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The Causal Effect of BMI on Hypertension: A Copula Model Approach with Genetic Risk Instruments

Robinson Dettoni
Universidad de Santiago de Chile

Petri Böckerman
University of Jyväskylä
and IZA@LISER

Cliff Bahamondes
Universidad de Santiago de Chile

Jose Vasquez
Universidad de Santiago de Chile

Carlos Yévenes
Universidad de Santiago de Chile

Olli Raitakari
University of Turku
and Turku University Hospital

Jutta Viinikainen
University of Jyväskylä

Terho Lehtimäki
Tampere University
and Pirkanmaa Hospital District

Jaakko Pehkonen
University of Jyväskylä

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Abstract

This paper estimates the effect of body mass index (BMI) on hypertension risk using data from the Young Finns Study. The empirical framework combines genetic instruments for body mass with a triangular copula model for a binary outcome and a continuous endogenous treatment. The model allows the unobserved determinants of BMI and hypertension to be dependent through alternative symmetric and asymmetric copula specifications. Identification is interpreted within a maintained parametric triangular copula model, while sensitivity analyses examine alternative copula families and nested BMI genetic-score constructions designed to address specific prior pleiotropy concerns. The results indicate that higher BMI increases hypertension risk across ordered blood-pressure severity categories. Probability-scale treatment effects reveal a nonlinear pattern, with marginal risk concentrated in clinically relevant regions of the BMI distribution and shifting from early blood-pressure elevation toward more severe hypertension as body mass increases. The findings show how genetic instruments and copula-based triangular models can be combined to study endogenous continuous treatments in nonlinear health outcomes, while also highlighting where along the BMI distribution marginal increases in body mass are most consequential for blood-pressure risk.

JEL classification

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Keywords

body mass index, hypertension, genetic instruments, copula models, endogeneity, mendelian randomization, latent dependence

Corresponding author

Robinson Dettoni
robinson.dettoni@usach.cl

1 Introduction

Obesity is a major public-health challenge worldwide. Its prevalence has risen sharply over recent decades, nearly tripling since 1975 [1]. In Europe, approximately one half of adults are overweight or obese, and one fifth meet the clinical threshold for obesity [2]. Finland has followed a similar trajectory [3]. Beyond its epidemiological significance, obesity imposes substantial economic costs through greater healthcare utilization, medication use, and productivity losses [4, 5, 6, 7, 8, 9, 10].

A central consequence of excess body weight is elevated blood pressure. Obesity is closely linked to hypertension through renal, vascular, metabolic, inflammatory, and neurohormonal mechanisms [11, 12, 13, 14, 15]. Hypertension, in turn, is a leading modifiable risk factor for cardiovascular disease and premature mortality [16, 17]. In Finland, body mass index (BMI) is an important predictor of hypertension [18, 19], and hypertension-related medical costs increase with body weight and metabolic risk [20, 21, 4]. Establishing whether BMI causally increases hypertension risk is therefore relevant for both clinical prevention and health-policy evaluation.

Although the association between BMI and hypertension is well documented, its causal interpretation remains difficult. BMI is not randomly assigned. It reflects genetic predisposition, early-life conditions, diet, physical activity, socioeconomic status, psychological stress, and other latent behavioral or physiological factors that may also affect blood pressure [e.g., 22, 23, 24, 25, 26]. These unobserved determinants can generate endogeneity in the BMI–hypertension relationship. Standard single-equation binary-response models may therefore confound the structural effect of BMI with latent selection into BMI. Linear instrumental-variable methods address endogeneity under strong linearity and additive-error restrictions, but they are not naturally suited to a binary hypertension outcome with a continuous endogenous treatment and potentially nonlinear dependence between unobservables.

This paper estimates the causal effect of BMI on hypertension in a triangular econometric framework with a binary outcome, a continuous endogenous treatment, and genetic instruments. Hypertension is modeled through a latent-index equation, while BMI is specified as a function of observed covariates and genetic risk scores. Endogeneity arises through dependence between the unobserved determinants of BMI and hypertension. Rather than imposing a single benchmark structure on that dependence, the paper adopts a parametric copula framework that separates the specification of the marginal distributions from the specification of their joint dependence. This allows the empirical analysis to estimate the model under a range of alternative dependence specifications and to assess the robustness of the estimated BMI effect to different assumptions about latent dependence [27, 28, 29, 30, 31, 32, 33].

Identification in the empirical analysis is based on BMI genetic risk scores used as excluded sources of variation in the triangular model. Starting from the 32 BMI-associated loci identified by Speliotes et al. [34], the paper constructs three nested BMI genetic scores. The nested scores exclude variants with specific prior concerns about blood-pressure-related or broader cardiometabolic pleiotropy, using prior evidence on blood-pressure and cardiometabolic pathways to screen specific loci [34, 35, 36, 37]. This instrument design is used as a transparent Mendelian-randomization sensitivity exercise: it examines whether the estimated BMI effect changes when variants with stronger prior pleiotropy concerns are removed, but it does not validate the exclusion restriction

[38, 39, 40]. The identifying claims are correspondingly limited. The triangular copula model is not presented as a globally nonparametric identification result. Rather, identification is local and model-based, resting on instrument exclusion and relevance, latent-scale normalization, common support, regularity, and a local rank condition [41, 42, 43, 44, 45, 46, 47, 48, 49, 50, 51].

The contribution of the paper is primarily empirical. It combines a triangular model for a binary outcome with a continuous endogenous treatment, a transparent genetic-instrument strategy based on nested BMI genetic scores, and probability-scale treatment-effect estimates for severity-specific hypertension outcomes. A Monte Carlo exercise complements the identification discussion by studying finite-sample recovery of the structural effect under controlled triangular designs. It is used as diagnostic numerical evidence on estimator performance, not as a replacement for full theoretical identification analysis.

Using data from the Young Finns Study, the paper finds evidence consistent with a positive model-based effect of BMI on hypertension risk. The estimated latent-dependence structure is specification-sensitive, with negative dependence appearing in several rotated-copula and Gaussian specifications. The genetic risk scores shift BMI in the expected direction, and the estimated BMI effect remains present under the main dependence structures used for sensitivity analysis. Translating the latent-index estimates into probability-scale treatment effects shows nonlinear effects that average roughly 2%–3% and are concentrated in clinically relevant regions of the BMI distribution. The profiles further indicate a severity gradient: early blood-pressure elevation is concentrated at lower and intermediate BMI values, whereas more severe hypertension risk becomes more relevant at higher BMI values.

These probability-scale results are relevant for health economics because they identify where along the BMI distribution marginal increases in body mass translate into the largest changes in hypertension risk. This matters for prevention, risk stratification, and the economic evaluation of interventions that shift BMI before severe hypertension becomes established. The main empirical message is therefore not only that BMI raises model-implied hypertension risk, but also that the marginal risk associated with BMI varies across both body-mass levels and blood-pressure severity categories.

The remainder of the paper is organized as follows. Section 2 presents the econometric framework, the identifying assumptions, the maximum likelihood estimator, and the construction of the genetic instruments. Section 3 reports the Monte Carlo evidence. Section 4 describes the Young Finns Study data and the variables used in the empirical analysis. Section 5 presents the empirical results. Section 6 discusses the findings, limitations, and implications.

2 Methodology

This section presents the econometric framework used to estimate the causal effect of body mass index on hypertension. The empirical strategy combines a triangular model with a binary outcome and a continuous endogenous treatment, a parametric copula representation of latent dependence, and genetic risk scores as excluded sources of variation in body mass index. Section 2.1 sets out the model, the maintained identifying conditions, and the estimation and inference framework used in the empirical analysis. Section 2.2 describes the construction of the genetic instruments.

2.1 Econometric Model

Let $W_i = (Y_i, T_i, X_i, Z_i)$ denote an i.i.d. observation, where Y_i is a binary indicator for hypertension, T_i is body mass index, X_i is a vector of observed covariates, and Z_i denotes the excluded genetic instrument. In the empirical implementation, one genetic risk score is used at a time. The instrument enters the treatment equation and is excluded from the structural hypertension equation.

The empirical framework is a triangular model with a binary outcome and a continuous endogenous treatment:

$$\begin{aligned} Y_i &= \mathbf{1}\{\gamma T_i + X_i'\beta + \varepsilon_{Yi} \geq 0\}, \\ T_i &= X_i'\alpha + Z_i'\pi + \varepsilon_{Ti}. \end{aligned} \tag{1}$$

For a fixed treatment level t, the corresponding potential outcome is obtained by replacing T_i with t in the hypertension equation. The central structural object is therefore the response probability

$$p(t, x) = \Pr(Y_i(t) = 1 \mid X_i = x). \tag{2}$$

Under the probit normalization used in the baseline specification, $p(t, x) = \Phi(\gamma t + x'\beta)$. In that specification, γ measures the effect of body mass index on the normalized latent propensity for hypertension, while $p(t, x)$ is the probability-scale object used for causal interpretation.

Endogeneity arises when the unobserved determinants of body mass index and hypertension are statistically dependent. In that case, a single-equation binary-response model will generally confound the structural effect of body mass index with latent selection into treatment. To accommodate this possibility, let F_Y and F_T denote the marginal distributions of the latent disturbances ε_{Yi} and ε_{Ti}, respectively. Their joint distribution is specified through a parametric copula C_θ such that

$$F_{\varepsilon_Y, \varepsilon_T}(e_Y, e_T) = C_\theta\{F_Y(e_Y), F_T(e_T)\},$$

where θ governs the strength and sign of latent dependence. Under the probit–Normal specification used in the empirical analysis, the binary-outcome margin is normalized as $F_Y = \Phi$, while the BMI margin is specified as Gaussian. The Gaussian copula provides the recursive bivariate normal benchmark, while Clayton, Gumbel, and their rotated versions allow alternative asymmetric and negative dependence patterns [27, 28, 52, 48, 49].

The identifying claims of the paper are intentionally limited. The model is not presented as delivering unrestricted global nonparametric identification. Rather, the empirical interpretation rests on maintained conditions under which identification can be understood in a local, model-based sense. These conditions are the following: the excluded genetic instrument must satisfy exclusion and relevance; the latent scale of the binary outcome must be normalized; the copula family must be treated as known up to a scalar dependence parameter and satisfy standard regularity conditions; and causal interpretation must be restricted to treatment values supported by the data and by the instrument-induced variation in body mass index. In addition, the parametric mapping from the model primitives to the observable distribution must satisfy a local rank condition [44, 45, 41, 42, 43, 46, 47, 51].

These conditions are standard in the literature on local identification in parametric and semiparametric models, triangular models with continuous endogenous treatments, and copula-based models with endogenous regressors. The purpose of this section is not to reproduce those formal arguments in full, but to state clearly the assumptions under which the empirical analysis is interpreted [41, 42, 43, 44, 45, 46, 47, 48, 49, 50].

Under these maintained conditions, the model supports a local and on-support interpretation of γ and, correspondingly, of the structural response probability $p(t, x)$. The empirical analysis therefore distinguishes three related objects: the coefficient on body mass index in the normalized latent hypertension equation, the structural probability of hypertension at treatment level t, and probability-scale contrasts constructed from that structural probability. The results reported below are interpreted within this parametric triangular framework and under the maintained validity of the excluded genetic instruments, the binary-outcome normalization, the copula specification, and the available support in body mass index.

Estimation follows from the joint triangular model. The parameters are estimated by full-information maximum likelihood using the generalized joint regression framework implemented in `GJRM` [53, 31]. The empirical specification uses a probit margin for the hypertension equation and a Gaussian margin for the BMI equation. Let $\eta_{Yi} = \gamma T_i + X_i'\beta$ and $\eta_{Ti} = X_i'\alpha + Z_i'\pi$. The likelihood combines the density of BMI with the copula-implied conditional probability of the observed binary outcome, thereby estimating the outcome equation, treatment equation, treatment margin, and latent-dependence parameter jointly. Alternative copula families are used as sensitivity specifications for latent residual dependence. When reported, the dependence parameter is expressed as Kendall's τ, which summarizes dependence between the latent disturbances of the BMI and hypertension equations, not the observed association between BMI and hypertension.

The main probability-scale estimand is the Sample Average Treatment Effect (SATE). For two BMI levels t_1 and t_0, chosen within the empirical support of BMI, the SATE is the sample average difference between fitted structural probabilities evaluated at those two treatment levels, holding observed covariates fixed:

$$\widehat{\Delta}_{SATE}(t_1, t_0) = \frac{1}{n}\sum_{i=1}^{n}\left[\Phi\left(\widehat{\gamma}_{BMI} t_1 + X_i'\widehat{\beta}\right) - \Phi\left(\widehat{\gamma}_{BMI} t_0 + X_i'\widehat{\beta}\right)\right].$$

Thus, the SATE translates the latent-index BMI effect into an average change in predicted hypertension risk on the probability scale. Confidence intervals for the SATE are obtained by simulation from the asymptotic Gaussian approximation to the maximum likelihood estimator, using the observed information matrix, in line with the inferential strategy used in `GJRM` [31]. All treatment-effect estimates are interpreted within the maintained triangular copula model and conditional on the instrument, margin, normalization, support, and copula assumptions.

2.2 Genetic Risk Scores as Instruments for Body Mass Index

The excluded variation in the triangular model is provided by BMI genetic risk scores. The instrumental-variable logic follows the Mendelian-randomization framework: because genetic variants are assigned at conception, they can provide predetermined sources of variation in BMI, provided that they are relevant for BMI, conditionally independent of unobserved determinants of hypertension, and excluded from the hypertension equation except through BMI [38, 39, 40].

In the present paper, these are maintained assumptions rather than testable implications of the econometric model.

The starting point is the set of 32 BMI-associated loci identified by Speliotes et al. [34]. From this set, the empirical analysis constructs three nested BMI genetic risk scores: the baseline score, denoted `GRSBMI32`; a blood-pressure-clean score, denoted `GRSBMI31`; and a stricter cardiometabolic-clean score, denoted `GRSBMI29`. The `GRSBMI31` score excludes the locus with the clearest prior evidence of blood-pressure-related pleiotropy, while `GRSBMI29` further excludes loci with stronger prior links to broader cardiometabolic pathways, including lipid traits, glucose metabolism, and insulin response [36, 37]. This nested design is used as a sensitivity exercise: it does not validate the exclusion restriction, but examines whether the estimated BMI effect changes when variants with stronger prior pleiotropy concerns are removed.

Formally, for individual i, each score is constructed by aggregating BMI-increasing alleles across a prespecified SNP set. In the weighted specification used here, the score is formed as a weighted sum of allele dosages, with weights taken from external genome-wide association estimates [34, 35]. The empirical analysis compares `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` one at a time, holding fixed the general instrument strategy while varying the exclusion of loci with specific prior pleiotropy concerns. Appendix Table A1 reports the full SNP composition of the three scores, together with the variants removed in each cleaning step and the rationale for their exclusion.

This instrument design plays a limited but important role in the paper. It supplies excluded variation for the BMI equation within the triangular model and provides a transparent sensitivity exercise for specific pleiotropy concerns. At the same time, excluding selected variants does not eliminate pleiotropy by construction, nor does it establish the validity of the exclusion restriction. Stability across `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` is therefore interpreted as sensitivity evidence, not as proof of instrument validity.

3 Simulation study

This section reports a Monte Carlo exercise designed to evaluate the finite-sample performance of the triangular copula estimator used in the empirical analysis. The objective is not to prove identification formally, but to assess whether the full-information maximum likelihood estimator can recover the structural coefficient γ under controlled data-generating processes that reproduce the main features of the empirical model.

The simulation follows a triangular binary-continuous design with one observed covariate and one excluded instrument. In each replication, the covariate is drawn from a standard normal distribution, while the excluded instrument is drawn from a normal distribution with reduced variance, corresponding to a weak-support instrument design. The true structural coefficient is fixed at $\gamma_0 = 2.0$. The latent outcome equation uses an intercept of -0.5 and a covariate coefficient of 0.5, while the treatment equation uses an intercept of 0.5 and a covariate coefficient of 0.7. Instrument relevance is varied through the first-stage coefficient, taking values 1.0, 0.5, and 0.2, which represent strong, medium, and weak instruments, respectively. The sample sizes are 500, 1000, and 1500, and each design is based on 500 replications. The full set of simulation parameters is reported in Appendix A.2.

Two dependence designs are considered. The first uses a standard Clayton copula, denoted `C0`, with Kendall's $\tau_0 = 0.10$, representing positive latent dependence between the unobserved components of the treatment and outcome equations. The second uses a 270-degree rotated Clayton copula, denoted `C270`, with Kendall's $\tau_0 = -0.10$, representing negative cross-tail latent dependence. This second design is particularly relevant for the empirical application, where several specifications suggest negative residual association between the BMI and hypertension equations.

