Clinical Medicine & Pharmacology

Research Article

https://doi.org/10.70731/ypcfxc33

CYP2C19 Gene Polymorphism in Patients with Digestive Disease from Hezhou Area

Min Deng [a], **Wenjian Zhou** [b], **Qiuyan Wei** [b], **Peng Li** [c,*], **Nianyi Ye** [a]

[a] *Department of Digestive Diseases, The People's Hospital of Hezhou, Hezhou 542800, China*
[b] *Department of Pharmacy, The People's Hospital of Hezhou, Hezhou 542800, China*
[c] *School of Design, Hezhou University, Hezhou 542800, China*

KEYWORDS

Vitamin D Receptor;
Polymorphism;
Breast Cancer;
Meta-Analysis

ABSTRACT

The CYP2C19 gene, characterized by high polymorphism, plays a key role in interindividual variability in drug response, particularly for proton pump inhibitors (PPIs). However, data on CYP2C19 genetic distribution in the local Hezhou population of Guangxi Province remain limited. This study aimed to identify CYP2C19 polymorphisms in digestive disease patients in Hezhou and compare genotype prevalence across populations to clarify allele distribution patterns and optimize prescription strategies. A total of 95 patients with digestive disorders were analyzed using high-throughput sequencing to detect CYP2C19 variants, and genotype distribution was further compared across different ethnic groups. The allele frequencies of CYP2C19*1, *2, *3, and *17 were 93.68%, 40%, 8.42%, and 7.37%, respectively. In terms of metabolic phenotypes, normal metabolizers (*1/*1) comprised 47.37%, rapid metabolizers (*1/*17) 6.32%, intermediate metabolizers (*1/*2, *1/*3, *2/*17) 41.05%, and poor metabolizers (*2/*2, *2/*3) 5.26%. Statistical analysis revealed no significant differences in genotype distribution between males and females (p=0.548), across six digestive disease categories (p=0.956), or between *Helicobacter pylori*-positive and -negative groups (p=0.160). These findings suggest that CYP2C19 polymorphism is prevalent in the Hezhou population but shows no significant association with sex, digestive disease subtype, or *H. pylori* infection status. The results provide insight into the genetic landscape of CYP2C19 in this region and highlight the importance of considering genetic background in guiding rational PPI use, while also serving as a reference for personalized treatment strategies in populations with similar genetic structures.

INTRODUCTION

Precision medicine has revolutionized patient care by tailoring treatments to individuals' genetic profiles, maximizing drug efficacy while minimizing adverse effects[1–3]. A key strategy involves analyzing genetic variations in cytochrome P450 (CYP) enzymes, crucial players in drug metabolism that either activate pro-drugs or break down active compounds during phase-1 processing[4–6]. Take CYP2C19, for example—this liver enzyme metabolizes drugs so extensively that barely half reach circulation, with certain genetic variants further reducing bioavailability. The CYP2C19*2 and CYP2C19*3 mutations account for most poor metabolizer (PM) cases, while CYP2C19*1 indicates normal metabolic function. Research demonstrates that proton pump inhibitors (like omeprazole and lansoprazole) combined with antibiotics show superior H. pylori eradication rates in patients carrying the *2 and *3 variants[7, 8]. Interestingly, the CYP2C19*17

* Corresponding author. E-mail address: 295548270@qq.com

Received 16 August 2025; Received in revised from 27 August 2025; Accepted 29 August 2025; Published online 31 August 2025.

variant boosts enzyme activity, potentially speeding up drug metabolism—though its impact on H. pylori treatment success remains less documented[9].

Existing research indicates significant regional and ethnic variations in the prevalence of CYP2C19*2, CYP2C19*3, and CYP2C19*17 alleles[10]. While nationwide studies have mapped CYP2C19 genetic polymorphisms across China[11], a notable gap remains in our understanding of these variations within Hezhou's population. This study specifically addresses this knowledge gap by investigating the distribution patterns of CYP2C19 gene polymorphisms in the Hezhou region.

