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Causal associations between scapular morphology and shoulder condition estimated with Bayesian statistics

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ABSTRACT

Background and Objective: While there is a reported correlation between shoulder condition and scapular morphology, the precise impact of typical anatomical variables remains a subject of ongoing debate. This study aimed to evaluate this causal association, by emphasizing the importance of scientific modeling before statistical analysis.

Methods: We examined the effect of scapular anatomy on shoulder condition, and conditioning on sex, age, height, and weight. We considered the two most common pathologies: primary osteoarthritis (OA) and cuff tear arthropathy (CTA). We combined the other pathologies into a single category (OTH) and included a control category (CTRL) of adult subjects without pathology. We represented acromion and glenoid morphology by acromion angle (AA), acromion posterior angle (APA), acromion tilt angle (ATA), glenoid inclination angle (GIA), and glenoid version angle (GVA). GVA was negative for posterior orientation. These variables were automatically calculated from CT scans of 396 subjects in the 4 shoulder condition groups by a deep learning model. We applied do-calculus to assess the identifiability of the causal associations and used a multinomial logistic regression Bayesian model to estimate them. To isolate the effect of each anatomical variable on each shoulder condition, we increased it from -2 to 2 z-score while constraining all other variables to their average value, and reported the effect on shoulder condition probability as percentage points (pp) for females and males.

Results: Increasing AA reduced the probability of OA by 44 pp for females and 17 pp for males while increasing the probability of CTA by 36 pp for females and 33 pp for males. Increasing APA raised the probability of OA by 15 pp for females and 4 pp for males and increased the probability of CTA by 12 pp for females and 4 pp for males. Increasing ATA increased the probability of OA by 15 pp for females but decreased it by 25 pp for males, while also raising the probability of CTA by 11 pp for females and 21 pp for males. Increasing GIA decreased the probability of OA by 55 pp for females and 23 pp for males while increasing the probability of CTA by 45 pp for females and 31 pp for males. GVA (more anterior), decreased the probability of OA by 33 pp for females and 63 pp for males. The effects of APA and ATA were less important compared to the other variables. Overall, morphological effects were more pronounced for females than for males, except for GVA's impact on OA.

Conclusions: We developed a Bayesian causal model to answer interventional questions about the scapular anatomy's effect on shoulder condition. Our results, consistent with clinical knowledge, hold promise for aiding in early pathology detection and optimizing surgical planning within clinical settings.

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1. Introduction

The etiology of primary osteoarthritis (OA) and cuff tear arthropathy (CTA) as main shoulder pathologies is multifactorial and still not fully understood [1]. It is hypothesized that an unfavorable acromion and glenoid shape might initiate or contribute to the development of glenohumeral osteoarthritis and degenerative rotator cuff lesions [2–5]. Understanding acromion and glenoid variations is thus crucial for advancing the knowledge of these shoulder pathologies.

The acromion shape in the sagittal and coronal plane has been described by multiple metrics and their association with different shoulder disorders has been analyzed. Bigliani et al. classified the shape of the acromion on supraspinatus outlet (or lateral) radiographs into three distinct types, flat, curved, and hooked [6]. They reported a higher prevalence of rotator cuff tears for hooked compared to the other types. Aoki et al. [7] introduced another metric called the α angle, also known as acromial tilt [8]. This angle was measured between two lines: one line runs along the undersurface of the acromion, and the other line connects the back bottom edge of the acromion to the bottom edge of the coracoid process. They suggested that a low slope of the acromion might be an important factor in the pathogenesis of subacromial impingement. Nyffeler et al. proposed a method to quantify the lateral extension of the acromion on standardized true anteroposterior (AP) radiographs with the arm in neutral rotation [9]. This method aimed to address the common observation of enlarged acromion in patients with a rotator cuff tear. They introduced the concept of the acromion index (AI), calculated by dividing the distance from the glenoid to the acromion by the distance from the glenoid to the lateral aspect of the humeral head, with a high AI corresponding to a large lateral extension of the acromion. They reported statistically significant differences in AI between patients with a full-thickness rotator cuff tear and the two groups of individuals with intact rotator cuffs: one group with OA and the other as the CTRL group. Later Moor et al. introduced a novel radiological parameter to characterize the lateral extension of the acromion, which is not influenced by arm orientation, glenohumeral joint space width, or humeral head flattening [1]. This parameter termed the critical shoulder angle (CSA), was determined on standardized anteroposterior (AP) radiographs. It involves measuring the angle formed between a line connecting the superior and inferior borders of the glenoid fossa and another line drawn from the inferior border of the glenoid to the most lateral point of the acromion. They concluded that degenerative rotator cuff tears were associated with significantly larger CSAs, while primary glenohumeral osteoarthritis was associated with significantly smaller CSAs compared to asymptomatic shoulders without these pathologies.