The copula estimator is compared with two benchmark estimators. The first is a classical control-function probit estimator, which addresses endogeneity in a nonlinear binary outcome model but imposes a more restrictive residual-dependence structure. The second is a linear IV estimator, included only as a reference because it does not model the latent threshold structure of the binary outcome. The copula-based models are estimated using a probit margin for the binary outcome and a Gaussian margin for the continuous treatment. In addition to the correctly specified copula in each design, the simulations estimate alternative copula families used in the empirical analysis: the Gaussian copula `N`, the standard Clayton and Gumbel copulas `C0` and `G0`, and the rotated Clayton and Gumbel copulas `C90`, `C270`, `G90`, and `G270`. The labels `C90` and `C270` denote 90- and 270-degree rotations of the Clayton copula, while `G90` and `G270` denote the corresponding rotations of the Gumbel copula. These rotated copulas allow negative cross-tail dependence.

Tables 1 and 2 report the Monte Carlo mean and RMSE of $\widehat{\gamma}$, together with the mean and RMSE of the estimated Kendall's $\widehat{\tau}$. The performance of each estimator is evaluated relative to the known data-generating process. The main criterion is whether $\widehat{\gamma}$ approaches the true value $\gamma_0 = 2.0$ as the sample size increases and as the instrument becomes stronger. The dependence estimates are used to assess whether the copula likelihood recovers the correct sign and approximate magnitude of the latent dependence.

The results show a clear pattern. Under the Clayton `C0` data-generating process, the correctly specified `C0` estimator recovers the structural coefficient closely and estimates Kendall's τ around its true positive value. Under the rotated Clayton `C270` data-generating process, the `C270` estimator provides the closest recovery of γ_0 and correctly captures the negative latent dependence. Misspecified copulas can still recover γ_0 reasonably well in some designs, but they tend to perform worse in terms of RMSE and may fail to recover the correct sign or magnitude of the dependence parameter.

Estimator performance improves as the sample size increases and as the first-stage coefficient becomes larger. Larger values of n reduce sampling variability, while stronger instruments provide more informative variation in the endogenous treatment equation. Conversely, weak-instrument designs generate less stable estimates, especially for benchmark estimators that do not jointly model the nonlinear binary outcome and the latent dependence structure. The Monte Carlo evidence therefore supports the empirical use of copula-based triangular models, while keeping the distinction between simulation-based estimator performance and formal identification.

Table 1: Monte Carlo estimator comparison under `C0-DGP` with continuous instrument Z

		$n = 500$			$n = 1000$			$n = 1500$		
Estimator	**Statistic**	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$
Panel A: Copula-based estimators										
`G0`	$\widehat{\gamma}$	1.9772	1.9814	2.0265	2.0583	2.0347	2.0383	2.0775	2.0795	2.0498
	$RMSE_{\gamma}$	0.6257	0.4525	0.3224	0.4178	0.2986	0.2384	0.3391	0.2286	0.1957
	$\widehat{\tau}$	0.1118	0.1185	0.1041	0.0698	0.0858	0.0936	0.0628	0.0637	0.0809
	$RMSE_{\tau}$	0.2080	0.1665	0.1179	0.1539	0.1196	0.0945	0.1328	0.0979	0.0837
`G90`	$\widehat{\gamma}$	2.2300	2.2390	2.2690	2.1990	2.2150	2.3400	2.2500	2.2510	2.2670
	$RMSE_{\gamma}$	0.2620	0.2840	0.3510	0.2350	0.2660	0.3230	0.2820	0.2960	0.3500
	$\widehat{\tau}$	-0.0470	-0.0610	-0.0890	-0.0140	-0.0190	-0.0320	-0.1100	-0.1360	-0.1640
	$RMSE_{\tau}$	0.1660	0.1920	0.2380	0.1180	0.1250	0.1470	0.2610	0.2960	0.3400
`G270`	$\widehat{\gamma}$	2.2150	2.2300	2.2700	2.1950	2.2110	2.2370	2.2400	2.2460	2.2900
	$RMSE_{\gamma}$	0.2490	0.2780	0.3580	0.2320	0.2610	0.3270	0.2730	0.2940	0.3660
	$\widehat{\tau}$	-0.0130	-0.0220	-0.0460	-0.0060	-0.0080	-0.0240	-0.0240	-0.0300	-0.0650
	$RMSE_{\tau}$	0.1170	0.1330	0.1770	0.1070	0.1100	0.1370	0.1370	0.1480	0.2090
`C0`	$\widehat{\gamma}$	1.9597	1.9951	1.9944	1.9629	1.9830	2.0112	1.9882	1.9835	2.0026
	$RMSE_{\gamma}$	0.3952	0.3617	0.3068	0.3044	0.2426	0.2123	0.2605	0.2165	0.1772
	$\widehat{\tau}$	0.1301	0.1140	0.1162	0.1163	0.1080	0.1045	0.1055	0.1066	0.1033
	$RMSE_{\tau}$	0.1364	0.1163	0.1049	0.1082	0.0901	0.0770	0.0951	0.0824	0.0657
`C90`	$\widehat{\gamma}$	2.3087	2.2809	2.2421	2.2561	2.2358	2.2138	2.2482	2.2201	2.1978
	$RMSE_{\gamma}$	0.3823	0.3665	0.3315	0.3035	0.2827	0.2643	0.2825	0.2531	0.2346
	$\widehat{\tau}$	-0.0503	-0.0421	-0.0234	-0.0279	-0.0217	-0.0084	-0.0235	-0.0128	-0.0066
	$RMSE_{\tau}$	0.1731	0.1642	0.1338	0.1382	0.1293	0.1106	0.1305	0.1159	0.1080
`C270`	$\widehat{\gamma}$	2.2905	2.2696	2.2349	2.2584	2.2375	2.2168	2.2539	2.2298	2.2006
	$RMSE_{\gamma}$	0.3607	0.3504	0.3239	0.2987	0.2820	0.2662	0.2836	0.2615	0.2368
	$\widehat{\tau}$	-0.1163	-0.0745	-0.0338	-0.0868	-0.0548	-0.0195	-0.0745	-0.0425	-0.0151
	$RMSE_{\tau}$	0.2670	0.2134	0.1504	0.2229	0.1802	0.1270	0.2042	0.1592	0.1200
`N`	$\widehat{\gamma}$	1.5374	1.9188	2.0041	1.7022	1.9358	2.0131	1.7381	1.9621	2.0156
	$RMSE_{\gamma}$	1.0368	0.5493	0.3594	0.8320	0.4120	0.2605	0.7497	0.3494	0.2055
	$\widehat{\tau}$	0.0061	0.0807	0.0981	0.0561	0.1021	0.1019	0.0884	0.0969	0.0980
	$RMSE_{\tau}$	0.5414	0.2750	0.1620	0.4178	0.1955	0.1136	0.3638	0.1701	0.0945
Panel B: Benchmark estimators										
IV	$\widehat{\gamma}$	-0.0516	0.2580	0.2516	0.2855	0.2529	0.2519	0.2563	0.2553	0.2513
	$RMSE_{\gamma}$	4.7052	1.7438	1.7488	1.7907	1.7479	1.7483	1.7497	1.7453	1.7488
CF	$\widehat{\gamma}$	0.0832	2.1073	2.0880	2.0979	2.0419	2.0675	2.0320	2.0483	2.0596
	$RMSE_{\gamma}$	27.0988	0.5777	0.3407	2.5843	0.3657	0.2337	1.0412	0.3095	0.1845

Notes: The table reports Monte Carlo means and root mean squared errors under a `C0` copula data-generating process with positive latent dependence. The true structural coefficient is $\gamma_0 = 2.0$, and the true Kendall's dependence parameter is $\tau_0 = 0.10$. The first-stage coefficient π indexes instrument relevance. Copula-based estimators are implemented using `GJRM`. IV denotes the linear instrumental-variables benchmark, and CF denotes the control-function probit benchmark.

Table 2: Monte Carlo estimator comparison under `C270-DGP` with continuous instrument

		$n = 500$			$n = 1000$			$n = 1500$		
Estimator	**Statistic**	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$	$\pi = 0.2$	$\pi = 0.5$	$\pi = 1.0$
Panel A: Copula-based estimators										
`G0`	$\widehat{\gamma}$	1.7727	1.8393	1.9301	1.8412	1.8872	1.9136	1.8573	1.9028	1.9159
	$RMSE_\gamma$	0.4582	0.3314	0.2190	0.2507	0.1932	0.1706	0.2196	0.1611	0.1420
	$\widehat{\tau}$	0.0810	0.0501	0.0235	0.0382	0.0229	0.0110	0.0300	0.0153	0.0066
	$RMSE_\tau$	0.2353	0.1822	0.1344	0.1576	0.1342	0.1156	0.1471	0.1219	0.1088
`G90`	$\widehat{\gamma}$	2.0160	2.0090	2.0100	2.0010	2.0030	2.0390	2.0010	1.9950	2.0100
	$RMSE_\gamma$	0.1170	0.1310	0.1950	0.1140	0.1390	0.1950	0.1070	0.1350	0.1900
	$\widehat{\tau}$	-0.1240	-0.1300	-0.1430	-0.1090	-0.1070	-0.1310	-0.1410	-0.1450	-0.1690
	$RMSE_\tau$	0.1010	0.1220	0.1570	0.0730	0.0800	0.1230	0.1310	0.1490	0.1970
`G270`	$\widehat{\gamma}$	1.9480	1.9580	1.9700	1.9620	1.9700	2.0180	1.9230	1.9320	1.9770
	$RMSE_\gamma$	0.1310	0.1510	0.2090	0.1330	0.1530	0.2040	0.1330	0.1570	0.2010
	$\widehat{\tau}$	-0.0200	-0.0400	-0.0570	-0.0370	-0.0430	-0.0700	-0.0140	-0.0270	-0.0580
	$RMSE_\tau$	0.1000	0.1130	0.1340	0.0870	0.0890	0.1140	0.1070	0.1130	0.1390
`C0`	$\widehat{\gamma}$	1.6554	1.7424	1.8957	1.6703	1.8098	1.8949	1.7161	1.8142	1.9009
	$RMSE_\gamma$	0.5499	0.4167	0.2596	0.4590	0.2970	0.1882	0.3832	0.2690	0.1604
	$\widehat{\tau}$	0.1267	0.0911	0.0395	0.1104	0.0579	0.0206	0.0920	0.0571	0.0147
	$RMSE_\tau$	0.2734	0.2236	0.1567	0.2417	0.1797	0.1279	0.2170	0.1753	0.1193
`C90`	$\widehat{\gamma}$	1.9800	1.9663	2.0094	1.9262	1.9497	1.9606	1.9219	1.9429	1.9509
	$RMSE_\gamma$	0.2058	0.2179	0.2027	0.1576	0.1528	0.1545	0.1338	0.1309	0.1315
	$\widehat{\tau}$	-0.0355	-0.0311	-0.0414	-0.0111	-0.0178	-0.0253	-0.0062	-0.0100	-0.0205
	$RMSE_\tau$	0.1067	0.1047	0.0929	0.0958	0.0938	0.0859	0.0967	0.0943	0.0880
`C270`	$\widehat{\gamma}$	2.0168	2.0087	2.0366	1.9954	2.0040	2.0027	1.9996	2.0111	2.0010
	$RMSE_\gamma$	0.1918	0.1959	0.1969	0.1345	0.1293	0.1368	0.1039	0.1141	0.1123
	$\widehat{\tau}$	-0.1375	-0.1231	-0.1216	-0.1190	-0.1048	-0.1011	-0.1132	-0.1061	-0.1025
	$RMSE_\tau$	0.1492	0.1285	0.1102	0.1060	0.0894	0.0730	0.0895	0.0792	0.0647
`N`	$\widehat{\gamma}$	1.4440	1.8530	2.0265	1.6136	1.9732	2.0168	1.7731	1.9871	2.0214
	$RMSE_\gamma$	0.9556	0.4487	0.2442	0.7238	0.2630	0.1726	0.5390	0.2235	0.1451
	$\widehat{\tau}$	-0.0732	-0.0711	-0.1003	-0.0677	-0.1049	-0.0942	-0.1195	-0.0942	-0.0990
	$RMSE_\tau$	0.5191	0.2794	0.1599	0.4161	0.1910	0.1043	0.3527	0.1633	0.0915
Panel B: Benchmark estimators										
IV	$\widehat{\gamma}$	0.7427	0.2741	0.2704	0.2969	0.2790	0.2686	0.3045	0.2752	0.2671
	$RMSE_\gamma$	8.8557	1.7283	1.7300	1.7169	1.7218	1.7316	1.7086	1.7253	1.7331
CF	$\widehat{\gamma}$	6.0130	2.0815	2.1309	2.1274	2.1087	2.0700	2.2617	2.0856	2.0709
	$RMSE_\gamma$	73.1134	0.5920	0.3547	1.4939	0.3734	0.2178	1.3979	0.3032	0.1961

Notes: The table reports Monte Carlo means and root mean squared errors under `C270` copula data-generating process with negative latent dependence. The true structural coefficient is $\gamma_0 = 2.0$, and the true Kendall's dependence parameter is $\tau_0 = -0.10$. The first-stage coefficient π indexes instrument relevance. Copula-based estimators are implemented using `GJRM`. IV denotes the linear instrumental-variables benchmark, and CF denotes the control-function probit benchmark.

4 Data and Variables

This study uses data from the Cardiovascular Risk in Young Finns Study (YFS), a nationally representative longitudinal cohort initiated in 1980 to investigate the determinants of cardiovascular health across the life course. The YFS integrates demographic, behavioral, clinical, and genetic information and has been widely used to study cardiovascular risk, socioeconomic health gradients, and the interaction between genetic and behavioral determinants [37, 54, 55]. The 2001 wave contains adult participants with information on blood pressure, BMI, covariates, and genetic data. After imposing the common sample restriction required by the full set of covariates and BMI-related genetic instruments used in the empirical models, the final analytical sample contains 1,278 individuals.

The dependent variables are based on measured systolic and diastolic blood pressure. As shown in Table 3, mean systolic blood pressure is 121.57 mmHg and mean diastolic blood pressure is 73.13 mmHg. Following the clinical thresholds defined by the World Health Organization [56], we construct three binary outcomes: pre-hypertension, stage 1 hypertension, and stage 2 hypertension. The three outcomes are constructed as mutually exclusive binary indicators. Each individual belongs to only one blood-pressure category, so pre-hypertension, Hypertension T1, and Hypertension T2 are interpreted as severity-specific outcomes rather than overlapping indicators. In the estimation sample, 50.7% of participants are classified as pre-hypertensive, 31.1% as having stage 1 hypertension, and 12.1% as having stage 2 hypertension. These prevalences indicate that elevated blood pressure is already empirically relevant in this young adult cohort.

Body mass index (BMI) is the key endogenous regressor. It is measured continuously in kg/m^2, with mean 24.93 and standard deviation 4.35. Treating BMI as a continuous treatment preserves within-sample variation and aligns the empirical specification with the triangular model developed in Section 2. This is important because discretizing BMI into overweight or obesity indicators would discard variation relevant for the latent-index model and could introduce additional threshold dependence. Previous research identifies BMI as an important determinant of hypertension and related cardiovascular outcomes [57, 58, 59, 60].

The excluded instruments are BMI genetic risk scores constructed from the same original set of BMI-associated SNPs. The instrument strategy is described in detail in Section 2.2. In the present empirical analysis, we use three nested scores: `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. The score `GRSBMI32` is the baseline BMI genetic score. The score `GRSBMI31` excludes the SNP with the clearest prior evidence of blood-pressure-related pleiotropy, while `GRSBMI29` further excludes loci with stronger prior links to broader cardiometabolic pathways. This design keeps the genetic-instrument strategy transparent by focusing on a common original BMI score and then examining whether the estimates are sensitive to excluding variants with specific prior pleiotropy concerns. In the estimation sample, the mean values of `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` are 4.045, 3.579, and 3.427, respectively.