There's no doubt that the phenotypes of CYP2C19 play a significant role in the success rate of eradicating H.pylori[12, 13]. The polymorphism status of CYP2C19 can also increase the risk of developing gastric cancer in individuals harboring H.pylori[14]. Furthermore, according to Tong Tie Gang and colleagues, this same polymorphism status may predispose gastric cancer patients to H.pylori infection[15]. Yet, the exact role of CYP2C19's polymorphism status in H.pylori infections among those with digestive disorders remains a mystery. This study delved into the potential link between H.pylori infection, as assessed by the 14C-urea breath test, and the CYP2C19 gene variants.

Wang Xianghui and colleagues observed that omeprazole worked better in the intermediate metabolizer (IM) and poor metabolizer (PM) groups compared to the extensive metabolizer (EM) group, although there wasn't a noticeable difference between the IM and PM groups[16]. On the other hand, it seems CYP2C19 gene variations don't really impact how well rabeprazole treats GERD[17]. That being said, another study indicated that esomeprazole's effectiveness in treating GERD did, in fact, differ significantly among the EM, IM, and PM groups[18]. So, whether there's a real connection between CYP2C19 gene diversity and digestive issues is still up for debate. This study aims to explore the possible link between digestive diseases and these CYP2C19 gene polymorphisms.

MATERIALS AND METHODS

Subjects

This research employed a retrospective design, analyzing data from 95 patients diagnosed with digestive disorders (average age: 57.16±14.46 years; age range: 21-89 years) who received treatment at Hezhou People's Hospital from December 2022 through October 2024. All participants were of Han Chinese descent with at least three generations of paternal lineage rooted in Hezhou, Guangxi Province. We excluded individuals with hepatic conditions, advanced cardiac insufficiency, or renal impairment. None of the selected subjects were biologically related. The investigation adhered strictly to the ethical guidelines outlined in the Declaration of Helsinki. Hezhou People's Hospital's Institutional Review Board granted ethical approval for this study (approval number: 2025040242), and we obtained written informed consent from every participant prior to their inclusion.

DNA Extraction

Blood samples were drawn from each participant and collected in 2 mL EDTA tubes to prevent coagulation. Using the MagPure Fast Blood DNA KF Kit (manufactured by Shenzhou Medical Biotechnology Co., Ltd. in Beijing, China), genomic DNA was extracted following the protocol provided by the supplier.

DNA Genotyping

The CYP2C19*1, *2, *3, and *17 alleles were identified using a commercially available test kit manufactured by Shenzhou Medical Data Technology in Beijing, China. The procedure involved combining 5 μl of sample DNA with 19 μl of either CYP2C19 amplification solution 1 or 2, along with 1 μl of reaction solution A from the kit. Each PCR run incorporated both positive and negative controls for quality assurance. Thermal cycling parameters consisted of an initial 5-minute hold at 50°C, followed by 5 minutes of denaturation at 94°C. This was succeeded by 35 amplification cycles (94°C for 25 seconds, 48°C for 40 seconds, and 72°C for 30 seconds per cycle), culminating in a final 5-minute extension at 72°C. The resulting PCR products were then subjected to hybridization in a specialized reaction chamber. For final analysis, the processed gene chips were scanned using Xi'an Tianlong Technology's reading instrument, with allele detection performed through BaiO Technology's BaioRBE-2.0 software (Shanghai, China). All experimental runs included appropriate positive and negative controls to validate the results.

Definition

The polymorphisms pinpointed were christened according to the nucleotide sequence in reference[19–21]. We categorized them following the Clinical Pharmacogenetics Implementation Consortium (CPIC) protocols; we detected the CYP2C19 wild-type gene along with three variants, specifically CYP2C19 *2, CYP2C19 *3, and CYP2C19 *17[13]. Furthermore, those with two copies of the hyperactive alleles, such as CYP2C19*17/*17, were labeled as potential CYP2C19 ultrarapid metabolizers (UM). Individuals with one hyperactive allele and one standard allele, like CYP2C19*1/*17, were deemed as potential rapid metabolizers (RM). Those with two copies of the standard allele, like CYP2C19*1/*1, were categorized as possible normal metabolizers (NM). People carrying two different copies of alleles with diminished or non-functioning properties, such as any combination of *2 and *3 alleles, were identified as potential poor metabolizers (PM). Moreover, those with one standard allele and one altered or increased function allele, or vice versa, such as CYP2C19*1/*2, CYP2C19*1/*3, CYP2C19*2/*17, or CYP2C19*3/*17, were classified as potential intermediate metabolizers (IM).

