IJHSR
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REVIEW ARTICLE

Beyond Flavor: A Systematic Review of the Maillard Reaction's Impact on Health and Link to Carcinogenesis

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ABSTRACT: This review explores the emerging link between the Maillard reaction (MR) and carcinogenesis, focusing on key products and their influence on gastrointestinal and breast cancers. While the MR is essential for enhancing food quality, evidence increasingly suggests that some resulting compounds may contribute to cancer risk and alter treatment outcomes. Using PubMed and Google Scholar, human-based studies were analyzed to identify how MR products — including heterocyclic amines, acrylamide, melanoidins, and carboxymethyl lysine (CML) — affect cancer development and progression. The findings reveal that certain byproducts, such as acrylamide and CML, are linked to genotoxic and inflammatory processes, whereas melanoidins demonstrate positive effects. The review also highlights mitigation strategies to mitigate exposure to harmful MR products, including enzymatic interventions and suggested cooking practices. On the whole, the evidence suggests associations with dualistic characteristics of the Maillard reaction in health and disease, rather than causal proof; additional long-term human case studies are necessary to discern causal relationships and to refine dietary guidance aligned with cancer prevention goals.
KEYWORDS: Medical and Health Sciences, Oncology, Food Science, Carcinogenesis, Maillard.

Introduction

Cancer Background:

Cancer remains one of the leading causes of mortality globally, with nearly 9.7 million deaths in 2022 alone, and about 1 in 5 people developing it in their lifetime.[1] Despite advancements in modern medicine and increased awareness, the incidence and prevalence of some of these cancers, including gastrointestinal (e.g., stomach, liver, esophagus, pancreas, and colorectal) and breast, have continued to increase over the last few decades.[2] Multiple risk factors have been identified, including *Helicobacter pylori* infection, age, environmental exposures, and processed foods. Western diets, rich in MR-processed foods, correspond to rising colorectal cancer in both Europe and North America.[3]

The Maillard Reaction:

With rapid global population growth, innovations in food processing have become crucial for meeting global nutritional demands. Additionally, food serves as a cornerstone of culture and identity, and globalization has enabled the rapid distribution of culinary dishes worldwide, allowing people to experience new cultures. A naturally occurring chemical reaction, the Maillard reaction (MR) is an innovative discovery harnessed to improve the coloration of food, taste, and aroma, and its breakthrough allows for rapid production of food with an emphasis on consumer-experience quality. The reaction is an organic chemical reaction between amino groups (protein) and reducing groups (reducing sugars) initiated by high temperatures; through a non-enzymatic process, several major chemical byproducts (the four most abundant groups being heterocyclic amines, acrylamide, melanoidins, and carboxymethyl lysine) are produced, and emerging data suggests beneficial or harmful health effects of these compounds.[4]

The Maillard Reaction and Carcinogenesis:

Numerous studies have investigated Maillard reaction byproducts in relation to cancer risk; however, the data are mixed, and the results are inconclusive. For example, a prospective cohort analysis by Li *et al.* evaluated the effects of end glycation byproducts on pancreatic cancer risk and found that men who consumed processed red meats had a dose-dependent increased risk of pancreatic cancer, where men in the highest quintile of consumption had the highest risk.[5] However, women had a nonsignificant increase in pancreatic risk in this same study. In contrast, in a 1977 study conducted by Sugimura *et al.*, it was found that heterocyclic amines (HCAs) were active in the rapid carcinogenesis bioassays, supporting biologic plausibility for pancreatic cancer. Additionally, some Maillard byproducts, such as melanoidins, are shown to be protective against cancers.[6] In this systematic literature review, we synthesize human-focused evidence linking Maillard reaction byproducts with gastrointestinal cancers and evaluate options for interventions to reduce dietary byproduct exposure.

Methods

To evaluate Maillard reaction in food processing and its relationship to cancer, we identified a selection of papers regarding the subject for analysis. We included research papers with primarily collected data using PubMed and Google Scholar. We focused our search using the terms: "Maillard reaction", "cancer" (e.g., "gastric cancer", "colon cancer", "pancreatic cancer"), "carcinogenesis", "Maillard reaction byproducts", "food processing." This yielded 210 papers. We then narrowed our search further

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DOI: 10.36838/IJHSR87.238

by filtering for papers published in English, excluding animal studies and editorials. We further refined our search to only include recent articles from between 2000 and the present, gastrointestinal or breast cancers associated with the Maillard reaction, and studies reporting estimates or mechanistic endpoints relevant to humans. This resulted in a final of 20 papers after removing duplicates.

