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A Copula-Based Piecewise Health Concentration Curve for Modeling Stress-Related Performance Inequalities

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Abstract ■ Health concentration curves are essential tools for quantifying inequalities in health outcomes across socioeconomic or biological gradients. In this study, we propose a novel copula-based piecewise health concentration curve constructed with the gluing copula technique and use it to reveal important insights into how stress-related biomarkers relate to inequalities in concentration performance. We employ stress-related concentration performance data for modeling localized changes in the dependence structure between concentration performance and stress-related variables, including cortisol, alpha-amylase responses, state anxiety, and cognitive stress appraisal. Through the application of this technique to experimental data from stress and unstress groups, we demonstrate how health concentration curves diverge from the line of equality and highlight stress-related biomarker ranges where concentration differences in performance are most noticeable. Our results show significant variations in concentration inequalities across stress biomarkers, emphasizing the significance of taking nonlinear dependence into account when evaluating how stress affects cognitive function. This copula-based approach offers an effective instrument for measuring and comparing health disparities in situations where the ability to focus under pressure is crucial, providing information for focused interventions aimed at minimizing performance gaps.

Keywords ■ Health concentration curve, gluing copula, concentration perfomance, cortisol, stress..

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Introduction

In health economics, a concentration curve can be used to quantify the inequality in health outcomes (such as life expectancy, access to care, etc.) between various socioeconomic categories. In order to evaluate the equitable distribution of health outcomes across the population, a health concentration curve can be emloyed. From lowest to the richest, the curve ranks the population by socioeconomic variables, then plots the cumulative proportion of the health variable against the cumulative percentage of the population.

A concentration measure that considers a particular weight function that reflects the aversion to socioeconomic health inequalities was first presented by Wagstaff et al. (1989). In the study by Wagstaff et al. (1991), a comprehensive evaluation of the several approaches used to quantify health disparities was provided. Kakwani et al. (1997) clarified the relationship between two widely used indices of health inequality and explained the significant role that demographic standardization plays in the analysis of socioeconomic inequalities in health. Laskowska (2012) utilized the health concentration index to measure health status distribution inequality caused by economic status.

Bishop et al. (1994) presented an estimation process for Lorenz and concentration curves from individual-level data. Concentration curves often use rank-based or linear connections, which may not adequately represent complicated, asymmetric, or nonlinear dependencies between variables. With this motivation, Bouezmarni et al. (2022) presented the health concentration curve (CH) in terms of copula and derived its semi/non-parametric estimator.

They calculated the disparities in COVID-19 infections and deaths and determined which socioeconomic groups were most vulnerable to contracting the virus and passing away. Using copula in health concentration curve has some advantages, such as they can capture nonlinear and tail dependence and also they are flexible across distributions.

Let H be a health random variable with its marginal distribution function F_H, Y be a socioeconomic random variable with its marginal distribution function F_Y and $F_{H|Y}$ be the conditional distribution of H given Y, then the general case of health concentration curve for $p \in (0,1)$ is given as

$$CH(p) = \frac{\int_0^p E[H \mid Y = F_Y^{-1}(u)]\, du}{\int_0^1 E[H \mid Y = F_Y^{-1}(u)]\, du} \tag{1}$$

where $u \in (0,1)$ and the quantile function of Y is given by $F_Y^{-1}(u) = \inf\{t : F_Y(t) \geq u\}$.

The dependence relation between H and Y is very essential in health concentration curve $CH(p)$. Health data often contain complex, nonlinear dependencies (e.g., diabetes and cardiovascular disease risk). So, copula models can be used in epidemiology, biostatistics, and health economics for comprehending dependencies and inequities in health-related data. Copulas can explain the relationships between various health issues among socioeconomic categories, whereas health concentration curves can demonstrate which groups suffer the most.

By Sklar's theorem (Sklar, 1959), any joint distribution function F of (H, Y) can be represented in terms of its marginal distribution functions and a copula function C as

$$F(h, y) = C(F_H(h), F_Y(y)) = C(u, v),$$

where $u = F_H(h)$ and $v = F_Y(y)$). Here, $C : [0,1]^2 \to [0,1]$ is a bivariate copula function, which is unique when the marginal distributions are continuous.

If F_H and F_Y are absolutely continuous, the joint density function can be written as

$$f(h, y) = c(u, v)\, f_H(h)\, f_Y(y),$$

where f_H and f_Y denote the marginal density functions, and $c(u, v)$ is the copula density defined as

$$c(u, v) = \frac{\partial^2 C(u, v)}{\partial u\, \partial v}.$$

Following the study by Bouezmarni et al. (2022), we define the health concentration curve in terms of copula as,

$$CH(p) = \frac{\int_0^1 F_H^{-1}(u) C^{(u)}(u, p) du}{E(H)}, p \in (0,1), \tag{2}$$

where $C^{(u)}(u, p)$ is the partial derivative of the copula given as

$$C^{(u)}(u, v) = \frac{\partial C(u, v)}{\partial u} = \int_0^v c(u, z) dz.$$

Compared to conventional linear methods, a copula-based approach to piecewise health concentration curve construction provides a more nuanced picture of health inequalities by precisely modeling localized changes in dependence (e.g., different relations in different quantile ranges) through the gluing of copulas. In copula modeling, the glue copula approach can be employed to combine several dependence structures for distinct dataset segments.