The dataset also includes demographic, family-background, behavioral, and dietary controls. The average age is 31.88 years, and 43.5% of the sample is male. Average years of education are 14.81. Parental hypertension is reported for 29.6% of fathers and 27.7% of mothers. Behavioral covariates include alcohol consumption and smoking status, while dietary controls include bread

Table 3: Descriptive statistics and variable definitions (Young Finns Study, 2001 wave)

Variable	**Definition / Unit**	**Mean**	**SD**	**N**
Dependent variables				
Pre-hypertension	1 if $120 \leq$ SBP < 140 or $80 \leq$ DBP < 90 (mmHg)	0.507	–	1,278
Stage 1 hypertension	1 if $140 \leq$ SBP < 160 or $90 \leq$ DBP < 100 (mmHg)	0.311	–	1,278
Stage 2 hypertension	1 if SBP ≥ 160 or DBP ≥ 100 (mmHg)	0.121	–	1,278
Blood pressure measures				
Systolic blood pressure	SBP, mmHg	121.569	14.296	1,278
Diastolic blood pressure	DBP, mmHg	73.129	8.831	1,278
Key endogenous regressor				
Body Mass Index (BMI)	kg/m^2	24.927	4.347	1,278
Genetic instruments				
GRSBMI32	Baseline BMI genetic risk score (32 SNPs)	4.045	0.513	1,278
GRSBMI31	BP-clean BMI genetic risk score (31 SNPs)	3.579	0.496	1,278
GRSBMI29	Cardiometabolic-clean BMI genetic risk score (29 SNPs)	3.427	0.488	1,278
Demographic covariates				
Age	Years	31.878	4.966	1,278
Male	1 = male	0.435	–	1,278
Years of education	Continuous	14.808	2.996	1,278
Parental hypertension (father)	1 = diagnosed hypertension	0.296	–	1,278
Parental hypertension (mother)	1 = diagnosed hypertension	0.277	–	1,278
Behavioral and lifestyle covariates				
Smoking	Categorical smoking measure	See notes	–	1,278
Alcohol consumption	Portions per day	0.825	1.060	1,278
Dietary controls				
Bread consumption	Slices per day	4.675	2.285	1,278
Milk intake	Glasses per day	1.638	1.788	1,278
Coffee consumption	Cups per day	3.262	2.586	1,278
Salt added hardly ever	Share (%)	0.774	–	1,278
Salt added after tasting first	Share (%)	0.218	–	1,278
Salt added before tasting first	Share (%)	0.009	–	1,278

Notes: SBP = systolic blood pressure; DBP = diastolic blood pressure; GRS = genetic risk score; SNP = single-nucleotide polymorphism. Hypertension categories follow Zhou et al. [56]. Statistics refer to the final analytical sample from the 2001 wave of the Young Finns Study. Sex composition is 56.5% female and 43.5% male. Father hypertension is 70.4% No and 29.6% Yes; mother hypertension is 72.3% No and 27.7% Yes. Smoking categories have shares of 19.2%, 4.8%, 5.9%, 18.6%, and 51.4%. Salt-use shares are 77.4% hardly ever, 21.8% after tasting first, and 0.9% before tasting first.

consumption, milk intake, coffee consumption, and salt-use frequency. These variables are included because they are documented determinants of BMI, blood pressure, and cardiovascular risk [61, 62, 19, 63, 64, 57, 65, 66]. Together, the YFS data provide measured clinical outcomes, continuous BMI, detailed covariates, and genetic instruments suitable for estimating the triangular copula model.

4.1 Control Variables and Empirical Specifications

Causal interpretation within the instrumental-variable and copula-based framework relies on the validity of the excluded genetic instruments and on the specification of an appropriate conditioning set. Including relevant pre-treatment covariates can reduce confounding due to observed heterogeneity, whereas adjusting for variables that are not confounders, or that may themselves be affected by BMI, can introduce overadjustment. The empirical specification therefore uses a parsimonious set of controls selected to account for demographic, family-background, behavioral, and dietary factors plausibly related to both body mass index and blood pressure.

The endogenous regressor is body mass index, BMI, and the excluded instruments are the three BMI genetic risk scores defined in Section 2.2: `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. These scores are constructed from the same original set of BMI-associated loci. The `GRSBMI32` is the baseline BMI genetic score, while `GRSBMI31` and `GRSBMI29` are nested scores that exclude variants with specific prior concerns about blood-pressure and broader cardiometabolic pleiotropy, respectively. The dependent variables are binary indicators for pre-hypertension, stage 1 (T1) hypertension, and stage 2 (T2) hypertension, defined according to systolic and diastolic blood-pressure thresholds.

The adjusted specification includes the following control variables in both the treatment and outcome equations. Demographic and family-background controls include age (`age`), sex (`sex`), years of education (`educ`), maternal history of hypertension (`mother_hypt`), and paternal history of hypertension (`father_hypt`). Behavioral controls include smoking status (`smoke`) and alcohol consumption (`alcohol_pd`). Dietary controls include salt added to food (`salt_add`), bread consumption (`bread_pd`), coffee intake (`coffee_pd`), and milk consumption (`milk_pd`). These variables are included because they may be associated with BMI, blood pressure, or both, and therefore help make comparisons across individuals more homogeneous.

The empirical analysis reports two main specifications. The first is a naive specification without additional controls, included as a descriptive benchmark. The second is an adjusted specification including the full set of controls described above. Comparing these two specifications provides evidence on the sensitivity of the estimated BMI effect to observed demographic, family, behavioral, and dietary heterogeneity.

5 Empirical Results

This section presents the empirical evidence on the causal relationship between BMI and hypertension. The analysis proceeds in five steps. Section 5.1 examines residual and pseudo-observation dependence to motivate the use of copula specifications. Section 5.2 quantifies latent residual dependence using Kendall's τ. Section 5.3 reports the copula-based BMI effects and the model-based first-stage coefficients. Section 5.4 examines whether the extended controls enter the BMI and hypertension equations with stable and interpretable signs under the negative-dependence specifications. Finally, Section 5.5 translates the latent-index estimates into probability-scale Sample Average Treatment Effects, showing how changes in BMI affect predicted hypertension risk over the observed BMI distribution.

5.1 Residual Dependence Analysis: Motivation for Copula Models

A central challenge in modelling the relationship between BMI and hypertension is that body mass is an imperfect proxy for the biological risk relevant for blood pressure. BMI summarizes body size, but it does not distinguish fat mass from lean mass, does not measure visceral adiposity, and does not capture metabolic health. Individuals with the same body mass may therefore differ substantially in their underlying hypertension risk. Clinical and epidemiological evidence supports this point: fat distribution, lean body mass, physical fitness, abdominal obesity, and cardiometabolic risk-factor clustering all contain information that is not fully captured by BMI alone [67, 68, 69, 70, 71]. This motivates examining whether the unobserved component of body mass and the unobserved component of latent hypertension risk move together, and in which direction.

Figures 1–3 suggest non-random residual-dependence patterns across outcomes and genetic instruments. In the naive specifications, the contour lines display a pronounced downward-sloping shape, consistent with negative association between the BMI residual and the latent hypertension residual. After adding extended control variables, the residual mass becomes less concentrated and more clustered. This pattern suggests that observed covariates absorb part of the shared residual structure, but do not fully eliminate dependence between the treatment and outcome equations. Economically, this is plausible: the BMI residual may contain unmeasured body composition, physical fitness, diet, and behavioral factors, whereas the hypertension residual may contain unobserved vascular, renal, metabolic, and socioeconomic determinants of blood pressure. These latent components need not move in the same direction.

The possibility of negative residual dependence is not specific to this paper. Dettoni et al. [72] estimate a flexible recursive copula model for obesity and chronic diseases in the United States and report negative Kendall's τ for hypertension, hyperlipidemia, and diabetes. They interpret this as evidence that the unobserved component of the chronic-disease equation is negatively associated with the unobserved component of the obesity equation, and relate this result to the earlier findings of Costa-Font and Gil [22]. Their proposed explanation is directly relevant here: if BMI measures obesity or cardiometabolic risk with error, then individuals with high lean mass may have high measured BMI but lower disease risk, while individuals with normal BMI may still have high abdominal adiposity or adverse metabolic profiles. In both cases, the residual component of BMI can move in the opposite direction from the residual component of hypertension risk.

This residual evidence motivates the use of copula models as sensitivity models for latent dependence. Copulas are useful because they separate the marginal specifications from the dependence structure. This is particularly relevant in the present setting, where hypertension is modeled through a latent binary-response equation and BMI is continuous. The mixed binary-continuous copula framework of Klein et al. [73] provides a natural way to represent this joint structure. The pseudo-observation analysis in Section A.3 provides a complementary view by placing residuals on a common unit-square scale, closer to the object modeled by the copula.

We therefore estimate several copula families that encode different assumptions about the sign and location of the dependence between unobservables. The Gaussian copula provides a symmetric benchmark for average dependence. The standard Clayton and Gumbel copulas capture positive asymmetric dependence. The standard Clayton copula emphasizes positive lower-tail dependence, whereas the standard Gumbel copula emphasizes positive upper-tail dependence. Positive upper-tail dependence is clinically plausible if unobserved adiposity-related factors, such as visceral fat accumulation, jointly increase BMI and latent hypertension risk [67].

The rotated copulas are included to represent negative cross-tail dependence, a pattern suggested by the residual plots and by previous copula-based evidence. In the orientation used here, the binary hypertension equation is treated as the first margin and the BMI equation as the second margin. Under this convention, `C90` and `G270` capture the region in which the unobserved component of BMI is low while the unobserved component of latent hypertension risk is high. This configuration is compatible with cases in which measured BMI does not fully summarize cardiometabolic risk, for example because of abdominal obesity, low lean mass, low aerobic capacity, or metabolically unhealthy normal weight [70, 71]. Conversely, `C270` and `G90` capture the region in which the unobserved component of BMI is high while the unobserved component of latent hypertension risk is low. This configuration can be interpreted as a sensitivity-based representation of cases in which higher measured BMI may reflect greater lean mass, better physical fitness, or a less adverse fat distribution rather than uniformly higher cardiometabolic risk [68, 69].

The rotated Clayton and rotated Gumbel specifications should therefore be viewed as alternative statistical representations of negative latent dependence, rather than as evidence for a specific biological mechanism. The difference between Clayton and Gumbel rotations concerns how dependence is concentrated within the relevant cross-tail region, not a different substantive mechanism. Estimating both families allows the empirical analysis to assess whether the estimated BMI effect is robust to alternative representations of negative asymmetric dependence.

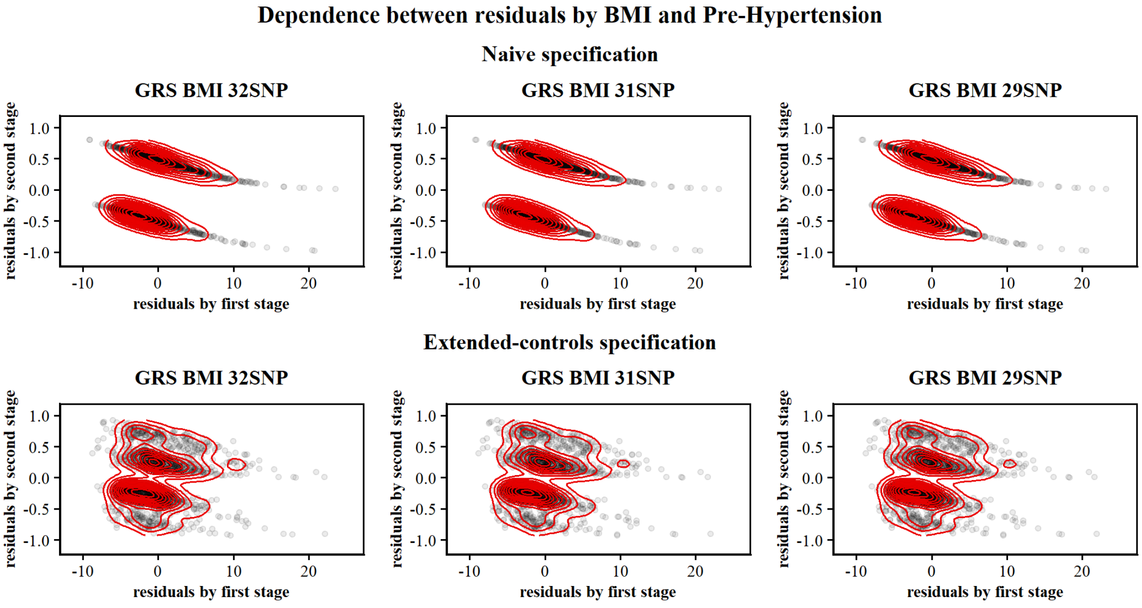
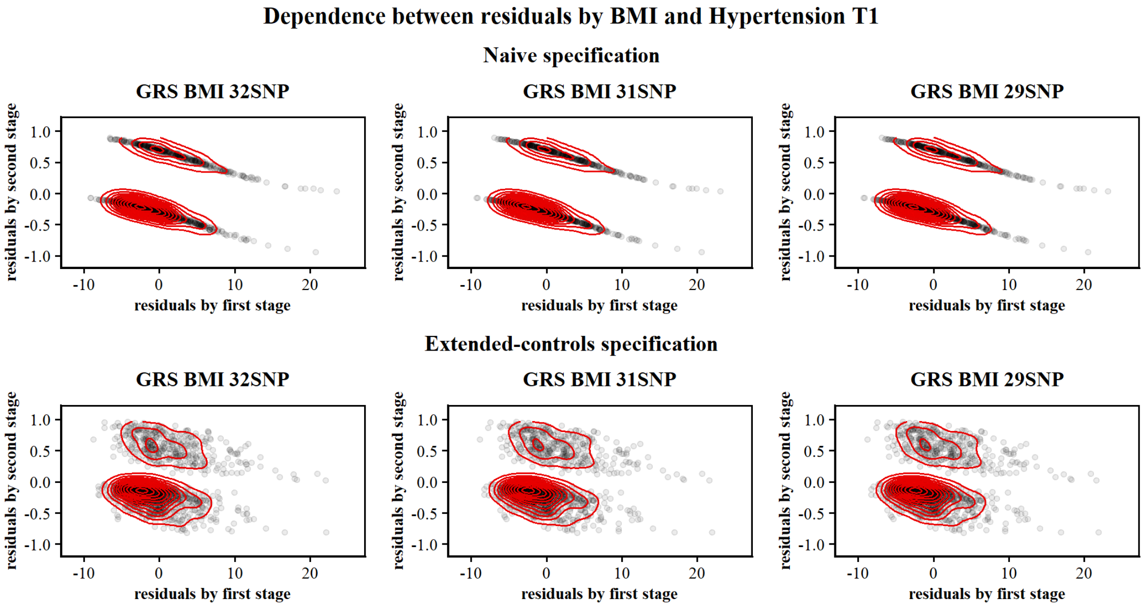


Figure 1: Residual dependence between first-stage BMI residuals and latent residuals for pre-hypertension. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.


Dependence between residuals by BMI and Hypertension T1

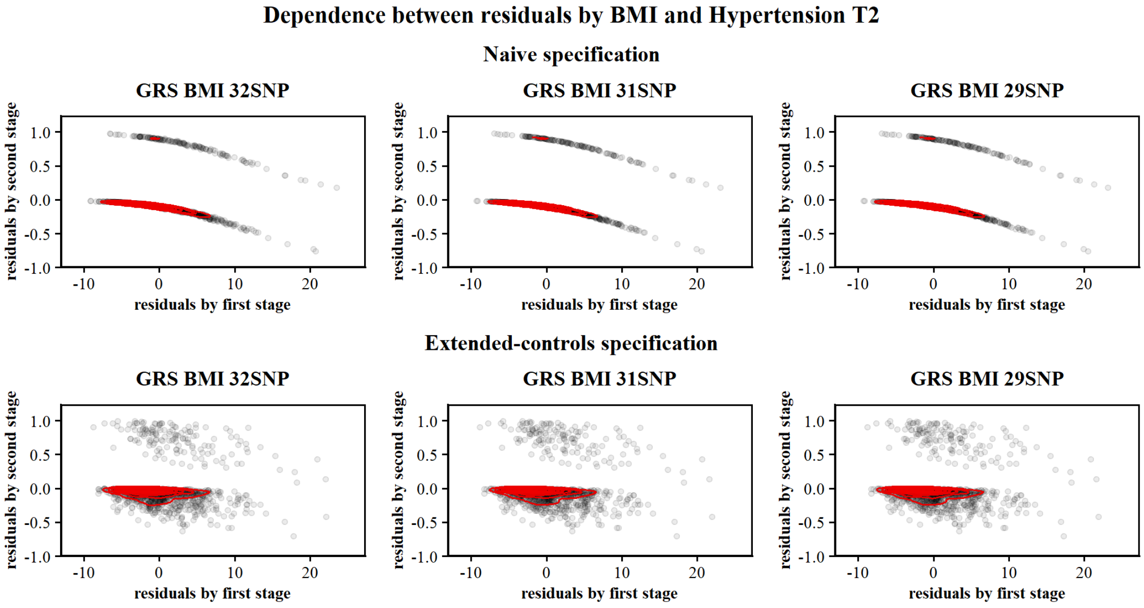
Naive specification

GRS BMI 32SNP GRS BMI 31SNP GRS BMI 29SNP

Extended-controls specification

GRS BMI 32SNP GRS BMI 31SNP GRS BMI 29SNP

Figure 2: Residual dependence between first-stage BMI residuals and latent residuals for stage 1 hypertension. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.


Dependence between residuals by BMI and Hypertension T2

Naive specification

GRS BMI 32SNP GRS BMI 31SNP GRS BMI 29SNP

Extended-controls specification

GRS BMI 32SNP GRS BMI 31SNP GRS BMI 29SNP

Figure 3: Residual dependence between first-stage BMI residuals and latent residuals for stage 2 hypertension. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.

5.2 Latent Residual Dependence and the Role of Control Variables

The dependence parameter in the triangular copula model summarizes the residual association between the unobserved component of BMI and the unobserved component of latent hypertension risk. This parameter is substantively important because it captures the direction and strength of endogeneity after conditioning on observed covariates and on the excluded genetic instrument. If the remaining unobservables jointly increase BMI and hypertension risk, the estimated dependence should be positive. If, instead, the residual component of measured BMI partly reflects features that are not necessarily harmful for cardiometabolic health, such as lean mass, physical fitness, or a less adverse fat distribution, then the dependence may be negative.