Discussion

Maillard reaction byproducts:

As previously mentioned, many aspects of food processing have relied on the Maillard reaction for its advantages in cultivating unique complexities of foods in a rapid, precise, and consistent fashion. Despite its benefits, MR byproducts may also relate to cancer risk. Below, we summarize and critically appraise the current data on the four major byproducts.

Heterocyclic amines (HCAs):

Heterocyclic amines (HCAs) represent a heterogeneous group of potentially carcinogenic compounds, which are mainly produced from proteinaceous foods during high-temperature heating processes and flavor-forming.[7] Specifically, a heterocyclic amine is any chemical compound containing at least one heterocyclic ring (atoms of at least two different elements), and one amine group (nitrogen-containing). While there are limited studies evaluating the relationship of heterocyclic amines to specific cancers, to date, 25+ unique types of heterocyclic amines have been identified in cooked foods.[4] Reported by the International Agency for Research on Cancer, multiple[8] types of HCAs — including PhIP — are categorized as possible, class 2A and 2B, carcinogens. In a crossover design study by Shaughnessy *et al.*, the authors fed subjects diets cooked at either high or low temperatures and then evaluated the mutagenicity of those diets on DNA in their colon epithelial cells using a modified Ames test.[9] Subjects were also fed diets prepared with meat fried at high temperature alone or in combination with three putative inhibitors of HCA-induced damage (cruciferous vegetables, chlorophyllin tablets, and yogurt). Overall, beef and sausage cooked at high temperatures were found to have high mutagenicity due to higher levels of HCA, compared to extracts of meat cooked at low temperatures.[10] Importantly, supplementation with vegetables, plant extracts, and yogurts showed protective effects against the mutagenic properties of HCAs.

Across cohorts, the association with colorectal endpoints is generally minimal and is confounded by overall dietary patterns, smoking, and cooking method. Though the crossover mutagenicity data support biological plausibility, the population-level causal relation remains unsure. Cancer risk appears to be concentrated in high-temperature and low-moisture cooking and may be modifiable through the intake of crucifers or chlorophyllin.[9,10]

Acrylamide:

Next, a well-known potential carcinogen is formed as an MR intermediate when reducing sugars and glyoxalase react with asparagine. Acrylamide is mainly present in coffee, baked goods, and processed snacks such as potato chips, breakfast cereals, and canned goods.[11] As previously mentioned, acrylamide's genotoxicity and mutagenicity have been well-studied in experimental toxicology models to establish plausible mechanisms related to carcinogenesis, yet these findings are not always relevant to human populations. This review summarizes large cohort findings that have evaluated acrylamide associations with cancer.

Hogervorst *et al.* investigated the association between dietary acrylamide and several urogenital malignancies in a large prospective cohort.[12] This study included 120,852 men and women aged 55-69 years and followed them over 13 years, with primary outcomes being rates of renal, bladder, and prostate cancer after acrylamide exposure. Acrylamide intake served as the proxy for exposure and was assessed with a food-frequency questionnaire at baseline that was based on chemical analysis of all relevant Dutch foods. In the final analysis using a multivariable-adjusted Cox proportional hazard model, the highest quintile exposure of acrylamide was associated with a 1.59-fold hazard of renal cancer, which can be interpreted as a 59% increased risk of developing cancer. (95% CI: 1.09, 2.30; P for trend = 0.04). The prospective design of this study, combined with a large sample size, provides suggestive evidence of acrylamide's possible cancer risk.

While the exact mechanism of acrylamide-induced carcinogenicity remains unclear and is likely case-dependent, genotoxic and nongenotoxic pathways of carcinogenesis have been proposed. Experimental studies demonstrate DNA adduct formation and oxidative-stress responses consistent with carcinogenic mechanisms; however, translating these signals precisely to population risk is limited by possible exposure misclassification (food-frequency questionnaire intake estimates) and co-exposures (smoking, diet).[12] Acrylamide has also been shown to interact with glutathione and negatively impact oxidation balance in cells, which is a known risk factor for carcinogenesis.[13] Biomarker-based studies (e.g., hemoglobin adducts) could potentially offer clearer, dose-response resolutions moving forward.

