INTRODUCTION

Cardiovascular diseases (CVDs) as the type of age-related disease including coronary artery disease (CAD), stroke, hypertension, and cerebral infarction, which are one of the main causes of morbidity and mortality in the world (Chen et al., 2017; Mozaffarian et al., 2016; Song et al., 2017; Sun et al., 2017; Yu et al., 2017; Yusuf, Reddy, Öunpuu, & Anand, 2001; Zhao et al., 2016). The clinical risk factors about CVDs have been established for many years, including obesity,
hypothesis, diabetes, and an inactive lifestyle (Cooper et al., 2000; Kaess et al., 2015; Yusuf et al., 2001). However, the pathological statistical concept suggested that the complex molecular basis of CDV was related to a wide range of biological pathways, involving lipid and glucose metabolism, vascular repair and angiogenesis (Gaziano et al., 2006). Several studies have shown that the etiology and pathogenesis of CVDs were likely to comprise a multifactorial disorder resulting from environmental and genetic factors (Cooper, 1999). Apart this, more and more researches identified that inflammatory molecules might play a central role in the pathogenesis of Cardiovascular diseases as well (Marousi, Ellul, et al., 2011).

The occurrence and development of arterial thrombotic diseases are involved in the inflammation (Meuwissen et al., 2004). Inflammatory cytokines are recognized as dysregulated in aging and age-related disease (Liu, Wang, & Jiang, 2017). Interleukin 10 (IL-10, OMIM: 226,990), as a potent immunoregulatory cytokine, is a newly discovered cytokine in recent years, which is widely known for its anti-inflammatory and B-cell stimulating function (Franceschi & Campisi, 2014; Rea et al., 2018). According to the chromosomal location and functional relevance, IL-10 is a multifunctional cytokine that could not only inhibit the synthesis of proinflammatory cytokines but also downregulate antigen presentation and macrophage activation (Lee, Kim, & Song, 2014). Scientific literature reported that IL-10 was related to the pathogenesis and development of coronary artery inflammation (Heeschen et al., 2003a), and have been considered a candidate gene for kind of cardiovascular diseases (CVD) (Liu, Hui-Min, Yang, & Geriatrics, 2017), including vasculitis, cerebral infarction, atrial fibrillation, atherosclerotic, and stroke, but the results are still controversial. Therefore, this meta-analysis was performed to obtain a more precise conclusion and to make a better understanding of IL-10 (−1082 G/A) single-nucleotide polymorphism (SNP) with CVDs risk.

Currently, many epidemiological studies have focused on the relationship between IL-10 (−1082 G/A) SNP and CVDs risk, including CAD, stroke, hypertension, and cerebral infarction, but the results are still controversial (Afzal et al., 2012; Ben-Hadj-Khalifa et al., 2010; Karaca, Kayıkçıoğlu, Onay, Gündüz, & Ozkinay, 2011b; Marousi, Ellul, et al., 2011; Zhang, Pan, Ran, & Bing-Xun, 2007). Furthermore, a single study might be too underpowered to provide accurate conclusion because of relatively small sample size. In order to reach a reliable conclusion, we designed this meta-analysis to assess the relationship of IL-10 (−1082 G/A) SNP with the risk of CVDs.

2 MATERIALS AND METHODS

2.1 Inclusion and exclusion criteria

We executed an extensive search for databases including PubMed, Embase, and Web of Science to confirm relevant research to analyze the association between the IL-10 polymorphisms and CVDs risk. The last report was updated on April 17, 2017. When included in the analysis, qualified researches must meet the following criteria: (1) the association studies about the IL-10 polymorphism and susceptibility to related CVDs must follow case–control study strategies; (2) all patients meet the diagnostic criteria for CVDs in the candidate studies; (3) There is enough available data to calculate the odds ratio (OR) and 95% confidence interval (CI). The major exclusion criteria for studies were: (a) not a case–control study; (b) studies that have been republished; and (c) no feasibility data studies. This meta-analysis was performed conforming to the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines. Finally, data for meta-analysis were available from 31 studies, including 10,502 cases and 7,865 controls 61 (Afzal et al., 2012; Benhadjkhalifa et al., 2010; Cruz et al., 2013; Donger et al., 2001; Elsaid, Abdelaziz, Elmougy, & Elwaseef, 2014; Fragas et al., 2011; He et al., 2015; Ianni et al., 2012; Jiang, Lin, Zhang, Chen, & Liu, 2015; Jin, Peihua, Jiemin, Yi, & Hailiang, 2011; Karaca, Kayıkçıoğlu, Onay, Gündüz, & Ozkinay, 2011a; Koch et al., 2001; Kumar et al., 2016; Li, Gao, He, & Zhang, 2016; Lin, 2009; Lio et al., 2004; Liu, Li, Zhu, & He, 2017; Lorenzová et al., 2007; Marousi, Ellul, et al., 2011; Munshi et al., 2010; O'Halloran, Stanton, O'Brien, & Shields, 2006; Ozkan, Silan, Uludağ, Degirmenci, & Karaman, 2015; Seifart et al., 2005; Babu et al., 2012; Sultana et al., 2011; Tabrez et al., 2017; Tuttolomondo et al., 2012a; Yu et al., 2012; Zhang et al., 2007; Zheng et al., 2014).