Statistical Analysis

Data was analyzed using Excel and SPSS 24.0. Allele and genotype frequencies were computed via the gene counting approach. To assess disparities in allele frequencies among our sample and external populations, we relied on the chi-

Table 1 | Different CYP2C19 allele and genotype frequencies in 95 patients

Metabolic phenotype	Genotype	Sample size(N)	Genotype frequency (%)
RM	*1*17	6	6.32
NM	*1*1	45	47.37
IM	*1*2	32	33.68
	*1*3	6	6.32
	*2*17	1	1.05
PM	*2*2	3	3.16
	*2*3	2	2.11

Table 2 | Distribution of different genotypes in different gender

Gender		*1*17	*1*1	*1*2	*1*3	*2*17	*2*2	*2*3
male	47	4 (8.5)	22 (46.8)	16 (34.0)	4 (8.5)	0 (0.0)	0 (0.0)	1 (2.1)
female	48	2 (4.2)	23 (47.9)	16 (33.3)	2 (4.2)	1 (2.1)	3 (6.3)	1 (2.1)
P[a]					0.548			

Table 3 | Allele frequencies of CYP2C19 in different populations

Population	Number	CYP2C19 allele frequencies(%)				Reference
		*1	*2	*3	*17	
Asian						
Chinese Hezhou	95	93.68	40	8.42	7.37	Present study
Chinese Foshan	1231	63.89	30.46	5.65	-	11
Chinese Ningxia	1050	63.2	31.7	4.5	2.1	22
Chinese Hakka	6686	64.33	31.06	4.61	-	23
Chinese Dai	193	66.30	30.30	3.40		24
Chinese Tibetan	96	78.13	15.10	5.21	1.56	25
Chinese Li	100	74.00	24.50	1.50	-	26
Chinese Han	136	56.25	38.60	5.15	-	27
Chinese Uighur	214	65.42	32.48	2.1	-	27
Chinese Hui	164	45.43	49.39	5.18	-	27
Chinese Mongolian	158	54.11	41.46	4.43	-	27
Korean	103	67	21	12	-	28
Japanese	186	58.06	28.76	13.17	-	29
Vietnamese	90	62	24	14	-	30
Thai	1051	63	27	1	-	31
Malaysian	54	72	23	5	-	32
Caucasians						
Swedish	175	77	23	0	-	33
Italian	360	89	11	0	-	34
Russian	290	88	11	0	-	35
Bolivian	778	92	8	0	-	36
Faroese	312	97	3	0	-	37
Mexican	238	77	8	-	-	38
Turkish	404	88	12	0	-	39
Africans						
Tanzanian	251	81	18	1	-	40
Ethiopian	114	84	14	2	-	41
Zimbabwean	84	87	13	0	-	42

square test and Fisher's exact test. We deemed any p-values less than 0.05 to indicate statistically significant findings. The chi-square test was employed to determine Hardy-Weinberg equilibrium for each allele.

RESULTS

CYP2C19 genotype and allele frequencies of all subjects.

The study enrolled 95 participants (47 male, 48 female) diagnosed with digestive disorders. Genetic analysis revealed the following allele frequencies: CYP2C19*1 at 93.68%, CYP2C19*2 at 40%, CYP2C19*3 at 8.42%, and CYP2C19*17 at 7.37%. Notably, the CYP2C19*3 variant occurred roughly four times less frequently than CYP2C19*2. Based on genetic polymorphisms, the cohort was categorized into four phenotypic groups: 6 rapid metabolizers (RM, CYP2C19*1/*17), 45 normal metabolizers (NM, CYP2C19*1/*1), 39 intermediate metabolizers (IM, comprising CYP2C19*1/*2, CYP2C19*1/*3, or CYP2C19*2/*17), and 5 poor metabolizers (PM, CYP2C19*2/*2 or CYP2C19*2/*3). The genotypes CYP2C19*3*3, CYP2C19*3*17, and CYP2C19*17*17 were not detected in any participants. The distribution of CYP2C19 alleles and genotypes across the study population maintained Hardy-Weinberg equilibrium ($\chi^2 = 1.936$, $p = 0.925$).

