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Expert Opinion **15/06/2025**

Subject: Medical expert opinion in oncology regarding the risks associated with exposure to frying oils as contributors to neoplastic mutations in body tissues, with particular emphasis on cancers of the gastrointestinal system

Academic and Clinical Background:

- Graduate of the Faculty of Medicine, Ben-Gurion University of the Negev, License No. 17896
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- Associate Professor, Department of Oncology, Faculty of Medicine, Tel Aviv University
- Associate Professor, School of Continuing Medical Education, Faculty of Medicine, Tel Aviv University
- Member of national and international oncology associations (Israel, Europe, USA)
- Fellowship at Memorial Sloan Kettering Cancer Center, New York, USA
- Fellowship at Cookridge Oncology Hospital, Leeds, UK
- Appointed medical expert for Israeli labor courts
- Senior Oncology Consultant, Oncology Institute, Barzilai Medical Center

Introduction

Repeated use of frying oil is a common practice in industrial kitchens, restaurants, and food production facilities. However, subjecting oils to high temperatures over multiple cycles leads to the breakdown of triglycerides and the formation of hazardous chemical byproducts. These substances—ranging from aldehydes and trans fats to polycyclic aromatic hydrocarbons (PAHs), heterocyclic amines (HCAs), and acrylamide—have been shown in numerous studies to possess mutagenic and carcinogenic potential.

The purpose of this review is to present:

- The **mechanisms** by which repeated frying leads to the generation of **carcinogenic compounds**, and
- How these substances—byproducts of oil frying—affect **cells and organs of the gastrointestinal system**.
- A technological innovation—**Beyond Oil**—that may mitigate these health risks by filtering harmful compounds and extending oil usability.

Chemistry of Oils During Frying

Frying is a process in which food is immersed in hot oil, typically between 150–190°C. During frying, interaction occurs between oil, air, and food. The external surface of the food, its moisture content, and the type of oil used all influence the amount of oil absorbed.

Key nutritional components in oil—such as tocopherols, essential fatty acids, and amino acids—are degraded during frying. As oil is heated repeatedly, several chemical reactions occur: **hydrolysis**, **oxidation**, and **polymerization**. These reactions produce both **volatile** and **non-volatile** degradation products.

- **Volatile compounds** may evaporate or be absorbed into the fried food.
- **Non-volatile compounds** accumulate and alter the physical and chemical characteristics of both the oil and the food.

One important indicator of oil degradation is the level of **Total Polar Materials (TPM)**, also referred to as **Total Polar Compounds (TPC)**. These are formed through hydrolysis, oxidation, and polymerization processes and include:

- Free fatty acids (FFA)
- Low molecular weight degradation products
- Polymerized triglycerides

High frying temperatures, an increased number of frying cycles, the presence of FFAs, unsaturated fatty acids, and multivalent metals all reduce oxidative stability and the sensory quality of oil.

The European Union has set a **maximum acceptable TPM value of 25–27%**. Oils exceeding this level are considered unsuitable for frying. High TPM levels indicate accumulation of degraded materials such as peroxides, aldehydes, ketones, alcohols, and other short-chain fatty acids—many of which are harmful.

Chemical Reactions Occurring During Repeated Frying

Hydrolysis

Moisture released from the food interacts with the oil and oxygen, initiating breakdown of triglyceride ester bonds. This results in the formation of **free fatty acids (FFA)**, **monoglycerides**, and **diglycerides**. The FFA level increases with frying duration and serves as a key quality marker.

Oxidation

Oxygen in the air reacts with oil at high temperatures, especially in the presence of light, metals (e.g., iron), and pre-existing oxidation products. This promotes the formation of **hydroperoxides**, which then degrade into:

- Volatile aldehydes and ketones
- Short-chain carboxylic acids and alkanes

These compounds contribute to rancidity and reduce oil quality.

Polymerization

Thermal stress causes unsaturated fatty acids to polymerize, forming **non-volatile polar compounds**, including **dimers and polymers of triglycerides**. These compounds:

- Increase viscosity
- Reduce heat transfer efficiency
- Cause foaming
- Impair food quality
- Promote further oil breakdown

The extent of polymerization depends on the oil type, temperature, and number of frying cycles. More cycles and higher temperatures accelerate polymer formation and oil degradation.

Factors Affecting Oil Quality During Deep Frying

Several parameters influence the degree of oil degradation during frying:

- Oil replacement frequency
- Frying temperature and duration
- Type of heating device
- Initial oil composition and quality
- Type of food being fried
- Frying equipment

- Antioxidant content
- Oxygen exposure

Studies show that frequent addition of fresh oil improves frying quality, delays accumulation of degradation products, and reduces the formation of FFAs, diacylglycerols, and polymers.

Harmful Chemical Compounds Formed During Repeated Frying

During repeated use of oil, several hazardous compounds accumulate. These substances are associated with oxidative stress, mutagenesis, and carcinogenesis. Key compounds include:

Free Fatty Acids (FFA):

Formed through hydrolysis, FFAs increase with frying time. The FFA value serves as a critical marker for oil quality deterioration.

Total Polar Materials (TPM):

Includes FFAs, oxidation products, and polymerized compounds. The **European Union** has established **25–27% TPM** as the upper threshold for acceptable oil quality. Beyond this, oil is considered degraded and unsuitable for further use.

Polymers and Dimers:

Formed through polymerization of triglycerides during extended heating. These non-volatile compounds increase oil viscosity, reduce heat transfer, and contribute to foaming during frying.

Volatile Compounds:

Include **aldehydes, ketones, and alcohols**, formed from oxidative degradation of oil. These compounds contribute to both sensory deterioration and potential health hazards.

Studies Evaluating the Impact of Repeated Frying

Numerous studies have demonstrated a **direct linear relationship** between oil degradation and frying conditions—specifically, the **number of frying cycles**, **heating duration**, and **temperature**. As these variables increase, so does the concentration of polar and toxic compounds with significant health implications.

- **Giuffrè et al. (2017)[1]:**

Studied the effects of temperature and heating time on sunflower oil. Oil was heated at 180°C, 210°C, and 240°C for 15–120 minutes. Parameters such as peroxide value (PV), p-anisidine value (p-AnV), total oxidation (TOTOX), and phenol content were assessed.

Findings:

- Oil oxidation worsened with increased temperature and time.
- TOTOX and PV increased significantly.
- A 50% reduction in antioxidants was observed.
- Free acidity rose from 0.15% to 0.35%, indicating triglyceride breakdown.

Conclusion: Extended heating and high temperatures significantly degrade oil and generate harmful oxidation products. Heating duration should be minimized for safe frying use.

