ISSN 2218-4333 (online)

World Journal of *Clinical Oncology*

World J Clin Oncol 2024 September 24; 15(9): 1117-1255

Published by Baishideng Publishing Group Inc

World Journal of Clinical Oncology

Contents

Monthly Volume 15 Number 9 September 24, 2024

World Journal of Clinical Oncology

Monthly Volume 15 Number 9 September 24, 2024

Contents

CASE REPORT

LETTER TO THE EDITOR

Contents

NAME OF JOURNAL
World Journal of Clinical Oncology

ISSN
ISSN 2218-4333 (online)

LAUNCH DATE
November 10, 2010

FREQUENCY
Monthly

EDITORS-IN-CHIEF
Hiten RH Patel, Jian-Hua Mao, Ken H Young, Stephen Safe

EDITORIAL BOARD MEMBERS
https://www.wjgnet.com/2218-4333/editorialboard.htm

PUBLICATION DATE
September 24, 2024

INSTRUCTIONS TO AUTHORS
https://www.wjgnet.com/bpg/gerinfo/204

GUIDELINES FOR ETHICS DOCUMENTS
https://www.wjgnet.com/bpg/GerInfo/287

GUIDELINES FOR NON-NATIVE SPEAKERS OF ENGLISH
https://www.wjgnet.com/bpg/gerinfo/240

PUBLICATION ETHICS
https://www.wjgnet.com/bpg/GerInfo/288

PUBLICATION MISCONDUCT
https://www.wjgnet.com/bpg/gerinfo/208

ARTICLE PROCESSING CHARGE
https://www.wjgnet.com/bpg/gerinfo/242

STEPS FOR SUBMITTING MANUSCRIPTS
https://www.wjgnet.com/bpg/GerInfo/239

ONLINE SUBMISSION
https://www.f6publishing.com

World Journal of Clinical Oncology

Submit a Manuscript: https://www.f6publishing.com

World J Clin Oncol 2024 September 24; 15(9): 1117-1121

DOI: 10.5306/wjco.v15.i9.1117

ISSN 2218-4333 (online)

EDITORIAL

Advanced glycation end products in gastric cancer: A promising future

Meng-Hui Wang, Hui Fang, Chuan Xie

Specialty type: Oncology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade C, Grade D

Novelty: Grade B, Grade D

Creativity or Innovation: Grade B, Grade C

Scientific Significance: Grade B, Grade C

P-Reviewer: Otsuka Y

Received: March 13, 2024

Revised: July 19, 2024

Accepted: August 2, 2024

Published online: September 24, 2024

Processing time: 168 Days and 23.2 Hours

Meng-Hui Wang, Hui Fang, Chuan Xie, Department of Gastroenterology, The First Affiliated Hospital of Nanchang University, The Jiangxi Medical College, The Nanchang University, Nanchang 330006, Jiangxi Province, China

Co-first authors: Meng-Hui Wang and Hui Fang.

Corresponding author: Chuan Xie, MD, Chief Doctor, Teacher, Department of Gastroenterology, The First Affiliated Hospital of Nanchang University, The Jiangxi Medical College, The Nanchang University, No.17 Yongwaizheng Street, Nanchang 330006, Jiangxi Province, China. xcsghhz@ncu.edu.cn

Abstract

In this editorial, we delve into the article and offer valuable insights into a crucial aspect of gastric cancer aetiology. Gastric cancer is a malignancy emanating from the epithelial lining of the gastric mucosa and one of the most prevalent forms of cancer worldwide. The development of gastric cancer is associated with multiple risk factors, including *Helicobacter pylori* infection, advanced age, a diet rich in salt, and suboptimal eating patterns. Despite notable reductions in morbidity and mortality rates, gastric cancer remains a formidable public health concern, impacting patients' lives. Advanced glycation end products (AGEs) are complex compounds arising from nonenzymatic reactions within living organisms, the accumulation of which is implicated in cellular and tissue damage; thus, the levels are AGEs are correlated with the risk of diverse diseases. The investigation of AGEs is of paramount importance for the treatment of gastric cancer and can provide pivotal insights into disease pathogenesis and preventive and therapeutic strategies. The reduction of AGEs levels and suppression of their accumulation are promising avenues for mitigating the risk of gastric cancer. This approach underscores the need for further research aimed at identifying innovative interventions that can effectively lower the incidence and mortality rates of this malignancy.

