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Agricultural and Industrial Carcinogens as Cancer Resulting: Part II Consumption Goods

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Abstract

This paper is an integrative review and hypothesis paper based on observation and practical analytical experiments of Dr. Larysa Karaliova. The second part of a review series examines agricultural and industrial-consumption goods such as carcinogens and their contribution to cancer development. Part II focuses on consumer products, food-related exposures, and materials unsuitable for food contact that may contribute to carcinogenic risk. Particular attention is given to dietary, environmental, and industrial factors potentially involved in the development of colorectal (CRC)-, lung cancer addressing its multifactorial nature, including genetic susceptibility, metabolic status, lifestyle, and chronic exposure to exogenous carcinogens. The review analyzes agricultural, industrial, and consumer-product carcinogens, as well as endocrine-disrupting compounds and agents capable of suppressing immune function, as potential contributors to the increasing global incidence of malignant diseases. The analysis is based on peer-reviewed scientific literature, selected analytical experiments, documented real-world cases, and laboratory investigations conducted at the Norwegian Drug Control and Drug Discovery Institute. The relative importance and interaction of diet, environmental conditions, genetic predisposition, and chronic exposure to industrial and agricultural carcinogens are discussed. Comparative observations of cancer occurrence in selected animal species are also considered to support the evaluation of environmental carcinogenic risk.

Keywords

Agricultural Carcinogens, Industrial Carcinogens, Cancer Resulting Substances

1. Introduction

The essay is an integrative review with experimental observations and hypotheses

mainly based on texts from Larysa Karaliova manuscript to book "*History and development of American and European politics and industrial progress*".

The increasing global burden of malignant diseases has attracted growing attention from researchers across multiple disciplines. The present work reflects such interdisciplinary cooperation, combining perspectives from history, policy analysis, healthcare practice, and toxicology.

Despite significant advances in oncology, the etiology of malignant tumors remains incompletely understood. In some cases, established risk factors such as tobacco smoking and alcohol consumption play a dominant role; in others, occupational and environmental exposures have proven decisive. A well-known historical example is the Norwegian "Stor Norsem saken" of the early 1980s, in which asbestos-exposed workers developed lung cancer. During the legal proceedings, the primary causal factors emphasized were tobacco and alcohol consumption, while other environmental determinants received limited consideration. Due to the lack of detailed verbatim documentation from the court process, only summary records remain available, indicating that the case concluded with a settlement in 1983, this example illustrates the methodological risk of attributing carcinogenesis to a restricted set of factors while insufficiently accounting for broader environmental and occupational exposures.

Contemporary industrial development, globalized markets, and the rapid dissemination of consumer products have further complicated risk assessment. Regulatory fragmentation between countries, aggressive marketing strategies, and limitations in risk communication have facilitated widespread exposure to products whose long-term health effects are not fully characterized. In this context, economic interests, regulatory gaps, and variability in healthcare quality may jointly influence population-level exposure to potentially harmful agents.

The present paper focuses on three categories of widely used consumer products: electronic cigarettes, selected oils, and kitchen equipment that are legally available for everyday use. These products are examined with respect to their potential role in carcinogenesis and their contribution to the increasing incidence of malignant disease.

Particular attention is given to so-called smoking surrogates, especially electronic cigarettes. Although frequently promoted as safer alternatives to conventional tobacco smoking, e-cigarettes generate aerosols containing a range of toxic and potentially carcinogenic substances. Inhalation of ultrafine aerosol particles, including heavy metals and carbonyl compounds, raises concerns regarding long-term health effects, including cancer risk. From a mechanistic perspective, these exposures share certain similarities with water-pipe (hookah, kalian) tobacco smoking, which has been associated with elevated risks of respiratory disease and malignancy.

Multiple types of electronic cigarettes are currently available on the market, including devices marketed for high vapor production, high nicotine delivery, or nicotine-free use. Regardless of nicotine content, most products contain propyl-

ene glycol (PG) and vegetable glycerol (VG) in varying proportions, commonly 60/40, 50/50, or 70/30 PG/VG ratios. These constituents, when heated, contribute to the chemical composition of the inhaled aerosol and are therefore relevant to toxicological evaluation.

1.1. E-Cigarettes: Mechanism of Action and Potential Health Risks

Electronic cigarettes (e-cigarettes), also referred to as vaping devices, are electronic systems designed to heat a liquid solution (e-liquid) and generate an inhalable aerosol. These products were initially introduced and marketed as less harmful alternatives to conventional tobacco smoking. The e-liquid typically contains nicotine (in most commercially available products), propylene glycol, vegetable glycerin, and a wide range of flavoring agents.

1.1.1. Potential Health Risks

A large proportion of e-cigarette products contain nicotine, a highly addictive substance with well-documented adverse effects on the cardiovascular system. In addition to nicotine, the generated aerosol contains multiple chemical constituents that may exert toxic effects on the respiratory tract. Experimental and clinical studies have shown that inhalation of e-cigarette aerosols can induce airway inflammation and damage to lung tissue. Nicotine is not classified as a carcinogen to humans by IARC (Mishra et al., 2015). Nicotine's primary documented effect is addiction, and IARC's evaluations focus on known carcinogens present in tobacco products, but not on nicotine itself as a carcinogenic agent (Sanner & Grimsrud, 2015).

The long-term health consequences of vaping remain insufficiently characterized, as e-cigarettes represent a relatively recent technology and long-term epidemiological data are still limited. Consequently, uncertainty persists regarding chronic outcomes, including cancer risk and cardiovascular disease.

1.1.2. Carcinogenic and Toxic Substances in E-Cigarette Aerosols

Chemical analyses of e-cigarette aerosols have identified a variety of substances with known or potential toxic and carcinogenic properties. Nicotine is consistently present in aerosols generated by numerous commercial products, including but not limited to Elf Bar, Lost Mary (including Lost Mary BM5000), Geek Bar, Elux Cyberover, NJOY, Vuse (RJ Reynolds), Vaporesso (XROS, Luxe XR), SMOK (Nord, RPM), Voopoo (Vmate, Drag), Innokin, Aspire, GeekVape, and Eleaf.

Nicotine itself is not classified as carcinogenic to humans by the International Agency for Research on Cancer (IARC). IARC evaluations emphasize that nicotine's primary documented effect is addiction, while carcinogenicity in tobacco products is attributed to other constituents. In contrast, tobacco-specific N-nitrosamines (TSNAs), particularly N-nitrosonornicotine (NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), are classified by IARC as Group 1 carcinogens (carcinogenic to humans). TSNAs are considered potential carcinogens affecting multiple organs and tissues (Sanner & Grimsrud, 2015; IARC,

2007; IARC, 2012b; Ogunwale et al., 2017).

Heavy metals such as lead, nickel, chrome and cadmium have also been detected in e-cigarette aerosols. These metals may originate from heating coils and other metallic components of the device. Inhalation exposure to heavy metals is associated with pulmonary toxicity and has been implicated in oxidative stress, inflammation, and carcinogenic processes (IARC, 2018a).