- **Nor Mahiran et al. (2023)[2]:**

Investigated chemical changes in reheated palm oil (RHPO) and their role in forming **glycidyl esters (GEs)** and **3-MCPD**—both potentially carcinogenic compounds. Oil was heated for 90–360 minutes and tested using peroxide (PV), p-anisidine (p-AnV), total oxidation (TOTOX), and spectroscopic methods (FTIR, NMR).

Findings:

- GEs and 3-MCPD levels rose significantly with heating duration.
- Between 180–360 minutes, 3-MCPD converted more into GE, indicating the accumulation of toxic compounds.

Conclusion: Prolonged heating of palm oil results in the formation of carcinogenic contaminants.

- **Szabo et al. (2022)[3]:**

Explored the effect of repeated heating on fatty acid profiles and oxidation in five vegetable oils: palm, canola, soybean, sunflower, and extra virgin olive oil. Each was heated at 180°C for 5 minutes across 1, 5, and 10 cycles.

Findings:

- Essential fatty acids (LA and ALA) decreased significantly.
- **Trans fatty acids (TFA)** increased sharply—canola oil showed a **233% rise** after 10 cycles.
- **Atherogenic Index (AI)** and **Thrombogenic Index (TI)** increased, especially in sunflower and soybean oils.

Conclusion: Repeated heating reduces nutritional value, promotes formation of harmful lipids, and may elevate cardiovascular disease risk.

Carcinogenic Compounds Formed During Repeated Frying

As detailed above, repeated frying causes chemical degradation of oil, including **oxidation, thermal breakdown, and polymerization of fatty acids**. These processes lead to the formation of toxic byproducts with **carcinogenic potential**. When oil is reused for frying various foods, additional harmful compounds may form due to prolonged exposure to high heat and reactive oxygen species.

Notable among these are:

- **Heterocyclic Aromatic Amines (HCAs)**
- **Polycyclic Aromatic Hydrocarbons (PAHs)**
- **Acrylamide (AA)**

- **Liu et al. (2024)** [4] **–** ***Effect of Oil Degradation on Formation of Carcinogens***

In a recent study, **Liu et al. (2024)** examined the effect of frying oil degradation on the formation of carcinogenic substances—including **acrylamide (AA)**, **heterocyclic aromatic amines (HCAs)**, and **polycyclic aromatic hydrocarbons (PAHs)**—during the frying of various foods (potatoes, tofu, and chicken wings) at temperatures between **150°C and 190°C**.

Findings:

- As frying temperature and duration increased, so did the accumulation of hazardous contaminants.
- **Potatoes** generated the highest levels of **acrylamide (AA)** (2.60 mg/kg),
- **Chicken wings** showed the highest levels of **HCAs** (5.06 µg/kg) and **PAHs** (5.18 µg/kg).
- A positive correlation was identified between oil degradation—measured via **oxidative indicators** such as AV, CV, and TPC—and the levels of these carcinogens.

Conclusion:
Repeated oil use significantly contributes to the production of toxic and carcinogenic substances in fried foods, reinforcing the health risk associated with degraded frying oils. *(Liu et al., 2024)*

- **Ganesan et al. (2019)** [5]**:**

Conducted a comprehensive review of clinical, epidemiological, and animal studies that evaluated the effects of consuming **repeatedly heated cooking oils (RCOs)** on cancer risk.

Findings:

- Reheating oils leads to the formation of carcinogenic compounds, including **PAHs**, **HCAs**, and **lipid oxidation products**.
- These compounds were associated with an increased incidence of **colorectal, lung, breast, and prostate cancers**.
- Animal studies demonstrated **genotoxic damage**, chromosomal aberrations, and systemic inflammation.
- Human studies showed elevated rates of **neoplastic changes** among individuals exposed to frying oil fumes or those who regularly consumed foods fried in reused oils.

Conclusion:
Reusing frying oil represents a significant and avoidable **risk factor for carcinogenesis**, warranting stronger **nutritional and industrial regulatory oversight**.
(Ganesan et al., 2019)

1. Acrylamide (AA)

Acrylamide is formed through the **Maillard reaction**, a chemical interaction between the amino acid **asparagine** and sugars during high-temperature cooking, especially in **starchy foods** like potatoes. Acrylamide is metabolized in the body into **glycidamide**, a known mutagen that damages DNA.

- The **International Agency for Research on Cancer (IARC)** [6] classified acrylamide as a **probable human carcinogen (Group 2A)** in 1994.

Key Studies:

- **Tareke et al. (2002)** [7]**:**
  Analyzed acrylamide formation in carbohydrate- and protein-rich foods using GC-MS and LC-MS/MS.
  Findings:
  - Acrylamide levels were high (150–4000 µg/kg) in starchy foods (potatoes, bread) heated above 120°C.
  - In contrast, protein-rich foods (meat, fish) showed significantly lower levels (5–50 µg/kg).
  - Boiling with water produced **no detectable acrylamide**.
    Conclusion: Heating of starchy foods is the main dietary source of acrylamide.

- **Palus (2024)** [8]**:**
  A recent review in *Nutrients* examined acrylamide's effects on the gastrointestinal system.
  Findings:
  - Acrylamide damages the gastric and intestinal epithelium, disrupts tight junctions, alters the microbiome, increases apoptosis, and harms the enteric nervous system.
  - Chronic intake—especially with oxidized fats—induced **precancerous changes** in the intestinal lining in animal models.
    Conclusion: Acrylamide is a hazardous compound with the potential to cause gastrointestinal inflammation, neurotoxicity, and carcinogenesis.

2. Heterocyclic Aromatic Amines (HCAs)

HCAs are primarily formed during **prolonged frying of animal-derived proteins**, such as meat and poultry. Key compounds include **PhIP, MeIQx**, and others. Their concentrations increase in reused oils used to fry meats.

- Experimental studies have shown that HCAs are **carcinogenic in animals**, inducing tumors in organs such as the **colon, breast, liver, lung, prostate**, and more. [9]

Key Studies:

- **Monti et al. (2001)** [10]**:**
  Investigated the effect of virgin olive oil (VOO) phenolic compounds on HCA formation.
  Findings:
  - Fresh olive oil, rich in antioxidants, significantly **reduced HCA formation by 30–50%** during meat/fish frying.
  - Stored oil had reduced phenolic content and was less protective.
    Conclusion: Reuse of oxidized oil increases carcinogen production due to the decline in natural antioxidants.
- **Sinha et al. (1995)** [11]**:**
  Studied the metabolism of HCAs by liver enzymes **CYP1A2 and NAT2** in humans.
  Findings:
  - Participants with high CYP1A2 activity had lower excretion of unmetabolized MelQx, suggesting conversion into **DNA-reactive carcinogens**.
    Conclusion: Metabolic activation of HCAs enhances their carcinogenicity and links genetic polymorphisms to cancer risk.

3. Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs are a group of carcinogenic organic compounds formed through **incomplete combustion** of organic materials, especially under **high-temperature frying** (180–200°C).

They result from the oxidation of unsaturated fatty acids and accumulation of polar degradation products.

- According to the **IARC (2010)** [12]:
  - **Benzo[a]pyrene (BaP)** is a **Group 1 carcinogen** (definitely carcinogenic to humans).
  - **Benzo[a]anthracene (BaA)**, **Benzo[b]fluoranthene (BbF)**, and **Chrysene (Chr)** are **Group 2B carcinogens** (possibly carcinogenic).

Key Studies:

- **Pan et al. (2008)** [13]**:**
  Studied PAH exposure in 288 Chinese restaurant workers.
  Findings:
  - Airborne PAH levels in kitchens were 5× higher than in dining areas.
  - Kitchen workers had significantly higher urinary **1-hydroxypyrene (1-OHP)** and **malondialdehyde (MDA)** levels—biomarkers for PAH exposure and oxidative damage.
    Conclusion: Inhalation of cooking oil fumes (COFs) increases systemic oxidative stress and cancer risk.
- **Li et al. (1994)** [14]**:**
  Found elevated PAH levels in fumes from refined oils used for prolonged frying in small eateries.
  Conclusion: A possible association between **COF exposure and lung adenocarcinoma** in non-smoking Chinese women.
- **Hao et al. (2016)** [15]**:**
  Analyzed 48 oil samples from Beijing restaurants.
  Findings:
  - Dishes cooked by deep frying and grilling had **Σ4PAHs levels up to 16× the EU limit** and BaP levels up to **10.6× the Chinese safety limit**.
    Conclusion: Extended frying at high temperatures leads to the accumulation of PAHs in oil and food, posing a serious dietary health risk.

4. Lipid Peroxidation Products Formed During Repeated Frying

During repeated frying, **unsaturated fatty acids** in the oil undergo **oxidation**, resulting in the formation of **toxic aldehydes** such as:

- **4-Hydroxynonenal (4-HNE)**
- **Malondialdehyde (MDA)**

These lipid peroxidation products are **highly reactive**, capable of damaging cellular membranes, altering protein function, and binding to DNA, thus contributing to **mutagenesis and carcinogenesis**.

Key Studies:

- **Chacko & Thankappan (2021)** [16]**:**

Studied the effects of repeatedly heated cooking oils (RHCOs) on rats fed a cholesterol-rich diet. Oils tested included coconut, mustard, and sunflower oils, all heated for 15 hours at 210°C.

Findings:

- Significant increase in lipid peroxidation markers (MDA) and protein oxidation
- Reduction in antioxidant defenses (SOD, catalase, thiol levels) in liver, heart, and kidney tissues
- Most pronounced toxic effects were observed with mustard and sunflower oils, which contain high levels of polyunsaturated fatty acids (PUFAs)

Conclusion: Repeated heating of oils high in PUFAs enhances the formation of toxic peroxidation products and depletes natural antioxidant defenses.

- **Grootveld (2022)** [17]**:**

Examined **lipid oxidation products (LOPs)** during high-temperature frying and their implications for chronic diseases.

Findings:

- Oils rich in PUFAs generated high levels of toxic aldehydes (e.g., **MDA, 4-HNE, acrolein**) during repeated heating
- These compounds demonstrated oxidative, mutagenic, and potentially carcinogenic properties
- Some LOPs were released into the air, suggesting that **inhalation of frying vapors** may also cause **respiratory tract damage**, inflammation, and increase the risk of lung disease and cancer in chronically exposed individuals.

- **Szabo et al. (2022)** [3]**:**

Repeated heating of vegetable oils (canola, soybean, sunflower, palm, and olive) at 180°C for 1, 5, and 10 cycles showed:

- **Significant depletion** of essential fatty acids (ALA and LA)
- **Sharp rise in trans fatty acids (TFAs)**, especially in canola oil (up to **233% increase**)
- Increased **Atherogenic Index (AI)** and **Thrombogenic Index (TI)** in sunflower and soybean oils, suggesting a shift toward cardiovascular risk factors

Conclusion: Lipid degradation during repeated frying transforms oils into sources of toxic and atherogenic fats, with implications for both **carcinogenesis and vascular disease**.

Carcinogenic Mechanisms of Compounds from Repeated Frying

The toxic compounds formed during repeated frying can contribute to cancer development via **three principal biological mechanisms**:

1. Genetic Damage (Mutagenicity)

Many of the substances formed—such as **acrylamide, HCAs, and PAHs**—are **mutagenic**, meaning they can induce genetic mutations and DNA damage.

- **Acrylamide** is metabolized into **glycidamide**, which forms **DNA adducts** that can alter the genome and disrupt tumor suppressor genes.
- HCAs and PAHs are enzymatically activated (e.g., by **CYP1A2**) in the liver into reactive metabolites that bind DNA, causing mutations and potential **oncogene activation** or **tumor suppressor inactivation**.

- **Hageman et al. (1991)[18]:**

Investigated the short-term effects (4 weeks) of consuming repeatedly heated frying oil in rats.

Findings:

- The heated oil contained **promutagenic HCAs and lipid oxidation products**, confirmed as mutagenic via the **Ames test**.
- Metabolites were detected in the urine.
- There was an increase in liver enzymes (GOT, GPT, ALP), indicating **hepatocellular toxicity**.
- Histological analysis showed **enhanced epithelial proliferation in the esophagus**.

Conclusion: Repeatedly heated oils promote systemic oxidative and genotoxic stress with early signs of carcinogenic activity.

- **Kawai & Kasai (2008)[19]:**

Identified a novel mutagenic compound—**4-Oxo-2-hexenal (4-OHE)**—formed during **omega-3 fatty acid oxidation** under heating/storage conditions.

Findings:

- 4-OHE formed DNA adducts with deoxycytidine and other bases.
- High levels were found in oxidized oils, fried food, and frying vapors.
- In mouse models, 4-OHE caused genetic damage after oral exposure.

Conclusion: 4-OHE is a potential genotoxin contributing to **gastrointestinal and systemic mutagenesis** in humans.

- **Hogervorst et al. (2010)**[20]**:**

A comparative review on dietary acrylamide exposure, combining data from animal and human studies.

Findings:

- Acrylamide was shown to induce tumors in animal models (thyroid, mammary gland, nervous system).
- In humans, higher intake of acrylamide was associated with elevated risk of **ovarian, endometrial, kidney, oral**, and some **breast cancers**—particularly in **non-smokers**.
- Mechanisms included formation of **glycidamide–DNA adducts**, **mutations**, and **micronuclei formation**.