Key Words: Advanced glycation end products; Gastric cancer; Receptor of advanced glycation end products; Prognosis; Therapeutic approaches

Core Tip: Gastric cancer is a widespread malignancy that is linked to multiple risk factors. Advanced glycation end products (AGEs) are closely correlated with gastric cancer pathogenesis. Decreasing AGEs levels and minimizing AGEs accumulation may slow gastric cancer progression. Future research endeavours should aim to discover novel interventions to reduce the burden of gastric cancer and improve human health.

Citation: Wang MH, Fang H, Xie C. Advanced glycation end products in gastric cancer: A promising future. *World J Clin Oncol* 2024; 15(9): 1117-1121
URL: https://www.wjgnet.com/2218-4333/full/v15/i9/1117.htm
DOI: https://dx.doi.org/10.5306/wjco.v15.i9.1117

INTRODUCTION

Gastric cancer is a prevalent and lethal malignancy of the digestive system that imposes a significant burden on patients and their families[1]. Gastric cancer ranks as the fourth most prevalent malignancy and the fifth leading cause of cancer-related mortality globally. Despite recent research indicating a favourable decline in its incidence and mortality rates, gastric cancer continues to pose a formidable public health challenge that necessitates unwavering attention[2]. Risk factors for gastric cancer include *Helicobacter pylori* (*H. pylori*) infection, age, high salt intake and dietary habits[3]. Among these factors, *H. pylori* infection is the main factor in gastric cancer development[4]. Notably, a randomized study conducted in a high-risk East Asian area demonstrated that targeted intervention against *H. pylori* significantly decreased the risk of gastric cancer[5]. Upon infecting the human host, *H. pylori* produces noxious substances and metabolites that elicit chronic inflammation within the gastric mucosa, a pivotal event in the multistep progression towards gastric cancer[6].

Furthermore, dietary practices, such as the excessive consumption of high-temperature foods, particularly those prepared through frying, barbecuing, or prolonged meat cooking, as well as nitrite-laden foods, are closely linked to the incidence of gastric cancer[7-9]. Notably, barbecued meat generates polycyclic aromatic hydrocarbons, such as acrylamide and heterocyclic amines, which possess mutagenic and carcinogenic properties in animals, thereby promoting the initiation and progression of gastric cancer[10]. Moreover, the enigmatic pathogenesis of gastric cancer, coupled with the fact that the majority of cases are diagnosed at an advanced stage, significantly compromises patient survival and quality of life[11].

Advanced glycation end products (AGEs) are complex chemical entities that are generated in living organisms *via* nonenzymatic reactions between free amino groups of macromolecules (proteins, amino acids, lipids, and nucleic acids) and the aldehyde moieties of reducing sugars[12]. This intricate cascade involves condensation, structural rearrangement, cleavage, and oxidative modifications, ultimately yielding a repertoire of stable end products termed AGEs[13]. The genesis of AGEs is a protracted and cumulative process that intensifies with advancing age and is further expedited in diabetic individuals experiencing hyperglycaemic conditions due to elevated blood glucose levels[14]. The accumulation of AGEs within biological systems has deleterious effects on cellular and tissue integrity, ultimately decreasing organ function and predisposing individuals to chronic diseases[15]. Consequently, AGEs have garnered significant interest within the realms of biology and medicine owing to their intricate associations with a broad spectrum of chronic illnesses, malig-nancies, and their accompanying complications. Notably, previous investigations have suggested that the overexpression of the receptor for AGEs (RAGE) in ovarian cancer could serve as a valuable biomarker for monitoring tumour progression and prognosis, underscoring the clinical significance of AGEs in disease pathogenesis[16]. Senavirathna *et al* [17] reported that abnormal accumulation of AGEs damages the cell proteome and promotes an AGEs-RAGE-driven proinflammatory signaling cascade.

