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Exploring the causal association between fatty acid-binding proteins and anaphylactic shock due to adverse reactions to medications

A two-sample Mendelian randomization study

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Abstract

Previous studies have identified a relationship between fatty acid-binding proteins (FABPs) and immune diseases. This study aimed to investigate whether a causal relationship exists between FABPs and anaphylactic shock resulting from adverse drug reactions. Single nucleotide polymorphisms associated with FABPs were utilized as instrumental variables, sourced from the National Human Genome Research Institute-European Bioinformatics Institute Catalog of human genome-wide association studies. Data on anaphylactic shock due to adverse effects of correctly administered drugs were obtained from the FinnGen database, which includes genomic and health data from 500,000 Finnish biobank donors. A two-sample Mendelian randomization analysis was conducted to explore the causality between FABPs and anaphylactic shock due to adverse drug reactions. The analysis revealed a negative causal relationship between FABP5 (odds ratio [OR] = 0.40; 95% confidence interval [CI] = 0.17–0.92; P = .032) and FABP12 (OR = 0.77; 95% CI = 0.63–0.94; P = .009) and anaphylactic shock due to adverse drug reactions. These findings were corroborated by Mendelian randomization-Egger, weighted median, and weighted mode methods. This study provides robust evidence supporting a protective relationship between FABP5 and FABP12 and anaphylactic shock due to adverse drug reactions. Further experimental studies are warranted to elucidate the causal mechanisms and associations between FABP5, FABP12, and anaphylactic shock in the context of adverse drug reactions.

Abbreviations: FABPs = fatty acid-binding proteins, GWAS = genome-wide association studies, IVs = instrumental variables, MR = Mendelian randomization, MR-PRESSO = MR Pleiotropy REsidual Sum and Outlier, SNPs = single nucleotide polymorphisms.

Keywords: adverse drug reactions, anaphylactic shock, causal association, fatty acid-binding proteins, mendelian randomization

1. Introduction

In recent years, with the increase in the variety and quantity of clinical medications, the incidence of drug allergic reactions has been on the rise,[1] especially the occurrence of anaphylactic shock. Anaphylactic shock is a severe systemic anaphylactic reaction triggered in a short period of time by immune mechanisms after certain external antigenic substances enter the sensitized organism, mainly caused by drugs, food, etc.[2] The occurrence of anaphylactic shock due to adverse reactions to medications is unpredictable and independent of the dosage of the medication. If not rescued in a timely manner after its occurrence, it will seriously affect the patient's health and even endanger the patient's life.[3]

Fatty acid-binding proteins (FABPs) are a class of low molecular weight polygenic proteins that function as transporters by binding to hydrophobic ligands (fatty acids) with different affinities and participate in fatty acid metabolism.[4] In humans, FABPs can be classified into 10 types based on their high expression in specific tissues (Table 1): FABP1/L-FABP (liver), FABP2/I-FABP (intestinal), FABP3/H-FABP (heart), FABP4/A-FABP (adipose), FABP5/E-FABP (epidermal), FABP6/IL-FABP (ileum), FABP7/B-FABP (brain), FABP8/M-FABP (myelin), FABP9/T-FABP (testis), and FABP12/R-FABP (retina).[5] FABPs as an important signaling molecule have been gradually emphasized in recent years. Multiple studies have confirmed the association of FABPs with diseases such as obesity and nonalcoholic liver disease (FABP1, FABP2,

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All data generated or analyzed during this study are included in this published article [and its supplementary information files].

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Supplemental Digital Content is available for this article.

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Table 1

Data sources.