Conclusion: Chronic dietary exposure to acrylamide—especially from fried foods—can initiate and promote cancer through **direct genotoxic effects**.

- **Perumalla Venkata (2024)**[21]**:**

Studied the genotoxicity of food fried in **sunflower oil reheated 3 or 5 times** in a mouse model.

Findings:

- Mice consuming reheated oil showed:
  - Elevated **DNA strand breaks** (Comet assay)
  - Increased **micronuclei formation**
  - Higher rates of **chromosomal abnormalities** in blood and bone marrow
- The severity of genetic damage **increased with the number of oil heating cycles**

Conclusion: Chronic ingestion of food fried in repeatedly heated oil leads to cumulative genetic damage and enhances **carcinogenic risk**.

2. Induction of Oxidative Stress

Compounds generated during repeated frying can increase the production of **free radicals** and weaken the **cellular antioxidant systems**.
Studies have shown that exposure to **acrylamide** leads to a **decrease in glutathione** (a key intracellular antioxidant) and an **increase in oxidative stress markers** such as **MDA** (a lipid peroxidation product) in **intestinal tissues of rats**.

In professional cooks occupationally exposed to **cooking oil fumes**, a rise was observed in:

- **8-hydroxy-2'-deoxyguanosine (8-OHdG)** — a urinary marker of **oxidative DNA damage**, and
- **Isoprostanes** — indicators of **lipid peroxidation**,
  linked to exposure to **PAHs** and other carcinogenic compounds released in frying vapors.

Additionally, experimental models demonstrated that exposure to substances present in cooking oil smoke leads to **oxidative DNA damage** in **lung and liver cells**, including the occurrence of **DNA strand breaks**.

Prolonged oxidative stress in cells can result in **cumulative damage** to genetic material and cellular components, and can **activate oncogenic signaling pathways**.

Key Studies:

- **Subramanyam & Perumalla (2016)[22]:**

Studied the effects of repeatedly heated cooking oil on gastrointestinal tissues in rats.

Findings:

- Oils heated three times had elevated **peroxide values (PV)**.
- Rats fed with these oils showed:
  - Increased **MDA** (lipid peroxidation marker)
  - Decreased **catalase activity**
  - Metabolic abnormalities (hyperglycemia, hypercholesterolemia)
  - Histopathological changes in the **colon and liver**, including **polyp formation**

Conclusion: Oxidative stress from reheated oils contributes to **intestinal injury** and **precancerous changes**.

- **Ke et al. (2009)[23]:**

Studied 122 Chinese men, including 86 cooks exposed to **COFs**, and 36 unexposed waiters.

Findings:

- Cooks had significantly higher urinary levels of:
  - **1-hydroxypyrene (1-OHP)** – a biomarker of PAH exposure
  - **8-OHdG** – a biomarker of oxidative DNA damage
- The highest damage was in cooks **without ventilation systems**.

Conclusion: Occupational exposure to COFs can lead to systemic oxidative stress and **DNA damage**, highlighting the **carcinogenic potential of frying vapors**.

- **Lai et al. (2013)**[24]**:**

A longitudinal study on **military kitchen workers** exposed to frying vapors.

Findings:

- Persistent oxidative damage was noted, even in facilities equipped with **exhaust hoods**.
- Chronic inhalation of COFs led to markers of **inflammation and DNA damage**.

3. Chronic Inflammation and Tumor-Promoting Environment

Repeated injury to the gastrointestinal epithelium, liver, and pancreatic tissues by toxic compounds formed during oil degradation may trigger **chronic inflammatory responses**.

- **Animal studies** have shown that **acrylamide** impairs the integrity of the intestinal barrier by disrupting **tight junctions between epithelial cells**, leading to the infiltration of inflammatory cells and histological signs of inflammation in the intestinal mucosa.
- Persistent inflammation in tissue is a known driver of carcinogenesis. **Immune cells** release **cytokines**, **growth factors**, and **reactive oxygen species** that promote:
  - Proliferation of damaged cells
  - DNA damage
  - A microenvironment conducive to cancer development

This creates a **"vicious cycle"**, in which continued exposure to toxic frying oil compounds causes inflammation and damage, which in turn promotes the formation of **preneoplastic cells**.

- **Zhang et al. (2019)**[25] **—** ***Preclinical Study***

Investigated the health effects of consuming **repeatedly heated canola oil**, used for commercial frying, on the development of **intestinal inflammation** and **colorectal cancer** in mice.

- The oil was collected from a university kitchen after multiple frying cycles and fed to mice using colitis and carcinogenesis models (DSS and AOM + DSS, respectively).

Findings:

- Mice consuming the degraded oil developed:
    - Severe colitis, crypt architecture disruption, and reduced **occludin** protein (marker of epithelial barrier damage)
    - Elevated **pro-inflammatory cytokines** (IL-1β, IL-6, MCP-1)
    - Inflammatory immune cell infiltration (CD45+, F4/80+)
- In the cancer model:
    - Increased number and size of tumors
    - Elevated expression of **pro-cancer genes** (Myc, Cyclin-D1)
    - Bacterial and **LPS translocation** into the intestinal mucosa — evidence of barrier breakdown and systemic inflammation

Additionally, polar compounds extracted from the degraded oil alone were found to aggravate inflammation, suggesting these are the primary **pathological drivers**.

Conclusion:
Consumption of reheated oils exacerbates gastrointestinal inflammation and contributes to the formation of a **tumor-promoting environment**.

- **Bastida et al. (2010)**[26] **—** ***Preclinical Study***

Assessed the impact of consuming **sunflower oil** after **40 actual frying cycles** on the antioxidant defense system in the small intestine of **Wistar rats**.

- Rats were divided by fasting status (15-hour fast vs. no fast) and received either fresh or repeatedly used oil.

Findings:

- Rats fed with reheated oil, particularly in the fasted state, showed:
    - Significant reduction in antioxidant enzyme activity and gene expression (**SOD, GPx**)
    - Increased oxidative stress markers (**TBARS**, oxidized/reduced glutathione ratio)
    - Upregulation of **TNFR** expression, indicating oxidative-inflammatory signaling in intestinal epithelial cells

Conclusion:
Consumption of repeatedly used frying oils compromises **intestinal antioxidant function**, increases oxidative stress, and **triggers local inflammation**, even in the absence of underlying disease.

- **Liu et al. (2024)**[27] **— *Preclinical Study***

Studied the **systemic effects of prolonged inhalation exposure** to **cooking-oil fumes (COFs)** from soybean oil heated to 200°C, on the **gut health** of mice.

- Mice were exposed to COFs for 3, 7, and 14 days and evaluated using metagenomics, metabolomics, and transcriptomics.