2.2 Data extraction

Primary investigators used standardization requirements to extract qualified research from the database. They organized the data into tables and submitted them to coauthors for review to ensure that data are strict and reasonable. We collected the following information from the retrieved literature: first author’s name, publication year, control source, country and ethnicity of population, the methods of genotype, the number of cases and controls, the distribution of genotype in cases and controls, and the p value for HWE (Hardy–Weinberg Equilibrium) in controls. Different ethnicity was categorized as Asians, Caucasians, and Mixed.

2.3 Statistical analyses

The IL-10 (−1082 G/A) polymorphisms and CVDs risk was assessed by OR and the corresponding 95% CI for each study. Different ORs (95% CI) were calculated using the following models: the allele model (G vs. A), the dominant model (GG + AG vs. AA), the recessive model (GG vs. AG + AA),
the homozygote model (GG vs. AA), and the heterozygote model (GA vs. AA). Using the Cochran’s $Q$ statistic and $I^2$ test to evaluate the statistical heterogeneity between eligible studies, which was considered as significant when $p_Q < 0.1$ or $I^2 > 50\%$. A fixed-effect model (the Mantel–Haenszel method) was adopted if $p > 0.1$ and $I^2 < 50\%$, and a random-effect model (DerSimonian–Laird method) was used if $p < 0.1$ and $I^2 > 50\%$. Furthermore, we conducted subgroup analysis based on ethnicity (Asian, Caucasian, or Mixed) and disease subtype (CAD, stroke, and cerebral infarction) to explore the sources of heterogeneity. All statistical analyses were performed using software STATA 15.0 (StataCorp LP).

3 | RESULTS

3.1 | Eligible studies

As shown in Figure 1, the process of literature retrieval and exclusion is listed. In general, the preliminary comprehensive search was identified 813 relevant articles. According to the inclusion and exclusion criteria, this meta-analysis is involving 31 case–control studies with 18,367 total sample sizes (10,502 cases and 7,865 controls).

The characteristics of the studies are listed in Table 1. $IL-10$ (−1082 G/A) genotype distributions in the controls from all studies has carried on HWE (Table 2). The 31 studies identified in this meta-analysis including 14 studies of Caucasians, 15 studies of Asians, and two studies from mixed population. Among these researches, patients were mostly recruited in referral centers with CAD, stroke, cerebral infarction and myocardial infarction, and the controls have not any obvious disease.

3.2 | Meta-analysis databases

As shown in Tables 3 and 4, the results including the association of the $IL-10$ (−1082 G/A) polymorphism and CVDs risk, as well as homozygote test and heterogeneity test. The combined results showed that the variant genotypes were related to increase the CVDs risk in different genetic models (OR = 1.06, 95% CI = 1.03–1.10 for the homozygote comparison model GG vs. AA; OR = 0.88, 95% CI = 0.73–1.06 for the heterozygote comparison model AG vs. AA; OR = 1.10, 95% CI: 1.04–1.15 for the allele model A vs. G; OR = 0.93, 95% CI: 0.87–1.04 for the dominant model GG + AG vs. AA; and OR = 1.03, 95% CI: 1.02–1.05 for the recessive model GG vs. AG + AA). The forest plots for −1082 G/A in the allele model are shown in Figure 2. In subgroup analyses by ethnicity, our results were similar to the Caucasian population.