The lack of studies exploring this approach reveals a significant gap in current research. The primary aim of this study is to close this gap by using gluing copula techniques to analyze health inequality. We present a gluing copula in the context of health concentration curves which is motivated by the necessity of appropriately capturing varied dependence structures in the analysis of health inequalities. Gluing copula was presented first in Siburg and Stoimenov (2008). Then Erdely (2017) presented the copula-based piecewise regression concept. By scaling and gluing a finite number of n-copulas, they introduced a method for creating n-copulas. For bivariate copulas, gluing results in a copula that aligns with the independence copula on a grid of vertical and horizontal segments.

Given two bivariate copulas, C_1 and C_2 and a fixed value $0 < \theta < 1$ which is a gluing point, C_1 with its dependence paremeter α_1 is scaled to $[0, \theta] \times [0, 1]$ and C_2 with its dependence paremeter α_2 is scaled to $[\theta, 1] \times [0, 1]$ and glue them into a single copula such as

$$C_{1,2,\theta}(u, v) = \begin{cases} \theta\, C_{1,\alpha_1}\left(u, \frac{v}{\theta}\right), & 0 \leq v \leq \theta, \\ (1-\theta)\, C_{2,\alpha_2}\left(u, \frac{v-\theta}{1-\theta}\right) + \theta u, & \theta \leq v \leq 1, \end{cases} \tag{3}$$

where the density function is given as

$$c_{1,2,\theta}(u, v) = \begin{cases} c_{1,\alpha_1}\left(u, \frac{v}{\theta}\right), & 0 \leq v \leq \theta, \\ c_{2,\alpha_2}\left(u, \frac{v-\theta}{1-\theta}\right), & \theta \leq v \leq 1, \end{cases} \tag{4}$$

and partial derivative of the given copula,

$$\frac{\partial C_{1,2,\theta}(u, v)}{\partial u} = \begin{cases} \theta\, \frac{\partial}{\partial u} C_{1,\alpha_1}\left(u, \frac{v}{\theta}\right), & 0 \leq v \leq \theta, \\ (1-\theta)\, \frac{\partial}{\partial u} C_{2,\alpha_2}\left(u, \frac{v-\theta}{1-\theta}\right) + \theta, & \theta \leq v \leq 1. \end{cases} \tag{5}$$

In this study, we apply health concentration curves to concentration performance data from groups experiencing stress and those not under stress. In the literature, there are many studies related to stress and concentration performance or control of attention. Bates and Lemay (2004) studied on attention comprises multiple active cognitive processes. The study examined the construct validity and internal consistency of the d2 Test of Attention. Krumm et al. (2008) investigated how various tests affected the correlation between two distinct sustained attention (SA) tests and a selective attention test.

Although the effects of acute stress on concentration performance are still unknown, it can have both positive and negative impacts on cognitive processing. See also Sandi (2013), Shields et al. (2016). The choice of a copula-based piecewise health concentration curve for this data may be required because complex, nonlinear and asymmetric dependencies between biological stress markers such as cortisol, alpha-amylase, anxiety and performance outcomes are likely to impact the ability to maintain concentration following stress. We can more accurately describe these dependence structures using copula models than with linear correlation or regression. In order to determine if particular stress response thresholds disproportionately degrade attention, piecewise gluing copulas can also be used to detect localized variations in association patterns across various stress biomarker ranges. Technically, we demonstrate methods for evaluating whether a health concentration curve deviates significantly from an equal copula, as well as for testing whether two concentration curves—potentially representing stress and unstress cases—differ significantly. To our knowledge, this is the first study to apply copula theory to health concentration curves. It fills an important gap in the measurement and interpretation of health inequalities across complex stress-response variables.

The rest of the paper is organized as follows: In the following section, we present a piecewise health concentration curve in the context of gluing copula for various dependence structures. We also give estimation and goodness-of-fit procedures. In the next section, we investigate the effects of acute stress on concentration performance using the piecewise health concentration curve on the data provided by Degroote et al. (2020). The last section is devoted to the conclusion and discussion.

Piecewise health concentration curve

The piecewise health concentration curve can be helpful in situations when health disparity varies across the socioeconomic spectrum. It is a useful tool because it allows for various slopes in different parts of the distribution, giving a more precise and detailed picture of health disparities.

Under frequently used parametric copula families, copula-based regression estimates are unable to replicate the qualitative characteristics of the regression function when it is non-monotone. This happens because such parametric copulas frequently result in monotone regression functions. However, if there is evidence that the underlying regression function is non-monotone, a piecewise regression approach may be used to determine the best copula fit for each piece after breaking down a non-monotone relationship into a piecewise monotonic one. By using copula based health concentration curve,

$$CH(p) = \frac{\int_0^1 F_H^{-1}(u) C_m^{(u)}(u,p)du}{E(H)},$$

the piecewise health concentation curve using glue copula is obtained as,

$$CH(p) = \begin{cases} \dfrac{\theta \displaystyle\int_0^1 F_H^{-1}(u)\, C_{1,\alpha_1}^{(u)}\left(u, \dfrac{p}{\theta}\right) du}{E(H)}, & 0 \le p \le \theta, \\ \dfrac{(1-\theta)\displaystyle\int_0^1 F_H^{-1}(u)\, C_{2,\alpha_2}^{(u)}\left(u, \dfrac{p-\theta}{1-\theta}\right) du + \theta \displaystyle\int_0^1 F_H^{-1}(u)\, du}{E(H)}, & \theta \le p \le 1. \end{cases} \tag{6}$$