This possibility is not only a statistical artifact of the copula specification. It is consistent with the interpretation developed in Section 5.1: BMI is an imperfect measure of cardiometabolic risk because it does not distinguish adiposity from lean mass, nor does it capture fat distribution or metabolic health [67, 68, 69, 70, 71]. It is also consistent with previous copula-based evidence in health economics. Dettoni et al. [72] estimate a flexible recursive copula model for obesity and chronic diseases in the United States and report negative Kendall's τ for hypertension, hyperlipidemia, and diabetes. They interpret this as negative association between the unobserved component of the chronic-disease equation and the unobserved component of the obesity equation, and relate this finding to the earlier evidence in Costa-Font and Gil [22]. The Young Finns results reported here point in the same direction: once the dependence between the BMI and hypertension equations is estimated directly, a negative residual dependence appears in several specifications.

Table 4 reports the dependence estimates for Hypertension T1. The results show that the estimated dependence structure changes once controls are added. In the naive models, the standard Clayton copula implies positive dependence, which is compatible with a conventional cardiometabolic-risk interpretation in which unobserved factors that increase BMI also raise latent hypertension risk. However, this positive same-tail dependence weakens in the extended-control specifications. Once demographic, family-background, behavioral, and dietary variables are included, the standard Clayton estimates move toward the independence boundary. This suggests that part of the positive dependence in the naive models reflects observed heterogeneity rather than residual latent association.

A more persistent pattern is negative dependence, especially in the rotated copula specifications. For Hypertension T1, the naive models display negative dependence under both rotated Gumbel specifications, `G90` and `G270`, and under the rotated Clayton specification, `C270`. After adding extended controls, the magnitudes decline, but the signs remain negative across several rotated specifications. This before-and-after pattern suggests that the controls absorb part of the shared variation between BMI and hypertension, while the remaining latent association is still compatible with negative cross-tail dependence. The rotated copulas should therefore be viewed as sensitivity specifications that allow residual variation in measured BMI and residual variation in latent hypertension risk to move in opposite directions.

The pre-hypertension results point in a similar direction, although the evidence is less concentrated in a single copula family; the corresponding estimates are reported in Appendix Table A3. In the naive models, `C0` suggests positive lower-tail dependence, whereas the Gaussian and rotated specifications often imply negative dependence. After adding controls, the positive Clayton dependence becomes smaller, while the Gaussian and rotated specifications continue to indicate negative residual association in several cases. This pattern is compatible with the interpretation that pre-hypertension combines early blood-pressure risk with substantial heterogeneity in the mapping between BMI and blood pressure.

For Hypertension T2, negative average dependence is most visible under the Gaussian copula; the corresponding estimates are reported in Appendix Table A4. Across instruments, the Gaussian specification implies large negative Kendall's $\widehat{\tau}$ in both naive and extended-control specifications. Since T2 is the most severe and least frequent outcome, this result suggests that the unobserved determinants of severe hypertension are not captured by BMI in a simple monotone way. The rotated specifications also allow negative cross-tail dependence, while the Gaussian estimates provide a useful benchmark for central residual dependence.

Table 4: Estimated copula parameter θ and Kendall's τ by Hypertension T1

Naive Models

Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)	`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)	`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)
`G90`	−1.8637 (−2.6547, −1.5029)	−0.4634 (−0.5664, −0.3262)	`G90`	−1.9506 (−2.8222, −1.5775)	−0.4873 (−0.5872, −0.3511)	`G90`	−1.9633 (−2.8378, −1.5871)	−0.4906 (−0.5897, −0.3550)
`G270`	−7.2100 (−17.0000, −2.5342)	−0.8613 (−0.9412, −0.6228)	`G270`	−10.7011 (−17.0000, −3.0198)	−0.9066 (−0.9412, −0.6852)	`G270`	−10.1694 (−17.0000, −2.9973)	−0.9017 (−0.9412, −0.6825)
`C0`	0.4130 (0.1546, 1.3447)	0.1711 (0.0656, 0.3488)	`C0`	0.4943 (0.2267, 1.2456)	0.1982 (0.0962, 0.3427)	`C0`	0.4816 (0.2128, 1.2683)	0.1941 (0.0901, 0.3449)
`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)	`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)	`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)
`C270`	−0.1187 (−0.3701, −0.0466)	−0.0560 (−0.1201, −0.0222)	`C270`	−0.1141 (−0.3718, −0.0436)	−0.0540 (−0.1174, −0.0205)	`C270`	−0.1150 (−0.3718, −0.0441)	−0.0544 (−0.1182, −0.0209)
`N`	−0.5432 (−0.8490, −0.0530)	−0.3656 (−0.6443, −0.0170)	`N`	−0.5844 (−0.9606, 0.4601)	−0.3973 (−0.8179, 0.3328)	`N`	−0.6626 (−0.9572, 0.0000)	−0.4611 (−0.8131, 0.0000)

Extended Controls Models

Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)	`G0`	1.0137 (1.0000, 17.0000)	0.0135 (0.0000, 0.9412)	`G0`	1.0088 (1.0000, 17.0000)	0.0087 (0.0000, 0.9412)
`G90`	−1.0069 (−3.5083, −1.0000)	−0.0069 (−0.8716, −0.0000)	`G90`	−1.0046 (−15.6690, −1.0000)	−0.0046 (−0.9412, −0.0000)	`G90`	−1.0049 (−13.8833, −1.0000)	−0.0049 (−0.9412, −0.0000)
`G270`	−1.0548 (−2.2454, −1.0028)	−0.0519 (−0.5496, −0.0028)	`G270`	−1.0319 (−2.2687, −1.0007)	−0.0309 (−0.7108, −0.0005)	`G270`	−1.0367 (−2.0459, −1.0011)	−0.0354 (−0.6575, −0.0008)
`C0`	0.0000 (0.0000, 28.0000)	0.0000 (0.0000, 0.9333)	`C0`	0.0000 (0.0000, 28.0000)	0.0000 (0.0000, 0.9333)	`C0`	0.0000 (0.0000, 28.0000)	0.0000 (0.0000, 0.9333)
`C90`	−0.0494 (−1.6820, −0.0012)	−0.0241 (−0.6150, −0.0005)	`C90`	−0.0262 (−14.3346, −0.0000)	−0.0129 (−0.9327, −0.0000)	`C90`	−0.0319 (−6.1499, −0.0001)	−0.0157 (−0.8830, −0.0000)
`C270`	−0.0350 (−0.4525, −0.0023)	−0.0172 (−0.2682, −0.0010)	`C270`	−0.0289 (−0.6205, −0.0011)	−0.0142 (−0.3552, −0.0004)	`C270`	−0.0298 (−0.5846, −0.0013)	−0.0147 (−0.3380, −0.0005)
`N`	−0.5165 (−0.7883, 0.0611)	−0.3455 (−0.6082, 0.0513)	`N`	−0.3907 (−0.8981, 0.6958)	−0.2555 (−0.7058, 0.4352)	`N`	−0.5398 (−0.8618, 0.3198)	−0.3630 (−0.7291, 0.2054)

Notes: The table reports the estimated copula parameter $\widehat{\theta}$ and Kendall's $\widehat{\tau}$, with 95% confidence intervals in parentheses. BMI is treated as an endogenous continuous variable and is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Kendall's $\widehat{\tau}$ measures latent residual dependence between the BMI equation and the binary-outcome equation, not the observed association between BMI and the outcome. Boundary estimates should be interpreted cautiously.

The interpretation of the rotated copulas follows directly from the health mechanism discussed above. In the orientation used here, `C90` and `G270` represent the cross-tail configuration in which the BMI residual is low and the latent hypertension residual is high. This configuration is consistent with individuals whose measured BMI is not especially high but whose cardiometabolic risk is elevated, for example because of abdominal obesity, low lean mass, low aerobic capacity, or metabolically unhealthy normal weight. Conversely, `C270` and `G90` represent the cross-tail configuration in which the BMI residual is high and the latent hypertension residual is low. This configuration is consistent with individuals whose BMI is high because of greater lean mass, better physical fitness, or a less harmful fat distribution rather than higher cardiometabolic risk.

Taken together, the dependence estimates show that the extended controls change the structure of latent selection without fully eliminating residual dependence. Positive same-tail dependence appears mainly in the naive specifications and often weakens or moves toward the independence boundary after conditioning on observed covariates. Once demographic, family-background, behavioral, and dietary factors are included, several Gaussian and rotated-copula specifications remain compatible with negative residual association, either through central dependence under the Gaussian copula or through cross-tail dependence under rotated Clayton and rotated Gumbel copulas. This pattern is consistent with the broader empirical rationale for allowing flexible copula-based dependence in applications linking obesity-related measures to chronic disease outcomes [72, 22]. However, the sign and location of latent dependence should be interpreted as specification-sensitive rather than as evidence for a single true dependence structure. Estimates close to the independence boundary or to copula-parameter boundaries should therefore be interpreted cautiously. The relevant question is not which copula is the true model, but whether the estimated BMI effect changes materially when the latent dependence between BMI and hypertension is allowed to take empirically motivated alternative forms.

5.3 Copula-Based Causal Effects and First-Stage Relevance

This subsection reports the copula-based estimates of the BMI coefficient and the corresponding first-stage relevance of the genetic instruments. The purpose is to evaluate whether the estimated effect of BMI on the latent propensity for hypertension is sensitive to alternative assumptions about the dependence between the unobserved components of the BMI and hypertension equations. This is the main empirical role of the copula framework: if the BMI coefficient remains positive and statistically significant across copulas that encode different forms of residual dependence, then the result is less likely to be driven by a particular parametric specification of the joint distribution of unobservables.

The estimates are reported in Appendix A.5. Table 5 and Appendix Tables A5–A6 present, for each hypertension outcome, genetic instrument, covariate specification, and copula family, two key parameters. The first is $\widehat{\gamma}_{BMI}$, the coefficient of BMI in the normalized latent hypertension equation. The second is $\widehat{\pi}_{GRS}$, the coefficient of the genetic score in the BMI equation. The latter is the model-based first-stage coefficient and measures whether the excluded genetic variation shifts BMI within the same joint likelihood used to estimate the outcome equation.

The central result is that $\widehat{\gamma}_{BMI}$ is positive across all hypertension outcomes, genetic instruments, and copula specifications, and is statistically significant in the main extended-control and negative-dependence specifications. This pattern holds for pre-hypertension, Hypertension T1, and Hypertension T2, and it remains present after the inclusion of demographic, family-background, behavioral, and dietary controls. In the latent-index specification, a positive $\widehat{\gamma}_{BMI}$ implies that an increase in BMI shifts individuals toward a higher latent propensity for hypertension. Although this coefficient is not itself a probability-scale marginal effect, its sign and statistical significance provide direct evidence, within the maintained triangular model, that BMI increases hypertension risk. The magnitude of the coefficient varies across outcomes and dependence structures, as expected in a nonlinear latent-index model, but the sign and statistical conclusion are stable.

The rotated-copula specifications provide an important sensitivity check. Section 5.1 motivates `C90`, `C270`, `G90`, and `G270` as specifications that allow negative cross-tail dependence between the unobserved component of BMI and the unobserved component of latent hypertension risk. The estimates show that the positive BMI coefficient is not eliminated when this form of negative latent dependence is allowed. Under both rotated Clayton and rotated Gumbel specifications, $\widehat{\gamma}_{BMI}$ remains positive in the extended-control specifications and when the nested `GRSBMI31` and `GRSBMI29` scores are used. This pattern suggests that the estimated BMI effect is not simply an artifact of imposing positive same-tail dependence between unobservables. Rather, it remains present when the model allows residual variation in measured BMI and residual variation in hypertension risk to move in opposite directions.

The comparison between naive and extended-control specifications further supports this interpretation. In the naive models, part of the estimated dependence may reflect observed heterogeneity that has not yet been conditioned out. Once the extended controls are included, the dependence parameters generally move closer to the independence boundary or become smaller in magnitude, as documented in Section 5.2. Nevertheless, the BMI coefficient remains positive and statistically significant. This indicates that the control variables reshape the estimated residual dependence structure without eliminating the positive structural relationship between BMI and latent hypertension risk.

The first-stage estimates provide complementary evidence on the relevance of the BMI genetic scores. Across outcomes and copula families, $\widehat{\pi}_{GRS}$ is positive, indicating that higher values of the genetic score predict higher BMI in the treatment equation of the joint model. This sign is consistent with the construction of the scores and with their intended role as excluded sources of BMI variation. As expected, `GRSBMI32` generally provides the strongest first-stage signal, while `GRSBMI31` and `GRSBMI29` are weaker because they exclude variants with specific prior concerns about blood-pressure or broader cardiometabolic pleiotropy. This reduction in first-stage strength is part of the sensitivity design: the nested scores do not validate the exclusion restriction, but they allow the analysis to assess whether the estimated BMI effect changes when variants with stronger prior pleiotropy concerns are removed.

Importantly, the positive BMI effect remains present when moving from `GRSBMI32` to `GRSBMI31` and `GRSBMI29`. This pattern suggests that the result is not driven exclusively by the baseline BMI genetic score. It should nevertheless be interpreted as sensitivity evidence from nested genetic-score constructions, not as evidence that the exclusion restriction has been proven.

The Gaussian copula provides an additional benchmark. Because it imposes symmetric central dependence, it is useful for assessing whether the estimated BMI effect survives a conventional dependence structure. The results show that $\widehat{\gamma}_{BMI}$ is also positive and statistically significant under the Gaussian specification, especially for the more severe hypertension outcomes. However, the Gaussian copula does not distinguish whether dependence is concentrated in cross-tail regions. It should therefore be interpreted as complementary to the rotated copulas, which are motivated by the residual-dependence patterns documented in Section 5.1.

The empirical results are also consistent with the Monte Carlo evidence in Section 3. The simulation shows that, when the excluded instrument is informative, the triangular copula estimator

Table 5: Copula-based estimates for Hypertension T1

Naive Models								
Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)
G0	0.1005***	0.8597***	G0	0.1005***	0.4899*	G0	0.1005***	0.5284*
	(0.0091)	(0.2358)		(0.0091)	(0.2449)		(0.0091)	(0.2485)
G90	0.2531***	0.6282**	G90	0.2560***	0.2927	G90	0.2563***	0.3446†
	(0.0079)	(0.1940)		(0.0075)	(0.1832)		(0.0075)	(0.1852)
G270	0.2400***	0.1651	G270	0.2385***	0.0675	G270	0.2388***	0.0909
	(0.0051)	(0.1309)		(0.0057)	(0.0686)		(0.0056)	(0.0810)
C0	0.0586**	0.7660**	C0	0.0514*	0.4016†	C0	0.0525*	0.4242†
	(0.0214)	(0.2384)		(0.0201)	(0.2419)		(0.0204)	(0.2472)
C90	0.1005***	0.8597***	C90	0.1005***	0.4899*	C90	0.1005***	0.5284*
	(0.0091)	(0.2358)		(0.0091)	(0.2449)		(0.0091)	(0.2485)
C270	0.1330***	0.8663***	C270	0.1321***	0.4815*	C270	0.1323***	0.5227*
	(0.0171)	(0.2348)		(0.0171)	(0.2438)		(0.0171)	(0.2474)
N	0.2085***	0.8615***	N	0.2158***	0.4868*	N	0.2274***	0.5302*
	(0.0409)	(0.2353)		(0.0654)	(0.2460)		(0.0444)	(0.2459)
Extended Controls Models								
Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)
G0	0.0900***	0.8418***	G0	0.0841**	0.4542†	G0	0.0862**	0.4994*
	(0.0097)	(0.2267)		(0.0288)	(0.2364)		(0.0290)	(0.2404)
G90	0.0945***	0.8433***	G90	0.0931***	0.4571†	G90	0.0933***	0.5024*
	(0.0177)	(0.2268)		(0.0177)	(0.2360)		(0.0177)	(0.2393)
G270	0.1057***	0.8697***	G270	0.0995***	0.4781*	G270	0.1009***	0.5281*
	(0.0240)	(0.2289)		(0.0216)	(0.2388)		(0.0222)	(0.2428)
C0	0.0900***	0.8418***	C0	0.0900***	0.4576†	C0	0.0900***	0.5026*
	(0.0097)	(0.2267)		(0.0097)	(0.2359)		(0.0097)	(0.2393)
C90	0.0969***	0.8562***	C90	0.0938***	0.4658*	C90	0.0946***	0.5141*
	(0.0165)	(0.2281)		(0.0165)	(0.2377)		(0.0166)	(0.2415)
C270	0.1001***	0.8456***	C270	0.0984***	0.4557†	C270	0.0987***	0.5019*
	(0.0176)	(0.2267)		(0.0176)	(0.2359)		(0.0176)	(0.2392)
N	0.1997***	0.8724***	N	0.1762†	0.4959*	N	0.2046**	0.5583*
	(0.0451)	(0.2197)		(0.1004)	(0.2262)		(0.0647)	(0.2228)

Notes: The table reports copula-based estimates of the BMI coefficient in the latent binary-outcome equation and the genetic-score coefficient in the BMI equation. The parameter $\widehat{\gamma}_{BMI}$ denotes the BMI coefficient, and $\widehat{\pi}_{GRS}$ denotes the coefficient of the excluded BMI genetic score. BMI is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Standard errors are in parentheses. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.

can recover the structural effect under both positive and negative latent dependence. The empirical findings follow the same logic: the genetic-score coefficients are positive, the BMI coefficients are positive and statistically significant, and the conclusion is preserved across copula families that allow different signs and shapes of residual dependence. This consistency between the simulation design and the empirical estimates supports the interpretation that the estimated BMI effect is not merely a consequence of imposing a particular copula specification.