The investigation of AGEs holds paramount importance in elucidating the underlying mechanisms of gastric cancer pathogenesis and fostering the development of innovative therapeutic strategies. By implementing targeted interventions aimed at mitigating the formation and accumulation of AGEs, we can aspire to prevent or delay the onset and progression of gastric cancer. As research on AGEs progresses, we anticipate the discovery of more potent interventions that will significantly reduce the incidence and mortality rates associated with gastric cancer, thereby safeguarding human health and well-being.

A REFLECTION ON AND FUTURE PERSPECTIVES FOR AGES RESEARCH IN GASTRIC CANCER

AGEs, which are produced by nonenzymatic glycosylation reactions, are ubiquitous in both human metabolic processes and food processing[18]. Although the origins of foodborne and nonfoodborne AGEs differ, they elicit equivalent detrimental effects. For example, methylglyoxal-H1 (MG-H1) is derived from in vitro foodborne pathogens, and compound methionine leucine (CML) impacts metabolic processes within the body[19]. CML and compound methionine leucine are predominantly found within red blood cells, and their levels remain unchanged regardless of dietary intake, suggesting endogenous production within the body[20]. Conversely, MG-H1 is derived primarily from dietary sources [21]. The accumulation of AGEs is associated with diabetic gastrointestinal motility disorders. Yu *et al*[22] found that advanced glycosylation end products interfere with the expression of markers of gastric smooth muscle contraction *via* the AGEs/RAGE/NF-kB pathway. Therefore, whether AGEs are ingested through food or accumulate in the gast-

rointestinal tract due to dietary habits, they may contribute to the risk of gastric cancer. The seminal work by Brownlee [23] indicated that biochemical alterations in receptors regulate AGEs levels. The intracellular generation of AGEs precursors initiates inflammatory infiltration by modifying intracellular proteins, the extracellular matrix, and plasma proteins, altering their structures and receptor interactions. Furthermore, the accumulation of the extracellular matrix disrupts matrix-matrix and matrix-cell interactions, impeding intercellular communication and fostering cancer development. The increase in AGEs promotes the activation of the RAGE axis, subsequently triggering a myriad of signaling cascades, including the JAK/STAT and AKT/mTOR pathways[24]. The activation of the RAGE axis promotes the development of key processes in oncology, such as genomic instability and aberrant interference with inflammatory processes[25]. Given the structural and functional attributes of AGEs and RAGE, the pursuit of small-molecule inhibitors or eliminators targeting these entities has emerged as a promising research direction. By inhibiting or eradicating their activities, we can potentially unravel novel and efficacious avenues for the prevention and treatment of chronic diseases and cancer.

AGEs, which are hallmarks of chronic hyperglycaemia in individuals with diabetes, are implicated in enhanced biofilm formation[26]. The generation and accumulation of AGEs are to some extent linked to the invasion, spread, metastasis and prognosis of gastric cancer cells. AGEs can induce processes such as oxidative stress, the inflammatory response, and apoptosis, thereby promoting gastric cancer development and progression[27,28]. High-mobility group box 1 (HMGB1), a RAGE-cognate ligand, is upregulated in gastric cancer cells, promoting their proliferation and growth. Notably, *H. pylori* infection potentiates HMGB1 expression and AGEs accumulation, stimulating gastric epithelial inflammation and ultimately carcinogenesis[29,30]. Intriguingly, HMGB1, *via* RAGE-mediated mechanisms, facilitates the release of autophagic gastric cancer cells, supporting their survival and decreasing their chemosensitivity[12].