Trait	Tissue site of initial discovery of exposures	Population		PMID
		Sample size case/control	Decent	
Serum levels of protein FABP1 (FABP1)	Liver	5368	European	35078996
Intestinal fatty acid-binding protein, levels (FABP2)	Intestine	1301	European	33303764
Serum levels of protein FABP3 (FABP3)	Heart, skeletal muscle, etc.	5368	European	35078996
Serum levels of protein FABP4 (FABP4)	Adipose tissue	5368	European	35078996
Serum levels of protein FABP5 (FABP5)	Epithelial tissue	5368	European	35078996
Serum levels of protein FABP6 (FABP6)	Ileum (anatomy)	5368	European	35078996
Level of FABP 9 in blood serum (FABP9)	Testicular tissue	466	American	35870639
Level of FABP 12 in blood serum (FABP12)	Testes, retina	466	American	35870639
Anaphylactic shock due to adverse effect of correct drug or medicament properly administered		132/218,660	European	FinnGen

FABP = fatty acid-binding proteins, MR = Mendelian randomization.

and FABP4),[6–8] cardiovascular disease (FABP3),[9] and cancer (FABP5 and FABP7).[10,11] In addition, Cao et al[12] found an increase in FABP2 in the serum of patients with episodes of idiopathic anaphylaxis. From this we hypothesized, is there an association between FABPs and anaphylactic shock due to adverse reactions to medications? There is currently a lack of sufficient evidence.

To gain a more detailed understanding of the potential causal relationship between FABPs and anaphylactic shock due to adverse reactions to medications, the present study employed a two-sample Mendelian randomization (MR) for an in-depth study. MR is a statistical model with genetic variation as instrumental variables (IVs), which uses single nucleotide polymorphisms (SNPs) as a genetic tool to estimate the effect of exposures on outcomes.[13] Summary data usually come from large-scale genome-wide association studies (GWAS) since many large GWAS consortia have been built to obtain public and free statistics.[14] Genetic variation has randomness, thus MR analysis can help reduce the possible confounding factors in observational studies and analyze the correlation between exposure and outcome factors from a genetic perspective to provide reliable results,[15] so that we can more accurately evaluate the causal association between FABPs and anaphylactic shock due to adverse reactions to medications.

2. Materials and methods

2.1. Data sources

SNPs associated with FABPs were obtained as IVs from the National Human Genome Research Institute-European Bioinformatics Institute Catalog of human genome-wide association studies (https://www.ebi.ac.uk/gwas/). FABP1, FABP3–6 were obtained from a large-scale proteogenomic study of 5368 individuals that revealed 4035 independent associations between genetic variants and 2091 serum proteins.[16] FABP2 was obtained from a high-depth (22.5-fold) whole-genome sequencing study of 1328 individuals that comprehensively assessed the genetic structure of 257 circulating proteins associated with cardiac metabolic.[17] FABP9 and FABP12 were obtained from a study that identified 969 protein quantitative trait loci from the Americans.[18] We integrated 8 FABPs for which serum level evidence had been found in mammals as exposures in the study. Anaphylactic shock due to adverse effect of correct drug or medicament properly administered as outcome obtained from FinnGen database (https://www.finngen.fi/en) that has collected and analyzed genome and health data from 500,000 Finnish biobank donors to understand the genetic basis of diseases.

2.2. Selection of the genetic instruments

The IVs of exposures included in our study were required to meet the following criteria: SNPs significantly associated with fatty acid-binding proteins genome-wide with criterion of $P < 5 \times 10^{-6}$; all SNPs independent (10,000 kb pairs apart, $R^2 < 0.001$) and the linkage disequilibrium effect among SNPs was excluded; when the screened IVs were not present in the summary data of the outcome, SNPs with high linkage disequilibrium ($R^2 > 0.8$) with the IVs were searched for as a proxy SNP to be replaced; and F-values were calculated for each SNP in the IVs to assess the strength of the IVs and exclude possible weak instrumental variable bias between the IVs and exposure factors, calculated as follows: $F = R^2*(N-2)/(1-R^2)$ with R^2 being the proportion of variation in the exposures explained by the SNPs in the IVs, and the requirement for the F-value being > 10.[19] The detailed flowchart of this study is shown in Figure 1.