In **Table 2**, we can see a side-by-side comparison of the CYP2C19 genotype distribution across male and female participants. The analysis yielded no discernible discrepancy in the distribution between the genders in the study population suffering from gastrointestinal disorders, suggesting a non-significant difference ($P > 0.05$).

CYP2C19 allele frequency in our study compared with previous reports.

We conducted a comparative analysis of the CYP2C19 allele frequencies in our dataset, contrasting it with findings from various international studies encompassing different countries and ethnic backgrounds (refer to **Table 3**). Our findings indicated that the prevalence of the CYP2C19*1 allele within our demographic matched closely with that observed in Caucasian populations and was notably higher than in Asian populations. Furthermore, the CYP2C19*2 and CYP2C19*3 allele frequencies among our participants aligned with those found in Asian studies, yet were notably more common than in Caucasian studies.

Comparison of genotype among patients with different clinical diseases

As detailed in **Table 4**, patients were categorized by the specific digestive ailment they were suffering from, ultimately falling into one of six clinical disease classifications. The data revealed no statistically significant link ($P > 0.05$) between a patient's CYP2C19 genotype and their metabolic phenotype, regardless of the particular digestive disease they had.

Table 4 | Distribution of different genotypes in different digestive diseases

Clinical illness	n	Genotype						
		*1*17	*1*1	*1*2	*1*3	*2*17	*2*2	*2*3
gastritis	41	3 (7.3)	15 (36.6)	16 (39.0)	4 (9.8)	1 (2.4)	1 (2.4)	1 (2.4)
peptic ulcer	10	0 (0.0)	5 (50.0)	3 (30.0)	0 (0.0)	0 (0.0)	2 (20.0)	0 (0.0)
gastroesophageal reflux	5	1 (20.0)	2 (40.0)	2 (40.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
polyp of stomach	12	1 (8.3)	7 (58.3)	4 (33.3)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
UGB	18	1 (5.5)	9 (50.0)	5 (27.8)	2 (11.1)	0 (0.0)	0 (0.0)	1 (5.5)
esophageal cancer	2	0 (0.0)	1 (50.0)	1 (50.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
P price					0.956			

Table 5 | CYP2C19 comparison of the different genotypes and metabolic phenotypes

H.pylori	n	Genotype						
		*1*17	*1*1	*1*2	*1*3	*2*17	*2*2	*2*3
positive	11	0 (0.0)	3 (27.3)	5 (0.0)	1 (0.0)	0 (0.0)	2 (0.0)	0 (0.0)
negative	37	2 (5.4)	18 (48.6)	13 (35.1)	3 (8.1)	1 (2.7)	0 (0.0)	0 (0.0)
P[a]					0.160			
H.pylori	**n**	**Metabolic phenotype**						
		RM		**NM**		**IM**		**PM**
positive	47	0 (0.0)		3 (27.3)		6 (54.5)		2 (18.1)
negative	48	2 (5.4)		16 (43.2)		17 (45.9)		0 (0.0)
P[a]					0.092			

Comparison of genotype and metabolic phenotype between patients with positive and negative C14 breath test

In the H. pylori-positive and negative cohorts, the genotypes showed no significant disparity (p=0.160), as indicated in **Table 5**. Among those with H. pylori, the distribution of RM, NM, IM, and PM was 0%, 27.3%, 54.5%, and 18.1%, respectively. For those without H. pylori, the split was 5.4%, 43.2%, 45.9%, and 0% for RM, NM, IM, and PM, respectively. A lack of statistical variation was detected across the various metabolic types (p=0.092), as detailed in Table 5.

DISCUSSION

As of June 2020, the Pharmacogenomics Knowledge Base (PharmGKB), a key resource in genetic pharmacology and pharmacogenomics, put out a list of must-know drug-related genes. These genes are classified into three tiers of clinical relevance. CYP2C19 tops the list as a first-tier gene, and rightly so, given the wealth of clinical evidence highlighting its pharmacogenomic significance[21].

The CYP2C19 gene exhibits significant genetic diversity, with 38 distinct alleles currently identified. These variants are categorized into five functional groups:

1) **Normal Function Alleles**: *1, *11, *13, *15, *18, *28, *38
2) **Non-Functional Alleles**: *2–*8, *22, *24, *35–*37
3) **Reduced Function Alleles**: *9, *10, *16, *19, *25, *26
4) **Alleles with Uncertain Function**: *2, *14, *23, *29–*34
5) **Enhanced Function Allele**: *17

This classification helps clarify the varying enzymatic activity associated with each allele.