3.3 | Test of heterogeneity

Using the chi-square test and fixed effects model, we observed the statistically significant heterogeneity in trials (Allele model G vs. A: $p = .000$, $I^2 > 50\%$; dominant model
| First author (year) | Disease                                      | Control source | Country        | Ethnicity | Matching  | Genotyping method |
|---------------------|----------------------------------------------|----------------|----------------|-----------|-----------|------------------|
| Koch et al. (2001)  | Coronary artery disease and myocardial infarction | HB             | Germany        | White     | Age, sex  | AS-PCR           |
| Donger et al. (2001) | Myocardial infarction                         | NA             | Mixed          | White     | Age, sex  | PCR-SSCP         |
| Liu et al. (2004)   | Cardiovascular diseases                       | HB             | North Italy    | White     | Age       | PCR-SSP          |
| Liu et al. (2004)   | Coronary heart disease                        | HB             | South Italy    | White     | Age       | PCR-SSP          |
| Seifert et al. (2005)| Cardiovascular diseases                      | PB             | Germany        | White     | NA        | PCR-RFLP         |
| O’Halloran et al. (2009)| Coronary artery disease                  | NA             | Ireland        | White     | NA        | AS-PCR           |
| Zhang et al. (2007) | Cerebral infarction                          | PB             | China          | Asian     | NA        | PCR-RFLP         |
| Lorenzová et al. (2007)| Myocardial infarction                      | PB             | Czech Republic | White     | Age       | PCR-RFLP         |
| Lin et al. (2009)   | Cerebral infarction                          | HB             | China          | Asian     | Age, sex  | ARMS-PCR         |
| Ben-Hadj-Khalifa et al. (2010) | Coronary artery disease                  | NA             | Tunisia        | White     | Age, sex  | AS-PCR           |
| Munshi et al. (2010) | Ischemic stroke                              | PB             | India          | Asian     | Age, sex  | ARMS-PCR         |
| Kanaca et al. (2011b)| Coronary artery disease                      | NA             | Turkey         | White     | Age       | PCR-RFLP         |
| Marouisi, Ellul, et al. (2011)| Ischemic stroke                           | PB             | Greece         | White     | Age, sex  | RT-PCR           |
| Sultan et al. (2011) | Cerebral infarction                          | PB             | India          | Asian     | Age       | ARMS PCR          |
| Jin et al. (2011)   | Cerebral infarction                          | HB             | China          | Asian     | Age       | RFLP-PCR         |
| Fragoso et al. (2011)| Acute coronary syndrome                      | HB             | Mexico         | Mixed     | Age, sex  | RT-PCR           |
| Babu et al. (2012)  | Cardiovascular diseases                      | NA             | India          | Asian     | Age, sex  | ARMS-PCR         |
| Afzal et al. (2012) | Coronary artery disease                      | HB             | Pakistan       | Asian     | Age       | ARMS-PCR         |
| Ianni et al. (2012) | Myocardial infarction                        | NA             | South Italy    | White     | NA        | TaqMan           |
| Tuttolomondo et al. (2012a) | Ischemic stroke                             | HB             | Italy          | White     | Age       | ASO-PCR          |
| Yu et al. (2012)    | Ischemic heart disease                       | PB             | Korea          | Asian     | NA        | Pyrosequencing   |
| Cruz et al. (2013)  | Myocardial ischemia                          | NA             | Mexico         | Mixed     | NA        | TaqMan           |
| Elsaid et al. (2014) | Cardiovascular                              | NA             | Egypt          | White     | NA        | PCR              |
| Zheng et al. (2014) | Atrial fibrillation                          | PB             | China          | Asian     | NA        | PCR-RFLP         |
| He et al. (2015)    | Ischemic stroke                              | PB             | China          | Asian     | Age       | PCR-RFLP         |
| Jiang et al. (2015) | Ischemic stroke                              | HB             | China          | Asian     | Age, sex  | PCR-RFLP         |
| Ozkan et al. (2015) | Ischemic stroke                              | HB             | Italy          | White     | Age       | RT-PCR           |
| Kumar et al. (2016) | Ischemic stroke                              | PB             | India          | Asian     | Age       | PCR-RFLP         |