Thermal degradation of propylene glycol and vegetable glycerin during aerosol generation can lead to the formation of carbonyl compounds, including formaldehyde, acetaldehyde, and acrolein. These substances are known as respiratory irritants and have demonstrated carcinogenic or potentially carcinogenic properties (Nitzkin et al., 2015). In addition, benzene and other aromatic hydrocarbons have been detected in some e-cigarette aerosols, particularly under high-temperature or high-power operating conditions (Kosmider et al., 2020; Ogunwale et al., 2017).

Benzene is unequivocally classified by IARC as a Group 1 carcinogen, with strong epidemiological and mechanistic evidence linking exposure to hematological malignancies, particularly leukemia. Aromatic hydrocarbons, including polycyclic aromatic hydrocarbons (PAHs), represent a broad class of compounds, several of which exhibit mutagenic and carcinogenic activity. Benzo[a]pyrene, one of the most extensively studied PAHs, is also classified as carcinogenic to humans (Group 1). Its metabolic activation leads to the formation of DNA-reactive intermediates capable of inducing mutagenic DNA adducts (IARC, 2018b).

Although e-cigarettes are frequently promoted as "less harmful" than conventional cigarettes, experimental studies indicate that heating elements, flavoring agents, and contaminants can generate measurable quantities of benzene and PAH-type aromatic hydrocarbons under certain conditions (IARC, 2010). These findings raise concerns regarding long-term carcinogenic risk associated with chronic vaping.

1.1.3. Public Health Considerations and Prevalence of Using E-Cigarettes

According to estimates reported by the World Health Organization, more than 100 million individuals worldwide use e-cigarettes. Approximately 86 million users are adults, while at least 15 million are adolescents aged 13 - 15 years. The high prevalence of use among young people is of particular concern, given the addictive potential of nicotine and the possibility of long-term health consequences.

E-cigarettes are not harmless products. They deliver nicotine and expose users to a mixture of toxic and potentially carcinogenic substances. While concentrations of many toxicants in e-cigarette aerosols are generally lower than those found in conventional cigarette smoke, this reduction does not necessarily imply proportionally lower biological risk.

E-cigarette aerosols are characterized by a high proportion of fine and ultrafine particles, which exhibit enhanced pulmonary penetration and systemic bioavailability. Exposure to such particles, combined with carcinogenic metals and reactive carbonyl compounds, may contribute to cumulative, multistage carcinogenesis

through mechanisms involving oxidative stress, genotoxicity, and epigenetic alterations.

A comparative overview of key hazards associated with e-cigarette use and conventional tobacco smoking is presented in Table 1.

Table 1. Comparison of e-cigarettes (vaping) and conventional tobacco cigarettes.

Aspect	E-Cigarettes (Vaping)	Conventional Tobacco Cigarettes (Smoking)
Nicotine delivery	Usually present; dose varies by device and user behaviour	Present; highly addictive
Primary process	Heating of liquid to generate aerosol (no combustion)	Combustion of tobacco (burning)
Main constituents of inhaled aerosol/smoke	Nicotine; propylene glycol; vegetable glycerine; carbonyl compounds (formaldehyde, acetaldehyde, acrolein); metals (Ni, Cr, Pb, Cd); occasional benzene and PAHs*	Nicotine; carbon monoxide; thousands of combustion products including benzene, PAHs, TSNAs, and oxidant gases**
Carcinogenic substances present	Fewer identified carcinogens, including formaldehyde, some heavy metals, and trace amounts of benzene and PAHs	Numerous established carcinogens, including multiple IARC Group 1 agents
Carbon monoxide (CO)	Minimal or absent	High
Particle characteristics	Fine and ultrafine aerosol particles	Fine and ultrafine combustion-derived particles
Cardiovascular effects	Increased cardiovascular stress associated with nicotine exposure and oxidative mechanisms	Strongly increased cardiovascular risk due to nicotine, CO, and oxidative stress
Cancer risk	Long-term risk not yet fully established; biologically plausible concern based on toxicant profile	High and well established
Second-hand exposure	Environmental exposure to aerosol containing nicotine and toxicants	Environmental tobacco smoke
Regulatory status	Regulated or restricted in many countries; bans in some jurisdictions	Regulated worldwide; mandatory health warnings

*E-cigarette aerosols have been shown to contain toxic substances, including known and suspected carcinogens (e.g., carbonyl compounds, metals, nitrosamines), depending on device type, operating conditions, and analytical methods. Estimates suggest that approximately 10 - 30 such substances have been identified across studies. **Conventional cigarette smoke contains approximately 400 chemical compounds, of which about 70 are classified as carcinogens; at least 20 are recognized human carcinogens (IARC Group 1).

1.1.4. Tobacco Smoking Versus Vaping: A Comparative Perspective

Combustible tobacco smoking and electronic cigarette (vaping) use differ fundamentally in the mechanisms of aerosol generation and chemical exposure. Tobacco smoking involves high-temperature combustion, resulting in the formation of thousands of chemical compounds, including polycyclic aromatic hydrocarbons, tobacco-specific nitrosamines, carbon monoxide, and heavy metals, many of which are established human carcinogens. In contrast, vaping generates an aerosol through heating of liquids containing nicotine, solvents (propylene glycol

and glycerol), and flavoring agents, without combustion. Although vaping aerosols generally contain fewer and lower concentrations of classical combustion-derived carcinogens, they are not chemically inert. Thermal degradation of solvents and additives can produce reactive carbonyl compounds (e.g., formaldehyde, acetaldehyde, acrolein), metals originating from device components, and ultrafine particles capable of inducing oxidative stress and inflammatory responses. Consequently, while vaping may reduce exposure to certain carcinogens compared with conventional smoking, it represents a distinct source of potentially harmful chemical exposure, and its long-term health effects, including carcinogenic risk, remain incompletely characterized.

2. Nutrients Like Fats and Oils and Their Role in Carcinogenesis

Humans require several classes of nutrients for normal physiological function, including water, carbohydrates, fats, minerals, vitamins, and phytoncides. In this section, non-thermally heated fats and oils are described. Their physical properties and fatty acid profiles are presented in Table 2. The abbreviations used are as follows: SFA—saturated fatty acids; MUFA—monounsaturated fatty acids; PUFA—polyunsaturated fatty acids.

Thermally processed fats and oils are discussed in the subsequent section.

Table 2. Physical properties and fatty acid profile of selected oils and fats.