Let $C_1 = W(u,v) = \max\left(u+v-1, 0\right)$ where H and Y are countermonotonic variables which are used to model Fréchet-Hoeffding lower bound copula. Let $C_2 = M(u,v) = \min\left(u,v\right)$ where H and Y are comonotone random variables which are used to model Fréchet-Hoeffding upper bound copula. Then,

$$CH(p) = \begin{cases} \dfrac{\theta \int_{1-\frac{p}{\theta}}^{1} F_H^{-1}(u)du}{E(H)} & 0 \le p \le \theta \\ \dfrac{(1-\theta)\int_{0}^{\frac{p-\theta}{1-\theta}} F_H^{-1}(u)du + \theta \int_{0}^{1} F_H^{-1}(u)du}{E(H)} & \theta \le p \le 1 \end{cases}$$

$$= \begin{cases} 1 - \theta L_H(1 - \dfrac{p}{\theta}) & 0 \le p \le \theta \\ (1-\theta)L_H(\dfrac{p-\theta}{1-\theta}) + \theta L_H(1) & \theta \le p \le 1 \end{cases}$$

where L_H is the Lorenz curve of H. It means $CH(p)$ is the percentage of the total overweightness of the (100 p)% of the wealthiest population if H stands for overweightness. Then, piecewise health concentration curve in terms of comonotonic and countermonotonic copula is obtained as

$$CH(p) = \begin{cases} CH_1(p) = \dfrac{\theta \sum_{i=1}^{n} H_i \, C_{1,\alpha_1}^{(u)}\left(\hat{F}_H(H_i), \dfrac{p}{\theta}\right)}{\sum_{i=1}^{n} H_i}, & 0 \le p \le \theta, \\ CH_2(p) = \dfrac{(1-\theta)\sum_{i=1}^{n} H_i \, C_{2,\alpha_2}^{(u)}\left(\hat{F}_H(H_i), \dfrac{p-\theta}{1-\theta}\right) + \theta \sum_{i=1}^{n} H_i}{\sum_{i=1}^{n} H_i}, & \theta \le p \le 1, \end{cases} \tag{7}$$

where

$$\hat{F}_H(h) = \frac{1}{n+1} \sum_{i=1}^{n} \mathbf{1}\{H_i \le h\}.$$

Also Gini health index which is used to estimate the dispersion of health disparities within a specific socioeconomic group for gluing copula of C_1 and C_2 is defined as

$$G = \begin{cases} G_1 = 2\int_0^{\theta} \left(p - CH_1(p)\right) dp, & 0 \le p \le \theta, \\ G_2 = 2\int_{\theta}^{1} \left(p - CH_2(p)\right) dp, & \theta \le p \le 1. \end{cases} \tag{8}$$

Estimation and Goodness-of-fit procedures

This section describes how to fit and assess a gluing copula model for the piecewise health concentration curve. Let $\{(H_i, Y_i),\ i = 1, \ldots, n\}$ be an independent and identically distributed sample of n copies of the vector (H, Y). The health concentration curve with respect to Y, denoted by $\mathrm{CH}(p)$, can be estimated using various ways by estimating the bivariate copula function $C_m^{(u)}(u, p)$. A random pair (H, Y) has a unique copula that is invariant under monotonic rising transformations of the marginals. The pair of ranks $\{(R_i, S_i), i = 1, \ldots, n\}$ of $H(X, Y)$ are associated with the sample are the statistics that preserve the maximum amount of information; see Genest and Favre (2007)

and Oakes (1982).

The pseudo-observations are then obtained using the ranks $\{(R_i, S_i), i = 1, \ldots, n\}$ as

$$\hat{U}_i = \frac{R_i}{n+1}, \qquad i = 1, \ldots, n,$$

and

$$\hat{V}_i = \frac{S_i}{n+1}, \qquad i = 1, \ldots, n.$$

Denoting the two component copulas by C_{1,α_1} and C_{2,α_2} and the glue-point by θ, we estimate the parameter vector $(\alpha_1, \alpha_2, \theta)$. Under a composite null hypothesis $H_0 : C \in \{C_\theta : \theta \in \Theta\}$, the copula parameter θ is estimated nonparametrically by maximising the *pseudo* log-likelihood

$$\ell(\theta) = \sum_{i=1}^{n} \log\big[c_\theta\big(\hat{U}_i, \hat{V}_i\big)\big],$$

where Θ is the parameter space. To test H_0, the empirical copula is obtained as

$$C_n(\mathbf{u}) = \frac{1}{n} \sum_{i=1}^{n} \mathbf{1}\{\hat{U}_i \leq u, \hat{V}_i \leq v\},$$

and then the Cramér–von Mises statistic is computed as

$$S_n = \int_{[0,1]^d} \big[C_n(\mathbf{u}) - C_{\hat{\theta}}(\mathbf{u})\big]^2 \, dC_n(\mathbf{u}).$$

Large values of S_n lead to rejection of H_0; approximate p-values are obtained via a tailored parametric bootstrap as described in Genest et al. (2008).