The glenoid shape and its association with shoulder condition have been analyzed in numerous studies. The literature showcases various methodologies for defining and measuring glenoid inclination. Churchill et al. examined 172 pairs of dry scapulae, using a line connecting the midpoint of the glenoid surface to the scapular spine-vertebral border junction as Ref. [10]. They found the inclination ranged from −7.0° to +15.8°. Hughes et al. analyzed eight cadaver shoulders using AP radiographs, noting higher inclination in shoulders with full-thickness rotator cuff tears compared to intact ones [11]. Conversely, Kandemir et al. found no significant difference in glenoid inclination between the intact cuff and tear shoulders in their study using a 3D digitizing system on 24 cadaveric shoulders [12]. Bishop et al. examined patient-specific CT-based bone models of 21 patients with rotator cuff tears, finding significantly lower inclination in repaired shoulders compared to asymptomatic ones [13]. The glenoid version defines the orientation of the glenoid cavity in relation to a plane perpendicular to the scapular body [14]. Posteriorly inclined glenoids have been reported to be associated with OA [1,15].

Although various studies have attempted to estimate the effect of scapular anatomical factors on shoulder condition, none have applied a causal model in their analyses. It is necessary to assume a causal model to find the causal effect of anatomy on shoulder condition. A review of the multivariable models in orthopedic research reported that only 3 out of 193 studies published in seven orthopedic journals with the highest-ranked impact factors in 2019 used causal models to interpret the causal inference [16]. This is currently the recommended method for confounder selection.

In this study, we address this gap by considering a causal model for the effect of the anatomical variables on shoulder condition, applying do-calculus [17] to find the minimal adjustment set, and utilizing Bayesian statistics for estimation. Since sex, age, height, and weight were included in the adjustments and hold clinical significance, we also developed statistical models to estimate their impact on the shoulder condition based on the causal model under consideration.

2. Method

2.1. Subjects

From our institution's picture archiving and communication system (PACS), we retrospectively identified and retrieved CT scans of trauma patients for the CTRL group. These CTs were performed between 2014 and 2018 in the emergency department. Patients were excluded when the CT scan, all reviewed by an attending musculoskeletal radiologist, showed signs of shoulder pathology (e.g., OA, rotator cuff tear, fracture or instability/dislocation, rheumatic disease, cancer, or history of surgery). Exclusion criteria were incomplete scapular bone coverage (at least one side's scapula had to be fully included) and a CT protocol deviating from the standardized protocol below. In this way, we obtained CT scans from 242 unique subjects (73 females, 169 males), aged 40 to 84 years (Table 1). From the same PACS, we collected CT scans covering the entire scapula for OA, CTA, and other shoulder pathologies, performed between 2002 and 2018. Identification of pathology was performed by a musculoskeletal radiologist and shoulder surgeon based on CT scans and clinical evaluation. OA cases with apparent glenoid wear were excluded, based on glenoid sphericity and visual inspection by the musculoskeletal radiologist and shoulder surgeon [18]. We obtained 86 CT scans from unique OA subjects (57 females, 29 males), aged 38 to 88 years, 50 CT scans from unique CTA subjects (37 females, 13 males), aged 63 to 89 years, and 18 other pathologies (13 females, 5 males) aged 35 to 85 years (Table 1). The 18 subjects with other pathologies included a range of conditions: 10 fractures (acute or sequelae), 3 osteonecrosis, 3 rheumatoid arthritis, 1 secondary osteoarthritis, and 1 septic arthritis.

2.2. Radiological data

CT scans were performed on an 8-, 64- or 256-detector row CT system (Lightspeed VCT or Revolution CT for CTRL, and Lightspeed Ultra, Lightspeed VCT or Discovery CT750 HD for OA and CTA; GE Healthcare). The relevant standardized data acquisition parameters were: tube potential, 120 kVp; tube current, 150–400 mA; automatic exposure control, enabled; gantry revolution time, 0.5–0.6 s. The relevant parameters for image reconstruction were the field of view, $18 \times 18 - 40 \times 40$ cm^2; section thickness, 1.25 mm; yielding non-isotropic voxels of $0.35 \times 0.35 \times 1.25$–$0.78 \times 0.78 \times 1.25$ mm^3.