AGEs have emerged as pivotal players in gastric cancer progression, engaging receptors such as RAGE and toll like receptor to activate diverse signaling pathways that stimulate gastric cancer cell proliferation, migration, and invasion [31]. This RAGE-dependent intracellular signaling orchestrates the activation of activator protein-1, cAMP-responsive element binding protein, and other transcription factors, ultimately transcriptionally activating cancer-promoting genes [16,32]. In addition to these direct effects, AGEs profoundly modulate the metabolic processes and energy supply mechanisms of gastric cancer cells, further exacerbating their neoplastic progression[12]. Indeed, AGEs intricately disrupt the homeostatic metabolic pathways of gastric cancer cells through multifaceted interactions with cellular molecules, culminating in a perturbation of energy production and allocation dynamics[33,34]. This metabolic imbalance not only undermines the viability of gastric cancer cells but also promotes their malignant transformation and proliferation, resulting in a more aggressive disease phenotype.

In the future, exploring the intricate relationship between AGEs and gastric cancer will necessitate a multifaceted approach. First, a meticulous investigation into the precise mechanisms underlying the involvement of AGEs in gastric carcinogenesis and progression is imperative to elucidate their role in modulating the biological behaviour of gastric cancer cells. Second, unravelling the intricate interplay between AGEs and other gastric cancer-associated factors will enhance our understanding of the intricate processes driving gastric cancer initiation and progression. Last, the development of targeted interventions aimed at modulating AGEs, including strategies to reduce their formation and inhibit their receptor binding, holds immense potential for offering novel insights and therapeutic avenues for gastric cancer prevention and treatment. Collectively, the presented findings indicate that the intricate relationship between AGEs and gastric cancer is a compelling research frontier and that further research on this topic could provide profound insights into the mechanistic underpinnings of AGEs in gastric carcinogenesis and progression to pave the way for innovative strategies to combat this disease.

CONCLUSION

Previous investigations have focused predominantly on traditional risk factors such as *H. pylori* infection and dietary habits. In contrast, our study innovatively elucidates the pivotal role of AGEs, which are emerging biomarkers, in the initiation and progression of gastric cancer. These findings not only enrich our understanding of the underlying mechanisms of gastric cancer but also highlight a novel strategy for its prevention and management.

First, our work systematically consolidates the intricate relationship between AGEs and gastric cancer through meticulous analysis, fostering a more coherent and exhaustive research landscape in this domain. Second, by integrating the latest scientific advancements, we delve into the contribution of AGEs receptors to gastric cancer pathogenesis, offering a fresh perspective for future investigations into the AGE–gastric cancer nexus.

Moreover, we introduce a novel preventative strategy aimed at mitigating the formation and accumulation of AGEs, which represents a theoretically ground-breaking approach while also presenting tangible prospects for gastric cancer prevention and treatment. Our findings underscore the pivotal role of AGEs in gastric cancer aetiology, thereby establishing a solid theoretical foundation for its prevention and management strategies. Moreover, these findings highlight the feasibility and promise of developing AGE-targeted interventions to reduce the incidence and mortality rates of gastric cancer. Notably, the pivotal involvement of both AGEs and their receptor RAGE in gastric cancer progression-related processes, including cell growth, invasion, and metastasis, underscores their importance. Furthermore, RAGE has emerged as a potential prognostic biomarker in gastric cancer patients, emphasizing the need for further exploration, particularly in East Asia, where the incidence of gastric cancer is high. To fully elucidate the specific roles of AGEs and RAGE in gastric cancer, additional research endeavours and investments are warranted.

Future endeavours should emphasize conducting comprehensive clinical trials to advance the therapeutic application of AGEs and RAGE in gastric cancer, promising breakthroughs in oncological treatment. Such studies will propel gastric

cancer research and enhance human health by providing novel therapeutic avenues. By elucidating the roles of AGEs and RAGE, we aim to improve patient outcomes and alleviate the disease burden.