Our investigation identified four distinct alleles and seven genotypes. Previous research indicates that the CYP2C9*2 allele occurs most frequently in Asian populations (15.1–49.39%) and least often among Caucasians (3–23%)[11, 22–42]. Interestingly, the prevalence of CYP2C19*2 in our sample exceeded rates documented in Caucasian, African, and certain Asian groups (Korean, Japanese, Vietnamese, Thai, Malaysian)[28–32, 33–42]. As Table 3 demonstrates, our findings for CYP2C19*2 frequency align more closely with data from other Asian populations.

Regarding CYP2C19*3, the Hezhou population showed an elevated frequency of 8.42% compared to other Chinese groups[11, 22–27]. However, our observed frequency for this allele fell below reported levels in Japanese, Korean, and Vietnamese populations [28–30], while still exceeding those recorded in Thai, Malaysian, African, and Caucasian groups [31–42].

The CYP2C19*17 allele stands out as the sole functionally enhanced variant within the CYP2C19 drug metabolism enzyme family. While existing research primarily focuses on how this allele amplifies drug efficacy or triggers adverse effects through enhanced enzymatic activity, our investigation breaks new ground by mapping its population distribution in Hezhou for the first time. These findings establish a crucial reference point for future clinical studies examining this genetic variant within the Hezhou population.

Notably, the prevalence of the CYP2C19*17 allele in our study cohort exceeded reported frequencies in both Ningxia and Tibetan populations[22,25].

Research by Sanford JC et al. suggests that additional regulatory mechanisms could contribute to mRNA expression imbalances in the livers of individuals of African descent with the CYP2C19*17 variant, which is associated with heightened metabolic activity of CYP2C19 enzymes[43]. This implies that factors beyond genetic variation may modulate the impact of CYP2C19*17 on digestive disorders. Conversely, Negovan Anna et al. demonstrated that the CYP2C19*2 and *3 variants showed no statistically significant association with digestive diseases[44]. Our findings align with this observation, revealing no gender-based or disease-specific variations in CYP2C19 genotype distribution. Nevertheless, further investigation is warranted to determine whether distinct CYP2C19 alleles exert differential effects across various digestive pathologies.

When it comes to gastric cancer, the majority of patients exhibit *Helicobacter pylori* infection accompanied by intense active gastritis or atrophic gastritis. This suggests that the potential carcinogen metabolized by CYP2C19 requires the inflammatory or atrophic damage triggered by *H. pylori* to kickstart cancerous changes in gastric epithelial cells[45]. Consequently, we hypothesize that *H. pylori* infection amplifies the direct impact of this CYP2C19-processed carcinogen on the stomach lining[45]. Our research examined CYP2C19 genetic variations in both *H. pylori*-positive and *H. pylori*-negative groups, yet no meaningful link emerged between the infection and these polymorphisms.

Our findings indicate that the CYP2C19 allele and its corresponding genotype and metabolic profile may not correlate with H. pylori susceptibility, casting doubt on the necessity of factoring in CYP2C19 gene variants when assessing H. pylori susceptibility risks[46]. The occurrence of H. pylori infection is a complex interplay of various elements, including where one lives and their daily lifestyle[47]. It's crucial to present evidence from a substantial, multifaceted study to validate the link between H. pylori susceptibility and CYP2C19 gene polymorphisms.

In summary, this research examined 95 individuals suffering from gastrointestinal issues in Hezhou, revealing that the distribution of CYP2C19 gene polymorphisms closely mirrors trends seen across broader Asian demographics. While pharmacogenomic studies linking CYP2C19 variations to proton pump inhibitor efficacy have gained traction globally, China's clinical landscape still lags behind, with insufficient data from comprehensive trials to draw definitive conclusions. Moving forward, the medical community should prioritize investigating how these genetic differences influence PPI dosing requirements, ultimately paving the way for tailored treatment protocols that optimize therapeutic outcomes..

Disclosure statement No potential conflict of interest was reported by the author(s).

Funding This study was supported by Scientific research project funded by

Guangxi Zhuang Autonomous Region Health Commission (grant number Z-J20221785).