Oil/Fat	Solidification Range (˚C)	Smoke Point (˚C)*	Protein (%)	SFA (%)	MUFA (%)	PUFA (%)
Palm oil	33 - 39	~230 (refined)	~0	48 - 52	38 - 42	8 - 10
Coconut oil	23 - 26	175 (virgin)/230 (refined)	~0	82 - 90	6 - 8	1 - 2
Avocado oil	−5 to +5	190 - 270	~0	10 - 15	70 - 75	10 - 15
Olive oil (extra virgin)	−6 to +7	180 - 190	~0	12 - 15	70 - 78	7 - 10
Soybean oil	−16	~230 (refined)	~0	14 - 16	22 - 26	55 - 60
Corn oil	−10	~230 (refined)	~0	12 - 14	25 - 30	55 - 60
**Sunflower oil (refined)	−16 to −18	~230	~0	9 - 11	18 - 25	63 - 70
Flaxseed oil	−18 to −24	~107	~0	8 - 10	15 - 20	65 - 75
Butter (cow)	28 - 32	~150	0.5 - 1.0	60 - 65	~30	3 - 5
*Ghee (clarified butter)	30 - 34	~250	~0	60 - 65	30 - 35	3 - 5
*Lard (rendered pork fat)	36 - 42	190 - 200	~0	38 - 42	45 - 50	8 - 12
Tail fat (rendered)	42 - 47	190 - 200	~0	48 - 52	40 - 45	5 - 7
Sesame oil	−6	175 - 230	~0	13 - 15	38 - 42	40 - 45
Chicken fat (schmaltz)	30 - 35	190 - 200	~0	30 - 35	45 - 50	15 - 20

*Rendered fats are animal fats obtained by slow heating to remove water and protein residues, resulting in a stable, purified fat suitable for cooking. Ghee is clarified butter produced by the same process, yielding pure milk fat without lactose or milk proteins. Smoking points depend on refinement and free fatty acid content. **Although refined sunflower oil has a relatively high smoke point, its high polyunsaturated fatty acid content renders it particularly susceptible to thermal oxidation. Prolonged heating results in the formation of reactive aldehydes such as 4-hydroxynonenal and malondialdehyde, which are mechanistically implicated in carcinogenic processes.

2.1. Oxidative Stability of Oils during Heating and Markers Relevant to Carcinogenesis

Table 3 presents the oxidative stability of selected oils during heating and cancer-relevant risk markers. The abbreviations used in Table 3 are as follows: 4-HNE-4-hydroxynonenal; MDA—malondialdehyde.

SFA, MUFA, and PUFA differ markedly in thermal stability and susceptibility to oxidation. Oxidation of PUFA leads to the formation of reactive aldehydes such as 4-hydroxynonenal (4-HNE) and malondialdehyde (MDA), which are implicated in oxidative stress and carcinogenic mechanisms.

Palm oil itself is not carcinogenic. However, high-temperature refining may lead to the formation of glycidyl esters and 3-MCPD esters (3-monochloropropane-1,2-diol esters). Glycidol, released from glycidyl esters during digestion, is classified by International Agency for Research on Cancer (IARC) as probably carcinogenic to humans (Group 2A).

3-MCPD is classified by IARC as possibly carcinogenic to humans (Group 2B) and is also known to be nephrotoxic and associated with reproductive toxicity (IARC, 2000; EFSA Panel on Contaminants in the Food Chain (CONTAM), 2017).

In addition, prolonged or repeated heating of oils during frying may generate reactive lipid-derived aldehydes. Compounds such as 4-HNE (Esterbauer et al., 1991) and MDA, formed during thermal oxidation, particularly when oils rich in PUFA are reheated or heated for extended periods, are genotoxic and mutagenic, even if not individually classified as human carcinogens. These aldehydes are capable of forming DNA adducts and have been mechanistically implicated in carcinogenic processes (Chung et al., 1996; IARC, 1995; IARC, 2012a).

Table 3. Oxidative stability during heating and cancer-relevant risk markers.

Oil/Fat Type	Oxidative Stability During Heating	Major Oxidation Products	Cancer-Relevant Concern
Flaxseed oil	Very low	Lipid hydroperoxides, aldehydes	High formation of reactive aldehydes
Soybean oil	Low	4-HNE, MDA, acrolein	DNA damage, mutagenicity
Corn oil	Low	α,β-unsaturated aldehydes	Lipid peroxidation products
**Sunflower oil (refined)	Low	4-HNE, MDA, acrolein	High aldehyde formation under prolonged heating
Sesame oil	Moderate	Aldehydes (lower levels)	Moderate oxidative stress
Olive oil (extra virgin)	Moderate–high	Limited aldehyde formation	Relatively protective phenolics
Avocado oil	High	Low aldehyde generation	Low oncogenic risk markers
Palm oil	High	Minimal PUFA oxidation	Low aldehyde formation

Continued

Coconut oil	Very high	Minimal oxidation products	Very low aldehyde generation
Butter	Low-moderate	Protein pyrolysis products	Not suitable for high heat
Ghee	High	Low aldehyde content	Thermally stable
Lard/animal fats	Moderate	Aldehydes if overheated	Moderate risk if reused

In Table 3, the abbreviation used are: 4-HNE is 4-Hydroxynonenal, MDA is Malondialdehyde. **Although refined sunflower oil has a relatively high smoke point, its high polyunsaturated fatty acid content renders it particularly susceptible to thermal oxidation. Prolonged heating results in the formation of reactive aldehydes such as 4-hydroxynonenal and malondialdehyde, which are mechanistically implicated in carcinogenic processes.

2.2. Chronic Exposure to Industrially Processed Foods and Their Potential Role in Carcinogenesis

Over the past century, profound social and economic changes have reshaped daily life patterns. Increasing demands related to education, employment, and social engagement have reduced the time available for food preparation and shared meals in private households. As a result, meals consumed outside the home, particularly in restaurants, fast-food establishments, and take-away outlets, have become increasingly common. Even foods prepared at home differ substantially from those consumed a century ago, reflecting the widespread use of industrially processed ingredients.

Modern diets rely heavily on foods produced through large-scale industrial technologies. Even traditional products such as milk, butter, and vegetable oils are now manufactured using processes designed to improve shelf life, appearance, texture, and economic efficiency. These technological modifications, while beneficial for food stability and distribution, may also introduce chemical contaminants or promote the formation of biologically active compounds with relevance to carcinogenesis.

Milk intended for commercial consumption is commonly homogenized. In non-homogenized milk, fat droplets typically range from approximately 1 to 10 μm in diameter. Homogenization reduces these droplets to approximately 0.2 - 1 μm, increasing their surface area and stabilizing them by adsorption of milk proteins and phospholipids. Although intact fat globules are not absorbed directly into the bloodstream, structural modification of dietary lipids may influence digestion kinetics, lipid oxidation susceptibility, and interactions with intestinal immune cells. Chronic low-grade inflammation and altered lipid signaling have been implicated as tumor-promoting conditions, particularly in colorectal and metabolic-associated cancers.

Dietary fats are normally emulsified in the intestine and hydrolyzed by pancreatic lipases into free fatty acids and monoglycerides, which are absorbed, re-esterified, and transported via the lymphatic system as chylomicrons. However, industrial processing and repeated thermal treatment of fats and oils-common in both

food manufacturing and domestic cooking, can generate reactive lipid oxidation products, including α,β-unsaturated aldehydes such as 4-hydroxynonenal (4-HNE) and malondialdehyde (MDA). These compounds are genotoxic, capable of forming covalent adducts with DNA and proteins, inducing mutations, and activating pro-carcinogenic signaling pathways. Persistent exposure to such aldehydes has been mechanistically linked to carcinogenesis, even in the absence of direct classification as human carcinogens.