5.4 Extended Control Variables under Negative-Dependence Specifications

Table 6 reports the extended-control coefficients for Hypertension T1 using `GRSBMI29`, the most restrictive score in the nested sensitivity design, the negative-dependence copulas, and the Gaussian benchmark. The corresponding estimates for pre-hypertension and Hypertension T2 are reported in Appendix Tables A7 and A8. These coefficients are not interpreted as causal effects. They are used as a diagnostic check on whether the observed covariates capture plausible demographic, family-background, behavioral, and dietary heterogeneity in the outcome and BMI equations.

Table 6: Control-variable coefficients under alternative copulas (Hypertension T1, `GRSBMI29`)

		Copula specification				
Control	Eq.	G90	G270	C90	C270	N
Age	Outcome	0.023* (0.011)	0.019 (0.011)	0.016† (0.009)	0.015† (0.009)	0.001 (0.014)
	BMI	0.096*** (0.025)	0.097*** (0.025)	0.096*** (0.025)	0.097*** (0.025)	0.096*** (0.025)
Gender: Male	Outcome	0.757*** (0.119)	0.716*** (0.123)	0.687*** (0.089)	0.679*** (0.090)	0.505* (0.221)
	BMI	0.567* (0.266)	0.562* (0.265)	0.567* (0.266)	0.567* (0.266)	0.564* (0.266)
Mother-hypt	Outcome	0.393*** (0.109)	0.366*** (0.108)	0.185* (0.088)	0.184* (0.088)	0.123 (0.104)
	BMI	0.265 (0.262)	0.270 (0.261)	0.264 (0.262)	0.268 (0.262)	0.261 (0.262)
Father-hypt	Outcome	0.282** (0.108)	0.232* (0.114)	0.169† (0.089)	0.173* (0.086)	0.042 (0.136)
	BMI	0.805** (0.256)	0.799** (0.256)	0.808** (0.256)	0.803** (0.256)	0.798** (0.256)
Education	Outcome	−0.015 (0.017)	−0.012 (0.017)	−0.023† (0.014)	−0.024† (0.014)	−0.005 (0.020)
	BMI	−0.114** (0.041)	−0.115** (0.041)	−0.114** (0.041)	−0.114** (0.041)	−0.115** (0.041)
Smoke	Outcome	0.107** (0.038)	0.101** (0.037)	0.102*** (0.029)	0.103*** (0.029)	0.072† (0.042)
	BMI	0.124 (0.083)	0.129 (0.083)	0.126 (0.083)	0.123 (0.083)	0.126 (0.083)
Alcohol-pd	Outcome	0.113* (0.045)	0.105* (0.045)	0.099* (0.040)	0.096* (0.040)	0.054 (0.056)
	BMI	0.234† (0.121)	0.220† (0.121)	0.232† (0.121)	0.236† (0.121)	0.233† (0.121)
Bread-pd	Outcome	−0.017 (0.022)	−0.023 (0.022)	−0.008 (0.018)	−0.008 (0.018)	−0.021 (0.019)
	BMI	0.121* (0.054)	0.123* (0.054)	0.122* (0.054)	0.119* (0.054)	0.119* (0.054)
Coffee-pd	Outcome	0.026 (0.021)	0.022 (0.021)	0.004 (0.017)	0.005 (0.017)	−0.011 (0.020)
	BMI	0.122* (0.050)	0.121* (0.050)	0.122* (0.050)	0.121* (0.051)	0.122* (0.051)
Milk-pd	Outcome	0.011 (0.028)	0.001 (0.029)	0.024 (0.023)	0.023 (0.023)	−0.010 (0.034)
	BMI	0.257*** (0.069)	0.253*** (0.068)	0.257*** (0.069)	0.259*** (0.069)	0.258*** (0.069)
Salt add 2	Outcome	−0.122 (0.127)	−0.123 (0.121)	−0.289** (0.100)	−0.287** (0.100)	−0.288** (0.098)
	BMI	0.369 (0.285)	0.390 (0.285)	0.375 (0.286)	0.365 (0.285)	0.365 (0.285)
Salt add 3	Outcome	0.529 (0.526)	0.573 (0.506)	0.122 (0.436)	0.128 (0.436)	0.357 (0.430)
	BMI	−2.044 (1.271)	−2.034 (1.266)	−2.024 (1.273)	−2.052 (1.272)	−2.050 (1.273)

Notes: The table reports estimated coefficients for the extended controls in the binary-outcome and BMI equations, using `GRSBMI29` as the excluded genetic score. Columns compare the rotated Gumbel copulas `G90` and `G270`, the rotated Clayton copulas `C90` and `C270`, and the Gaussian copula `N`, under probit–Normal margins. Standard errors are in parentheses. The BMI coefficient in the outcome equation, the `GRSBMI29` coefficient in the BMI equation, and intercepts are omitted. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.

The outcome equation shows a coherent pattern across the negative-dependence specifications.

Male sex and parental history of hypertension generally enter with positive signs, consistent with their role as observed predictors of blood-pressure risk. Smoking and alcohol consumption also tend to be positively associated with Hypertension T1, especially under the rotated copulas. Education enters mostly with a negative sign, although with weaker precision. By contrast, the dietary variables are less stable and should be interpreted cautiously as conditional associations rather than as structural dietary effects. The negative coefficient on one salt-addition category, for example, may reflect reporting behavior, health awareness, or broader lifestyle differences rather than a direct protective effect of salt use.

The BMI equation is more stable across copula specifications. Age and male sex tend to enter positively, education enters negatively, and paternal history of hypertension is consistently positive. Behavioral and dietary variables also show broadly stable signs, although they are best interpreted as descriptive controls for the observed environment associated with body mass in this cohort. The appendix tables show a similar broad structure across the other severity-specific outcomes: family-background and behavioral variables are more prominent for the hypertension outcomes, while the BMI equation remains comparatively stable across copula families.

Taken together, the extended-control results support the use of the adjusted specification as a richer conditioning set. The controls absorb systematic observed heterogeneity in both equations and help reduce reliance on a sparse BMI–instrument specification. At the same time, the heterogeneity captured by these controls should not be overinterpreted. Their role in the main analysis is to improve comparability across individuals and to discipline the residual-dependence analysis, while the causal interpretation remains attached to the BMI effect within the maintained triangular copula framework.

5.5 Sample Average Treatment Effects (SATE)

The positive latent-index estimates have a clear probability-scale counterpart. Across the observed BMI distribution, higher BMI increases the predicted probability of pre-hypertension, Hypertension T1, and Hypertension T2. The SATE profiles are computed as described in Section 2.1 and show that the effect of BMI is not constant across either BMI values or hypertension severity.

The main Sample Average Treatment Effects analysis focuses on `GRSBMI29`, the most restrictive score in the nested sensitivity design. This score excludes variants with stronger prior concerns about blood-pressure and broader cardiometabolic pleiotropy, but it is not interpreted as validating the exclusion restriction. The figures focus on copula specifications that allow negative residual dependence, together with the Gaussian copula as a central-dependence benchmark. This follows the residual-dependence evidence in Sections 5.1 and 5.2. The SATE exercise is therefore interpreted as a probability-scale sensitivity analysis rather than as a copula-selection exercise.

Figure A4 shows that the effect on pre-hypertension is positive but concentrated at lower and intermediate BMI values. Across the reported specifications, the average effects are approximately 2.15%–2.92%. Under the extended-control models, the largest effects occur around BMI 24–25, ranging from about 2.97% under `C90` to 4.37% under `G90`, with the Gaussian benchmark reaching 8.63%. The decline at higher BMI values is consistent with pre-hypertension operating as an early-risk margin, after which marginal risk begins to shift toward more severe outcomes.

Figure 4 shows positive effects on Hypertension T1, with average effects of approximately

2.51%–2.94%. Relative to pre-hypertension, the T1 profiles peak later in the BMI distribution. In the extended-control specifications, the maximum effects under the rotated copulas are concentrated around BMI 30–32 and range from about 3.40% to 3.68%, while the Gaussian benchmark peaks around BMI 27–28 with a maximum effect of 7.80%. At higher BMI values, the T1 profiles flatten or decline, consistent with probability mass shifting from stage 1 toward stage 2 hypertension rather than with a weakening of the harmful effect of BMI.

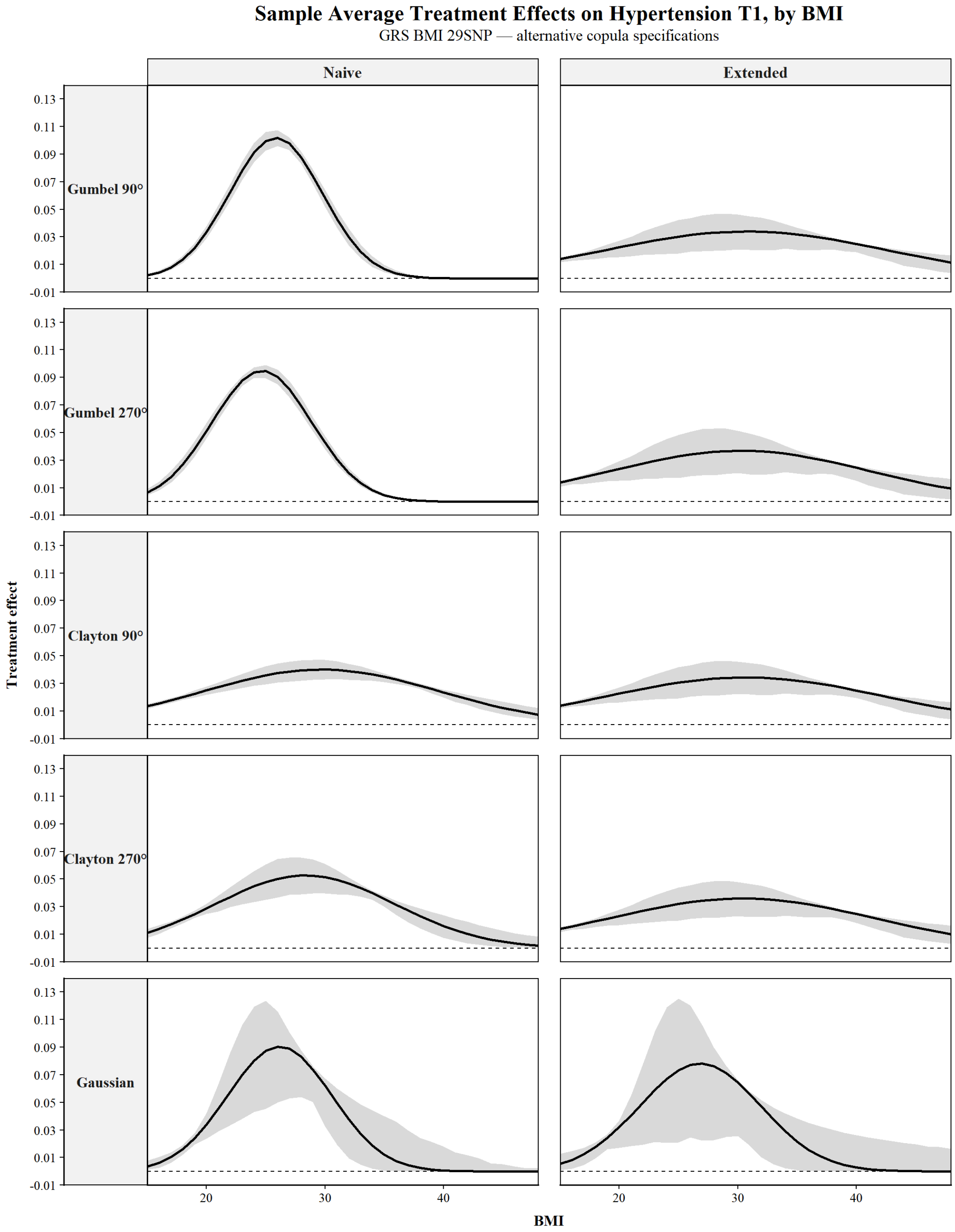


Figure 4: Sample average treatment effects of BMI on Hypertension T1, by BMI. SATE estimates are reported using the `GRSBMI29` instrument across alternative copula specifications. Black lines denote point estimates, shaded areas confidence bands, and dashed lines zero effect.

Figure A5 completes this severity-gradient pattern. The effects on Hypertension T2 are positive, averaging approximately 2.08%–2.94%. Under the extended-control models, the largest effects under the negative-dependence copulas occur at higher BMI values: around BMI 37–42, with maximum effects between 3.11% and 4.03%. The Gaussian benchmark peaks earlier, around BMI 27–28, with a maximum effect of 9.64%. This pattern indicates that severe hypertension becomes the relevant marginal outcome in the upper part of the BMI distribution.

The comparison between naive and extended-control specifications is also informative. In the naive models, the SATE profiles are often sharper and sometimes peak at lower BMI values, reflecting residual heterogeneity that has not yet been conditioned out. After adding controls, the profiles become smoother, but the effects remain positive. Thus, conditioning on demographic, family-background, behavioral, and dietary variables does not eliminate the probability-scale effect of BMI.

Taken together, the SATE profiles support a nonlinear severity-gradient interpretation. The estimated probability effects are concentrated at lower and intermediate BMI values for pre-hypertension, around the overweight and early-obesity range for Hypertension T1, and at higher BMI values for Hypertension T2. For a relatively young adult sample, average probability effects of roughly 2.00%–3.00% are economically meaningful, especially because maximum effects are larger in the BMI ranges where marginal risk is most concentrated. The results therefore complement the latent-index estimates by showing that the positive BMI effect is also relevant on the predicted-probability scale.

6 Discussion

This paper estimates the effect of BMI on hypertension using a maintained triangular copula model with a binary outcome, a continuous endogenous treatment, and genetic instruments. The empirical strategy combines the logic of Mendelian randomization with a parametric sensitivity analysis for latent dependence between the BMI and hypertension equations. This flexibility is important because BMI is an imperfect proxy for cardiometabolic risk: it does not distinguish fat mass from lean mass, nor does it capture fat distribution, physical fitness, or metabolic health.

The main finding is that BMI increases model-implied hypertension risk across pre-hypertension, Hypertension T1, and Hypertension T2. The BMI coefficient is positive in the main specifications used for the sensitivity analysis, and the model-based first-stage estimates indicate that the genetic scores predict BMI in the expected direction. The result is not driven exclusively by the baseline `GRSBMI32` score: it remains present when using the nested `GRSBMI31` and `GRSBMI29` scores, which exclude variants with stronger prior concerns about blood-pressure or broader cardiometabolic pleiotropy. This pattern provides sensitivity evidence with respect to specific prior pleiotropy concerns, although it does not validate the exclusion restriction.

The copula results show that the positive BMI effect is not eliminated when the model allows for negative residual dependence. Positive same-tail dependence is mainly concentrated in some naive specifications and becomes weaker after adding demographic, family-background, behavioral, and dietary controls. Several rotated-copula and Gaussian specifications remain compatible with negative residual association between the BMI and hypertension equations. These estimates should not be interpreted as direct evidence of a biological mechanism or as evidence that one copula is the true model. Rather, the copula framework is used to assess whether the estimated BMI effect is sensitive to alternative parametric representations of latent residual dependence. The extended controls and Monte Carlo evidence support this interpretation: the controls reshape residual dependence without eliminating it, and the simulations show that the triangular copula estimator can recover the structural effect under both positive and negative latent dependence when the excluded instrument is informative.

The SATE analysis translates the latent-index results into the probability scale. Using `GRSBMI29`, the most restrictive score in the nested sensitivity design, the estimated probability effects remain positive and economically meaningful under the dependence structures considered in the probability-scale analysis. Average SATEs are roughly 2%–3%, which is relevant in a relatively young adult sample. The profiles reveal a nonlinear severity gradient: estimated probability effects are concentrated at lower and intermediate BMI values for pre-hypertension, around the overweight and early-obesity range for Hypertension T1, and at higher BMI values for Hypertension T2. Thus, the probability-scale risk associated with BMI varies across both body-mass levels and blood-pressure severity categories.

Several limitations remain. The causal interpretation depends on the validity of the genetic instruments and on the maintained triangular structure, including exclusion, relevance, support conditions, the binary-outcome normalization, and the copula specification. The `GRSBMI31` and `GRSBMI29` specifications address specific prior pleiotropy concerns but cannot rule out all possible biological pathways from genetic variants to hypertension. The copulas are used as parametric sensitivity models rather than as the true data-generating process, and BMI remains a coarse measure that cannot identify the specific biological mechanisms linking body composition to blood pressure. External validity is also limited because the analysis is based on a relatively young Finnish cohort observed in 2001; the genetic instruments, BMI distribution, cardiometabolic risk profiles, and healthcare environment may not generalize directly to older populations, other ancestral groups, or health systems with different prevention and treatment patterns.