Findings:

- After 3 days: Pulmonary inflammation and elevated **pro-inflammatory cytokines** in both lung and intestinal tissues
- After 7 days: Classic **IBD-like changes**, including:
  - Disruption of the epithelial barrier
  - Altered gut microbiota composition
  - Upregulation of inflammatory genes (e.g., IL-12Rβ1)
  - Decrease in anti-inflammatory metabolites
- After 14 days: Partial **adaptive immune response** observed

Conclusion:
These findings support the **lung–gut axis hypothesis**, indicating that **prolonged environmental or occupational exposure** to oil fumes can induce **chronic inflammation** in both the respiratory and gastrointestinal systems via systemic **immunometabolic disruption**.

- **Zhu et al. (2021)**[28] **— *Comprehensive Experimental Study***

Evaluated the long-term consumption of **used frying oil** over **34 weeks** in mice.

- The oil, collected from restaurants after reuse, was filtered and reheated to simulate human dietary exposure. Comprehensive chemical analysis confirmed high levels of **peroxides**, **aldehydes**, and **FFAs** — markers of advanced oxidation and thermal degradation.

Findings:

- Mice exhibited:
  - Multisystem injury, including **intestinal DNA damage** (γ-H2AX marker)
  - **Lung inflammation**
  - **Pancreatic apoptosis** in 100% of animals
  - **Esophageal hyperplasia** in 80% of cases — a clear **preneoplastic lesion**

Conclusion:
Chronic consumption of reused oils triggers **inflammatory, genotoxic, and preneoplastic** changes across multiple organ systems.

4. Disruption of Intracellular Signaling Pathways

Certain oxidation products of fats, especially **4-Hydroxynonenal (4-HNE)**, are capable of interfering with intracellular defense and repair mechanisms.

- **4-HNE** binds to key cellular proteins and alters their function.
- It affects the **Nrf2/Keap1 antioxidant signaling pathway**, disrupting the balance between **protective responses** and **pro-carcinogenic signals**.
- Aldehydes like 4-HNE and **MDA** can impair **DNA repair mechanisms**, bind to **regulatory proteins**, and promote **survival of damaged or transformed cells**.
- Additional components in frying vapors—such as **benzene** and **formaldehyde**—are known carcinogens and may induce **epigenetic modifications** and **abnormal gene expression** over time.

- **Guéraud et al. (2017)**[29] **—** ***Scientific Review***

Explored the role of **4-Hydroxynonenal (4-HNE)** in carcinogenesis, particularly in **colorectal cancer**.

- 4-HNE is a **reactive electrophilic aldehyde** formed during the oxidation of omega-6 fatty acids, both endogenously and in processed/heated food.
- It forms **covalent DNA adducts** (e.g., propano-ethanol adducts), inducing mutations at key genomic sites such as **p53**, and disrupts DNA repair.
- 4-HNE also interacts with **Nrf2/Keap1/ARE**, **NF-κB**, and **PKC** signaling pathways, promoting cell proliferation and **resistance to oxidative stress**.

Findings in colorectal cancer models:

- 4-HNE was found in fecal water of animals fed red meat or oxidized oils.
- It was **cytotoxic to normal intestinal cells** but promoted survival of **preneoplastic cells**.

Conclusion:
4-HNE acts as a **tumor promoter** by combining **genotoxic stress**, **oxidative injury**, and **disruption of apoptosis and gene regulation**.

Conclusion of Mechanisms Section

Compounds formed in frying oils subjected to **prolonged and repeated heating** act as **potential carcinogens** through a combination of:

- **Direct genotoxicity**
- **Oxidative stress induction**
- **Suppression of cellular defense mechanisms**
- **Chronic inflammatory responses**

These are **key drivers of cancer initiation and progression**.

Thank you for your patience. Here is the **full English translation** of the **"Impact on Gastrointestinal Cancer Incidence"** section (pages 19–27 of your Hebrew article), preserving the **tone, structure, and academic fluency** of the original:

Impact on Gastrointestinal Cancer Incidence

In recent years, an increasing body of scientific literature has revealed a disturbing trend: **diets rich in fried foods—especially those prepared using reused oils—are associated with an elevated risk of gastrointestinal (GI) cancers**. Repeated heating of oil causes significant chemical changes that generate carcinogenic compounds, such as **acrylamide, heterocyclic amines (HCAs), polycyclic aromatic hydrocarbons (PAHs), and toxic aldehydes**. These substances may be absorbed by the mucosal tissues along the GI tract, cause oxidative DNA damage, and induce chronic inflammation—well-established processes that promote carcinogenesis.

The impact varies across GI organs depending on tissue type, exposure duration, and the types of food and oil involved.

A comprehensive scientific review published in *Critical Reviews in Food Science and Nutrition* (Ganesan et al.[5], 2019; IF ≈ 10.3, cited over 90 times) evaluated the link between repeatedly heated oils and cancer development, integrating findings from both experimental and epidemiological studies. The review highlighted the formation of toxic compounds—including PAHs, HCAs, electrophilic aldehydes (e.g., 4-HNE, trans-2,4-decadienal), and lipid peroxidation products—which are implicated in:

- DNA adduct formation
- Chromosomal breaks
- Impaired genetic repair mechanisms
- Activation of pro-carcinogenic signaling pathways (e.g., NF-κB, MAPKs)
- Promotion of oxidative stress and chronic inflammation

The review found a clear association between repeated consumption of heated oils and increased incidence of various cancers—especially **colorectal, liver, pancreatic, lung, and**

breast cancers—in both humans and animal models. Occupational exposure (e.g., among chefs) was also associated with higher cancer risk, independent of other risk factors. These findings reinforce the idea that heated oils represent a potential genotoxic and carcinogenic exposure, particularly within the digestive system.

Evidence by Specific GI Cancer Sites:

1. Esophagus

Several studies report a higher risk of esophageal cancer in individuals who frequently consume fried foods. A case-control study in Australia (Ibiebele et al., 2010) found that frequent consumption of commercially fried take-away foods was associated with a significantly higher risk of adenocarcinoma at the esophagogastric junction (EGJ), with an odds ratio (OR) of 1.44 (95% CI: 1.01–2.05).

Other studies suggest that **dietary acrylamide**—a compound formed during high-temperature frying of carbohydrate-rich foods—may increase the risk of esophageal squamous cell carcinoma (ESCC), particularly among **non-smokers and overweight individuals**. Proposed mechanisms include direct mucosal irritation from frying smoke or reflux-mediated delivery of carcinogens to the esophageal lumen.

- In a large case-control study in Australia (Ibiebele et al., 2010) [30], frequent **consumption of takeaway fried foods was associated with an increased risk of adenocarcinoma in the esophagogastric junction (EGJ).**

  The odds ratio **increased to ~2.05** in the older age group and with intake of more than 2–3 servings per week. The risk was specifically **related to industrial-style frying—especially repeated heating**—rather than home-fried foods, and is likely mediated by exposure to acrylamide and other carcinogens formed during deep frying.