Author contributions Deng Min, Zhou Wenjian,Li Peng coordinated and performed all sample analyses. Deng Min, Wei Qiuyan and Ye Nianyi drafted and revised the manuscript. All the authors read and approved the final manuscript.

Availability of data and materials The data collected and examined in this study are not openly accessible to safeguard participant confidentiality; however, they can be obtained from the lead researcher upon justified request.

References

1. Velmovitsky PE, Bevilacqua T, Alencar P, Cowan D, Morita PP. Convergence of Precision Medicine and Public Health Into Precision Public Health: Toward a Big Data Perspective. Front Public Health. 2021;9:561873.
2. Döhner H, Wei AH, Löwenberg B. Towards precision medicine for AML. Nat Rev Clin Oncol. 2021;18(9):577–90.
3. Weldy CS, Ashley EA. Towards precision medicine in heart failure. Nat Rev Cardiol. 2021;18(11):745–62.
4. Ahmad T, Valentovic MA, Rankin GO. Effects of cytochrome P450 single nucleotide polymorphisms on methadone metabolism and pharmacodynamics. Biochem Pharmacol. 2018;153:196–204.
5. Stajnko A, Runkel AA, Kosjek T, Snoj Tratnik J, Mazej D, Falnoga I, et al. Assessment of susceptibility to phthalate and DINCH exposure through CYP and UGT single nucleotide polymorphisms. Environ Int. 2022;159: 107046.
6. Gao B, Tan T, Cao X, Pan M, Yang C, Wang J, et al. Relationship of cytochrome P450 gene polymorphisms with blood concentrations of hydroxychloroquine and its metabolites and adverse drug reactions. BMC Med Genom. 2022;15(1):23.
7. Sapone A, Vaira D, Trespidi S, Perna F, Gatta L, Tampieri A, et al. The clinical role of cytochrome p450 genotypes in Helicobacter pylori management. Am J Gastroenterol. 2003;98(5):1010–5.
8. Ozdil B, Akkiz H, Bayram S, Bekar A, Akgöllü E, Sandikçi M. Influence of CYP2C19 functional polymorphism on Helicobacter pylori eradication. Turk J Gastroenterol. 2010;21(1):23–8.
9. Kurzawski M, Gawrońska-Szklarz B, Wrześniewska J, Siuda A, Starzyńska T, Droździk M. Effect of CYP2C19*17 gene variant on Helicobacter pylori eradication in peptic ulcer patients. Eur J Clin Pharmacol. 2006;62(10):877–80.
10. Shuldiner AR, O'Connell JR, Bliden KP, Gandhi A, Ryan K, Horenstein RB, et al. Association of cytochrome P450 2C19genotype with the antiplatelet effect and clinical efficacy of clopidogrel therapy. JAMA. 2009;302(8):849–57.
11. Yuan XW, Yuan SY, Wu GX, Wu ZX, Guan ZY. Genetic polymorphism of clopidogrel metabolism related gene CYP2C19 gene in Chinese from Foshan area of Guangdong Province. Hematology. 2022;27(1):1056–61.
12. Zhang YD, Dong QW, Zhang SH, Gu F, Zhang Y, Song HB, et al. Effectiveness of eradication regimen based on the bacterial susceptibility and CYP2C19 genotype in children with refractory Helicobacter pylori infection. Zhonghua Er Ke Za Zhi. 2020;58(1):41–5 (Chinese).
13. Ima JJ, Thomas CD, Barbarino J, Desta Z, Van Driest SL, El Rouby N, et al. Clinical pharmacogenetics implementation consortium (CPIC) Guideline for CYP2C19 and proton pump inhibitor dosing. Clin Pharmacol Ther. 2021;109(6):1417–23.
14. Wang P, Feng SB, Jin Y. CYP2C19 gene polymorphism and susceptibility to gastric cancer after helicobacter pylori infection. Chinese J Nosocomiol. 2020;30(2):170–3.
15. Tong TG, Zhang RL, Fu GF. The distribution characteristics of CYP2C19 gene polymorphism in Hp positive gastric cancer patients. Modern Diag Treat. 2017;28(11):2116–7.
16. Wang Xianghui, Yu Jing, Gao Ruofei.Effect of CYP2C19 gene polymorphism on the efficacy of omeprazole in the treatment of pediatric gastric ulcer [J]. Experimental and Clinical Medicine, 2020,38(2):286-288.
17. Cao Shaohua, Gao Xuede, Zhang Jinran, et al.Relationship between CYP2C19 gene polymorphism and the efficacy of proton pump inhibitor therapy in gastroesophageal reflux disease [J]. Modern Digestive and Interventional Therapy, 2020,25(7):889-892.
18. Peng Jinwei, Zhou Baozhu, Liao Guobin et al.Effect of CYP2C19 gene polymorphism on the efficacy of esomeprazole in treating gastroesophageal reflux disease [J]. Chinese Journal of Modern Applied Pharmacy, 2020,37(18):2254-2257.