Melanoidins:

Conversely, dietary melanoidins, a group of late-stage Maillard reaction products, may confer protection against carcinogenesis.[14-16] A study by Langner *et al.* investigated whether roasted potato extracts, known to be rich in melanoidins, can inhibit the proliferation of human colon cancer cells *in vitro*.[17] LS180 colon cancer cell lines were treated with roasted potato fiber extract (AM4) as well as with high (HMW) and low (LMW) molecular weight fractions at varying concentrations. Ultimately, the authors found that compound concentrations of 1000 μg/mL reduced colon cancer cell growth by as much as 69%. Interestingly, the authors were able to identify specific cell cycle pathways that were likely targets of the melanoidins. For example, deregulated ERK1/2 signaling was revealed upon treatment, and ERK1/2 is a well-known proliferative cell cycle pathway implicated in carcinogenesis. Additionally, multiple alterations in other cell cycle regulators, such as cyclin-depen-

dent kinases, lead to cancer cell cycle arrest in G0, which is another anti-carcinogenesis mechanism.

Another study by Kamei *et al*. found that the extract of melanoidin cultured together with gastric and colon cancer cell lines led to suppression of cancer cell line growth by 50% after four days.[15] This mechanism was similarly found to be related to the blockage of the S phase and G2 phase in cancer cells. Although mechanistic and *in vitro* findings are consistent with antioxidative and antiproliferative properties and indicate possible contribution of melanoidins to natural chemoprevention in colorectal cancer, human outcome data are scarce, thus no causation can be drawn. Additionally, typical intakes of ~10g per day and diverse food sources create difficulty in making attributions. Thus, melanoidins seem promising, but lack evidence for clinical protection in humans.

Melanoidins are also shown to have antioxidative, antimicrobial, antihypertensive, and antiallergenic properties; they have exhibited the direct ability to scavenge free radicals both *in vitro* and *in vivo*.[4]

Studies have shown that average diets consist of ~10g of melanoidins a day[18] and are found in a variety of foods, including cocoa beans, carob kibbles, and acorns.[16] These studies may support counselling at-risk cancer patients on incorporating these diets rich in melanoidins. Counselling patients to consume melanoidin-rich foods in their overall diet is reasonable, but future clinical studies are warranted to further evaluate the benefits regarding cancer prevention.

Carboxymethyl lysine (CML):

Carboxymethyl lysine (CML) is an advanced glycation end product (AGE) formed during the late stages of the Maillard reaction, whose formation is driven by glycolytic byproducts following combustion; it is also demonstrated to form from oxidative stress in cancer cells. Similar to other AGEs, CML is implicated in tumorigenesis as evidenced by *in vitro* and population studies. Biologically, CML activates the receptor for advanced glycation end products (RAGE), a single transmembrane, multiligand receptor expressed on a variety of cells, including immune cells, neurons, vascular smooth cells, and cancer cells.[19] Activation of these pathways triggers and enhances downstream signaling pathways that can contribute to breast cancer cell migration, growth, angiogenesis, metastasis, and drug resistance.[20]

Furthermore, high CML accumulation was shown to correlate with estrogen receptor expression in breast cancer cell lines;[21] estrogen-positive receptors are known to play a key role in the promotion of breast cancer, and upregulation of these estrogen receptors by CML may initiate and or promote tumorigenesis. While only a few studies have evaluated CML's association with breast cancer in humans, Peterson *et al*. demonstrated that the highest quintile of CML levels in women was significantly associated with breast cancer risk in postmenopausal women.[22] Similarly, in the Prostate, Lung, Colorectal, and Ovarian Cancer Screening Trial (PLCO), higher CML-AGE intake was associated with increased risk of breast cancer in women of all ages.[23] Furthermore, these population studies note that CML accumulation in estrogen-negative tumors is associated with poor relapse-free survival after chemotherapy. Thus, high levels of CML and AGEs may not only create a microenvironment that promotes tumor formation but may also directly impede the positive treatment effects of cancer therapy.

The direction of effect in human cohort trends is adverse, but variables in exposure measurement (dietary intake versus circulating AGEs) and introduced bias from metabolic comorbidities (e.g., insulin resistance) defer to causal claims. Larger cohorts are crucial to support a causal claim.