2.3. Anatomical measurements

We used a deep learning model for scapula segmentation and landmarking [19]. This model demonstrated high accuracy, achieving an average Dice similarity coefficient of 0.97 ± 0.01 when comparing automatic and manual scapula segmentations, effective across both normal and pathological cases. Furthermore, the automatic landmark identification showed an average deviation of only 1.0 mm to 2.5 mm from the reference points for all landmarks. We defined our regional coordinate system based on these landmarks: the x-axis as posterior-anterior, the

Table 1
Subjects characteristics by shoulder condition and sex.

	OA		CTA		CTRL		OTH	
	Female	Male	Female	Male	Female	Male	Female	Male
n	57	29	37	13	73	169	13	5
Age (years)	73 ± 8	66 ± 11	78 ± 6	75 ± 6	57 ± 12	54 ± 10	67 ± 12	56 ± 11
Height (cm)	161 ± 7	174 ± 6	156 ± 6	168 ± 6	164 ± 6	177 ± 7	162 ± 6	178 ± 6
Weight (kg)	74 ± 14	88 ± 14	63 ± 12	75 ± 14	66 ± 14	83 ± 14	68 ± 16	78 ± 3
AA (degree)	23 ± 7	23 ± 6	27 ± 7	29 ± 7	24 ± 6	25 ± 8	27 ± 7	27 ± 4
APA (degree)	15 ± 3	15 ± 4	15 ± 4	15 ± 3	12 ± 2	13 ± 2	13 ± 3	13 ± 3
ATA (degree)	27 ± 8	20 ± 8	30 ± 8	33 ± 10	27 ± 8	27 ± 8	25 ± 12	30 ± 7
GIA (degree)	6 ± 7	5 ± 6	15 ± 10	10 ± 8	10 ± 5	5 ± 8	11 ± 8	4 ± 4
GVA (degree)	−5 ± 12	−11 ± 11	−3 ± 7	0 ± 7	1 ± 5	1 ± 5	1 ± 8	4 ± 8