Data and Empirical results

In this study, we use the data of the study by Degroote et al. (2020). They examined the potential of acute psychosocial stress on concentration ability. The effects of acute stress on concentration performance can have both positive and negative impacts on cognitive processing. Our motivation is to uncover the relation between acute stress and concentration ability using piecewise health concentration curve for both of the direction. The study sample consisted of 47 male subjects in good health. They were assigned at random to either a psychosocial stress scenario (Trier Social Stress Test) or a neutral control task. Prior to and 30 minutes following the stress or control exercise, concentration performance was assessed using the d2 Test of Attention. To facilitate reproducibility, we provide an example R code in the Appendix for computing the copula-based health concentration curve and its pointwise bootstrap confidence intervals.

Alpha amylase and salivary cortisol levels were continuously assessed prior to and for up to one hour following stress. State Trait Anxiety Inventory (STAI) is used to measure state anxiety on multiple occasions, and the Primary Appraisal Secondary Appraisal (PASA) questionnaire to measure anticipatory cognitive stress. Their findings demonstrated better concentration performance following acute psychosocial stress induction, which was anticipated based on lower cortisol levels and higher state anxiety rises. This study, however, is based on simple correlations and regressions, which might not help quantify inequalities in concentration performance.

We model the copula of post-task percentage changes in concentration performance ($\mathrm{D2.2.CP.changes}$) pairwise with cortisol maximum change ($\mathrm{s.cort.change}$), alpha-amylase maximum change ($\mathrm{s.AA.change}$), state anxiety change ($\mathrm{stai.S.2.changes}$) and cognitive stress appraisal ($\mathrm{pasa.stressindex}$) for both stress and unstress groups. Before fitting the copula models, the raw observations are transformed into pseudo-observations based on marginal ranks, namely $\hat{U}_i = R_i/(n+1)$ and $\hat{V}_i = S_i/(n+1)$. Figure 1 presents the pseudo-observation scatter plots for the four pairs of variables. These plots provide a preliminary visual description of the rank-based dependence structure in the unit square and help motivate the use of copula models, since the relationships are not purely linear and may vary across different regions of the distribution.

Normal copula, as a symmetric copula, captures linear dependence well. Similar to the Normal copula, the Student-t copula is also symmetric but has heavier tails and can model joint extreme events. Frank copula is a member of the Archimedean copula family; it is also symmetric and is flexible in moderate dependence scenarios. We obtain the Cramér–von Mises (CvM) goodness-of-fit test results for the given health variable (H) with the selected Y variables pairwise in Table 1. The copula models are selected according to the higher p-values of the CvM test statistic, and the selected copula models are shown in bold in Table 1.

In their work, Degroote et al. (2020) used multiple linear regression analysis with percentage changes in concentration performance as the dependent variable and as independent variables, maximum cortisol and alpha-amylase changes as potential physiological predictors in addition to cognitive stress appraisal and percentage changes in post-task state anxiety as potential psychological predictors. They found cortisol maximum change and state anxiety change to be significant variables in the model, whereas alpha amylase maximum change and cognitive stress appraisal were insignificant variables.

In our models, we model how percentage changes in concentration performance behave together with cortisol maximum change, alpha-amylase maximum change, post-task state anxiety and cognitive stress appraisal for both stress and unstress groups, separately via health concentration curves. The results are given in Figure 2. When the

Table 1 ■ Goodness-of-Fit Test for Gluing copula models of percentage changes in concentration performance (H) and various stress-related variables (Y).