| Li et al. (2016)    | Ischemic heart disease                       | PB             | China          | Asian     | Age, sex  | PCR-RFLP         |
| Liu, Hui-Min, et al. (2017)| Ischemic heart disease                | PB             | China          | Asian     | Age, sex  | PCR-LDR          |
| Tabrez et al. (2017) | Cardiovascular diseases                      | HC             | KAUH           | Asian     | NA        | PCR              |
| First author (year)               | Sample size (Case/Control) | Case AA | AG | GG | Control AA | AG | GG | HWE  |
|----------------------------------|----------------------------|--------|----|----|------------|----|----|------|
| Koch et al. (2001)               | 1,791/340                  | 540    | 874 | 377 | 105        | 161 | 74 | 0.407 |
| Donger et al. (2001)             | 1,107/1,082                | 242    | 486 | 256 | 231        | 477 | 244 | 0.944 |
| Lio et al. (2004)                | 142/153                    | 60     | 52  | 30  | 30         | 75  | 48  | 0.942 |
| Lio et al. (2004)                | 90/110                     | 44     | 29  | 17  | 28         | 56  | 26  | 0.846 |
| Seifart et al. (2005)            | 104/243                    | 19     | 59  | 25  | 86         | 115 | 42  | 0.739 |
| O’Halloran et al. (2006)         | 1,598/386                  | 324    | 784 | 490 | 77         | 138 | 117 | 0.004 |
| Zhang et al. (2007)              | 204/131                    | 202    | 2   | 0   | 120        | 14  | 0   | 0.523 |
| Lorenzová et al. (2007)          | 284/568                    | 90     | 98  | 40  | 207        | 255 | 106 | 0.083 |
| Lin (2009)                       | 181/90                     | 153    | 28  | 0   | 83         | 32  | 0   | 0.083 |
| Ben-Hadj-Khalifa et al. (2010)   | 291/291                    | 76     | 108 | 101 | 52         | 100 | 76  | 0.088 |
| Munshi et al. (2010)             | 480/470                    | 92     | 241 | 147 | 63         | 218 | 189 | 0.991 |
| Karaca et al. (2011b)            | 86/88                      | 20     | 44  | 22  | 21         | 44  | 23  | 0.996 |
| Marousi, Antonacopoulou, et al. (2011) | 145/145                | 47     | 71  | 27  | 53         | 71  | 21  | 0.723 |
| Sultana et al. (2011)            | 238/226                    | 154    | 44  | 40  | 163        | 47  | 16  | 0.000 |
| Jin et al. (2011)                | 189/92                     | 161    | 27  | 1   | 78         | 12  | 2   | 0.087 |
| Fragoso et al. (2011)            | 389/302                    | 211    | 142 | 36  | 164        | 113 | 25  | 0.38  |
| Babu et al. (2012)               | 651/432                    | 318    | 260 | 73  | 170        | 188 | 74  | 0.079 |
| Afzal et al. (2012)              | 93/99                      | 6      | 77  | 10  | 4          | 92  | 3   | 0.000 |
| Ianni et al. (2012)              | 267/321                    | 68     | 141 | 56  | 78         | 88  | 73  | 0.000 |
| Tuttolomondo et al. (2012a)      | 96/48                      | 58     | 14  | 24  | 20         | 17  | 11  | 0.065 |
| Yu et al. (2012)                 | 173/313                    | 150    | 22  | 1   | 275        | 38  | 0   | 0.253 |
| Cruz et al. (2013)               | 149/248                    | 55     | 83  | 11  | 125        | 106 | 17  | 0.387 |
| Elsaid et al. (2014)             | 108/143                    | 2      | 49  | 22  | 8          | 85  | 5   | 0.000 |
| Zheng et al. (2014)              | 117/100                    | 84     | 27  | 6   | 55         | 35  | 10  | 0.221 |
| He et al. (2015)                 | 260/260                    | 41     | 124 | 95  | 29         | 108 | 123 | 0.475 |
| Jiang et al. (2015)              | 181/115                    | 153    | 28  | 0   | 83         | 32  | 0   | 0.083 |
| Ozkan et al. (2015)              | 42/48                      | 11     | 26  | 5   | 19         | 18  | 11  | 0.113 |
| Kumar et al. (2016)              | 250/250                    | 11     | 77  | 162 | 4          | 37  | 209 | 0.127 |
| Li et al. (2016)                 | 335/335                    | 54     | 151 | 130 | 34         | 143 | 158 | 0.844 |
| Liu, Hui-Min, et al. (2017)      | 386/386                    | 313    | 68  | 5   | 308        | 75  | 3   | 0.498 |
| Karami, Zabihzadeh, Shams, and Saki Malehi (1002) | 75/50                  | 1      | 66  | 8   | 40         | 1   | 9   | 0.000 |
GG/AG vs. AA: \( p = .000, \, I^2 > 50\% \); recessive model GG vs. AG/AA: \( p = .000, \, I^2 > 50\% \); homozygote comparison model GG vs. AA: \( p = .000, \, I^2 > 50\% \); heterozygote comparison model GA vs. AA: \( p = 0.000, \, I^2 > 50\% \) (Tables 3 and 4). Thus, the wider CIs will be generated by the random-effect model.