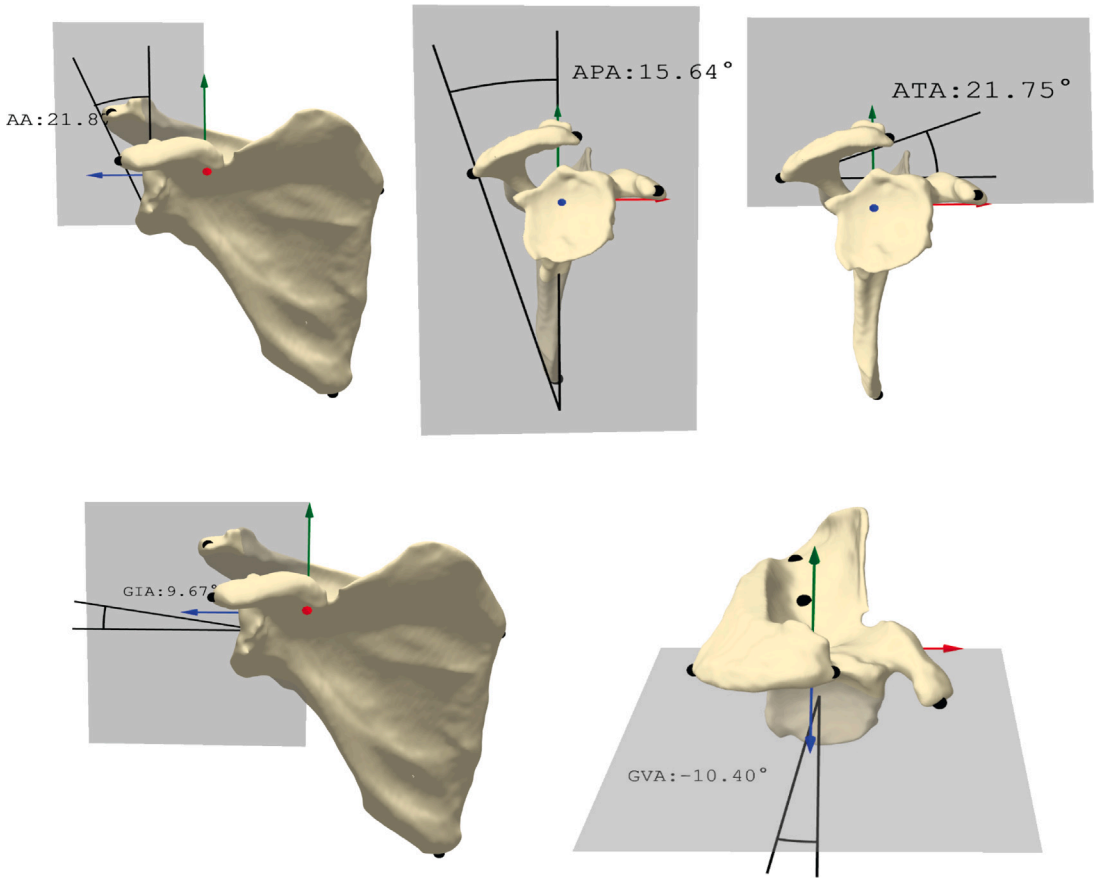


Fig. 1. Illustrative diagram of definitions of acromion angle (AA), acromion posterior angle (APA), acromion tilt angle (ATA), glenoid inclination angle (GIA), and glenoid version angle (GVA).

y-axis as inferior-superior, and the *z*-axis as the medial-lateral axis of the scapula [18]. The origin of the coordinate system corresponds to the spinoglenoid notch projected on the medial-lateral scapular axis. We calculated the acromion angle (AA) and glenoid inclination angle (GIA) by the definitions provided here [4], acromion posterior angle (APA), acromion tilt angle (ATA), and glenoid version angle (GVA) by the definitions provided here (Fig. 1) [20].

2.4. Biomechanical model

Since there is a reported association, we hypothesized that an unfavorable acromion and glenoid shape might initiate or contribute to the development of glenohumeral osteoarthritis and degenerative rotator cuff lesions [2–5,21]. Accordingly, this study assessed the effect of the acromion and glenoid morphology, represented by five angles, AA, APA, ATA, GIA, and GVA, on shoulder condition. These specific parameters were chosen because they have been shown in previous research to correlate with various shoulder conditions, providing insights into the relationship between morphology and shoulder condition [6,8,9,11,15,20]. Since it is not possible to compute the effect of anatomy on shoulder condition from observational data without causal assumptions [22], we delineated our assumed biomechanical model of the anatomy effect on shoulder condition in terms of a DAG (Fig. 2) [23]. To justify the arrows from GIA and GVA to shoulder condition and not the other way around, we removed cases with clear glenoid wear, caused by pathology. For this, we evaluated the sphericity of the glenoid and removed cases below a certain threshold of sphericity which showed clear wear of the glenoid.

To have a correct estimate of the effect that AA, APA, ATA, GIA, and GVA have on the shoulder condition, the common causes of them and shoulder condition must also be adjusted for in the statistical analysis [17]. We first described the logic of the arrows from the common causes toward the exposures (morphological angles), then from the common causes toward the outcome (shoulder condition). The

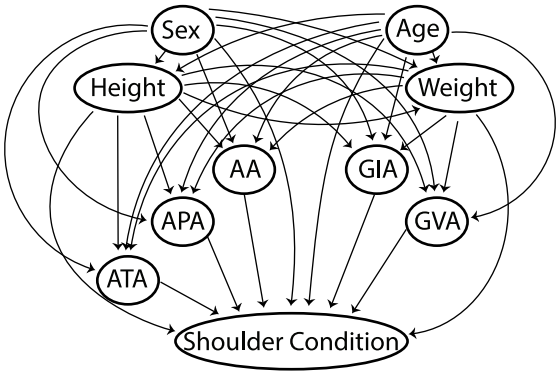


Fig. 2. Directed acyclic graph (DAG) for describing the relationships between analyzed variables.