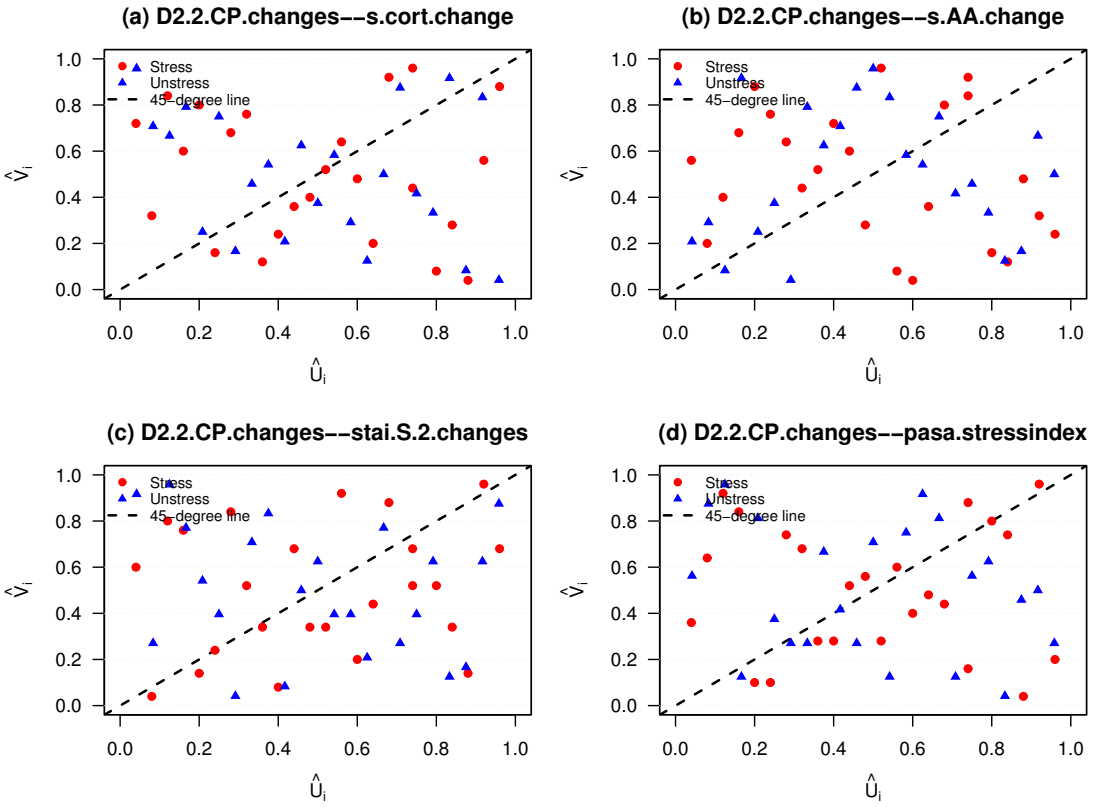
Group	Data(H—Y)	(C_1, C_2)	$\hat{\alpha}_1$	$\hat{\alpha}_2$	$\hat{\theta}$	Test stat.	P-value
Stress	D2.2.CP.changes—s.cort.change	Normal	-0.5320	-0.0259	0.4399	0.0411	0.246
		Student-t	-0.5583	-0.0423	0.3806	0.0355	**0.276**
		Frank	-3.0235	-0.2964	0.4399	0.0396	0.253
	D2.2.CP.changes—s.AA.change	Normal	0.7823	-0.0455	0.1724	0.0248	**0.677**
		Student-t	0.2966	-0.1515	0.2418	0.0293	0.443
		Frank	5.2865	-0.4821	0.1600	0.0260	0.574
	D2.2.CP.changes—stai.S.2.changes	Normal	0.5404	0.2100	0.2398	0.0432	0.241
		Student-t	0.5372	0.1264	0.3840	0.0371	**0.327**
		Frank	-0.3463	-0.1327	0.2800	0.0594	0.081
	D2.2.CP.changes—pasa.stressindex	Normal	0.3730	0.0567	0.2000	0.0285	0.469
		Student-t	0.4168	0.1712	0.2024	0.0262	**0.588**
		Frank	2.1520	1.2868	0.2000	0.0278	0.499
Unstress	D2.2.CP.changes—s.cort.change	Normal	-0.5801	-0.1851	0.4166	0.0235	0.638
		Student-t	-0.5040	-0.0381	0.4043	0.0199	**0.733**
		Frank	-7.1843	-0.8656	0.3333	0.0232	0.581
	D2.2.CP.changes—s.AA.change	Normal	-0.3415	-0.2791	0.2916	0.0712	**0.072**
		Student-t	0.1994	-0.6515	0.4882	0.0770	0.067
		Frank	-0.3749	-4.6385	0.3790	0.0999	0.032
	D2.2.CP.changes—stai.S.2.changes	Normal	0.5999	-0.2014	0.3090	0.0290	0.464
		Student-t	0.4755	-0.1224	0.3504	0.0274	**0.481**
		Frank	5.8915	-1.3292	0.2389	0.0303	0.426
	D2.2.CP.changes—pasa.stressindex	Normal	-0.5024	-0.5417	0.4523	0.0483	**0.194**
		Student-t	-0.4274	0.3713	0.3444	0.0512	0.114
		Frank	-0.4991	-2.2729	0.2708	0.0651	0.043

curve deviates from the equality line, it indicates the presence of inequality: worse (or better) health outcomes are not evenly distributed but are disproportionately concentrated among particular subgroups defined by the ranking variable.

Figure 2 displays copula-based piecewise health concentration curves of health variable H (D2.2.CP) with stress-related variables Y across the stress and unstress subgroups. The health variable H is more concentrated among the lower ranks of the Y variable (lower cortisol, for example) if the curve is above the equality line; conversely, a curve below the equality line indicates concentration among the higher ranks (greater cortisol or anxiety, for example). Figure 2 is constructed based on the selected glue-copula models reported in Table 1, where $\hat{\theta}$ denotes the estimated glue-point and $\hat{\alpha_1}$ and $\hat{\alpha_2}$ represent the estimated dependence parameters of C_{1,α_1} and C_{2,α_2}, respectively. The glue point indicates a change in the dependence structure between the selected variables for both the stress and non-stress groups. These points are generally similar across models, with the exception of D2.2.CP and pasa.stressindex. Furthermore, for most dependence structures, the first segment of the copula model exhibits stronger dependence than the second segment that is $\hat{\alpha_1} > \hat{\alpha_2}$, again with the exception of D2.2.CP and pasa.stressindex.