Taken together, the analysis supports a local and model-based conclusion that higher BMI increases hypertension risk across severity-specific blood-pressure outcomes. The main empirical contribution is to show that this risk varies across the BMI distribution and across hypertension severity, while remaining present under alternative latent-dependence specifications and nested BMI genetic-score constructions used as pleiotropy-sensitivity checks. More broadly, the paper illustrates how genetic instruments and triangular copula models can be combined to study endogenous continuous treatments in nonlinear health outcomes, while keeping the identifying assumptions and external-validity limits explicit.

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Data availability

The data used in this study are from the Cardiovascular Risk in Young Finns Study. Individual-level data are not publicly available because they contain sensitive health, genetic, and personal information, and access is restricted by participant consent, ethical approvals, and data-protection regulations. Requests for data access may be directed to Robinson Dettoni, the corresponding author, and will be considered subject to approval by the relevant Young Finns Study data controllers and ethics committees.

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A Appendix

A.1 SNP Composition of the BMI Genetic Risk Scores

Table A1: SNP composition and exclusion rationale for the BMI genetic risk scores

SNP	Gene / region	GRSBMI32	GRSBMI31	GRSBMI29	Status	Main rationale for exclusion
`rs10150332`	*NRXN3*	Yes	Yes	Yes	Included	–
`rs10767664`	*BDNF*	Yes	Yes	Yes	Included	–
`rs10938397`	*GNPDA2*	Yes	Yes	Yes	Included	–
`rs10968576`	*LRRN6C*	Yes	Yes	Yes	Included	–
`rs11847697`	*PRKD1*	Yes	Yes	Yes	Included	–
`rs12444979`	*GPRC5B/IQCK*	Yes	Yes	Yes	Included	–
`rs13078807`	*CADM2*	Yes	Yes	Yes	Included	–
`rs13107325`	*SLC39A8*	Yes	No	No	BP-clean excluded	Associated with diastolic blood pressure and lipid traits after adjustment for BMI, raising direct blood-pressure and cardiometabolic pleiotropy concerns.
`rs1514175`	*TNNI3K*	Yes	Yes	Yes	Included	–
`rs1555543`	*PTBP2*	Yes	Yes	Yes	Included	–
`rs1558902`	*FTO*	Yes	Yes	Yes	Included	–
`rs206936`	*NUDT3/HMGA1*	Yes	Yes	Yes	Included	–
`rs2112347`	*FLJ35779/POC5/HMGCR*	Yes	Yes	No	CM-clean excluded	Located in a region associated with LDL cholesterol and total cholesterol after adjustment for BMI, suggesting a possible lipid-mediated pathway.
`rs2241423`	*MAP2K5/LBXCOR1*	Yes	Yes	Yes	Included	–
`rs2287019`	*QPCTL/GIPR*	Yes	Yes	No	CM-clean excluded	Located in a region linked to incretin biology, glucose metabolism, insulin response, and lipid traits.
`rs2815752`	*NEGR1*	Yes	Yes	Yes	Included	–
`rs2867125`	*TMEM18*	Yes	Yes	Yes	Included	–
`rs2890652`	*LRP1B*	Yes	Yes	Yes	Included	–
`rs29941`	*KCTD15*	Yes	Yes	Yes	Included	–
`rs3810291`	*TMEM160*	Yes	Yes	Yes	Included	–
`rs3817334`	*MTCH2/NDUFS3*	Yes	Yes	Yes	Included	–
`rs4771122`	*MTIF3/GTF3A*	Yes	Yes	Yes	Included	–
`rs4836133`	*ZNF608*	Yes	Yes	Yes	Included	–
`rs4929949`	*RPL27A/TUB*	Yes	Yes	Yes	Included	–
`rs543874`	*SEC16B*	Yes	Yes	Yes	Included	–
`rs571312`	*MC4R*	Yes	Yes	Yes	Included	–
`rs713586`	*RBJ/ADCY3/POMC*	Yes	Yes	Yes	Included	–
`rs7138803`	*FAIM2*	Yes	Yes	Yes	Included	–
`rs7359397`	*SH2B1*	Yes	Yes	Yes	Included	–
`rs887912`	*FANCL*	Yes	Yes	Yes	Included	–
`rs9816226`	*ETV5*	Yes	Yes	Yes	Included	–
`rs987237`	*TFAP2B*	Yes	Yes	Yes	Included	–

Notes: The table reports the SNP dosage variables used to construct the three nested BMI genetic risk scores. `GRSBMI32` is the baseline 32-SNP BMI genetic score. `GRSBMI31` is the blood-pressure-clean score, which excludes `rs13107325`. `GRSBMI29` is the stricter cardiometabolic-clean score, which further excludes `rs2112347` and `rs2287019`. The individual identifier `tutkn001` is excluded from the score construction. “BP-clean excluded” refers to exclusion from both `GRSBMI31` and `GRSBMI29`. “CM-clean excluded” refers to additional exclusion from `GRSBMI29`.

A.2 Monte Carlo design

The simulation parameters are chosen to generate a transparent and demanding finite-sample environment that mirrors the main features of the empirical application without calibrating the data-generating process to any particular set of estimates. The sample sizes $n \in \{500, 1000, 1500\}$ are selected to cover small and moderate samples and to bracket the empirical sample size used in the Young Finns application. This allows the simulations to assess whether estimator performance improves as the amount of information approaches and exceeds the empirical setting.

Table A2: Monte Carlo simulation design

Component	Specification
Panel A. Sample design and structural parameters	
Monte Carlo replications	500 replications for each design.
Sample sizes	$n \in \{500, 1000, 1500\}$.
Observed covariates	One exogenous covariate, $X_i \sim N(0, 1)$.
Excluded instrument	One excluded instrument, $Z_i \sim N(0, 0.4^2)$, corresponding to a weak-support design.
Structural treatment effect	$\gamma_0 = 2.0$.
Outcome equation parameters	Intercept equal to -0.5 and covariate coefficient equal to 0.5.
Treatment equation parameters	Intercept equal to 0.5 and covariate coefficient equal to 0.7.
Instrument relevance	First-stage coefficient $\alpha_z \in \{1.0, 0.5, 0.2\}$, interpreted as strong, medium, and weak relevance, respectively.
Panel B. Latent-dependence designs	
Positive latent dependence	Clayton $C0$ copula with Kendall's $\tau_0 = 0.10$.
Negative latent dependence	Rotated Clayton $C270$ copula with Kendall's $\tau_0 = -0.10$.
Latent margins	Standard normal margins for both unobserved components.
Panel C. Estimated models	
Outcome margin	Probit margin for the binary outcome.
Treatment margin	Gaussian margin for the continuous treatment.
Estimated copulas	Gaussian N, Clayton $C0$, rotated Clayton $C90$, rotated Clayton $C270$, Gumbel $G0$, rotated Gumbel $G90$, and rotated Gumbel $G270$.
Benchmark estimators	Linear instrumental variables and classical control-function probit.
Random seed	88.

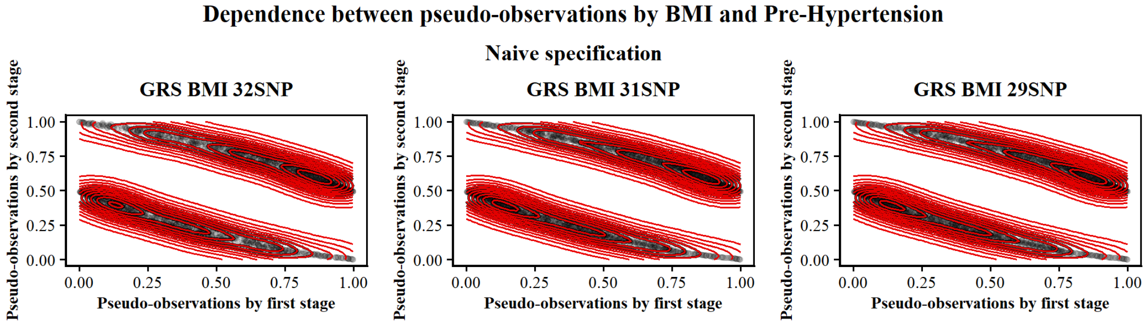
Notes: The table summarizes the data-generating processes used in the Monte Carlo simulations. The weak-support design restricts the empirical variation of the excluded instrument by drawing Z_i from a normal distribution with variance 0.4^2. The coefficient α_z indexes first-stage relevance. The positive- and negative-dependence designs differ only in the copula used to generate the latent disturbances. For each data-generating process, the same set of copula-based models is estimated.

The covariate X_i is drawn from a standard normal distribution to provide a normalized source of observed heterogeneity. The excluded instrument Z_i is drawn from $N(0, 0.4^2)$ in order to impose limited support variation, which makes the design more demanding for identification and estimation. Conditional on this restricted support, instrument strength is varied through the first-stage coefficient $\alpha_z \in \{1.0, 0.5, 0.2\}$. These values generate strong, medium, and weak first-stage relevance while keeping the support of the instrument fixed. Thus, the simulations separate the role of instrument support from the role of instrument relevance.

The structural coefficient is fixed at $\gamma_0 = 2.0$ to ensure a clear latent treatment effect in the binary-response equation, while the intercepts and covariate coefficients are chosen to produce non-degenerate outcome probabilities and a continuous treatment with meaningful variation. The dependence parameter is set to $|\tau_0| = 0.10$, representing modest latent endogeneity. This choice is intentionally close to independence, making the recovery of the dependence structure nontrivial, while still allowing the estimator to distinguish positive from negative residual association. The positive-dependence design uses a standard Clayton copula, whereas the negative-dependence design uses a 270-degree rotated Clayton copula. Holding the absolute value of Kendall's τ_0 fixed across the two designs isolates the effect of the sign and orientation of latent dependence.

Finally, the estimated copula set includes both the correctly specified copula and several misspecified alternatives. This allows the Monte Carlo exercise to evaluate not only recovery under correct specification, but also the sensitivity of the structural coefficient to alternative assumptions about latent dependence. The linear IV and control-function probit estimators are included as benchmarks because they represent common alternatives that do not fully model the mixed binary-continuous triangular structure with flexible copula-based dependence.

A.3 Pseudo-observations of Residual Dependence



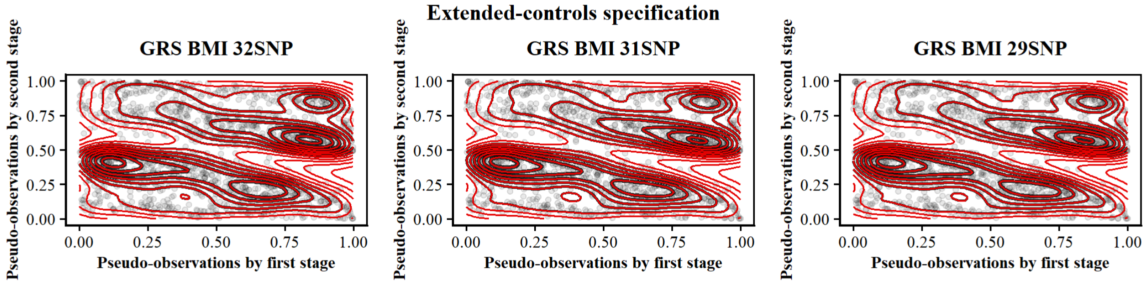


Figure A1: Pseudo-observation dependence between the BMI and pre-hypertension residuals. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.

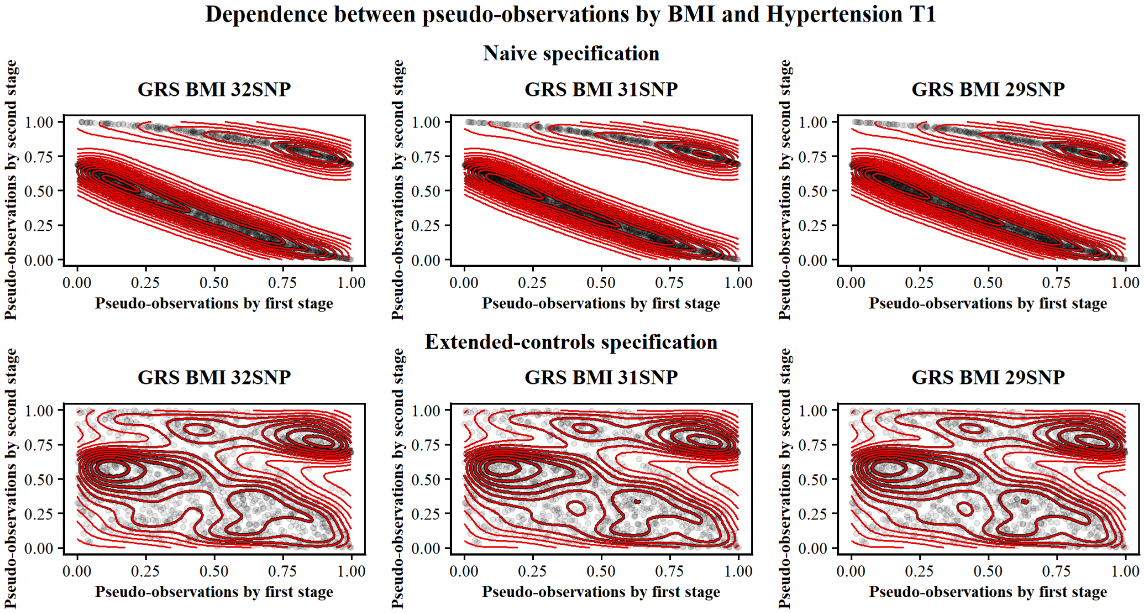


Figure A2: Pseudo-observation dependence between the BMI and stage 1 hypertension residuals. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.

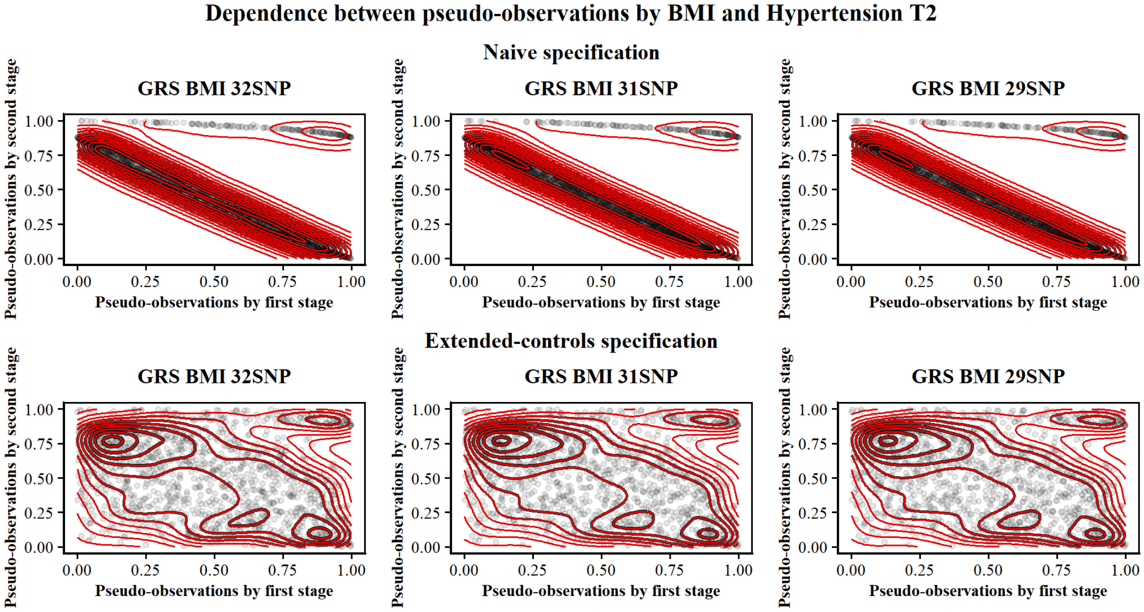


Figure A3: Pseudo-observation dependence between the BMI and stage 2 hypertension residuals. Panels compare naive and extended-control specifications across the `GRSBMI32`, `GRSBMI31`, and `GRSBMI29` instruments.