DAG contains arrows from sex and age to the morphological angles since variations in acromion and glenoid morphology between females and males across various age groups have been reported [24]. There is however limited evidence to link BMI with alterations in acromion and glenoid morphology. Existing studies have primarily focused on the association between BMI and conditions such as osteoarthritis and rotator cuff tendinitis [25,26]. Nonetheless, we assumed there could be an effect of the BMI on the acromion and glenoid morphology, shown by an arrow from height and weight to morphological angles in the DAG. This could be through mechanical loading on the joint, or inflammatory processes that might have systemic effects on joints and connective tissues, potentially impacting the morphology of the acromion and glenoid. Age is reported as a significant risk factor for both triggering and advancing osteoarthritis [27]. It also plays a crucial role in the development of cuff tears [28], potentially due to an increase in intrinsic degeneration and a diminished healing response [29]. Sex can influence shoulder issues, potentially through various mechanisms such as physical activity. Furthermore, research suggests that sex hormones may also have an impact on osteoarthritis [30,31]. Moreover, the incidence of rotator cuff tears tends to be higher during the post-menopausal period [32]. This is why we incorporated an arrow from sex to shoulder condition. Additionally, there is evidence suggesting that BMI may influence the occurrence of shoulder osteoarthritis [25] and rotator cuff tendinitis [26]. This is the rationale behind including an arrow from height and weight to shoulder condition.

Eventually, assuming that our DAG (Fig. 2) correctly described the causal relationships between the variables as observed in reality, the effect of morphological variables on shoulder condition was identifiable [17], by adjusting for the sufficient minimal adjustment set: sex, age, height, and weight. For a discussion on the conditional independencies implied by the DAG, refer to the section titled "DAG implications test" in the supplementary materials.

Moreover, we evaluated the total effect of sex, age, height, and weight on shoulder condition as it can be clinically useful. Based on the assumed DAG, no adjustment set was needed for estimating the total effect of sex and age on shoulder condition. For the height effect on shoulder condition, the adjustment set consisted of sex and age. For weight effect on shoulder condition, the adjustment set consisted of sex, age, and height.

In the following section, we outlined four distinct statistical models for estimating the effect of various exposures, (1) sex and age, (2) height, (3) weight, and (4) anatomical variables on shoulder condition.

2.5. Statistical models

In this section, we used the assumed causal model (Fig. 2) and Bayesian statistics to estimate the effect of different exposures on the shoulder condition.

2.5.1. Sex and age

For estimating the effect of sex, and age on shoulder condition there was no adjustment set. We modeled the shoulder condition of each subject with a multinomial logistic regression distribution (categorical) [33] as

$$p_i \sim Categorical(\theta) \qquad for\ i = 1...N(number\ of\ subjects) \tag{1}$$

The θ is a vector of probabilities provided by the multinomial logit (softmax) function

$$\theta = softmax(\gamma) \tag{2}$$

The input of the softmax function is a vector of scores predicted by a linear model of sex and age as

$$\gamma_i = \alpha_{s[i],p[i]} + \beta^a_{s[i],p[i]}(a_i - \overline{a}) \tag{3}$$

where the s[i] and p[i] are the sex and shoulder condition of each subject, $\overline{a}$ is 70 years, the average age of the pathological subjects. In this model, we considered an average effect of sex, α, for each shoulder condition, and an age effect, β, for each sex and shoulder condition. The prior distribution of the parameters of the linear model was considered as

$$\alpha_{s,p} \sim Normal(0, 0.1) \tag{4}$$

$$\beta^a_{s,p} \sim Normal(0, 0.01) \tag{5}$$

The (arbitrary) choice of the average ($\overline{a}$) simplified the definition of the prior distribution, as of the amount of change in shoulder condition probability by deviating one year from the average (70 years).

2.5.2. Height

For estimating the effect of height on shoulder condition, we need to adjust for sex and age in the model. We used a similar model to the one above and we just added the height term to the linear model as

$$p_i \sim Categorical(\theta) \qquad for\ i = 1...N(number\ of\ subjects) \tag{6}$$

$$\theta = softmax(\gamma) \tag{7}$$

$$\gamma_i = \alpha_{s[i],p[i]} + \beta^a_{s[i],p[i]}(a_i - \overline{a}) + \beta^h_{s[i],p[i]}(h_i - \overline{h}) \tag{8}$$

$$\alpha_{s,p} \sim Normal(0, 0.1) \tag{9}$$

$$\beta^a_{s,p}, \beta^h_{s,p} \sim Normal(0, 0.01) \tag{10}$$

$\overline{h}$ is the average height (170 cm) of the subjects.