- In another study conducted in Sweden (Lin et al., 2011) [31], a **significant association was found between dietary acrylamide intake and esophageal cancer,** particularly squamous cell carcinoma (ESCC), with a 23% increased risk (OR = 2.82) among overweight non-smokers. These findings strengthen the proposed link between dietary acrylamide and esophageal cancer development and support the call to reduce exposure to fried food as part of public health efforts against GI tract cancers.

2. Stomach

Epidemiological evidence points to a relationship between fried food consumption and stomach cancer. A 2023 meta-analysis (Zhang et al., 2023) summarizing 18 case-control

studies with over 5,700 gastric cancer patients found that high consumption of fried foods was associated with a **52% increase in gastric cancer risk** (OR = 1.52; 95% CI: 1.23–1.87). The risk was significant across both Asian and Western populations. Potential mechanisms include increased exposure to **PAHs**, nitrosamines, and oxidative compounds that damage the gastric mucosa and promote chronic gastritis.

- A recent meta-analysis from 2023 (Zhang et al., 2023) [32] reviewed 18 case-control studies involving over 5,700 gastric cancer patients and found that **high consumption of fried foods was associated with a 52% increase in gastric cancer risk** (OR = 1.52; 95% CI: 1.23–1.87). The risk was consistent across subgroups (Asian and non-Asian), and especially prominent when fried food was consumed more than three times per week. **Researchers emphasize that deep frying—particularly when oil is reused—leads to the formation of acrylamide and heterocyclic amines (HCAs), known genotoxic and pro-inflammatory carcinogens** that damage the gastric mucosa and promote chronic gastritis.

3. Duodenum and Small Intestine

These types of cancer are relatively rare, and therefore the nutritional connection has been less studied and is not well understood.
To date, there is no conclusive evidence directly linking the consumption of repeatedly heated frying oils to cancer of the **duodenum or small intestine**.
However, it is worth noting that **small intestine cancer shares biological and pathological characteristics with colorectal cancer**, and thus **dietary risk factors**—such as diets high in **animal fats and fried foods**—may also contribute to risk in these organs to some extent.

That said, the **very low incidence rate** of tumors in these organs makes epidemiological associations difficult to establish.
Large population-based studies are needed to **accurately examine such potential links**.
In general, it is advisable to assume that the **same carcinogenic compounds** that are absorbed in the upper gastrointestinal tract **may also adversely affect the mucosa of the small intestine**, even if **direct proof is still lacking**.

4. Colon and Rectum

The association between fried food and colorectal cancer has been examined in several studies, and while the findings are mixed, they point to a **potential risk**.
In some studies, **no strong association** was found with overall fried food consumption.
However, the **method of meat preparation** appears to be a key factor.

For example, in one case-control study, frequent consumption of **well-done fried meat**—rich in **heterocyclic amines (HCAs)** and **polycyclic aromatic hydrocarbons (PAHs)**—was significantly associated with an increased risk of colorectal cancer (**OR ≈ 2.6**) for very high vs. low intake. In terms of **mechanism of action**, consumption of well-done fried meat

exposes the colon to HCAs and PAHs formed during cooking, which can create **DNA adducts in colonic epithelial cells**.
In animal models, exposure to such compounds led to the development of intestinal tumors, and **similar findings were demonstrated in humans** with respect to colorectal adenomas.
Based on current knowledge, a **diet high in fried meats** may contribute to an **increased incidence of colorectal cancer**.

- In a Dutch study (Moonen et al., 2005)[34], the association was examined between **a genetic polymorphism in the enzyme CYP1A2**—involved in the metabolic activation of heterocyclic amines (HCAs) formed during high-temperature meat frying and cooking—and the risk of developing **colorectal adenomas**. It was found that carriers of the **CYP1A2*1F allele** (A/A genotype), which confers higher metabolic activity toward HCAs, were at **3.7-fold increased risk** of having pre-cancerous lesions in the colon. These findings support a **biological mechanism** whereby consumption of fried or charred meat exposes the intestinal mucosa to **carcinogenic compounds**, which are **bioactivated into toxic metabolites in the body**—potentially encouraging malignant transformation, especially in **genetically susceptible individuals**.

5. Liver

The connection between repeated oil frying and **liver cancer** is complex and manifests primarily **indirectly**, through **non-alcoholic fatty liver disease (NAFLD)** and **obesity**.
In **preclinical experiments**, there is clear evidence: in rats fed with **edible oil that had been repeatedly heated**, **pre-neoplastic liver damage** was demonstrated — including:

- Increased **micronuclei** in the liver (a marker of genotoxic damage)
- Decreased **antioxidant enzyme activity**
- Increased levels of **free radicals** and **lipid peroxidation** in liver tissue

Furthermore, **exposure to oxidized oil** promoted the appearance of **preneoplastic hepatic foci** when administered together with a known carcinogen.
These findings indicate that **repeated frying** may **initiate and promote carcinogenic processes** in the liver, especially in the presence of **additional risk factors**.

In humans, it is difficult to isolate the dietary contribution of fried foods to liver cancer, since most cases of hepatocellular carcinoma develop in the context of **cirrhosis** (resulting from viral or fatty liver disease).
Nevertheless, it is well established that **high consumption of fried and fast food** contributes to **obesity** and **NAFLD**, which is itself a **significant risk factor** for the development of **hepatocellular carcinoma (HCC)** in the long term.

Studies have shown that **young adults consuming a high-calorie, fried food–rich diet** developed **abnormal liver function tests** within just a few weeks — evidence of **subclinical liver injury**. Moreover, **current treatment guidelines for NAFLD explicitly recommend reducing fried and fast food consumption** as a strategy to prevent progression to severe liver disease. Therefore, it may be indirectly inferred that **chronic consumption of fried foods** could contribute to liver cancer development — through induction of fatty liver, inflammation, and fibrosis, creating a pro-carcinogenic environment. **Direct exposure** of hepatocytes to absorbed compounds such as **acrylamide**, **HCAs**, and others may also contribute to **mutations in liver cells** themselves.

In conclusion, oil that has been **reheated repeatedly** is **hepatotoxic** and may **promote carcinogenic processes** in the liver, as evidenced by animal studies.
As part of **primary prevention**, it is recommended to **reduce fried food intake** to support **liver health**.