A.4 Copula Dependence Estimates by Hypertension Outcome

Table A3: Estimated copula parameter θ and Kendall's τ by Pre-Hypertension

Naive Models								
Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)	`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)	`G0`	1.0000 (1.0000, 17.0000)	0.0000 (0.0000, 0.9412)
`G90`	−1.4598 (−2.4270, −1.1873)	−0.3150 (−0.4810, −0.1478)	`G90`	−1.4755 (−2.4832, −1.1960)	−0.3223 (−0.4940, −0.1524)	`G90`	−1.4963 (−2.4229, −1.2174)	−0.3317 (−0.4934, −0.1676)
`G270`	−1.0000 (−17.0000, −1.0000)	0.0000 (−0.9412, −0.0000)	`G270`	−1.0000 (−17.0000, −1.0000)	0.0000 (−0.9412, −0.0000)	`G270`	−1.0000 (−17.0000, −1.0000)	0.0000 (−0.9412, −0.0000)
`C0`	0.4742 (0.2528, 0.9980)	0.1917 (0.1035, 0.3054)	`C0`	0.5305 (0.3022, 1.0161)	0.2096 (0.1243, 0.3121)	`C0`	0.5233 (0.2957, 1.0213)	0.2074 (0.1204, 0.3131)
`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)	`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)	`C90`	0.0000 (−28.0000, −0.0000)	0.0000 (−0.9333, −0.0000)
`C270`	−0.2266 (−0.5008, −0.1134)	−0.1018 (−0.1784, −0.0592)	`C270`	−0.2160 (−0.4791, −0.1080)	−0.0975 (−0.1714, −0.0567)	`C270`	−0.2189 (−0.4870, −0.1089)	−0.0987 (−0.1744, −0.0573)
`N`	−0.6177 (−0.8694, −0.2304)	−0.4239 (−0.6663, −0.1444)	`N`	−0.9332 (−0.9853, −0.7965)	−0.7660 (−0.8848, −0.5439)	`N`	−0.9342 (−0.9900, −0.7116)	−0.7677 (−0.9101, −0.4663)
Extended Controls Models								
Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0008 (1.0000, 17.0000)	0.0008 (0.0000, 0.9412)	`G0`	1.0281 (1.0003, 2.9874)	0.0273 (0.0002, 0.8078)	`G0`	1.0213 (1.0001, 7.0586)	0.0208 (0.0000, 0.9291)
`G90`	−1.0655 (−1.1843, −1.0216)	−0.0615 (−0.1436, −0.0209)	`G90`	−1.0579 (−1.1644, −1.0189)	−0.0547 (−0.1315, −0.0185)	`G90`	−1.0596 (−1.1689, −1.0195)	−0.0562 (−0.1338, −0.0189)
`G270`	−1.0774 (−1.5787, −1.0092)	−0.0718 (−0.3100, −0.0086)	`G270`	−1.0541 (−1.6185, −1.0041)	−0.0513 (−0.4929, −0.0034)	`G270`	−1.0627 (−1.8790, −1.0070)	−0.0590 (−0.3727, −0.0089)
`C0`	0.1041 (0.0076, 1.2381)	0.0495 (0.0031, 0.4950)	`C0`	0.1489 (0.0185, 0.9715)	0.0693 (0.0110, 0.3334)	`C0`	0.1366 (0.0137, 1.0485)	0.0639 (0.0087, 0.3553)
`C90`	−0.0240 (−20.9300, −0.0000)	−0.0119 (−0.9333, −0.0000)	`C90`	−0.0038 (−28.0000, −0.0000)	−0.0019 (−0.9333, −0.0000)	`C90`	−0.0099 (−28.0000, −0.0000)	−0.0049 (−0.9333, −0.0000)
`C270`	−0.1299 (−0.3581, −0.0629)	−0.0610 (−0.1408, −0.0246)	`C270`	−0.1195 (−0.3436, −0.0557)	−0.0564 (−0.1373, −0.0217)	`C270`	−0.1218 (−0.3481, −0.0572)	−0.0574 (−0.1384, −0.0223)
`N`	−0.5927 (−0.8266, −0.1405)	−0.4039 (−0.6414, −0.0472)	`N`	−0.6412 (−0.8787, 0.0702)	−0.4431 (−0.7391, 0.0489)	`N`	−0.7123 (−0.9073, −0.1367)	−0.5047 (−0.7384, −0.0719)

Notes: The table reports the estimated copula parameter $\widehat{\theta}$ and Kendall's $\widehat{\tau}$, with 95% confidence intervals in parentheses. BMI is treated as an endogenous continuous variable and is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Kendall's $\widehat{\tau}$ measures latent residual dependence between the BMI equation and the binary-outcome equation, not the observed association between BMI and the outcome. Boundary estimates should be interpreted cautiously.

Table A4: Estimated copula parameter θ and Kendall's τ by Hypertension T2

Naive Models

Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0000 $(1.0000, 17.0000)$	0.0000 $(0.0000, 0.9412)$	`G0`	1.0000 $(1.0000, 17.0000)$	0.0000 $(0.0000, 0.9412)$	`G0`	1.0000 $(1.0000, 17.0000)$	0.0000 $(0.0000, 0.9412)$
`G90`	-1.4598 $(-2.4270, -1.1873)$	-0.3150 $(-0.4810, -0.1478)$	`G90`	-1.4755 $(-2.4832, -1.1960)$	-0.3223 $(-0.4940, -0.1524)$	`G90`	-1.4963 $(-2.4229, -1.2174)$	-0.3317 $(-0.4934, -0.1676)$
`G270`	-1.0000 $(-17.0000, -1.0000)$	0.0000 $(-0.9412, -0.0000)$	`G270`	-1.0000 $(-17.0000, -1.0000)$	0.0000 $(-0.9412, -0.0000)$	`G270`	-1.0000 $(-17.0000, -1.0000)$	0.0000 $(-0.9412, -0.0000)$
`C0`	0.0000 $(0.0000, 28.0000)$	0.0000 $(0.0000, 0.9333)$	`C0`	0.0776 $(0.0000, 28.0000)$	0.0374 $(0.0000, 0.9333)$	`C0`	0.0623 $(0.0000, 28.0000)$	0.0302 $(0.0000, 0.9333)$
`C90`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$	`C90`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$	`C90`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$
`C270`	-0.0499 $(-0.4748, -0.0040)$	-0.0243 $(-0.1909, -0.0026)$	`C270`	-0.0395 $(-0.6593, -0.0017)$	-0.0193 $(-0.2449, -0.0012)$	`C270`	-0.0400 $(-0.6472, -0.0018)$	-0.0196 $(-0.2416, -0.0013)$
`N`	-0.8188 $(-0.9518, -0.5696)$	-0.6107 $(-0.7781, -0.3582)$	`N`	-0.9016 $(-0.9853, -0.6237)$	-0.7151 $(-0.8830, -0.3598)$	`N`	-0.9013 $(-0.9839, -0.6421)$	-0.7148 $(-0.8786, -0.3781)$

Extended Controls Models

Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$	`Copula`	$\widehat{\theta}$	$\widehat{\tau}$
`G0`	1.0000 $(1.0000, 17.0000)$	0.0000 $(0.0000, 0.9412)$	`G0`	1.0114 $(1.0000, 4.9994)$	0.0112 $(0.0000, 0.9120)$	`G0`	1.0085 $(1.0000, 15.8639)$	0.0084 $(0.0000, 0.9412)$
`G90`	-1.0655 $(-1.1843, -1.0216)$	-0.0615 $(-0.1436, -0.0209)$	`G90`	-1.0579 $(-1.1644, -1.0189)$	-0.0547 $(-0.1315, -0.0185)$	`G90`	-1.0596 $(-1.1689, -1.0195)$	-0.0562 $(-0.1338, -0.0189)$
`G270`	-1.0774 $(-1.5787, -1.0092)$	-0.0718 $(-0.3100, -0.0086)$	`G270`	-1.0541 $(-1.6185, -1.0041)$	-0.0513 $(-0.4929, -0.0034)$	`G270`	-1.0627 $(-1.8790, -1.0070)$	-0.0590 $(-0.3727, -0.0089)$
`C0`	0.0000 $(0.0000, 28.0000)$	0.0000 $(0.0000, 0.9333)$	`C0`	0.0000 $(0.0000, 28.0000)$	0.0000 $(0.0000, 0.9333)$	`C0`	0.0000 $(0.0000, 28.0000)$	0.0000 $(0.0000, 0.9333)$
`C90`	-0.1941 $(-0.7371, -0.0463)$	-0.0885 $(-0.2438, -0.0220)$	`C90`	-0.1525 $(-0.7161, -0.0290)$	-0.0708 $(-0.2376, -0.0140)$	`C90`	-0.1554 $(-0.7165, -0.0301)$	-0.0721 $(-0.2382, -0.0146)$
`C270`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$	`C270`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$	`C270`	0.0000 $(-28.0000, -0.0000)$	0.0000 $(-0.9333, -0.0000)$
`N`	-0.8353 $(-0.9397, -0.4800)$	-0.6294 $(-0.7907, -0.3734)$	`N`	-0.8489 $(-0.9703, -0.2301)$	-0.6454 $(-0.8482, -0.2430)$	`N`	-0.8533 $(-0.9681, -0.3020)$	-0.6508 $(-0.8430, -0.2795)$

Notes: The table reports the estimated copula parameter $\widehat{\theta}$ and Kendall's $\widehat{\tau}$, with 95% confidence intervals in parentheses. BMI is treated as an endogenous continuous variable and is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Kendall's $\widehat{\tau}$ measures latent residual dependence between the BMI equation and the binary-outcome equation, not the observed association between BMI and the outcome. Boundary estimates should be interpreted cautiously.

A.5 Copula-Based Estimates by Hypertension Outcome

Table A5: Copula-based estimates for Pre-hypertension

Naive Models

Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)
C0	0.0374*	0.6955**	C0	0.0317†	0.3137	C0	0.0324†	0.3285
	(0.0184)	(0.2349)		(0.0177)	(0.2389)		(0.0179)	(0.2445)
C90	0.0948***	0.8597***	C90	0.0948***	0.4899*	C90	0.0948***	0.5284*
	(0.0089)	(0.2358)		(0.0089)	(0.2449)		(0.0089)	(0.2485)
C270	0.1540***	0.9082***	C270	0.1520***	0.5249*	C270	0.1526***	0.5720*
	(0.0175)	(0.2317)		(0.0173)	(0.2406)		(0.0174)	(0.2442)
N	0.2159***	0.8657***	N	0.2539***	0.2896†	N	0.2528***	0.3390
	(0.0315)	(0.2336)		(0.0059)	(0.1673)		(0.0058)	(0.2145)
G0	0.0948***	0.8597***	G0	0.0948***	0.4899*	G0	0.0948***	0.5284*
	(0.0089)	(0.2358)		(0.0089)	(0.2449)		(0.0089)	(0.2485)
G90	0.2171***	0.8623***	G90	0.2185***	0.5395*	G90	0.2197***	0.5901**
	(0.0152)	(0.2157)		(0.0151)	(0.2136)		(0.0138)	(0.2148)
G270	0.0948***	0.8597***	G270	0.0948***	0.4899*	G270	0.0948***	0.5284*
	(0.0089)	(0.2358)		(0.0089)	(0.2449)		(0.0089)	(0.2485)

Extended Controls Models

Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)	Copula	$\widehat{\gamma}_{BMI}$ (SE)	$\widehat{\pi}_{GRS}$ (SE)
C0	0.0711***	0.8113***	C0	0.0653**	0.4093†	C0	0.0669**	0.4515†
	(0.0216)	(0.2305)		(0.0214)	(0.2399)		(0.0217)	(0.2451)
C90	0.0885***	0.8507***	C90	0.0858***	0.4593†	C90	0.0866***	0.5077*
	(0.0157)	(0.2291)		(0.0158)	(0.2396)		(0.0159)	(0.2437)
C270	0.1233***	0.8755***	C270	0.1208***	0.4765*	C270	0.1214***	0.5289*
	(0.0178)	(0.2257)		(0.0177)	(0.2349)		(0.0178)	(0.2383)
N	0.2096***	0.8896***	N	0.2184***	0.5531**	N	0.2296***	0.6165**
	(0.0352)	(0.2161)		(0.0441)	(0.2093)		(0.0297)	(0.2083)
G0	0.0850**	0.8414***	G0	0.0754**	0.4466†	G0	0.0777**	0.4912*
	(0.0259)	(0.2297)		(0.0260)	(0.2375)		(0.0262)	(0.2422)
G90	0.1282***	0.8800***	G90	0.1247***	0.4826*	G90	0.1255***	0.5342*
	(0.0200)	(0.2261)		(0.0190)	(0.2352)		(0.0192)	(0.2388)
G270	0.1076***	0.8873***	G270	0.1014***	0.4994*	G270	0.1038***	0.5560*
	(0.0242)	(0.2293)		(0.0234)	(0.2413)		(0.0241)	(0.2462)

Notes: The table reports copula-based estimates of the BMI coefficient in the latent binary-outcome equation and the genetic-score coefficient in the BMI equation. The parameter $\widehat{\gamma}_{BMI}$ denotes the BMI coefficient, and $\widehat{\pi}_{GRS}$ denotes the coefficient of the excluded BMI genetic score. BMI is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Standard errors are in parentheses. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.

Table A6: Copula-based estimates for Hypertension T2

Naive Models

Panel A: GRS 32SNP			*Panel B: GRS 31SNP*			*Panel C: GRS 29SNP*		
`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`	`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`	`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`
`G0`	0.0957***	0.8597***	`G0`	0.0957***	0.4899*	`G0`	0.0957***	0.5284*
	(0.0103)	(0.2358)		(0.0103)	(0.2449)		(0.0103)	(0.2485)
`G90`	0.2776***	0.8443***	`G90`	0.1057***	0.5014*	`G90`	0.1059***	0.5397*
	(0.0068)	(0.1773)		(0.0185)	(0.2454)		(0.0186)	(0.2490)
`G270`	0.2188***	0.7098**	`G270`	0.2265***	0.3767†	`G270`	0.2263***	0.4070*
	(0.0111)	(0.2298)		(0.0085)	(0.1991)		(0.0086)	(0.2064)
`C0`	0.0957***	0.8597***	`C0`	0.0898**	0.4815†	`C0`	0.0909**	0.5213*
	(0.0103)	(0.2358)		(0.0316)	(0.2488)		(0.0323)	(0.2529)
`C90`	0.0957***	0.8597***	`C90`	0.0957***	0.4899*	`C90`	0.0957***	0.5284*
	(0.0103)	(0.2358)		(0.0103)	(0.2449)		(0.0103)	(0.2485)
`C270`	0.1095***	0.8793***	`C270`	0.1067***	0.5026*	`C270`	0.1068***	0.5411*
	(0.0186)	(0.2366)		(0.0186)	(0.2454)		(0.0186)	(0.2490)
`N`	0.2422***	0.8847***	`N`	0.2486***	0.5117*	`N`	0.2485***	0.5452*
	(0.0127)	(0.2331)		(0.0069)	(0.2360)		(0.0069)	(0.2414)

Extended Controls Models

Panel D: GRS 32SNP			*Panel E: GRS 31SNP*			*Panel F: GRS 29SNP*		
`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`	`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`	`Copula`	$\widehat{\gamma}_{BMI}$ `(SE)`	$\widehat{\pi}_{GRS}$ `(SE)`
`G0`	0.0884***	0.8418***	`G0`	0.0810**	0.4470†	`G0`	0.0828**	0.4931*
	(0.0114)	(0.2267)		(0.0273)	(0.2383)		(0.0274)	(0.2428)
`G90`	0.0884***	0.8418***	`G90`	0.0884***	0.4576†	`G90`	0.0884***	0.5026*
	(0.0114)	(0.2267)		(0.0114)	(0.2359)		(0.0114)	(0.2393)
`G270`	0.1263***	0.9088***	`G270`	0.1115***	0.4937*	`G270`	0.1123***	0.5401*
	(0.0340)	(0.2299)		(0.0240)	(0.2369)		(0.0245)	(0.2403)
`C0`	0.0884***	0.8418***	`C0`	0.0884***	0.4576†	`C0`	0.0884***	0.5026*
	(0.0114)	(0.2267)		(0.0114)	(0.2359)		(0.0114)	(0.2393)
`C90`	0.1097***	0.8891***	`C90`	0.1063***	0.4907*	`C90`	0.1065***	0.5373*
	(0.0157)	(0.2268)		(0.0160)	(0.2360)		(0.0159)	(0.2393)
`C270`	0.0884***	0.8418***	`C270`	0.0884***	0.4576†	`C270`	0.0884***	0.5026*
	(0.0114)	(0.2267)		(0.0114)	(0.2359)		(0.0114)	(0.2393)
`N`	0.2468***	0.8971***	`N`	0.2489***	0.5570**	`N`	0.2492***	0.6022**
	(0.0120)	(0.2126)		(0.0136)	(0.2019)		(0.0125)	(0.2061)

Notes: The table reports copula-based estimates of the BMI coefficient in the latent binary-outcome equation and the genetic-score coefficient in the BMI equation. The parameter $\widehat{\gamma}_{BMI}$ denotes the BMI coefficient, and $\widehat{\pi}_{GRS}$ denotes the coefficient of the excluded BMI genetic score. BMI is instrumented, one score at a time, using `GRSBMI32`, `GRSBMI31`, and `GRSBMI29`. Models are estimated in `GJRM` with probit–Normal margins. Standard errors are in parentheses. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.