FOOTNOTES

Author contributions: Wang MH and Fang H wrote the manuscript; Xie C revised the manuscript; all authors have read and approved the final manuscript.

Supported by The National Natural Science Foundation of China, No. 82100599 and No. 81960112; The Jiangxi Provincial Department of Science and Technology, No. 20212ACB216003; The Science and Technology Plan of Jiangxi Provincial Administration of Traditional Chinese Medicine, No.2023Z021; and The Young Talents Project of Jiangxi Provincial Academic and Technical Leaders Training Program for Major Disciplines, No. 20204BCJ23022.

Conflict-of-interest statement: The authors declare no conflict of interest.

Country of origin: China

ORCID number: Chuan Xie 0000-0001-5748-3443.

S-Editor: Luo ML
L-Editor: A
P-Editor: Zhao YQ

REFERENCES

1 **Smyth EC**, Nilsson M, Grabsch HI, van Grieken NC, Lordick F. Gastric cancer. *Lancet* 2020; **396**: 635-648 [PMID: 32861308 DOI: 10.1016/S0140-6736(20)31288-5]
2 **Yoon JH**, Choi BJ, Nam SW, Park WS. Gastric cancer exosomes contribute to the field cancerization of gastric epithelial cells surrounding gastric cancer. *Gastric Cancer* 2022; **25**: 490-502 [PMID: 34993738 DOI: 10.1007/s10120-021-01269-3]
3 **Thrift AP**, El-Serag HB. Burden of Gastric Cancer. *Clin Gastroenterol Hepatol* 2020; **18**: 534-542 [PMID: 31362118 DOI: 10.1016/j.cgh.2019.07.045]
4 **Waldum H**, Fossmark R. Gastritis, Gastric Polyps and Gastric Cancer. *Int J Mol Sci* 2021; **22**: 6548 [PMID: 34207192 DOI: 10.3390/ijms22126548]
5 **Moss SF**. The Clinical Evidence Linking Helicobacter pylori to Gastric Cancer. *Cell Mol Gastroenterol Hepatol* 2017; **3**: 183-191 [PMID: 28275685 DOI: 10.1016/j.jcmgh.2016.12.001]
6 **Stewart OA**, Wu F, Chen Y. The role of gastric microbiota in gastric cancer. *Gut Microbes* 2020; **11**: 1220-1230 [PMID: 32449430 DOI: 10.1080/19490976.2020.1762520]
7 **Weisburger JH**, Raineri R. Dietary factors and the etiology of gastric cancer. *Cancer Res* 1975; **35**: 3469-3474 [PMID: 1192413]
8 **Ward MH**, Sinha R, Heineman EF, Rothman N, Markin R, Weisenburger DD, Correa P, Zahm SH. Risk of adenocarcinoma of the stomach and esophagus with meat cooking method and doneness preference. *Int J Cancer* 1997; **71**: 14-19 [PMID: 9096659 DOI: 10.1002/(sici)1097-0215(19970328)71:1<14::aid-ijc4>3.0.co;2-6]
9 **Friborg JT**, Melbye M. Cancer patterns in Inuit populations. *Lancet Oncol* 2008; **9**: 892-900 [PMID: 18760245 DOI: 10.1016/S1470-2045(08)70231-6]
10 **Pelucchi C**, Bosetti C, Galeone C, La Vecchia C. Dietary acrylamide and cancer risk: an updated meta-analysis. *Int J Cancer* 2015; **136**: 2912-2922 [PMID: 25403648 DOI: 10.1002/ijc.29339]
11 Study Offers Insights into Gastric Cancer. *Cancer Discov* 2021; **11**: 525-526 [PMID: 33526445 DOI: 10.1158/2159-8290.CD-NB2021-0305]
12 **Takase F**, Mifune Y, Inui A, Ueda Y, Kataoka T, Kokubu T, Kuroda R. Association of advanced glycation end products in Dupuytren disease. *J Orthop Surg Res* 2018; **13**: 143 [PMID: 29880057 DOI: 10.1186/s13018-018-0848-4]
13 **Sugiyama K**, Miura J, Shimizu M, Takashima A, Matsuda Y, Kayashima H, Okamoto M, Nagashima T, Araki T. Effects of advanced glycation end products on dental pulp calcification. *Oral Dis* 2022; **28**: 745-755 [PMID: 33539626 DOI: 10.1111/odi.13792]
14 **Scavello F**, Tedesco CC, Castiglione S, Maciag A, Sangalli E, Veglia F, Spinetti G, Puca AA, Raucci A. Modulation of soluble receptor for advanced glycation end products isoforms and advanced glycation end products in long-living individuals. *Biomark Med* 2021; **15**: 785-796 [PMID: 34236256 DOI: 10.2217/bmm-2020-0856]
15 **Sahi AK**, Verma P, Varshney N, Gundu S, Mahto SK. Revisiting Methodologies for In Vitro Preparations of Advanced Glycation End Products. *Appl Biochem Biotechnol* 2022; **194**: 2831-2855 [PMID: 35257316 DOI: 10.1007/s12010-022-03860-5]
16 **Rojas A**, Lindner C, Schneider I, González I, Morales MA. Contributions of the receptor for advanced glycation end products axis activation in gastric cancer. *World J Gastroenterol* 2023; **29**: 997-1010 [PMID: 36844144 DOI: 10.3748/wjg.v29.i6.997]
17 **Senavirathna L**, Pan S, Chen R. Protein Advanced Glycation End Products and Their Implications in Pancreatic Cancer. *Cancer Prev Res (Phila)* 2023; **16**: 601-610 [PMID: 37578815 DOI: 10.1158/1940-6207.CAPR-23-0162]