Butter and other dairy fats may also contain additives, depending on national regulations and manufacturing practices, making product-to-product comparisons difficult. Of particular oncological relevance are contaminants formed during high-temperature processing of fats and oils. Refining of vegetable oils, especially palm and sunflower oils—may lead to the formation of glycidyl esters and 3-monochloropropane-1,2-diol (3-MCPD) esters. Glycidol, released from glycidyl esters during digestion, is classified by International Agency for Research on Cancer (IARC) as probably carcinogenic to humans (Group 2A), while 3-MCPD is classified as possibly carcinogenic (Group 2B). These compounds exhibit genotoxic and organ-toxic properties and are considered relevant to long-term cancer risk assessment.

Most commercial seed oils, including sunflower oil, are produced by solvent extraction, typically using *n*-hexane. Although refining procedures are designed to remove residual solvents, trace amounts may persist. While dietary exposure to hexane from oils is generally low and regulated, chronic occupational exposure is well established as neurotoxic. Importantly, solvent extraction and subsequent high-temperature refining also reduce natural antioxidants, increasing the susceptibility of oils to oxidative degradation during storage and cooking, thereby indirectly enhancing exposure to carcinogenesis-relevant oxidation products.

Refining of seed oils commonly involves extraction with *n*-hexane, followed by degumming, neutralization, bleaching, and deodorization. These processes effectively remove pigments (e.g., chlorophyll), volatile aroma compounds, and a substantial fraction of naturally occurring minor components, including phenolic compounds and other antioxidants. As a consequence, refined seed oils not only lose their characteristic traditional flavour and aroma but also exhibit reduced resistance to oxidative and photo-oxidative degradation.

Packaging of hexane-extracted seed oils in transparent plastic bottles further exacerbates oxidative instability by allowing light penetration and promoting photo-oxidation during storage in retail environments. After opening, additional exposure to atmospheric oxygen in household kitchens accelerates autoxidation processes. Lipid peroxidation is initiated by the formation of hydroperoxides, which subsequently decompose into secondary oxidation products, including reactive aldehydes such as 4-hydroxynonenal and malondialdehyde.

These compounds are known to induce oxidative stress, inflammation, and DNA damage, all of which are mechanistically implicated in carcinogenesis.

Taken together, chronic exposure to industrially processed foods may contribute to carcinogenesis not through a single dominant carcinogen, but via a convergence of mechanisms, including oxidative stress, chronic inflammation, genotoxic lipid oxidation products, and exposure to processing-related contaminants. These factors act cumulatively over long periods, particularly in populations with high consumption of ultra-processed foods. Industrial food processing may contribute to carcinogenesis primarily through cumulative, low-dose exposure to genotoxic lipid oxidation products and processing-related contaminants that promote oxidative stress, inflammation, and DNA damage over time.

2.3. Air, Thermal and Light-Induced Lipid Oxidation and Cancer Risk

Over time, air, thermal and light-induced lipid oxidation reactions produce secondary oxidation products.

When edible oils are exposed to air and light, particularly in transparent plastic containers, polyunsaturated fatty acids (PUFAs) rapidly undergo oxidation and photo-oxidation. This process generates reactive oxygen species, leading to the formation of conjugated dienes and trienes, detectable at UV wavelengths (232 nm, 268 - 272 nm). Over time, these reactions produce secondary thermal and lipid oxidation products, such as aldehydes (e.g., 4-HNE, MDA)—which are known to induce oxidative stress, DNA damage, and chronic inflammation. These mechanisms are implicated in carcinogenesis (Choe & Min, 2006; Esterbauer et al., 1991; Guéraud et al., 2010).

Thus, prolonged exposure of oils to elevated temperatures, air and light may increase the risk of generating food-borne carcinogens, emphasizing the importance of proper storage conditions (Table 4).

Table 4. Relative photo-oxidation rates of oils stored in transparent plastic containers (light exposure).

Oil	PUFA content	Natural antioxidants	Relative photo-oxidation rate	Typical visible changes
Flaxseed oil	Very high (ALA)	Very low	**Very fast (hours-days)**	Rapid rancidity
Sunflower oil (refined)	High (LA)	Low	**Fast (days)**	↑ K232, off-odors
Soybean oil	High	Low–moderate	**Fast**	Flavor degradation
Corn oil	High	Moderate	**Moderate-fast**	Gradual oxidation
Canola oil	Moderate	Moderate	**Moderate**	Slower peroxide rise
Olive oil (EVOO)	Low PUFA	High phenolics	**Slow**	Relatively stable
Sesame oil	Moderate	High (lignans)	**Very slow**	Strong light stability
Avocado oil	Moderate	Pigments	**Moderate**	Risk of photo-sensitized oxidation
Fish oil	Extremely high	Very low	**Extremely fast**	Rapid peroxide formation

PUFA: Polyunsaturated Fatty Acids, ALA: Alpha-Linolenic Acid, LA: Linoleic Acid, EVOO: Extra Virgin Olive Oil, K232, K270: Specific UV absorption values adopted in the standards (ISO/AOCS) for the assessment of oil oxidation.

Edible oils do not exhibit strong intrinsic UV absorption; however, progressive Air, thermal and photo-oxidation leads to the formation of conjugated dienes and trienes with characteristic absorption maxima at approximately 232 nm and 268 - 272 nm. Storage of polyunsaturated oils in transparent plastic containers under air, elevated temperature and light exposure significantly accelerates oxidative degradation. UV Absorption Characteristics of Common Liquid Edible Oils are presented in Table 5.

Table 5. UV absorption characteristics of common liquid edible oils (200 - 400 nm).

Oil (liquid)	Main UV-absorbing components	Characteristic absorption (nm)	Practical notes
Olive oil (extra virgin)	Conjugated dienes, phenolics	**232 nm**, 268 - 270 nm	Used as quality indices (K232, K270)
Sunflower oil (refined)	Conjugated dienes	**232 - 234 nm**	Rapid increase on oxidation
Soybean oil	Conjugated dienes, trienes	**232 nm**, **268 - 272 nm**	Strong UV response
Corn oil	Dienic oxidation products	**232 - 234 nm**	Moderate baseline
Canola (rapeseed) oil	Dienic products	**232 nm**	Slower increase
Sesame oil	Lignans (sesamol, sesamin)	**290 - 320 nm**	Natural UV protection
Avocado oil	Chlorophyll derivatives, carotenoids	**270 - 350 nm (weak)**	Photosensitization possible
Flaxseed oil	Trienes	**232 nm**, **268 - 270 nm**	Very sensitive
Fish oil (liquid)	PUFA oxidation products	**232 nm**, **268 - 272 nm**	Extremely unstable

232 nm → conjugated dienes, 268 - 272 nm → conjugated trienes, 290 - 330 nm → phenols, lignans and secondary products.