2.3. Statistical analysis

We used two-sample MR analyses to explore the relationship between FABP and anaphylactic shock due to the adverse effect of correct drug or medicament properly administered. Since each IV of exposures contained multiple SNPs, we used 5 dominant MR methods: inverse-variance weighted (IVW) test,[20] weighted mode,[21] MR-Egger regression,[22] weighted median regression,[23] and MR Pleiotropy REsidual Sum and Outlier (MR-PRESSO).[24] Since the IVW approach yielded the most accurate results when all selected SNPs were valid IVs, it was mainly used for fundamental causal estimates.

2.4. Pleiotropy and sensitivity analysis

To assess the presence of horizontal pleiotropy we performed the MR-egger regression, whose intercept term indicates the average pleiotropic effect of IVs.[22] Moreover, the MR-PRESSO was also conducted to detect the presence of pleiotropy and potential outliers.[24] If both reported pleiotropy, outliers needed to be removed to determine whether there was a substantial difference before and after the causal effect. Not only that, we used Cochran's Q statistic to quantify the heterogeneity present in the IVW analysis[25] and "leave-one-out" analysis to test the robustness and consistency of the results.[26] Q statistics significant at a P value < 0.05 can imply the presence of heterogeneity.

All analyses were carried out using the TwoSampleMR package[27] and MR-PRESSO package[24] in R Software 3.6.1. A multiple-testing threshold of $P < .00625$ (0.05/8*1) was adopted to declare a statistical significance using the Bonferroni method.

3. Results

3.1. Included instrumental variables

We selected 14,15,11,11,12,12,34 and 18 SNPs associated with FABPs at FABP1-6, FABP9, and FABP12, respectively, at a significance level of $P < 5 \times 10^{-6}$. The F-statistics of these IVs are all greater than 10, which indicates that there is no weak instrumental bias (Table S1, Supplemental Digital Content, https://links.lww.com/MD/O747). Individual SNPs with echo structure were excluded from the analyses after being harmonized with outcome summary data. A total of 119 SNPs were finally included in the MR analysis.

3.2. Causal effects of fatty acid-binding proteins on anaphylactic shock due to adverse effect of correct drug or medicament properly administered

The MR estimates of different methods were presented in Table S2, Supplemental Digital Content, https://links.lww.com/MD/O748. We found a negative causal relationship between FABP5 (odds ratio [OR] = 0.40; 95% confidence interval [CI] = 0.17–0.92; $P = .032$) and FABP12 (OR = 0.77; 95% CI = 0.63–0.94; $P = .009$) and anaphylactic shock due to adverse effect of correct drug or medicament properly administered (Fig. 2). However, similar relationships were not significant between other FABPs and outcome (Table S2, Supplemental Digital Content, https://links.lww.com/MD/O748). In addition, the MR-Egger, the weighted median, and the weighted mode methods also argued these results. The scatter plot for effect sizes of SNPs for FABP5 and FABP12 on anaphylactic shock due to adverse effect of the correct drug or medicament properly administered were shown in Figure 3 and Figure S1, Supplemental Digital Content, https://links.lww.com/MD/O749.

3.3. Pleiotropy and sensitivity analysis

According to the results of MR-Egger and global test of MR-PRESSO, there was no horizontal pleiotropy in the study (Table 2), while MR-PRESSO did not report outliers, as can be seen in the funnel plot (Figure S3, Supplemental Digital Content, https://links.lww.com/MD/O750). Furthermore, there was no heterogeneity between the individual SNP according to the heterogeneity test. Leave-one-out analysis indicated that the causal estimates of FABP5 and FABP12 on anaphylactic shock due to the adverse effect of correct drug or medicament properly administered were not driven by any single SNP (Table 3). Based on the results of sensitivity analysis, we still believe that there may be a causal association between FABP5 and FABP12 on anaphylactic shock due to the adverse effect of correct drug or medicament properly administered. However, it is worth noting that the P value for FABP5 and anaphylactic shock due to the adverse effect of correct drug or medicament properly administered was not significant after Bonferroni correction, but a similar association was significant in FABP12, so the relationship should be interpreted with caution. The forest plots and leave-one-out analysis plots are shown in Figure S2 and Figure S4, Supplemental Digital Content, https://links.lww.com/MD/O751.