### 3.4 | Sensitivity analysis and Bias diagnostics

To evaluate the stability of the pooled results, sensitivity analysis was performed. The result showed that there were no substantial changes in ORs after canceling each study (Figure 3). As to the publication bias, it was estimated by the Begg’s funnel plots and Egger’s tests. The funnel plot shapes did not reveal obvious evidence of asymmetry (Figure 4). Additionally, according to the result of Egger’s tests, the \( p \) values greater than 0.05 (G vs. A: \( p = .814 \); GG/AG vs. AA: \( p = .789 \); GG vs. AG/AA: \( p = .253 \); GG vs. AA: \( p = .312 \); GA vs. AA: \( p = .855 \)), which providing statistical evidence to the funnel plots’ symmetry.

### 4 | DISCUSSION

Cardiovascular disease is broadly testified as atherosclerosis, underlying vascular aberrations, as well as formation of coronary plaques (Malik et al., 2004; Strazzullo, D’Elia, Kandala, & Cappuccio, 2009; Vasan, 2006). Many studies identified that the potential influence of inflammatory cascade in many kinds of CVD. Inflammatory responses could trigger and accelerate vascular injury and CVDs risk (Lakka et al., 2002; Malik et al., 2004). Therefore, the pathogenesis of CVDs have been deemed as both genetic and inflammatory pathways, which modulated by various inflammatory cytokines. Genetic factors are regarded as strong determinants of CVDs (Brigitta, 2007; Mckusick, 1959). According to the chromosomal location and functional relevance, \( IL-10 \) is a multifunctional cytokine that has been considered as a candidate gene for kinds of CVD (Couper, Blount, & Riley, 2008; Dopheide et al., 2015; Eskdale, Wordsworth, Bowman, Field, & Gallagher, 2010; Heeschen et al., 2003a). Therefore, this meta-analysis was performed to obtain a more precise

### TABLE 3

| Variables       | Allele model | Dominant model | Recessive model |
|-----------------|--------------|----------------|-----------------|
|                 | G/A          | (GG/AG vs. AA) | (GG vs. AG/AA)  |
|                 | OR (95% CI)  | OR (95% CI)    | OR (95% CI)     |
|                 | \( p_{OR} \)| \( p_{Het} \) | \( p_{OR} \)    | \( p_{Het} \)  |
| Total           | 1.10 (1.04–1.15) | 0.000           | 0.87 (0.72–1.04) | 0.036           |
|                 |              |                 |                 |                 |
| Ethnicity       |              |                 |                 |                 |
| Asian           | 1.27 (1.17–1.38) | 0.000           | 0.73 (0.54–0.99) | 0.000           |
| Caucasian       | 1.03 (0.96–1.09) | 0.446           | 0.95 (0.76–1.20) | 0.754           |
| Mix             | 0.87 (0.72–1.05) | 0.154           | 1.29 (0.76–2.22) | 0.118           |
| Source of control |             |                 |                 |                 |
| HB              | 1.10 (0.99–1.22) | 0.063           | 0.88 (0.76–1.02) | 0.080           |
| PB              | 1.18 (1.08–1.28) | 0.000           | 0.87 (0.76–0.99) | 0.032           |

Note: \( p_{OR} \): \( p \) value from the odd ratio and obtained from Z test; \( p_{Het} \): \( p \) value from the heterogeneity and obtained from the chi-square test.