Refining and light exposure markedly reduce the oxidative stability of seed oils by removing natural antioxidants and promoting lipid peroxidation, leading to the formation of reactive aldehydes implicated in carcinogenesis (Frankel, 1980; (Guillén & Cabo, 2002).

It should be emphasized that the present discussion addresses mechanistic plausibility and cumulative exposure scenarios, rather than asserting direct causal relationships between individual food products and cancer development.

Taken together, air-induced oxidation, thermal oxidation, and light-induced photo-oxidation of dietary lipids represent interconnected pathways leading to the formation of reactive lipid oxidation products with shared biological consequences.

2.4. Chronic Exposure to Processed Foods Prepared Using PTFE-Coated Cookware and Their Potential Role in Carcinogenesis

Polytetrafluoroethylene (PTFE), commercially known as Teflon™, is a synthetic fluoropolymer of tetrafluoroethylene widely used as a non-stick coating for cookware. PTFE is a high-molecular-weight polymer that is solid at room temperature

and highly hydrophobic (IARC, 2016). Owing to its low coefficient of friction and chemical inertness, PTFE has been extensively applied in domestic and industrial food preparation equipment. The physicochemical properties of PTFE have been thoroughly characterized by the manufacturer (DuPont, now Chemours) and are summarized in technical documentation on Teflon™ PTFE fluoropolymer resins. PTFE is chemically resistant to most acids, bases, and solvents and demonstrates high thermal stability under recommended conditions of use. Its melting point is approximately 327˚C, while the maximum continuous-use temperature for cookware applications is approximately 260˚C. The repeating structural unit of PTFE is shown in Figure 1:

$$-(CF_2-CF_2)_n-$$

Figure 1. Chemical structure of the PTFE polymer.

Despite its relative inertness under moderate thermal conditions, PTFE may pose ecotoxicological and health concerns when subjected to excessive overheating. At temperatures exceed approximately 300˚C - 350˚C, thermal degradation of PTFE may occur, resulting in the release of low-molecular-weight fluorinated decomposition products. Several of these compounds exhibit biological reactivity, and mechanistic studies have demonstrated tumorigenic potential of certain per- and polyfluoroalkyl substances (PFAS) *in vitro*, mediated through oxidative stress, mitochondrial dysfunction, and dysregulation of apoptotic pathways. Importantly, such adverse effects are associated with thermal decomposition products rather than with intact PTFE under normal conditions of use.

PTFE continues to be widely used in non-stick cookware because of its hydrophobic nature and relatively high heat resistance. However, empty cookware or insufficient temperature control may rapidly exceed recommended thermal limits, particularly in domestic kitchen environments. From a mechanical perspective, PTFE exhibits pronounced temperature-dependent behavior. Members of the fluorocarbon polymer family (PTFE, PCTFE, FEP) retain a measurable degree of ductility even at cryogenic temperatures. Nevertheless, PTFE does not display a sharp glass-transition temperature due to its high crystallinity; instead, practical brittleness develops at subzero temperatures, as summarized in Table 6.

Table 6. Mechanical behavior of PTFE as a function of temperature.

Temperature	Mechanical state
~−35˚C	Glass-transition-like region (amorphous phase contribution)
<−40˚C	Brittle, fracture-prone behavior
≤−70˚C	Pronounced brittleness; easily cracked under impact

2.5. Cryogenic Pulverization of PTFE

PTFE does not undergo melting, dissolution, or chemical degradation at cryo-

genic temperatures. However, below approximately −40˚C, the polymer becomes mechanically brittle. At liquid-nitrogen temperature (−196˚C), PTFE loses ductility and can be efficiently fragmented by mechanical impact or shear forces. Cryogenic grinding therefore represents a purely physical size-reduction process, allowing the production of PTFE powder without cleavage of C-F bonds, thermal decomposition, or formation of low-molecular-weight fluorinated by-products.

Cryogenic size reduction was performed using a cryogenic ball mill, in which samples were cooled with liquid nitrogen before and during milling. Grinding media and milling chambers were selected to withstand cryogenic temperatures and to minimize contamination. Particle formation under these conditions reflects physical fragmentation only and should not be conflated with thermal degradation or wear processes occurring at elevated temperatures during cookware use, which may involve chemical transformation and formation of biologically active fluorinated compounds.

3. Aim of the Experiment

The present experiment was motivated by observations made by Larysa Karaliova, Head of the laboratory. Following frying or repeated high-temperature exposure of PTFE-coated cookware in household kitchens, catering facilities, cafés, and restaurants, it was observed that residual lipid droplets may remain on PTFE surfaces even after washing with commonly used detergents. The experiment does not assess toxicity or carcinogenicity of the retained residues but demonstrates their physical persistence.

Thermal exposure, particularly repeated heating of oils and fats—can transform lipid residues into compounds capable of promoting oxidative stress, inflammatory responses, and DNA damage over time, processes mechanistically linked to carcinogenesis. Accordingly, the primary aim of this study was to determine whether lipid residues persist on PTFE-coated frying pans after conventional washing procedures.

The objective was not to characterize chemical transformations of oils or fats, as such processes are well documented and vary widely depending on oil type, temperature, and frequency of use. Instead, the focus was on assessing the physical persistence of lipid residues on PTFE surfaces following washing.

Residual material was evaluated semi-quantitatively based on differences observed between initial and repeated washing cycles. Thus, the aim of the experiment was to demonstrate that standard household washing procedures may be insufficient to completely remove lipid residues from PTFE-coated cookware.

3.1. PTFE Material

PTFE supplied by Norhage Industri/TEHI AS (Bryne, Norway) was selected due to its widespread use, including applications in the food industry. A PTFE film (0.5 mm thickness, supplied in rolls) was used in this study. According to the manufacturer's specifications, the material is suitable for operation over a tem-

perature range from −190˚C to +250˚C and is compliant with EU Regulation (EC) No. 1935/2004 for food-contact materials.

Prior to cryogenic grinding, the PTFE film was mechanically cut into fragments smaller than 1 mm^2 and washed with methanol pro analysis (PA) grade (Merck, Darmstadt, Germany).

3.1.1. HPLC Instrumentation and Conditions

High-performance liquid chromatography (HPLC) analyses were performed using an Agilent 1100 Series LC system equipped with a diode-array detector. A stainless-steel column (250 mm × 4.6 mm i. d.) packed with cryogenically pulverized PTFE was used as the stationary phase. Detection was performed at 232 nm, with simultaneous UV scanning from 190 to 400 nm.

The mobile phase consisted of methanol/deionized water (60/40, v/v) at a flow rate of 1 mL/ min. Nitrogen GC grade (Norsk Hydro ASA, Oslo, Norway) was used for stationary phase as drying gas delivered through column at flow rates of approximately 60 L/h. The washing phase was deionized water added 10% w/w commercial detergent "Hånd oppvask" (supermarket KIVI, Ski, Norway) followed by deionized water. The injection 20 µL *n*-hexane (Merck). Exposure of the oil was done in HPLC column packed with Teflon at temperature 180˚C during 30 min. Analyses were conducted at ambient temperature unless otherwise specified.