19. Caudle KE, Sangkuhl K, Whirl-Carrillo M, Swen JJ, Haidar CE, Klein TE, et al. Standardizing CYP2D6 genotype to phenotype translation: consensus recommendations from the clinical pharmacogenetics implementation consortium and dutch pharmacogenetics Working group. Clin Transl Sci. 2020;13(1):116–24.
20. PharmGKB. CYP2C19 clinical annotations. 2021. https://www.pharmgkb.org/gene/PA124/clinicalAnnotation.
21. PharmGKB. Drug Label Information and Legend. 2020. https://www.pharmgkb.org/
22. Zhen Yang,Yunqian Xie, Daya Zhang, et al.CYP2C19 gene polymorphism in Ningxia.Pharmacological Reports (2023) 75:705–714.
23. Zhong Z, Hou J, Li B, Zhang Q, Liu S, Li C, et al. Analysis of CYP2C19 genetic polymorphism in a large ethnic hakka population in Southern China. Med Sci Monit. 2017;23:6186–92.
24. He N, Yan FX, Huang SL, Wang W, Xiao ZS, Liu ZQ, et al. CYP2C19 genotype and S-mephenytoin 4'-hydroxylation phenotype in a Chinese Dai population. Eur J Clin Pharmacol. 2002;58(1):15–8.
25. Jin T, Zhang X, Geng T, Shi X, Wang L, Yuan D, et al. Genotypephenotype analysis of CYP2C19 in the Tibetan population and its potential clinical implications in drug therapy. Mol Med Rep. 2016;13(3):2117–23.
26. Ding Y, Xu D, Zhang X, Yang H, Geng T, He P, et al. Genetic polymorphisms and phenotypic analysis of drug-metabolizing enzyme CYP2C19 in a Li Chinese population. Int J Clin Exp Pathol.2015;8(10):13201–8.
27. Zuo LJ, Guo T, Xia DY, Jia LH. Allele and genotype frequencies of CYP3A4, CYP2C19, and CYP2D6 in Han, Uighur, Hui, and Mongolian Chinese populations. Genet Test Mol Biomarkers. 2012;16(2):102–8.
28. Roh HK, Dahl ML, Tybring G, Yamada H, Cha YN, Bertilsson L. CYP2C19 genotype and phenotype determined by omeprazole in a Korean population. Pharmacogenetics. 1996;6(6):547–51.
29. Kubota T, Chiba K, Ishizaki T. Genotyping of S-mephenytoin 4'-hydroxylation in an extended Japanese population. Clin Pharmacol Ther. 1996;60(6):661–6.
30. Yamada S, Onda M, Kato S, Matsuda N, Matsuhisa T, Yamada N, et al. Genetic differences in CYP2C19 single nucleotide polymorphisms among four Asian populations. J Gastroenterol. 2001;36(10):669–72.
31. Sukasem C, Tunthong R, Chamnanphon M, Santon S, Jantararoungtong T, Koomdee N, et al. CYP2C19 polymorphisms in the Thai population and the clinical response to clopidogrel in patients with atherothrombotic-risk factors. Pharmgenom Pers Med. 2013;6:85–91.
32. Pang YS, Wong LP, Lee TC, Mustafa AM, Mohamed Z, Lang CC. Genetic polymorphism of cytochrome P450 2C19 in healthy Malaysian subjects. Br J Clin Pharmacol. 2004;58:332–5.
33. Chang M, Dahl ML, Tybring G, Gotharson E, Bertilsson L. Use of omeprazole as a probe drug for CYP2C19 phenotype in Swedish Caucasians: comparison with S-mephenytoin hy-droxylation phenotype and CYP2C19 genotype. Pharmacogenetics. 1995;5:358–63.
34. Scordo MG, Caputi AP, D'Arrigo C, Fava G, Spina E. Allele and genotype frequencies of CYP2C9, CYP2C19 and CYP2D6 in an Italian population. Pharmacol Res. 2004;50:195–200.
35. Gaikovitch EA, Cascorbi I, Mrozikiewicz PM, Brockmöller J, Frötschl R, Köpke K, et al. Polymorphisms of drug-metabolizing enzymes CYP2C9, CYP2C19, CYP2D6, CYP1A1, NAT2 and of P-glycoprotein in a Russian population. Eur J Clin Pharmacol. 2003;59:303–12.
36. Bravo-Villalta HV, Yamamoto K, Nakamura K, Bayá A, Okada Y, Horiuchi R. Genetic polymor-phism of CYP2C9 and CYP2C19 in a Bolivian population: An investigative and comparative study. Eur J Clin Pharmacol. 2005;61:179–84.
37. Halling J, Petersen MS, Damkier P, Nielsen F, Grandjean P, Weihe P, et al. Polymorphism of CYP2D6, CYP2C19, CYP2C9 and CYP2C8 in the Faroese population. Eur J Clin Pharmacol. 2005;61:491–7.
38. Favela-Mendoza AF, Martinez-Cortes G, Hernandez-Zaragoza M, Salazar-Flores J, Muñoz-Valle JF, Martinez-Sevilla VM, et al. Genetic variability of CYP2C19 in a Mexican population: Contribution to the knowl-edge of the inheritance pattern of CYP2C19*17 to develop the ultrarapid metabolizer phenotype. J Genet. 2015;94:1–5.