### TABLE 4

| Variables       | Homozygote (GG vs. AA) | Heterogeneity (AG vs. AA) |
|-----------------|-------------------------|---------------------------|
|                 | OR (95% CI)             | \( p_{OR} \) | \( p_{Het} \) | OR (95% CI) | \( p_{OR} \) | \( p_{Het} \) |
| Total           | 1.06 (1.03–1.10)        | 0.009 | 0.000 | 0.88 (0.73–1.06) | 0.205 | 0.000 |
| Ethnicity       |                          |                 |                 |              |                 |                 |
| Asian           | 0.81 (0.50–1.31)        | 0.000           | 0.75 (0.56–1.00) | 0.003 | 0.000 |
| Caucasian       | 0.95 (0.74–1.21)        | 0.464           | 0.97 (0.74–1.26) | 0.853 | 0.000 |
| Mix             | 1.22 (0.77–1.92)        | 0.406           | 1.29 (0.72–2.33) | 0.138 | 0.028 |
| Source of control |                        |                 |                 |              |                 |                 |
| HB              | 0.84 (0.68–1.05)        | 0.14            | 0.90 (0.77–1.04) | 0.165 | 0.000 |
| PB              | 0.82 (0.69–0.99)        | 0.039           | 0.87 (0.75–0.99) | 0.047 | 0.005 |

Note: \( p_{OR} \): \( p \) value from the odd ratio and obtained from Z test; \( p_{Het} \): \( p \) value from the heterogeneity and obtained from the chi-square test.
conclusion and to make a better understanding of IL-10 (−1082 G/A) SNP with CVDs risk.

For this meta-analysis, we aimed to identify the dispute about the role of IL-10 (−1082 G/A) in CVD risk. The results showed that there exists a significant relationship between IL-10 (−1082 G/A) and CVDs risk. This study was conducted by critically evaluating 31 individual case–control studies involving the IL-10 (−1082 G/A) polymorphism and CVDs risk. We found that the IL-10 (−1082 G/A) was associated with an increased risk of CVDs among Asians. As far as we know, this updated meta-analysis includes the largest samples and the most cogent conclusions.

In our study, we observed that the allele and genotype frequencies of IL-10 (−1082A/G) were associated with CVDs risk, which was consistent with a present study, as reported by Yang et al. (Xuan, Wang, Zhi, Li, & Wei, 2016). One meta-analysis study observed that the allele and genotype frequencies of IL-10 (−1082A/G) were not associated with ischemic stroke risk (Liu, Hui‐Min, et al., 2017). However, the powerful relationship between IL-10 (−1082A/G) and ischemic stroke has been found in the Italian population (Tuttolomondo et al., 2012a). Li et al. reported that IL-10 −1082G/A was related to increase the atherosclerotic risk (Chao, Lei, & Fei, 2014). Wang et al. observed that IL-10 (−1082A/G) polymorphism

FIGURE 2  Forest plots for the IL-10-1082A/G polymorphism and cardiovascular disease in the allele model
was significantly associated with increasing the cerebral infarction risk in Asians (Fan et al., 2016). Based on the Asian population, several studies have indicated the positive or null relationship of IL-10 (−1082A/G) with CVDs, and the results remain controversial. Although previous individual studies have reported an association, the overall result of the present analysis support the relationship between IL-10−1082G/A polymorphism and CVD risk in all genetic models. There are three potential reasons can explain the difference among these findings. First, the relatively small sample sizes of included studies might lead to false positive or negative results. Second, the inconsistent results could result from different genotype frequencies among enrolled subjects, especially in different ethnic groups. Third, due to the complicated pathogenesis of CVDs, it is difficult to explain that the SNP in a single gene could increase risk of CVD without a contribution of other polymorphic susceptibility genes.

Limitations also exist in our study. First, the study lack of detailed information in patients. Second, the studies with small sample size (<100 cases and controls) may overestimate the relationship. Third, the origins of heterogeneity may contain many factors, such as the diverse characteristics of the control group and the different methods of diverse genotyping. Finally, the human IL-10 production is regulated by the complicated interaction between gene and environment, the effect of single genetic mutation is limited. So the evidence offered by this meta-analysis should be accepted with caution. However, our study also exist some advantages: the reasonable-designed search and selection method could increase the statistical power and the accuracy of the results, at the same time, the results did not show any evidence of publication bias.

In conclusion, the results of our study indicate that the IL-10−1082A/G polymorphism is associated with an increased risk of CVDs. In subsequent studies, we will conduct functional studies to confirm our conclusions.

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CONFLICT OF INTEREST

The authors have no conflict of interest to report.

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