3.1.2. Column Preparation with a Stable PTFE Stationary Phase

A stainless-steel HPLC column (250 mm length × 4.6 mm i.d.; Agilent ZORBAX) was mechanically opened, and the original sorbent was completely removed. The empty column housing was sequentially washed with ethanol and *n*-hexane to eliminate residual contaminants and then dried.

Cryogenically pulverized PTFE powder was introduced into the column as the stationary phase. Both column ends were secured with ceramic frits (pore size 5 µm) to retain the PTFE material. After packing, the column was flushed with ethanol followed by n-hexane to ensure uniform distribution of the PTFE phase and removal of loosely bound particles. Prior to analytical use, baseline stability was verified under mobile-phase flow conditions.

3.1.3. Preparation of the Stable PTFE Phase

PTFE was ground and pulverized using a Retsch CryoMill (Retsch GmbH, Haan, Germany; SKU RET_12020, GTIN 4064343315685, manufacturer no. 207490001) operating under liquid nitrogen (LN_2) cooling conditions. Cryogenic milling was employed to ensure purely mechanical fragmentation of PTFE without thermal degradation or chemical modification.

The pulverized PTFE material was thoroughly washed with ethanol at 40˚C to remove potential contaminants and then suspended in ethanol for column packing. After packing, the PTFE stationary phase was conditioned by flushing with the washing mobile phase for 30 min at 40˚C. Subsequently, the column was washed with deionized water, dried with the designated drying phase for 10 min

at 40˚C, and further dried under a nitrogen stream (N_2) for 30 min at 40˚C. Finally, the column was equilibrated with the analytical mobile phase prior to use.

3.1.4. Analysis of Oil Retained on the PTFE Phase

Exposure of PTFE to Oil

The PTFE-packed column was filled with olive oil purchased from a commercial supermarket (KIWI, Norway). Both ends of the column were tightly sealed to prevent leakage and external contamination. The column was placed in a laboratory oven and heated at 180˚C for 30 min, simulating typical domestic frying conditions. After thermal exposure, the column was allowed to cool to ambient temperature.

Following cooling, the bulk oil was removed from the column. The column was subsequently subjected to a standardized washing procedure consisting of flushing with the washing mobile phase for 30 min, followed by rinsing with deionized water for 30 min. To minimize oxidative effects, the column was dried under a nitrogen (N_2) stream for 30 min, thereby reducing contact with atmospheric oxygen.

After drying, the column was connected to the HPLC system. Prior to detector connection, nitrogen was displaced by pumping the mobile phase through the column. The column was equilibrated until baseline stabilization was achieved using a diode-array detector.

Analytical Cycles

After equilibration, 20 μL of *n*-hexane was injected into the column as the analytical probe. Upon completion of the first chromatographic run, the column was regenerated according to the following cycle:

1) flushing with deionized water for 5 min.

2) washing with the washing mobile phase at 40˚C for 15 min.

3) rinsing with deionized water for 5 min.

4) re-equilibration with the analytical mobile phase until baseline stabilization.

The column was then returned to ambient temperature. The same analytical and washing procedure was repeated for the second and third cycles under identical conditions.

The resulting chromatograms are presented in **Figure 2**, reproduced from the analytical journal of L. Karaliova. The primary objective of the experiment was to determine whether lipid residues persist on the PTFE phase after repeated washing cycles with a commercial detergent at 40˚C.

Quantification of residual oil was performed on a semi-quantitative basis, as precise quantification was not the objective of the study and because, under real-life conditions, a broad spectrum of edible oils is used. Semi-quantitative evaluation based on integrated peak areas indicated that the signal corresponding to the second washing cycle accounted for approximately 70% of the initial post-exposure signal, whereas the third washing cycle retained approximately 14%. These results demonstrate that lipid residues persist on the PTFE surface even after three consecutive washing cycles performed under conditions representative of house-

hold kitchen practice.

Representative HPLC chromatograms obtained from the PTFE-packed column after exposure to olive oil at 180˚C for 30 min and subsequent washing cycles. Cycles 1 following oil exposure after first washing cycles. Cycles 2 and 3 were recorded after the second and third washing cycles, respectively, performed with a commercial detergent solution at 40˚C. The results demonstrate the persistence of lipid residues on the PTFE phase despite repeated washing. Quantitative evaluation based on total 80.893% peak area indicates that peak 2 area is 30.676% representing approximately 70% or is (69.62%) of peak 1 area is 44.064%, whereas peak 3 area is 6.153% accounts for approximately 14% or is (13.96%) of peak 1.

4. Immunity and Carcinogenesis

The immune system is a complex, multilevel network comprising cellular components-such as macrophages, natural killer cells, cytotoxic T lymphocytes, and other phagocytes-as well as humoral factors including cytokines, chemokines, antibodies, and antimicrobial peptides as lysozyme. Proper immune function is essential for immune surveillance, elimination of transformed cells, and maintenance of tissue homeostasis.

Environmental exposures, including selected herbicides and pesticides, may exert immunosuppressive effects and thereby indirectly contribute to carcinogenesis. Such effects may involve suppression of immune responses, endocrine disruption, or impairment of immune surveillance mechanisms. Immune competence plays a crucial role not only in cancer prevention but also in disease progression, therapeutic response, and recovery.

Dietary factors further modulate immune function and cancer risk. Alcohol consumption is an established carcinogenic risk factor, largely due to its metabolic conversion to acetaldehyde. In addition, certain dietary components influence insulin-like growth factor signaling, which has been implicated in cancer promotion. The contribution of food additives, detergents, and stabilizers to long-term cancer risk remains insufficiently studied and warrants further investigation.

4.1. Nutrition and Carcinogenesis

Claims that established malignant tumors can be reversed through dietary interventions alone lack scientific support. While certain dietary components may modulate carcinogenic risk, inflammatory signaling, or immune competence, no food or nutritional strategy is capable of eliminating an existing malignancy. Nevertheless, avoidance of immunosuppressive exposures and consumption of foods that support immune function and metabolic balance remain important elements of cancer prevention and supportive care.

Food preparation methods play a critical role in determining the biological activity of plant-derived compounds. Cruciferous vegetables, such as broccoli, contain glucosinolate-derived metabolites, including sulforaphane, which exhibit chemopreventive properties. The release of these bioactive compounds depends

on the enzyme myrosinase, which is thermolabile and readily inactivated by boiling. Consequently, excessive thermal processing markedly reduces the bioavailability of these protective agents. As noted by (Ludikhuyze et al., 2000), "*the activity of myrosinase is influenced by intrinsic and extrinsic factors such as ascorbic acid, $MgCl_2$, pH, temperature, and pressure*".

The effects of thermal processing on vegetable-derived nutrients are nutrient-specific. While excessive heat may degrade heat-labile compounds such as vitamin C and certain folates, carotenoids frequently exhibit improved bioavailability after cooking. This effect is particularly pronounced when vegetables are consumed in combination with dietary lipids, which facilitate micellar solubilization and intestinal uptake of these lipophilic compounds.