4. Discussion

In this study, we used summary data from similar ancestry but 2 different population to explore the potential causal relationship between 8 types of FABPs and anaphylactic shock due to adverse reactions to medications by classical MR. The results of the study showed a negative causal relationship between FABP5 and FABP12 and anaphylactic shock due to adverse reactions to medications, that is, the higher the values of FABP5 and FABP12, the lower the risk of anaphylactic shock due to adverse

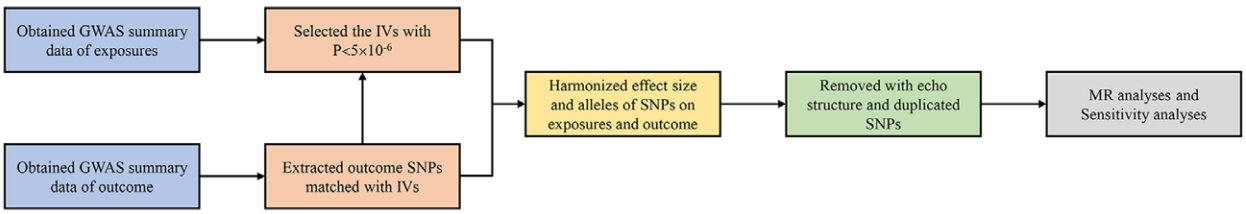

Figure 1. The schematic diagram of the study design. GWAS = genome-wide association studies, IVs = instrumental variables, MR = Mendelian randomization, SNPs = single nucleotide polymorphisms.

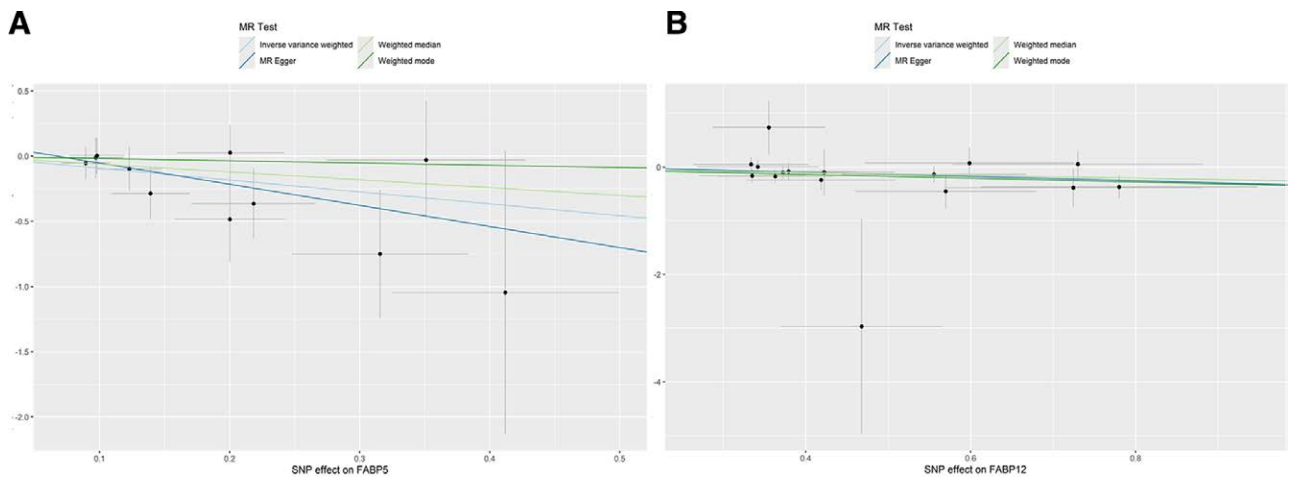

Figure 2. Forest of the causal relationship between FABP5 and FABP12 and anaphylactic shock due to the adverse effect of correct drug or medicament properly administered. MR = Mendelian randomization, SNPs = single nucleotide polymorphisms.