- In a comprehensive **preclinical study on rats** (Srivastava et al., 2010)[35] , the **genotoxic and carcinogenic effects** of **repeatedly boiled sunflower oil (RBSO)** were examined in Wistar rats, compared to fresh oil and once-boiled oil.
  It was found that **repeated boiling** produced **elevated concentrations of PAHs**, including **benzo[a]pyrene** and **dibenzo[a,h]anthracene**, at levels up to **7 times higher** than in fresh oil.

  Dietary exposure to this oil led to:

- Significant increase in **chromosomal aberrations** and **micronuclei** in bone marrow
- Decrease in **antioxidant enzyme activity** (SOD, catalase)
- Increase in **oxidative stress markers** (ROS, lipid peroxidation)
- Appearance of **preneoplastic lesions in the liver** (altered hepatic foci), even **in the absence of an added carcinogenic stimulus**, along with marked changes in the expression of **biomarkers** such as **GST-P** and **GGT**

  These findings support the conclusion that **repeated consumption of reheated or boiled oils** may trigger **genetic damage**, **suppression of antioxidant defenses**, and **pre-neoplastic liver changes**, **even without other risk factors**.

- Similar results were reported in another study that examined **reused coconut oil** (Srivastava et al., 2010) [36], in which consistent findings were observed: increased **genetic damage**, decreased **antioxidant activity**, and **preneoplastic liver lesions** — even **without exposure to a chemical carcinogen**.

6. Gallbladder

Gallbladder cancer is **strongly associated** with the presence of **gallstones** and with **obesity**. A **diet high in fats and fried foods** may influence both of these risk factors:

- **Excess calories and fat** contribute to obesity
- **High dietary fat intake** can promote the formation of **cholesterol gallstones**

According to expert reports, there is **strong evidence** that **overweight and obesity** increase the risk of gallbladder cancer. **Obesity** promotes **gallstone formation**, and **gallstones themselves** significantly elevate the risk of **malignant transformation** in gallbladder epithelial cells — due to **chronic irritation and inflammation**. Therefore, it can be inferred that **high consumption of fried foods** — for example, in a **Western diet** rich in high-calorie, fried foods — may **indirectly increase the incidence** of gallbladder cancer by **increasing rates of gallstone formation and obesity** in the population. In addition, a **2019 report** by the **World Cancer Research Fund (WCRF)** noted that **carcinogenic compounds from dietary sources** — including, as explained above, those derived from **oxidized or repeatedly heated oils** — may undergo **concentration and excretion via bile**, thereby **exposing the gallbladder mucosa directly** to **carcinogenic compounds** and increasing cancer risk.

- In a **comprehensive report** by the **World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR, 2019)**[37], which was based on an analysis of **approximately 13 million individuals**, **obesity**, **insulin resistance**, and **chronic inflammation** were identified as **established risk factors** for gallbladder cancer.

The report found that:

- Each **5-unit increase in BMI** raised the risk by approximately **25%**
- Moreover, the report emphasized that **environmental carcinogens**, including **dietary components**, may be **concentrated and excreted through the biliary system**, thereby **exposing the gallbladder epithelium to harmful compounds**

These findings support concerns that a **high-fat diet**, particularly **chronic consumption of fried or oxidized oils**, may contribute **indirectly** to **carcinogenic processes in the gallbladder** — both via **systemic metabolic effects** and through **direct exposure of the bile** to toxic compounds.

7. Pancreas

Pancreatic cancer is considered one of the most **lethal malignant tumors**, with **very low survival rates**. While **obesity**, **diabetes**, and **smoking** are established risk factors, **growing**

scientific evidence highlights the role of **diet**—particularly **fried food consumption**—in the risk of this disease.

A major study from the **NIH-AARP cohort**, conducted among more than **530,000 participants** in the United States, found that in **men**, **high consumption of red meat and food fried at high temperatures** (such as grilling, deep-frying, or pan-frying) was associated with an **up to 50% increase in the risk of pancreatic cancer**.

The highest risk was observed with elevated intake of **carcinogenic compounds** formed during high-temperature cooking:

- **Heterocyclic amines (HCAs)**, such as **PhIP**
- **Polycyclic aromatic hydrocarbons (PAHs)**, such as **benzo[a]pyrene**

In addition, a **classic experimental study** in mice by **Rao & Haseman (1993)** showed that **chronic administration of corn oil** as part of the diet led to a **significant increase** in the incidence of **acinar cell tumors** in the pancreas. This finding emphasizes the possibility that both the **fat content** and the **type of fat** in the diet—especially when **reheated**—may contribute to **pancreatic carcinogenesis**.

Beyond the **direct effect** of carcinogenic compounds on pancreatic cells, **indirect mechanisms** should also be considered: A **diet rich in fried foods** promotes **obesity** and **insulin resistance**, both of which have been **clearly shown** to contribute to the **development of pancreatic cancer**.

- In a **large-scale cohort study** (Marsh et al., 1999)[38] including **8,508 workers** across three chemical plants in the U.S., **cancer mortality** was monitored over a period of approximately **70 years** among workers **occupationally exposed to acrylamide**. While no significant association was found between acrylamide exposure and overall cancer mortality or many cancer types, there was a **statistically significant 2.26-fold increase** in **pancreatic cancer mortality** among workers with **very high cumulative exposure** (defined as >0.3 year·mg/m³). Although the researchers expressed caution regarding statistical significance due to epidemiological limitations (such as lack of smoking data), this finding aligns with prior experimental research on the **carcinogenic potential of acrylamide** in pancreatic tissue and raises **serious concern** regarding **chronic exposure to frying fumes** as a contributing factor.

- In a **broad preclinical epidemiological analysis** (Rao & Haseman, 1993)[39] of more than **6,000 mice** from long-term carcinogenesis studies, **chronic oral administration of fresh corn oil** over a period of two years led to a **significant increase** in the incidence of **pancreatic tumors**, especially **acinar cell tumors**, among **male mice** (8-fold higher compared to the control group).

The rise in tumors was attributed to:

- Chronic consumption of **polyunsaturated fatty acids (PUFAs)**
- Associated **body weight gain**

These results suggest that **even non-oxidized plant oils** may promote **neoplastic processes** in the pancreas.

Importantly, the study used **fresh corn oil**, yet it is reasonable to assume that exposure to **reheated or thermally degraded oils**, as is common in **household or industrial use**, would further **increase the harmful potential**—as previously demonstrated.

- In a **large prospective cohort study** by **NIH researchers (Stolzenberg-Solomon et al., 2007)** [40] , which included **over half a million participants** from the **AARP Diet and Health Study**, the relationship was examined between:

- **Meat consumption**
- **Cooking methods**
- **Exposure to carcinogenic compounds**

— and the **risk of pancreatic cancer**.

Over **five years of follow-up**, **836 pancreatic cancer cases** were identified.