Polyphenol-rich foods, including red onions, purple cabbage, and dark grapes, may exert beneficial physiological effects through microbial fermentation in the colon, resulting in the formation of bioactive phenolic acids. These metabolites can contribute to the maintenance of immune-related and metabolic homeostasis via interactions with the gut microbiota and host signaling pathways.

Some vegetables and fruits may be consumed raw; however, processing can modify the bioavailability of specific nutrients and alter their biological effects. Bananas are rich in potassium, which contributes to electrolyte balance and renal handling of fluids. Although urinary pH is typically neutral or slightly acidic, dietary composition can influence renal acid-base handling. When banana-based preparations are combined with acidic-tasting additives such as lemon or lime juice or apple cider vinegar, the metabolic processing of organic acids may yield bicarbonate, resulting in a net alkalinizing effect on urine. Such diet-induced modulation of urinary pH may facilitate the excretion of selected metabolites and influence renal acid-base balance. While systemic pH homeostasis is tightly regulated and remains largely unchanged by diet, modest shifts in urinary and local microenvironmental pH may contribute to patient comfort and metabolic handling during cytotoxic treatment.

4.2. Dr. Larisa Karaliova's Hypothesis on the Development of Accelerated Metastasis

Food-related additives and other exogenous compounds may influence carcinogenesis not only through direct genotoxic mechanisms but also by modifying the tumor microenvironment and processes related to metastasis (de Visser & Joyce, 2023; Nallasamy et al., 2022; Powter et al., 2021; Riaz et al., 2024).

Conceptual hypothesis

Dr. Larisa Karaliova proposed a hypothesis suggesting that certain physicochemical properties of selected food additives could indirectly facilitate metastatic dissemination under specific conditions.

It is well established that a wide range of substances can translocate from the intestinal lumen into systemic circulation via blood and lymphatic pathways. Recent reports have demonstrated the presence of microplastic particles in human

tissues, including arteries, cardiac tissue, and the brain, raising concerns regarding their potential impact on immune regulation and tissue integrity. The modern food industry employs numerous additives, such as stabilizers, emulsifiers, and surfactant-like compounds, most of which do not exhibit genotoxic, mutagenic, or carcinogenic properties at approved exposure levels.

Unlike endogenous metabolites, many exogenous compounds entering systemic circulation require hepatic detoxification or remain unmetabolized for prolonged periods. Dr. Karaliova hypothesized that compounds with surface-active properties might influence cell-cell and cell-matrix interactions. Cells at the surface of malignant tumors are often weakly attached and mechanically vulnerable. In this context, exposure to circulating substances with detergent-like physicochemical characteristics could theoretically reduce adhesion strength, increasing the likelihood of tumor cell detachment and subsequent dissemination.

Detached, viable tumor cells represent the cellular substrate for metastatic spread. Although direct epidemiological evidence linking dietary additives to metastatic incidence is currently lacking, this hypothesis highlights a plausible biophysical mechanism by which non-genotoxic agents might modulate metastatic risk. As such, it should be regarded as a conceptual framework warranting experimental investigation rather than a definitive causal model.

Although population-level statistics on metastatic cancer have been available for several decades, historical data are constrained by limited diagnostic sensitivity, likely leading to systematic underestimation of metastatic burden compared with contemporary datasets.

5. Genetics

Genetics, nutrition, environmental exposure, viral infections, intoxications (Shen et al., 2025), and timely diagnostics influence the probability of developing malignant tumors or metastatic disease, as well as survival outcomes.

Humans are mammalians, and it is of interest to look at differences in susceptibility among different mammalians to malignancy and their defenses against this illness. Large, long-lived mammals such as elephants (*Loxodonta africana*), (*Loxodonta pharaoensis*) and (*L. cyclotis*) exhibit a surprisingly low incidence of cancer relative to their body size and lifespan, a phenomenon known as Peto's paradox. Experimental studies have demonstrated that elephants possess multiple copies of the tumor suppressor gene TP53, resulting in enhanced apoptotic responses to DNA damage and improved suppression of malignant transformation (Abegglen et al., 2015).

In addition to multiple copies of TP53, elephants exhibit a constellation of cancer-protective mechanisms, including enhanced apoptotic sensitivity, stringent cell-cycle control, efficient DNA repair pathways, robust immune surveillance, reduced oxidative stress, and structural features that limit cellular invasion. These adaptations collectively contribute to a remarkably low cancer incidence despite large body size and long lifespan. Both African and Asian (*Elephas maximus*) el-

ephants exhibit enhanced cancer resistance mediated by multiple copies of the TP53 tumor suppressor gene.

Evolutionary Adaptation

African elephants possess a higher number of TP53 copies and demonstrate stronger apoptotic responses to DNA damage, suggesting a quantitatively augmented anticancer defense relative to Asian elephants, while maintaining a shared protective architecture across Proboscidea (Abegglen et al., 2015).

Genomic analyses indicate that woolly mammoths (*Mammuthus primigenius*) possessed multiple copies of the TP53 tumor suppressor gene, suggesting that enhanced cancer-protective mechanisms evolved prior to the divergence of mammoths and modern elephants. Thus, mammoths likely shared a similar baseline architecture of cancer resistance, although later population bottlenecks and genomic deterioration may have compromised overall physiological resilience.

Despite being one of the closest living relatives of elephants, the hyrax (*Procavia capensis*) does not exhibit the enhanced anticancer mechanisms observed in Proboscidea. Genomic analyses indicate that hyraxes retain a single functional TP53 gene, consistent with their small body size and limited lifespan, supporting the view that augmented cancer resistance in elephants evolved as an adaptive response to increased cellular mass rather than phylogenetic inheritance.

Phylogenetic and molecular clock analyses indicate that the evolutionary divergence between Proboscidea and Hyracoidea (*Procavia capensis*) occurred approximately 55 - 65 million years ago, implying that enhanced anticancer mechanisms observed in elephants evolved long after this split.

The enhanced anticancer defenses observed in African elephants are consistent with stronger selective pressure imposed by cumulative DNA-damaging factors. While ionizing radiation represents a potent source of genomic injury, humans and most modern species have lacked sufficient evolutionary time to develop specialized protective adaptations. Observations from the Chernobyl exclusion zone suggest limited physiological and epigenetic adjustments in wildlife populations exposed to chronic radiation; however, these changes do not constitute complete evolutionary adaptation and are accompanied by persistent biological costs.

6. Memoriam

Larysa Karaliova (November 2, 1961-January 7, 2025) graduated from the University of Oslo, Norway, with a degree from the Faculty of American and European Studies, Department of History and Policy. The present paper is based on a manuscript originally developed as part of her long-term book project. This project was encouraged in 2011 by former French President Valéry Giscard d'Estaing, who invited her to his family residence to discuss her thesis *La politique de Valéry Giscard d' Estaing en matière de condition féminine* (1974-1981) and subsequently recommended that she expand her work into a broader study of American and European history and public policy (**Figure 2**) (Brondz, 2025).