In Figure 2.a, percentage changes in concentration performance is given with maximum cortisol change

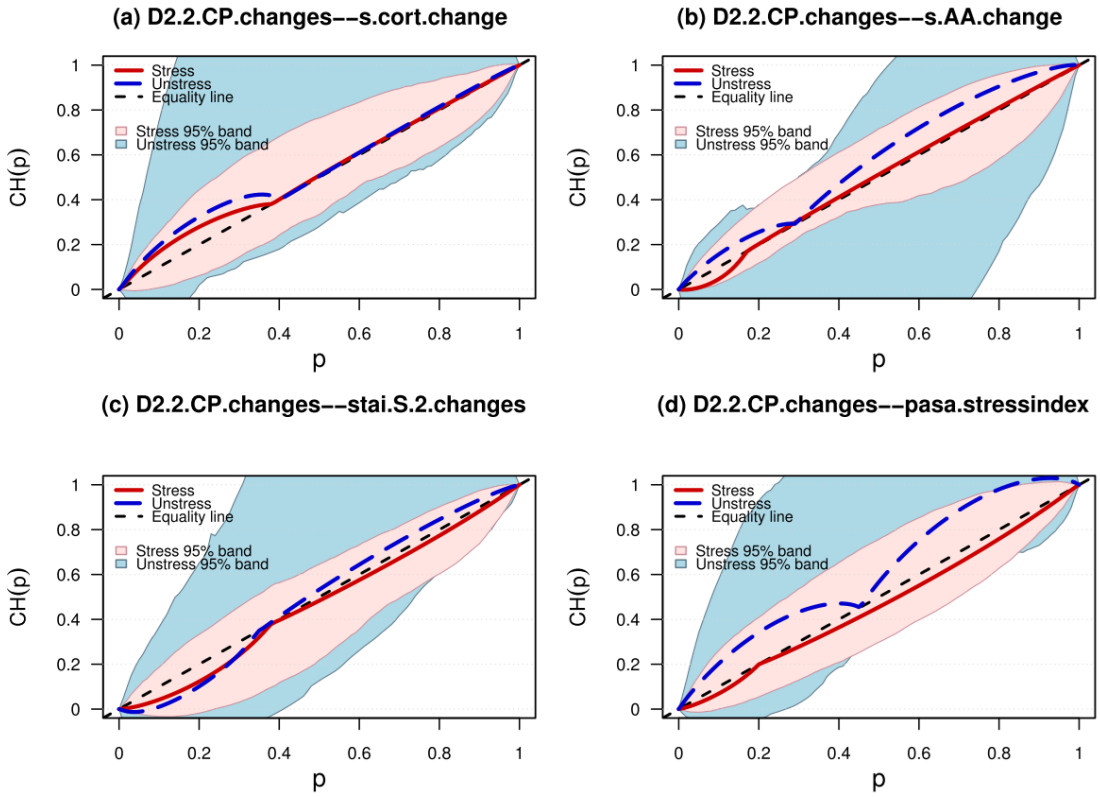
Figure 1 ■ Pseudo-observation scatter plots for the association between $(H = \mathrm{D2.2.CP.changes})$ and the selected stress-related variables (Y) in the stress and unstress groups.



(s.cort.change). Both stress (red) and unstress (blue) curves start above the line of equality until about $p = 0.3$–0.4, then equalized the line of equality before converging near $p = 1$. When we consider the above line, worse concentration performance is concentrated among lower ranks of cortisol change (i.e., those with smaller cortisol increases) and when we consider the below line, worse concentration performance is concentrated among higher ranks of cortisol change (i.e., those with larger cortisol increases). Individuals with smaller cortisol rises perform comparatively poorly (curve above equality) in the lower 30–40% of cortisol change for both stress and unstress groups, however unstress group differs much. The stress and unstress groups do not significantly differ from one another after $p > 0.4$. The bootstrap confidence bands are relatively wider in the lower and middle parts of the distribution, indicating greater uncertainty in the estimated concentration pattern over these ranges. Since the stress and unstress bands largely overlap after the mid-quantiles, the difference between the two fitted curves should be interpreted cautiously, especially beyond the glue-point region.

In Figure 2.b, percentage changes in concentration performance is given with alpha-amylase maximum change (s.AA.change). The unstress group (blue) curve has a greater bowing beneath the line of equality, suggesting that the higher alpha-amylase increases of unstress group are primarily responsible for poorer performance. The distribution of performance decline in the unstress group is considerably more uneven, with a small percentage of poorer performance being attributed to a small number of people with elevated alpha-amylase alterations. This disparity is less noticeable in the stress group. The bootstrap bands are wider for the unstress curve, particularly in the middle and upper parts of the distribution, which reflects the additional uncertainty associated with the stronger departure of the unstress curve from the equality line. Although the blue curve suggests a more pronounced inequality pattern, the broad confidence band implies that this conclu-

Figure 2 ■ Copula-based piecewise health concentration curves CH(P) for stress-unstress groups



sion should be viewed as suggestive rather than definitive.

