Khanal, J. S., and Anal, A. K. (2010). Acrylamide: thermally induced toxicant in foods and its control measures. *Journal of Food Science and Technology Nepal*, 6, 19-30.
- Van Dyk, J. C., Marchand, M. J., Pieterse, G. M., Barnhoorn, I. E., and Bornman, M. S. (2009). Histological changes in the gills of *Clarias gariepinus* (Teleostei: Clariidae) from a polluted South African urban aquatic system. *African Journal of Aquatic Science*, 34(3), 283-291.
- Wang, H., Huang, P., Lie, T., Li, J., Hutz, R. J., Li, K., and Shi, F. (2010). Reproductive toxicity of acrylamide-treated male rats. *Reproductive Toxicology*, 29(2), 225-230.
- Wei, Q., Li, J., Li, X., Zhang, L., and Shi, F. (2014). Reproductive toxicity in acrylamide treated female mice. *Reproductive Toxicology*, 46, 121-128.
- Wolf, M., Gasparin, B. C., and Paulino, A. T. (2018). Hydrolysis of lactose using  $\beta$ -d-galactosidase immobilized in a modified Arabic gum-based hydrogel for the production of lactose-free/low-lactose milk. *International Journal of Biological Macromolecules*, 115, 157-164.
- Yalçın, A., and Pekmez, H. (2020). Black carrot juice: A new approach to cure acrylamide-induced hepatotoxicity in rats. *Ankara Sağlık Bilimleri Dergisi*, 9(1), 207-216.
- Yilmaz, B. O., Yildizbayrak, N., Aydin, Y., and Erkan, M. (2017). Evidence of acrylamide and glycidamide-induced oxidative stress and apoptosis in Leydig and Sertoli cells. *Human and experimental toxicology*, 36(12), 1225-1235.

- 
- Zamani, E., Shokrzadeh, M., Fallah, M., and Shaki, F. (2017). A review of acrylamide toxicity and its mechanism. *Pharmaceutical and biomedical research*, 3(1), 1-7.