18 **Li X**, Bakker W, Sang Y, Rietjens IMCM. Absorption and intracellular accumulation of food-borne dicarbonyl precursors of advanced glycation end-product in a Caco-2 human cell transwell model. *Food Chem* 2024; **452**: 139532 [PMID: 38705120 DOI: 10.1016/j.foodchem.2024.139532]

19 **Akıllıoğlu HG**, Lund MN. Quantification of advanced glycation end products and amino acid cross-links in foods by high-resolution mass spectrometry: Applicability of acid hydrolysis. *Food Chem* 2022; **366**: 130601 [PMID: 34298391 DOI: 10.1016/j.foodchem.2021.130601]

20 **Nomi Y**, Kudo H, Miyamoto K, Okura T, Yamamoto K, Shimohiro H, Kitao S, Ito Y, Egawa S, Kawahara K, Otsuka Y, Ueta E. Free advanced glycation end product distribution in blood components and the effect of genetic polymorphisms. *Biochimie* 2020; **179**: 69-76 [PMID: 32946992 DOI: 10.1016/j.biochi.2020.09.010]

21 **Kato S**, Sugawa H, Tabe K, Ito K, Nakashima H, Nagai R. Rapid pretreatment for multi-sample analysis of advanced glycation end products and their role in nephropathy. *J Clin Biochem Nutr* 2022; **70**: 256-261 [PMID: 35692677 DOI: 10.3164/jcbn.21-175]

22 **Yu T**, Zheng Y, Wang Y, Xiong W, Lin L. Advanced glycation end products interfere with gastric smooth muscle contractile marker expression *via* the AGE/RAGE/NF-κB pathway. *Exp Mol Pathol* 2017; **102**: 7-14 [PMID: 27939576 DOI: 10.1016/j.yexmp.2016.12.002]

23 **Brownlee M**. Biochemistry and molecular cell biology of diabetic complications. *Nature* 2001; **414**: 813-820 [PMID: 11742414 DOI: 10.1038/414813a]

24 **DeChristopher LR**. Perspective: The Paradox in Dietary Advanced Glycation End Products Research-The Source of the Serum and Urinary Advanced Glycation End Products Is the Intestines, Not the Food. *Adv Nutr* 2017; **8**: 679-683 [PMID: 28916568 DOI: 10.3945/an.117.016154]