In Figure 2.c, percentage changes in concentration performance is given with state anxiety change ($\mathrm{stai.S.2.changes}$). Both curves start slightly below the equality line till $p < 0.4$. They have similar behaviour. Larger bow of blue curve (unstress group) below the equality line in the early percentiles indicates that those with higher state anxiety increases, particularly those in the unstressed group, tend to cluster with worse concentration performance. Throughout the range of p, both curves remain near the line of equality. The relation shows a converse pattern with cortisol change till $p < 0.4$. Changes in state anxiety are not strongly associated with inequalities in concentration performance. The bootstrap confidence bands support this moderate interpretation, since the fitted curves remain close to the equality line and the bands cover a relatively broad region around it.Thus, the uncertainty bands do not provide strong evidence of a marked separation between the stress and unstress concentration curves for state anxiety change.

In Figure 2.d, percentage changes in concentration performance is given with cognitive stress appraisal ($\mathrm{pasa.stressindex}$). For early percentiles $p < 0.4$, both curves, but especially the unstress group (blue), rise far above the equality line. It can be concluded that worse concentration performance is disproportionately concentrated among those with lower cognitive stress appraisal scores in this range. The line suggests that individuals with low perceived stress in the unstress group still experienced worse performance, possibly due to underestimating task demands. For higher percentiles $p > 0.5$, the unstress curve climbs dramatically above the equality line again, while the stress group curve approaches the line more gradually. In the unstress group, individuals with extreme high or low appraisal scores contribute disproportionately to performance disparities, while the stress group shows a steadier inequality. It can be concluded that individual differences in stress perception under unstressed conditions can drive big disparities in performance outcomes. The bootstrap confidence bands are particularly wide for the unstress group,indicating that the strong curvature of the blue curve is accompanied by considerable estimation un-

certainty. Nevertheless, the location of the fitted unstress curve above the equality line over a large part of the distribution suggests that cognitive stress appraisal may still be associated with notable heterogeneity in concentration performance, although this pattern should be interpreted with attention to the width of the bands.

We compute Gini Health Index for concentration performance (H) and stress-related variables (Y) given in Table 2. Low values of Gini index indicate small differences. (De Battisti et al., 2019). It can be concluded that concentration performance is mostly the same across stress biomarkers. The absolute magnitude tells the strength of inequality. The Gini calculated at each interval can vary, sometimes even reversing sign, depending on where the worst outcomes are concentrated in that particular part of the distribution. Intervals of Gini turning from negative to positive indicate regime changes: worse outcomes shift from being concentrated among low responders to being concentrated among high responders, or vice versa. Generally, the highest divergence is seen in the unstressed group. Gini values are compatible with the results given in Figure 2.

Although the effects of stress vary depending on the marker, generally speaking, stress tends to bring out disparities in particular biological responses.

Conclusion

Health outcomes become considerably more uneven under stressful environmental or socioeconomic conditions, with varying structures (bottom-heavy, middle-heavy) depending on the underlying connection between health and other variables. The concentration curve is a valuable tool for analyzing inequality in any relevant health sector variable, not only health outcomes. Moreover, it serves to evaluate disparities in health inequalities across different groups.

This study proposes and applies a copula-based piecewise health concentration curve framework to investigate the relationship between stress-related physiological indicators and post-task concentration performance. Gluing copula approaches which allow us to identify localized, nonlinear dependencies that are not captured by conventional methods are used. The findings show that, depending on the level of stress exposure, stress indicators like cortisol, alpha-amylase, and cognitive stress appraisal show differing degrees of inequity in their association with cognitive function. Some highlight points about the stress data application can be listed as:

- For cortisol changes, the unstress group curve exhibits greater inequality associated with changes in cortisol during stress, deviating more from equality. This suggests that poorer concentration ability is disproportionately prevalent among persons exhibiting lower cortisol changes, especially within the unstress group. Both groups show a nonlinear pattern. These models suggest that under non-stressed conditions, individual variability in cortisol response is still significant, with low responders (low cortisol) performing disproportionately worse; under acute stress, this disparity diminishes, presumably because stress affects performance equally across cortisol responses.
- For Alpha-amylase responses, in contrast to the stress group, which shows reduced inequality, alpha-amylase changes of the unstress group show significant inequality, with the worst performance being concentrated among those with the greatest alpha-amylase reactions. The stress group shows a flatter, more consistent curve that is closer to the equity line. It can be concluded that concentration performance under stress is more evenly distributed across alpha-amylase responses, likely because the stressor overrides individual differences in sympathetic nervous system activation.
- For state anxiety, the curve changes show moderate inequality in concentration performance, with poorer performance clustering in individuals with higher anxiety levels in the early percentiles, particularly under stress. However, both curves remain largely close to the equity line, indicating relatively small differences overall when compared with biomarkers such as cortisol or stress assessment. It can be concluded that state anxiety contributes to performance differences, but the degree of inequality is limited and the patterns of inequality are similar between stressl and unstress conditions.
- For cognitive stress appraisal, significant inequality is seen, especially in the unstress group, where the individuals with the highest perceived stress levels perform worse. The stress group exhibits a more moderate, consistent inequality with less dramatic departures from the equity line, suggesting that under actual stress, performance differences across rating levels are more evenly distributed. This curve suggests that misjudging stress can significantly impact cognitive performance even in the absence of acute stress, whereas exposure to actual stress reduces this extreme disparity.

Our findings indicate that the gluing copula technique can identify critical points where interventions may be most required and may provide a powerful new tool for analyzing health inequalities in complex, heterogeneous populations. Future research can extend this framework to longitudinal designs and broader health contexts to better capture stress–performance relations over time.