39. Aynacioglu AS, Sachse C, Bozkurt A, Kortunay S, Nacak M, Schröder T, et al. Low frequency of defec-tive alleles of cytochrome P450 enzymes 2C19 and 2D6 in the Turkish population. Clin Pharmacol Ther. 1999;66:185–92.
40. Herrlin K, Massele AY, Jande M, Alm C, Tybring G, Abdi YA, et al. Bantu Tanzanians have a decreased capacity to metabolize omeprazole and mephenytoin in relation to their CYP2C19 genotype. Clin Pharmacol Ther. 1998;64:391–401.
41. Persson I, Aklillu E, Rodrigues F, Bertilsson L, Ingelman-Sundberg M. S-mephenytoin hy-droxylation phenotype and CYP2C19 genotype among Ethiopians. Pharmacogenetics. 1996;6:521–6.
42. Masimirembwa C, Bertilsson L, Johansson I, Hasler JA, Ingelman-Sundberg M. Pheno-typing and genotyping of S-mephenytoin hy-droxylase (cytochrome P450 2C19) in a Shona population of Zimbabwe. Clin Pharmacol Ther. 1995;57:656–61.
43. Sanford JC, Guo Y, Sadee W, Wang D. Regulatory polymorphisms in CYP2C19 afecting hepatic expression. Drug Metabol Drug Interact. 2013;28(1):23–30.
44. Negovan Anca, Iancu Mihaela, Moldovan Valeriu, et al. Infuence of MDR1C3435T, CYP2C19*2 and CYP2C19*3 gene polymorphisms and clinical characteristics on the severity of gastric lesions: a case control study. J Gastrointest Liver Dis JGLD.2016; 25(2):258–60.
45. Sugimoto M, Furuta T, Shirai N, Nakamura A, Kajimura M, Sugimura H, et al. Poor metabolizer genotype status of CYP2C19 is a risk factor for developing gastric cancer in Japanese patients with Helicobacter pylori infection. Aliment Pharmacol Ther. 2005;22(10):1033–40.
46. Jiang X R, Zhang Y G, Lu J, et al. Analysis of CYP2C19 gene polymorphisms in gastrointestinal diseases among Han Chinese in northern China[J]. J Clin Experim Pathol. 2016;32(10):1101–1104,1109.
47. Lirong Mo. Investigation of the current status of Helicobacter pylori infection in the population of Wuzhong City. Ningxia and analysis of related factor: Ningxia Medical University; 2019.