A.6 Additional SATE Results for Pre-Hypertension and Hypertension T2

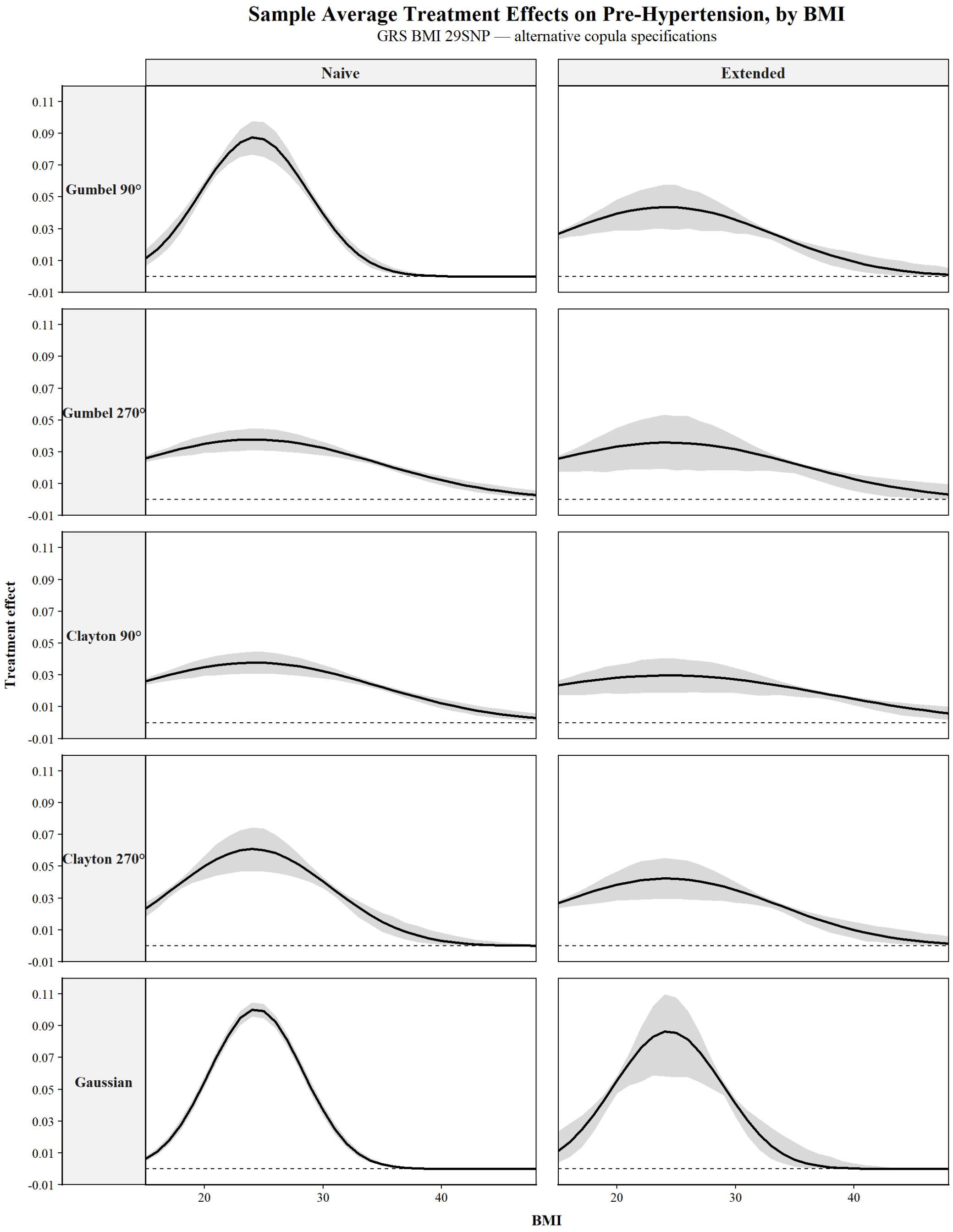


Figure A4: Sample average treatment effects of BMI on Pre-Hypertension, by BMI. SATE estimates are reported using the `GRSBMI29` instrument across alternative copula specifications. Black lines denote point estimates, shaded areas confidence bands, and dashed lines zero effect.

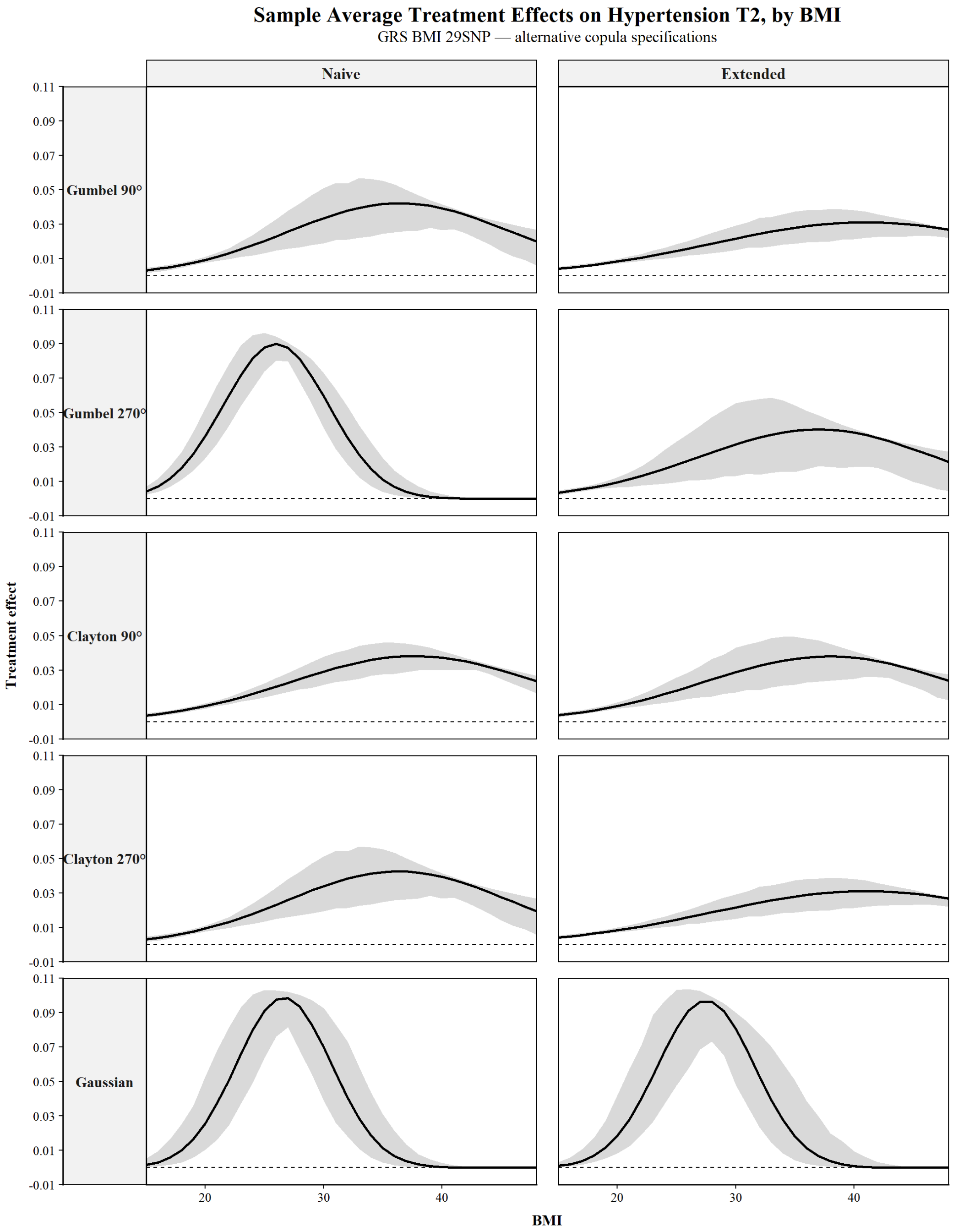


Figure A5: Sample average treatment effects of BMI on Hypertension T2, by BMI. SATE estimates are reported using the `GRSBMI29` instrument across alternative copula specifications. Black lines denote point estimates, shaded areas confidence bands, and dashed lines zero effect.

A.7 Additional Extended-Control Estimates

Table A7: Control-variable coefficients under alternative copulas (Pre-hypertension, `GRSBMI29`)

		Copula specification				
`Control`	`Eq.`	`G90`	`G270`	`C90`	`C270`	`N`
`Age`	Outcome	-0.002 (0.008)	0.000 (0.008)	0.002 (0.008)	-0.002 (0.008)	$-0.015^{\dagger}$ (0.008)
	BMI	0.096^{***} (0.025)	0.094^{***} (0.025)	0.096^{***} (0.025)	0.097^{***} (0.025)	0.096^{***} (0.025)
`Gender: Male`	Outcome	1.016^{***} (0.090)	1.041^{***} (0.091)	1.057^{***} (0.088)	1.017^{***} (0.090)	0.647^{**} (0.243)
	BMI	0.565^{*} (0.265)	0.565^{*} (0.265)	0.568^{*} (0.266)	0.566^{*} (0.265)	0.560^{*} (0.267)
`Mother-hypt`	Outcome	0.181^{*} (0.087)	0.178^{*} (0.087)	0.186^{*} (0.087)	0.182^{*} (0.086)	0.086 (0.089)
	BMI	0.254 (0.262)	0.270 (0.262)	0.266 (0.262)	0.249 (0.262)	0.257 (0.263)
`Father-hypt`	Outcome	0.236^{**} (0.086)	0.224^{*} (0.091)	0.248^{**} (0.087)	0.235^{**} (0.085)	0.036 (0.113)
	BMI	0.816^{**} (0.255)	0.808^{**} (0.256)	0.805^{**} (0.256)	0.806^{**} (0.255)	0.790^{**} (0.257)
`Education`	Outcome	-0.002 (0.014)	-0.002 (0.014)	-0.005 (0.014)	-0.002 (0.014)	0.016 (0.013)
	BMI	-0.117^{**} (0.041)	-0.115^{**} (0.041)	-0.114^{**} (0.041)	-0.115^{**} (0.041)	-0.117^{**} (0.041)
`Smoke`	Outcome	$0.050^{\dagger}$ (0.028)	$0.046^{\dagger}$ (0.027)	$0.049^{\dagger}$ (0.027)	$0.049^{\dagger}$ (0.027)	0.015 (0.028)
	BMI	0.128 (0.083)	0.130 (0.083)	0.125 (0.083)	0.121 (0.083)	0.127 (0.083)
`Alcohol-pd`	Outcome	0.051 (0.042)	0.062 (0.042)	0.064 (0.042)	0.051 (0.042)	0.007 (0.042)
	BMI	0.246^{*} (0.121)	$0.227^{\dagger}$ (0.121)	$0.234^{\dagger}$ (0.121)	0.244^{*} (0.121)	$0.232^{\dagger}$ (0.121)
`Bread-pd`	Outcome	-0.025 (0.018)	-0.022 (0.018)	-0.019 (0.018)	-0.023 (0.018)	-0.033^{*} (0.016)
	BMI	0.116^{*} (0.054)	0.123^{*} (0.054)	0.121^{*} (0.054)	0.117^{*} (0.054)	0.117^{*} (0.054)
`Coffee-pd`	Outcome	0.003 (0.017)	0.002 (0.017)	0.005 (0.017)	0.003 (0.017)	-0.017 (0.016)
	BMI	0.123^{*} (0.050)	0.122^{*} (0.050)	0.122^{*} (0.050)	0.122^{*} (0.051)	0.121^{*} (0.051)
`Milk-pd`	Outcome	0.001 (0.023)	-0.002 (0.023)	0.003 (0.023)	-0.002 (0.023)	$-0.040^{\dagger}$ (0.023)
	BMI	0.254^{***} (0.068)	0.257^{***} (0.069)	0.257^{***} (0.069)	0.254^{***} (0.069)	0.258^{***} (0.069)
`Salt add 2`	Outcome	-0.314^{***} (0.095)	-0.325^{***} (0.094)	-0.314^{***} (0.095)	-0.313^{***} (0.094)	-0.281^{**} (0.094)
	BMI	0.334 (0.285)	0.403 (0.287)	0.371 (0.286)	0.335 (0.285)	0.361 (0.286)
`Salt add 3`	Outcome	0.262 (0.407)	0.235 (0.410)	0.188 (0.412)	0.245 (0.407)	0.481 (0.368)
	BMI	-2.051 (1.268)	-2.023 (1.274)	-2.042 (1.271)	-2.059 (1.270)	-2.057 (1.275)

Notes: The table reports estimated coefficients for the extended controls in the binary-outcome and BMI equations, using `GRSBMI29` as the excluded genetic score. Columns compare the rotated Gumbel copulas `G90` and `G270`, the rotated Clayton copulas `C90` and `C270`, and the Gaussian copula `N`, under probit–Normal margins. Standard errors are in parentheses. The BMI coefficient in the outcome equation, the `GRSBMI29` coefficient in the BMI equation, and intercepts are omitted. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.

Table A8: Control-variable coefficients under alternative copulas (Hypertension T2, `GRSBMI29`)

		Copula specification				
Control	Eq.	G90	G270	C90	C270	N
Age	Outcome	0.023^{*} (0.011)	0.019 (0.011)	$0.020^{\dagger}$ (0.011)	0.023^{*} (0.011)	-0.008 (0.010)
	BMI	0.096^{***} (0.025)	0.097^{***} (0.025)	0.096^{***} (0.025)	0.096^{***} (0.025)	0.095^{***} (0.025)
Gender: Male	Outcome	0.757^{***} (0.119)	0.716^{***} (0.123)	0.743^{***} (0.118)	0.757^{***} (0.119)	0.278 (0.177)
	BMI	0.567^{*} (0.266)	0.562^{*} (0.265)	0.563^{*} (0.265)	0.567^{*} (0.266)	0.564^{*} (0.266)
Mother-hypt	Outcome	0.393^{***} (0.109)	0.366^{***} (0.108)	0.372^{***} (0.108)	0.393^{***} (0.109)	0.156 (0.112)
	BMI	0.265 (0.262)	0.270 (0.261)	0.274 (0.261)	0.265 (0.262)	0.261 (0.262)
Father-hypt	Outcome	0.282^{**} (0.108)	0.232^{*} (0.114)	0.246^{*} (0.110)	0.282^{**} (0.108)	-0.019 (0.107)
	BMI	0.805^{**} (0.256)	0.799^{**} (0.256)	0.792^{**} (0.256)	0.805^{**} (0.256)	0.795^{**} (0.256)
Education	Outcome	-0.015 (0.017)	-0.012 (0.017)	-0.012 (0.017)	-0.015 (0.017)	0.016 (0.014)
	BMI	-0.114^{**} (0.041)	-0.115^{**} (0.041)	-0.116^{**} (0.041)	-0.114^{**} (0.041)	-0.116^{**} (0.041)
Smoke	Outcome	0.107^{**} (0.038)	0.101^{**} (0.037)	0.099^{**} (0.037)	0.107^{**} (0.038)	0.033 (0.035)
	BMI	0.124 (0.083)	0.129 (0.083)	0.131 (0.083)	0.124 (0.083)	0.126 (0.083)
Alcohol-pd	Outcome	0.113^{*} (0.045)	0.105^{*} (0.045)	0.109^{*} (0.045)	0.113^{*} (0.045)	0.013 (0.044)
	BMI	$0.234^{\dagger}$ (0.121)	$0.220^{\dagger}$ (0.121)	$0.221^{\dagger}$ (0.121)	$0.234^{\dagger}$ (0.121)	$0.232^{\dagger}$ (0.121)
Bread-pd	Outcome	-0.017 (0.022)	-0.023 (0.022)	-0.021 (0.022)	-0.017 (0.022)	-0.033^{*} (0.016)
	BMI	0.121^{*} (0.054)	0.123^{*} (0.054)	0.122^{*} (0.054)	0.121^{*} (0.054)	0.117^{*} (0.054)
Coffee-pd	Outcome	0.026 (0.021)	0.022 (0.021)	0.022 (0.021)	0.026 (0.021)	-0.011 (0.017)
	BMI	0.122^{*} (0.050)	0.121^{*} (0.050)	0.123^{*} (0.050)	0.122^{*} (0.050)	0.120^{*} (0.051)
Milk-pd	Outcome	0.011 (0.028)	0.001 (0.029)	0.005 (0.028)	0.011 (0.028)	-0.044^{*} (0.022)
	BMI	0.257^{***} (0.069)	0.253^{***} (0.068)	0.252^{***} (0.069)	0.257^{***} (0.069)	0.259^{***} (0.069)
Salt add 2	Outcome	-0.122 (0.127)	-0.123 (0.121)	-0.132 (0.123)	-0.122 (0.127)	-0.137 (0.089)
	BMI	0.369 (0.285)	0.390 (0.285)	0.386 (0.285)	0.369 (0.285)	0.362 (0.285)
Salt add 3	Outcome	0.529 (0.526)	0.573 (0.506)	0.566 (0.513)	0.529 (0.526)	$0.684^{\dagger}$ (0.385)
	BMI	-2.044 (1.271)	-2.034 (1.266)	-2.056 (1.268)	-2.044 (1.271)	-2.054 (1.273)

Notes: The table reports estimated coefficients for the extended controls in the binary-outcome and BMI equations, using `GRSBMI29` as the excluded genetic score. Columns compare the rotated Gumbel copulas `G90` and `G270`, the rotated Clayton copulas `C90` and `C270`, and the Gaussian copula `N`, under probit–Normal margins. Standard errors are in parentheses. The BMI coefficient in the outcome equation, the `GRSBMI29` coefficient in the BMI equation, and intercepts are omitted. Significance: $^{***}p < 0.001$, $^{**}p < 0.01$, $^{*}p < 0.05$, and $^{\dagger}p < 0.10$.