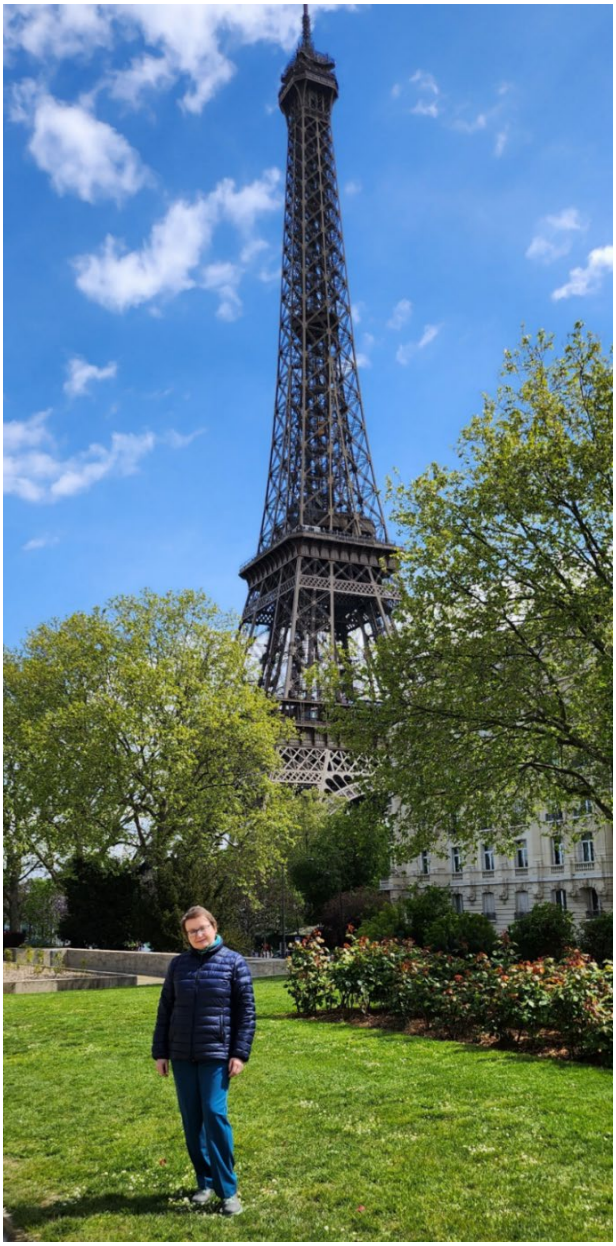

Figure 2. Larysa Karaliova April 2024, last months of the life, farewell Paris.

Larysa Karaliova published the papers (Karaliova, 2015; Brondz, Karaliova, & Ekeberg, 2006; Karaliova & Brondz, 2025). She was an Assistant Editor in Chief of the journal *Voice of the Publisher* since 2015. In addition to her academic background, Larysa Karaliova was also educated at Oslo Metropolitan University medical faculty and worked in healthcare settings involving patients with terminal illness. Her practical experience contributed to the conceptual framework of the manuscript, which seeks to examine environmental, technological, and societal factors potentially involved in carcinogenesis.

Larysa Karaliova passed away on January 7, 2025, in a palliative department of Ahus University Hospital, after a 2-year battle with aggressive cancer.

7. Results

Observations and analyses of oil-polytetrafluoroethylene (PTFE, Teflon) interactions in kitchen utensils, conducted by Dr. Larisa Karaliova, confirmed the reproducibility of the experimental findings from residuals of heated oil. It was demonstrated that following thermal interaction between cooking oils and PTFE-coated surfaces, cookware cannot be completely cleared of oil residues, even after multiple washing cycles. Thermally treated oil residues persist on the surface and remain as stable contaminants. The composition of remains was not analysed. The toxicological properties of these residues have not yet been systematically assessed and, to the best of the authors' knowledge, remain insufficiently characterized.

The effects of heat, oxygen, and light on a wide range of edible oils were examined and described (Choe & Min, 2006; Esterbauer et al., 1991; Guéraud et al., 2010). These factors promote oxidative degradation, polymerization, and the formation of secondary products such as aldehydes, ketones, and other reactive carbonyl compounds. Such transformations may contribute to biological reactivity and raise concerns regarding potential carcinogenic mechanisms. These observations are consistent with the experimental findings obtained from oil–PTFE interaction studies.

The long-term toxicity and carcinogenic potential of electronic cigarettes remain underestimated due to the relatively short period of clinical observation and the physicochemical characteristics of inhaled aerosols. A substantial fraction of e-cigarette aerosols consists of ultrafine particles (<5 μm), which can penetrate deep into the pulmonary alveoli, interact with alveolar macrophages, and enter systemic circulation. Both organic and inorganic components of these aerosols may induce inflammation, immune dysregulation, intoxication, and potentially contribute to carcinogenic processes.

Nutritional factors were also considered in relation to cancer development and metastasis, as discussed in Part I of this series (Karaliova & Brondz, 2025). Dietary exposure to thermally degraded lipids may represent an additional modifying factor in carcinogenesis. The evaluation of such effects requires consideration of genetic susceptibility and evolutionary aspects of species-specific metabolic adaptation.

8. Discussion

At first consideration, the present article may appear to address three distinct topics: thermal degradation of oils, toxic compound formation in e-cigarette aerosols, and genetic factors influencing tumor development. However, a closer analysis reveals a common mechanistic foundation.

Both heated edible oils and e-cigarette aerosols generate similar classes of reactive and potentially carcinogenic compounds, including aldehydes and lipid peroxidation products. This chemical similarity provides a rational link between these sections and supports the investigation of oil-derived residues on PTFE (Teflon) surfaces under repeated thermal exposure. The persistence of thermally

altered lipid compounds may represent a cumulative exposure pathway that warrants further investigation.

The genetic component discussed in this study is not presented as an alternative to environmental causation, but rather as a modifying factor. Environmental pollution and chemical exposure are globally distributed; however, individual susceptibility to malignant transformation may vary due to genetic background and evolutionary adaptation. The discussion of comparative cancer resistance and tumor microenvironment therefore serves to contextualize exposure-related risk within a broader biological framework.

Taken together, the findings suggest that low-dose, chronic exposure to thermally generated and industrially derived chemical agents may contribute to carcinogenic risk through cumulative and multifactorial mechanisms. Further research within this series of studies is required to refine quantitative assessment and clarify long-term implications.

Acknowledgments

The idea to publish the presented text came after the death of the main author, Karaliova Larysa, of the book "*History and development of American and European politics and industrial progress*".

The authors thank friends, schoolmates, and fellow students of Karaleva Larysa for their help and assistance. **Figure 2** is a photo kindly provided by the family from Larysa's archive. Additional thanks to the editorial team of Artificial Intellect for gathering information.

Conflicts of Interest

The authors have no conflicts of interest to declare regarding the publication of this paper.

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