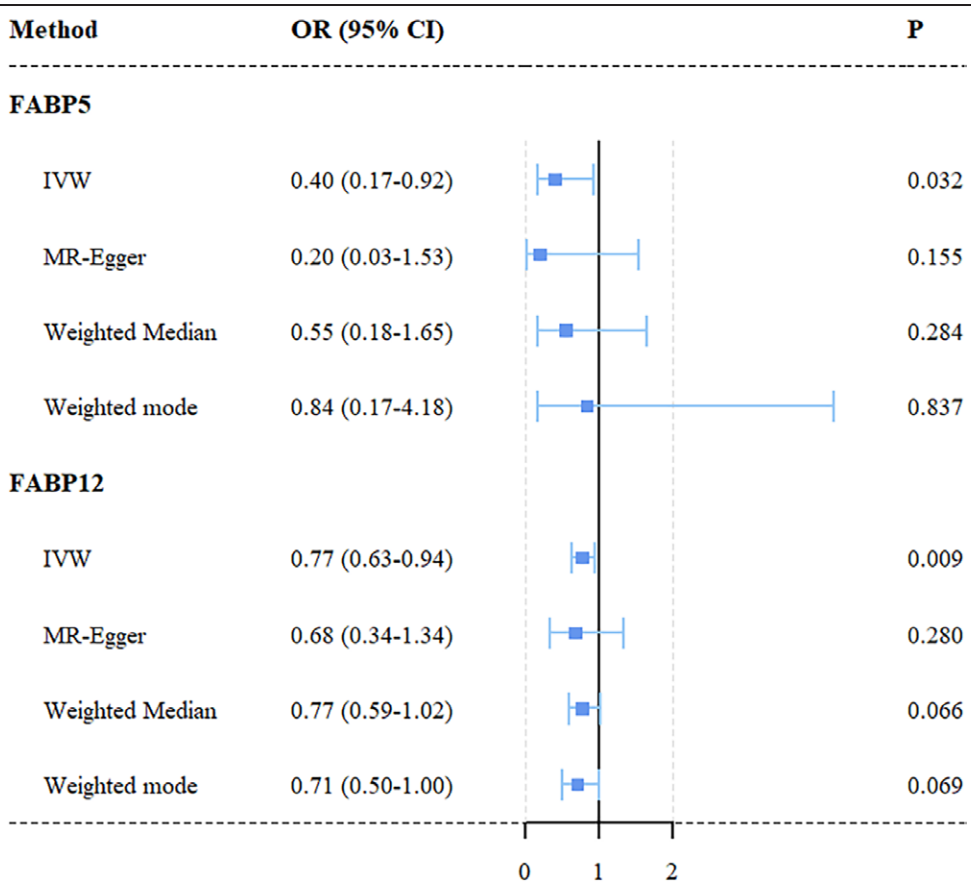

Figure 3. Scatter plot for effect sizes of SNPs for FABP5 and FABP12 on anaphylactic shock due to adverse effect of correct drug or medicament properly administered. FABP = fatty acid-binding proteins, IVW = inverse-variance weighted, MR = Mendelian randomization.

Table 2

Pleiotropy test using MR-Egger and MR-PRESSO.

		MR-Egger		MR-PRESSO				
Outcome	**Exposure**	**Intercept**	***P***	**Raw OR (CI%)**	**Raw *P***	**Global *P***	**Number of outliers**	**Distortion *P***
Anaphylactic shock due to adverse effect of correct drug or medicament properly administered	FABP5	0.110	.475	0.40 (0.22–0.74)	.01	.858	NA	NA
	FABP2	0.025	.733	0.88 (0.75–1.04)	.16	.826	NA	NA
	FABP3	–0.015	.925	0.78 (0.38–1.61)	.52	.807	NA	NA
	FABP4	–0.157	.251	0.40 (0.16–0.96)	.07	.501	NA	NA
	FABP12	0.059	.707	0.77 (0.65–0.91)	.01	.794	NA	NA
	FABP1	–0.043	.705	0.84 (0.57–1.24)	.4	.961	NA	NA
	FABP6	0.072	.735	0.95 (0.31–2.88)	.93	.063	NA	NA
	FABP9	–0.025	.785	0.89 (0.79–1.00)	.06	.211	NA	NA

FABP = fatty acid-binding proteins, MR = Mendelian randomization, NA = not applicable.

reactions to medications. The relative stability of the research results was confirmed through sensitivity analysis. These results were not significantly affected by a single SNP, enhancing the reliability of this study.