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Table 2 ■ Gini Health index results for concentration performance (H) and various stress-related variables (Y).

Group	Data(H—Y)	(C_1, C_2)	$\hat{\theta}$	$G_1(0 \leq p \leq \hat{\theta})$	$G_2(\hat{\theta} \leq p \leq 1)$
Stress	D2.2.CP.changes—s.cort.change	Student-t	0.3806	-0.0414	-0.0080
	D2.2.CP.changes—s.AA.change	Normal	0.1724	0.0124	-0.0158
	D2.2.CP.changes—stai.S.2.change	Student-t	0.3840	0.0404	0.0238
	D2.2.CP.changes—pasa.stressindex	Student-t	0.2024	0.0086	0.0544
Unstress	D2.2.CP.changes—s.cort.change	Student-t	0.4043	-0.071	-0.0114
	D2.2.CP.changes—s.AA.change	Normal	0.2916	-0.0254	-0.1226
	D2.2.CP.changes—stai.S.2.change	Student-t	0.3504	0.0502	-0.0438
	D2.2.CP.changes—pasa.stressindex	Normal	0.4523	-0.0908	-0.1440

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Appendix. Example R code

The following R code gives a simple implementation of the copula-based health concentration curve for a generic pair of variables (H, Y). The code first converts the raw observations into pseudo-observations for copula parameter estimation, estimates the copula parameter by maximum pseudo-likelihood, computes the concentration curve values $CH(p)$, and obtains pointwise 95% bootstrap confidence intervals. The same code can be used for Clayton, Gumbel, Frank, Normal and Student-t copulas.

```
library(copula)

## Data frame: dat
## H: health/outcome variable
## Y: ranking variable
H_name <- "H"
Y_name <- "Y"

d <- dat[, c(H_name, Y_name)]
d <- d[complete.cases(d), ]
colnames(d) <- c("H", "Y")
p_grid      <- seq(0.10, 0.90, by = 0.10)
B <- 200

make_cop <- function(fam, par) {
   if (fam == "Clayton")  return(claytonCopula(max(par, 1e-5), dim = 2))
   if (fam == "Gumbel")   return(gumbelCopula(max(par, 1 + 1e-5), dim = 2))
   if (fam == "Frank")
      return(frankCopula(ifelse(abs(par) < 1e-5, 1e-5, par), dim = 2))
   if (fam == "Normal")   return(normalCopula(max(min(par, 0.95), -0.95), dim = 2))
   if (fam == "Student-t")
      return(tCopula(max(min(par, 0.95), -0.95), dim = 2, df = 4, df.fixed = TRUE))
}

fit_par <- function(d, fam) {
   uv <- pobs(as.matrix(d))
   if (fam == "Clayton")  { lo <- 1e-5; hi <- 20; st <- c(0.2, 0.5, 1, 2) }
   if (fam == "Gumbel")   { lo <- 1+1e-5; hi <- 20; st <- c(1.05, 1.2, 1.5, 2) }
   if (fam == "Frank")    { lo <- -20; hi <- 20; st <- c(-5, -2, 2, 5) }
   if (fam %in% c("Normal", "Student-t")) {
      lo <- -0.95; hi <- 0.95; st <- c(-0.5, 0, 0.5)
   }
   nll <- function(par) {
      den <- dCopula(uv, make_cop(fam, par))
      -sum(log(pmax(den, 1e-10)))
   }
   vals <- lapply(st, function(s)
   try(optim(s, nll, method = "L-BFGS-B", lower = lo, upper = hi),
   silent = TRUE))
   vals <- vals[!sapply(vals, inherits, "try-error")]
   vals[[which.min(sapply(vals, function(z) z$value))]]$par
}
```

```r
CH_curve <- function(d, fam, par, p_grid) {
   H <- d[, "H"]
   U <- rank(H, ties.method = "average") / (length(H) + 1)
   cop <- make_cop(fam, par)

   sapply(p_grid, function(p) {
      cc <- cCopula(cbind(U, rep(p, length(U))), copula = cop)
      sum(H * cc[, 2]) / sum(H)
   })
}

boot_CH <- function(d, fam, p_grid, B = 200) {
   par_hat <- fit_par(d, fam)
   CH_hat <- CH_curve(d, fam, par_hat, p_grid)

   boot <- replicate(B, {
      db <- d[sample(1:nrow(d), nrow(d), replace = TRUE), ]
      pb <- try(fit_par(db, fam), silent = TRUE)
      if (inherits(pb, "try-error")) rep(NA, length(p_grid))
      else CH_curve(db, fam, pb, p_grid)
   })

   data.frame(
   p = p_grid,
   CH = CH_hat,
   lower95 = apply(boot, 1, quantile, 0.025, na.rm = TRUE),
   upper95 = apply(boot, 1, quantile, 0.975, na.rm = TRUE)
   )
}

families <- c("Clayton", "Gumbel", "Frank", "Normal", "Student-t")

for (fam in families) {
   cat("\nCopula:", fam, "\n")
   print(round(boot_CH(d, fam, p_grid, B), 4))
}
```

Open practices

The *Open Data* badge was earned because the data of the experiment(s) are available on doi.org/10.23668/psycharchives.2886

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