25 **Kheirouri S**, Alizadeh M, Maleki V. Zinc against advanced glycation end products. *Clin Exp Pharmacol Physiol* 2018; **45**: 491-498 [PMID: 29220086 DOI: 10.1111/1440-1681.12904]

26 **Ni L**, Shen R, Luo H, Li X, Zhang X, Huang L, Deng Y, Liao X, Wu Y, Duan C, Xie X. GlmS plays a key role in the virulence factor expression and biofilm formation ability of Staphylococcus aureus promoted by advanced glycation end products. *Virulence* 2024; **15**: 2352476 [PMID: 38741276 DOI: 10.1080/21505594.2024.2352476]

27 **Lin JA**, Wu CH, Yen GC. Perspective of Advanced Glycation End Products on Human Health. *J Agric Food Chem* 2018; **66**: 2065-2070 [PMID: 29421872 DOI: 10.1021/acs.jafc.7b05943]

28 **Carvalho A**, Lu J, Francis JD, Moore RE, Haley KP, Doster RS, Townsend SD, Johnson JG, Damo SM, Gaddy JA. S100A12 in Digestive Diseases and Health: A Scoping Review. *Gastroenterol Res Pract* 2020; **2020**: 2868373 [PMID: 32184815 DOI: 10.1155/2020/2868373]

29 **Lin HJ**, Hsu FY, Chen WW, Lee CH, Lin YJ, Chen YY, Chen CJ, Huang MZ, Kao MC, Chen YA, Lai HC, Lai CH. Helicobacter pylori Activates HMGB1 Expression and Recruits RAGE into Lipid Rafts to Promote Inflammation in Gastric Epithelial Cells. *Front Immunol* 2016; **7**: 341 [PMID: 27667993 DOI: 10.3389/fimmu.2016.00341]

30 **Kishi S**, Nishiguchi Y, Honoki K, Mori S, Fujiwara-Tani R, Sasaki T, Fujii K, Kawahara I, Goto K, Nakashima C, Kido A, Tanaka Y, Luo Y, Kuniyasu H. Role of Glycated High Mobility Group Box-1 in Gastric Cancer. *Int J Mol Sci* 2021; **22**: 5185 [PMID: 34068442 DOI: 10.3390/ijms22105185]

31 **Reddy VP**, Aryal P, Darkwah EK. Advanced Glycation End Products in Health and Disease. *Microorganisms* 2022; **10**: 1848 [PMID: 36144449 DOI: 10.3390/microorganisms10091848]

32 **Li T**, Qin W, Liu Y, Li S, Qin X, Liu Z. Effect of RAGE gene polymorphisms and circulating sRAGE levels on susceptibility to gastric cancer: a case-control study. *Cancer Cell Int* 2017; **17**: 19 [PMID: 28184178 DOI: 10.1186/s12935-017-0391-0]

33 **Zhou Y**, Liu SX, Zhou YN, Wang J, Ji R. Research on the relationship between RAGE and its ligand HMGB1, and prognosis and pathogenesis of gastric cancer with diabetes mellitus. *Eur Rev Med Pharmacol Sci* 2021; **25**: 1339-1350 [PMID: 33629304 DOI: 10.26355/eurrev_202102_24841]

34 **Zgutka K**, Tkacz M, Tomasiak P, Tarnowski M. A Role for Advanced Glycation End Products in Molecular Ageing. *Int J Mol Sci* 2023; **24**: 9881 [PMID: 37373042 DOI: 10.3390/ijms24129881]

Published by **Baishideng Publishing Group Inc**
7041 Koll Center Parkway, Suite 160, Pleasanton, CA 94566, USA
Telephone: +1-925-3991568
E-mail: office@baishideng.com
Help Desk: https://www.f6publishing.com/helpdesk
https://www.wjgnet.com