Moreover, CML is found to modify high mobility group box protein-1 (HMGB1), which is a cytokine linked to cancer progression due to its tumor-promoting effects.[24] With CML-modified HMBG1 (CML-HGB1) detected in 10 gastric cancer patient samples, all 10 patients exhibited distant metastasis, and patients with higher concentrations of the modified HMBG1 later showed greater resistance to neoadjuvant therapy. Overall, it is observed that CML-HMGB1 has stronger pro-tumor effects than unmodified or oxidized HMGB1, and patients with diabetes or metabolic dysfunctions of the like may exacerbate gastric cancer progression via CML-HMGB1.

Generally, major AGEs are linked to chronic inflammation, oxidative stress, and insulin resistance — all factors prompting cancer development.[4] As studied by Tamanna and Mahmood, high dietary AGEs are associated with increased risk of colorectal, breast, and pancreatic cancers due to their pro-inflammatory and oxidative properties in promoting DNA damage by impairing cellular repair mechanisms and further fostering tumor growth. Meanwhile, other studies show no direct causation between CML and cancer, but its role in metabolic disorders (diabetes and cardiovascular diseases) may elevate cancer risk. More human data and long-term studies are necessary to discern the role of CML and other AGEs in carcinogenesis.

Prevention and Applications of MRPs:

Acrylamide:

Maillard deglycases are enzymes that prevent protein glycation by degrading early Maillard adducts; specifically, members of the DJ-1/Park7 family can break down the bonds between glyoxals and amino groups.[25] Accordingly, thermophilic microorganisms (e.g., *Pyrococcus furiosus*) *and thermostable enzymes* (including β-glucosidases used in processing) function at high temperatures and can reduce acrylamide formation without depleting essential precursors such as asparagine and reducing sugars.

Another important application of the biological removal of Maillard reaction products is demonstrated in asparaginase. Asparaginase is an enzyme found in various mammals, plants, and vegetables, and can be isolated in concentrated form to be used in food processing.[26] Specifically, asparaginase conversion to aspartic acid reduces acrylamide levels by lowering levels of asparagine (a precursor of acrylamide) during the Maillard reaction.[27] This method has been well-studied and demonstrated to be effective at the biochemical level, where some studies have demonstrated over 80% reduction of acryl-

DOI: 10.36838/IJHSR87.238

amide in various products.[28] While this method is beneficial for lowering levels of toxic substances, the use of asparaginase may lower the sensory quality of food (color, flavor, texture), and these side effects make asparaginase less desirable to use in some instances. Future studies are warranted to investigate how asparaginase can be used while preserving the sensory components of food.

CML:

Since the accumulation of carboxymethyl lysine (CML) is significantly correlated with an unfavorable prognosis for estrogen-negative receptor breast cancer patients, standard chemotherapy might be a less effective treatment strategy. This suggests that identifying high CML concentrations could be crucial to preventing poor outcomes; this necessitates alternate, more aggressive therapeutic strategies for this specific patient subgroup to avoid unfavorable results associated with chemotherapy.

Otherwise, CML formation can be minimized by cooking at lower temperatures, using acidic marinades, or counteracted by consuming naturally antioxidant-rich foods (e.g., vegetables and spices).

Culinary mitigation strategies:

In order to reduce exposure to the discussed potential carcinogens, it is necessary to introduce modified cooking practices. In general, common low-risk food preparation methods include lower-temperature, higher-moisture approaches such as boiling, steaming, and microwaving — all methods of reducing HCA and acrylamide formation. Acidic marinades and avoiding charring further reduce HCA formation. Next, vacuum frying can retain antioxidants in vegetable or plant-based snacks, thus potentially buffering harmful free radicals and preventing tumor growth while keeping these foods tasty.

Overview:

As of 2022, gastrointestinal cancers and breast cancers are some of the most commonly occurring cancers, and make up 30.8%, approximately 2.99 million, of total cancer deaths.[1] These cancers are partly attributed to modern dietary patterns rich in processed foods. This review has outlined the current research and evidence on the dualistic role of MR products and byproducts in carcinogenesis.