Among men, **high consumption of red meat**, particularly meat prepared by **grilling or barbecue** (i.e., **high-temperature cooking methods**), was associated with an **up to 50% increase** in risk. Moreover, men who consumed **high levels of mutagenic HCAs** such as **PhIP** and **DiMeIQx** showed a **significant increase** in cancer risk.

These findings strengthen the link between **cooked meat**, **mutagenic compound formation**, and **pancreatic cancer development**, via a mechanism involving **systemic absorption of carcinogens**, **inflammatory pathways**, and **immune involvement in pancreatic tissue**.

Summary

The accumulated evidence from epidemiological, preclinical, and meta-analytic studies strongly supports a link between **reused frying oil** and **increased cancer risk**—especially in organs of the **gastrointestinal tract**. Carcinogenic compounds formed during thermal degradation (e.g., acrylamide, HCAs, PAHs, aldehydes) induce **DNA mutations, oxidative stress, and chronic inflammation**—hallmarks of tumorigenesis.

This risk is both **direct** (e.g., through mucosal absorption of toxins) and **indirect** (e.g., via obesity, NAFLD, and insulin resistance), which act synergistically to promote cancer. Thus,

reused oil exposure should be considered a modifiable carcinogenic risk factor, warranting attention in both public health and food safety strategies.

In light of this accumulating evidence, there is a clear **scientific rationale** for conducting an in-depth evaluation of **methods to reduce harmful compound levels in frying oil**—whether by **dietary practices** or **advanced technological interventions** (e.g., oil filtration).

Such technologies may not only **improve food safety**, but also **significantly reduce human exposure to harmful compounds** and support the goal of **primary cancer prevention** along the gastrointestinal tract.

Review of the Beyond Oil Solution for Preventing Reheated Oil Damage

In light of the accumulating evidence regarding the potential harm caused by the consumption of reheated oils—and the known health risks associated with their use, particularly in high-volume commercial kitchens - there is growing interest in technological solutions aimed at **reducing the concentration of toxic and carcinogenic compounds in frying oil**.

A technological solution developed in recent years and found to be highly effective in reducing exposure to toxins formed during repeated oil frying is a product called **Beyond Oil** – a unique active powder additive for filtering frying oils, intended for both home use and the restaurant/food industry sectors. This powder is based on a natural formulation (a blend of plants and minerals) and is added to the oil after each frying session. The process is simple: after addition, the oil is filtered using basic means, during which the powder binds to and removes a range of toxic degradation products – including **polycyclic aromatic hydrocarbons (PAHs)**, **unsaturated aldehydes**, **acrylamides**, and other known **carcinogenic substances**.

Studies have shown that even after more than 20 days of use with oil treated with the powder, parameters such as **TPM (Total Polar Materials – a breakdown index of oil)**, **anisidine value (oxidative damage index)**, and **acrylamide levels** remained significantly lower compared to untreated oil and stayed below public health regulation thresholds in Europe and the United States.

Product Description

Beyond Oil is an innovative filtration formulation based on natural powder, designed to:

- Reduce **Free Fatty Acids (FFA)**
- Reduce **Total Polar Materials (TPM)**
- Prevent the formation of **carcinogenic substances** such as **PAHs** and **acrylamide (AA)**

→ Thus **prolonging the frying oil's lifespan** and maintaining its health-related quality.

Studies and Tests Conducted on Beyond Oil

The assessments of **Beyond Oil's efficacy** included a series of controlled experiments in certified laboratories of high regulatory standard, including **Merieux NutriSciences (Switzerland)** and **Milouda Lab (Israel)**, operating under **ISO/IEC 17025** and **OECD-GLP** standards. As part of the tests, samples of canola oil that had undergone repeated frying for **22 and 51 days**, compared to fresh oil, were treated with Beyond Oil powder according to usage instructions. The oil was then filtered through simple filter paper, and a variety of toxic and biochemical markers were tested in both the oil and the fried food.

Among others, the following were measured:

- Levels of **PAHs (carcinogenic aromatic hydrocarbons)** using **GC-MS** (Gas Chromatography–Mass Spectrometry)
- **Acrylamide levels** in food using **LC-MS/MS**
- **TPM (Total Polar Materials)** using a dielectric electronic meter
- **Anisidine and peroxide values** according to **ISO standards**
- **Organoleptic tests** (taste, color, odor) were also conducted to assess the powder's impact on food quality

Summary of Key Results

Parameter	Fresh Oil	After 3 Days Frying Without Beyond Oil	After 22 Days Frying With Beyond Oil	After 51 Days Frying With Beyond Oil
FFA (%)	0.12%	1.69%	0.17%	0.38%
TPM (%)	10%	23%	16%	16%
PAHs$_4$ (µg/kg)	5	15.69	5.2	8.63
Acrylamide (AA)	ND	Trace	ND	ND

Notes:

- **PAHs$_4$** includes: *Benzo(a)pyrene, Benz(a)anthracene, Benzo(b)fluoranthene, Chrysene*
- **ND** = Not Detected (below analytical detection threshold)

Key Findings

- **Beyond Oil maintains low FFA levels**, even after dozens of frying days.
- **Significant reduction of carcinogens** (PAHs and AA) in oil.
- **Oil treated with Beyond Oil after extended use (22–51 days) shows quality close to fresh oil**.
- **Oxidation and polymerization indicators remain stable**.

These findings reinforce the **scientific and practical validity** of the product and indicate high potential to **reduce exposure to carcinogens** among both consumers and workers in kitchen environments.

Medical Conclusions

From a medical standpoint, the importance of this technology is especially evident in light of the above findings – both regarding **occupational exposure** among kitchen workers and for the general population that consumes fried foods. Substances such as **PAHs, benzene, heterocyclic amines, and unsaturated aldehydes** formed in overheated oil have been implicated in cancer development in multiple studies.

Therefore, based on research data and laboratory results for **Beyond Oil**:

- **Regular use of Beyond Oil may reduce dietary and inhalational exposure to carcinogens in frying oil**.
- **There is potential to reduce gastrointestinal cancer risk** among food service workers and consumers of fried foods, compared to standard frying without filtration.

Given the **totality of the scientific evidence**, the **quality of testing**, and the **high degree of reliability**, regular use of Beyond Oil may serve as an **effective means to reduce both dietary and occupational exposure** to a wide range of carcinogenic substances formed during repeated frying. The product's ability to **preserve critical parameters** such as **TPM, FFA, AA, PAHs**, and oxidation levels even after many frying cycles—**without altering the taste or smell of the food**—makes it a practical **preventive tool** not only for the food industry but also for home use.

Therefore, **there is reason to consider integrating this technology as a preventive recommendation** in high-risk settings – such as schools, hospitals, industrial kitchens – and even **promoting public health policies that limit the use of unfiltered reheated oils**.

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