FABP5 is most abundantly expressed in epidermal cells of the skin, responsible for the transport of long-chain fatty acids in cells. FABP5 bound to specific ligands can also undergo nuclear transfer, thereby regulating gene transcription.[28] A study conducted by Hou et al[29] found that FABP5 can selectively regulate the accumulation of long-chain unsaturated fatty acids in macrophages (M2), reprogramming lipid metabolism and regulating M2 polarization, which in turn modulates airway allergic inflammation. The study revealed the molecular mechanism by which FABP5 regulates macrophage alternative activation and allergic asthma for the first time, which provides a reference for the prevention and treatment of allergic asthma.

FABP12, as a novel protein, has multiple functions in organisms. FABP12 is a good drug-targeting protein, and its expression is influenced by regulation under a variety of physiological and pathological conditions, such as cell cycle, metabolism, and activation.[30] Furthermore, FABP12 also has various characteristics that respond to drug molecules, such as high potential binding ability in drug-protein complexes, and can bind to various drug molecules, such as chemotherapy drugs, anti-inflammatory drugs, and immunosuppressive.[31] By studying the function and mechanism of action of FABP12 in organisms, we can provide a more adequate theoretical basis for drug research and clinical applications.

Table 3

Heterogeneity test of MR-Egger and inverse-variance weighted.

Outcome	Exposure	Method	Q	Q_df	P
Anaphylactic shock due to adverse effect of correct drug or medicament properly administered	FABP5	MR-Egger	4.719	9	.858
	FABP5	Inverse-variance weighted	5.274	10	.872
	FABP2	MR-Egger	8.958	12	.707
	FABP2	Inverse-variance weighted	9.080	13	.767
	FABP3	MR-Egger	6.306	9	.709
	FABP3	Inverse-variance weighted	6.316	10	.788
	FABP4	MR-Egger	6.630	8	.577
	FABP4	Inverse-variance weighted	8.161	9	.518
	FABP12	MR-Egger	10.938	14	.691
	FABP12	Inverse-variance weighted	11.085	15	.747
	FABP1	MR-Egger	5.916	12	.920
	FABP1	Inverse-variance weighted	6.067	13	.944
	FABP6	MR-Egger	18.699	10	.044
	FABP6	Inverse-variance weighted	18.926	11	.062
	FABP9	MR-Egger	35.533	29	.188
	FABP9	Inverse-variance weighted	35.626	30	.221

FABP = fatty acid-binding proteins, MR = Mendelian randomization.

This study has several advantages. To the best of our knowledge, this is the first study to explore the causal relationship between FABPs and anaphylactic shock due to adverse reactions to medications. In contrast to observational studies, MR studies cleverly avoid traditional confounding factors and issues related to reverse causation. Furthermore, the use of large-scale GWAS data provides powerful and reliable IVs and confirms causality inference through sensitivity analyses. However, this study also has several limitations. First, the sample in this study comes from a European population, and it is unclear whether it is applicable to other ethnic groups. Second, MR did not consider the interaction between genes and the environment. Third, this study is an exploratory analysis, and the role of FABP5 and FABP12 in the occurrence and development of anaphylactic shock due to adverse reactions to medications needs to be further confirmed by prospective studies and basic experiments.

5. Conclusions

We reported robust evidence to support a protective relationship between FABP5 and FABP12 and anaphylactic shock due to adverse reactions to medications. Experimental studies are also required to further examine causality and mechanisms for FABP5 and FABP12 and anaphylactic shock due to adverse reactions to medications.

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Writing – review & editing: Yu Zhang, Guoping Peng.
Investigation: Guoping Peng.
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