The Maillard reaction, while key for processing and sensory appeal in our common diets, generates an array of compounds with significant and sometimes perceived detrimental biological effects. After a thorough review, associations for several MRPs, notably acrylamide and CML, have been reported in human studies, with effect sizes usually modest and context dependent. Acrylamide demonstrated indirect suggestive links with renal cancer through genomic alteration and toxicity, while CML aligns with pro-inflammatory signal pathways relevant to cancer cell progression. Contrarily, melanoidins show mechanistic chemopreventive properties; they reduce cancer cell proliferation by arresting the cell cycle and scavenging free radicals, but again, human data outcomes remain limited, and no direct linkage can be drawn.

Ultimately, we explored prospective interventions to mitigate exposure to harmful MRPs, including using enzymatic processes with asparaginase to limit acrylamide precursors and adopting cooking methods to minimize HCA and CML formation.

Common foods containing MRPs:

As previously mentioned, there are several food products that contain these Maillard Reaction products as a direct result of the chemical reaction. Studies show that meats (e.g., seared steak, barbecue), fried foods, cereals, and baked goods all contain the highest concentrations of byproducts.[4] One key example can be found in soybeans, which are used as flour, grits, concentrates, and also as cooking oil. Soybeans must be processed appropriately to ensure safe consumption, and this typically requires high cooking temperatures. Authors found that microwave heating for short times (1-2 min) generates high levels of acrylamide, whereas long-time heating (3–5 min) generates lower levels of acrylamide.[29] Thus, increasing awareness of home practices of preparing food could significantly reduce the amount of MRPs.

Socially, there is an increasing preference for instant meals rather than traditional cooking as societies have become busier and desire food "on the go." As a result, the consumption of processed meat, pizza, and snacks has led to an increase in insulin resistance and metabolic syndrome, compared to people having a high intake of vegetables and low processed food, which has dramatically increased.[30] MRPs that change during food processing might be one of the important factors for either disease progression or combating diseases. Public health initiatives should address increasing awareness of food product combinations that should be avoided or encouraged to lower overall exposure.

Strengths and limitations:

This review uses high-quality databases and includes the most current and relevant papers. Additionally, it covers one of the most critical medical concerns faced by our world today and directs the focus of future research and prevention to improve healthcare. Finally, the review raises awareness amongst the general population by offering relatable and digestible information.

This review is not fully exhaustive of resources outside of major databases, and some studies included may not be comparable due to variable differences.

Future directions:

Due to limited research, mixed results, and the mechanistic complexities of the MRPs' relationship to carcinogenesis, as highlighted in this review, it is critical to conduct further research on this topic. This may include extensive, long-term human epidemiological studies with larger populations and well-considered case-control groups, moving beyond toxicological models and *in vitro* experimentation to discern a clear association between specific MRPs and cancer risk in more diverse human populations. Furthermore, research should focus on optimizing interventions in daily life (e.g., culinary meth-

ods — baking temperatures, marinades, cooking time, etc.) to reduce the intake of potentially carcinogenic compounds in foods without compromising positive qualities, thus keeping it viable for industrial adoption. Of course, it may also prove significant to investigate how unique factors — like lifestyle, genetics, and medical history — in individuals might modulate cancer risk correlated with different MRPs; using the resulting data, personalized dietary recommendations can be implicated in cancer prevention as well.

Conclusion

This review paper highlights the association of the widespread Maillard reaction with cancer. For some MRPs discussed (HCA, acrylamide, CML), human evidence supports context-dependent associations with site-specific cancer risks and progression, as well as diabetes and cardiovascular diseases, whereas melanoidins exhibit antibiotic and antioxidative promise but lack robust data to support strong associations. Pragmatic risk reduction is feasible through simple cooking techniques and targeting processing interventions. Future studies should continue to evaluate associations between food groups containing high concentrations of Maillard reaction byproducts and help identify which food groups should be avoided to lower the risk of cancer. Priorities include increasing biomarker cohorts, controlled exposure metrics, and trials with culinary interventions using validated endpoints.

Acknowledgments

I would like to recognize my mother as an inspiration to investigate this topic, and express gratitude for Indigo Research and Dr. Frank Glover's mentorship and editorial guidance that ultimately encouraged my completion of this paper. I attest that the ideas, graphics, and writing in this paper are entirely my own.

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DOI: 10.36838/IJHSR87.238

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