2.5.3. Weight

For estimating the effect of weight on shoulder condition, we needed to adjust for sex, age, and height in the model

$$p_i \sim Categorical(\theta) \qquad for\ i = 1...N(number\ of\ subjects) \tag{11}$$

$$\theta = softmax(\gamma) \tag{12}$$

$$\gamma_i = \alpha_{s[i],p[i]} + \beta^a_{s[i],p[i]}(a_i - \overline{a}) + \beta^h_{s[i],p[i]}(h_i - \overline{h}) + \beta^w_{s[i],p[i]}(w_i - \overline{w}) \tag{13}$$

$$\alpha_{s,p} \sim Normal(0, 0.1) \tag{14}$$

$$\beta^a_{s,p}, \beta^h_{s,p} \sim Normal(0, 0.01) \tag{15}$$

$\overline{w}$ is the average weight (75 kg) of the subjects.

2.5.4. Anatomical variables

For estimating the effect of anatomical variables on shoulder condition, we needed to adjust for sex, age, height, and weight in the model

$$p_i \sim Categorical(\theta) \qquad for\ i = 1...N(number\ of\ subjects) \tag{16}$$

$$\theta = softmax(\gamma) \tag{17}$$

$$\gamma_i = \alpha_{s[i],p[i]} + \beta^{a}_{s[i],p[i]}(a_i - \overline{a}) + \beta^{h}_{s[i],p[i]}(h_i - \overline{h}) + \beta^{w}_{s[i],p[i]}(w_i - \overline{w}) + \beta^{AA}_{s[i],p[i]}(AA_i - \overline{\text{AA}}) + \beta^{APA}_{s[i],p[i]}(APA_i - \overline{\text{APA}}) + \beta^{ATA}_{s[i],p[i]}(ATA_i - \overline{\text{ATA}}) + \beta^{GIA}_{s[i],p[i]}(GIA_i - \overline{\text{GIA}}) + \beta^{GVA}_{s[i],p[i]}(GVA_i - \overline{\text{GVA}}) \quad (18)$$

$$\alpha_{s,p} \sim Normal(0, 0.1) \quad (19)$$

$$\beta^{a}_{s,p}, \beta^{h}_{s,p}, \beta^{w}_{s,p} \sim Normal(0, 0.01) \quad (20)$$

$$\beta^{AA}_{s,p}, \beta^{APA}_{s,p}, \beta^{ATA}_{s,p}, \beta^{GIA}_{s,p}, \beta^{GVA}_{s,p} \sim Normal(0, 0.1) \quad (21)$$

$\overline{\text{AA}}$, $\overline{\text{APA}}$, $\overline{\text{ATA}}$, $\overline{\text{GVA}}$, and $\overline{\text{GIA}}$, are the average of these angles for CTRL subjects.

The choice of prior distributions (Eqs. (4), (5), (9), (10), (14), (15), (19), (20), (21)) was analyzed by prior predictive simulation (section "Prior predictive simulation" in supplementary materials). We estimated the posterior distribution of the models using the No-U-Turn version of the Hamiltonian Monte Carlo (HMC) algorithm [34] provided by Stan [33], and we analyzed this distribution with R 4.1.0 [35]. The generated posterior distribution was based on 2000 iterations of 1000 warm-ups and 1000 sampling from four chains. Three different assessments confirmed the HMC convergence. First, we visually inspected the trace-plot (section "Hamiltonian Monte Carlo convergence" in supplementary materials). Second, we assessed the number of effective samples, which was always higher than 50% of the total number of samples drawn (1000 from each chain, a total of 4000 from the four chains) [36]. Third, we investigated the potential scale reduction factor, with a minimum of 0.999 and a maximum of 1.002 for all of the models, indicating HMC convergence [37].

2.6. Simulations with the posterior distributions

To estimate the causal effect of the exposures on the shoulder condition, we performed simulations with the posterior distributions of the models. For the sex and age model, we varied age from 50 to 90 years for each sex and found its effect on shoulder condition. For other models, we kept all of the variables constant (average value) and changed the exposure from −2 z-score to 2 z-score. We calculated the difference of the shoulder condition probability distribution at 2 z-score and −2 z-score and summarized this distribution of the difference by its median and 89% percentile interval (PI). The changes in shoulder condition probabilities were reported as percentage points (pp) to clarify the impact of the exposures.

3. Results

3.1. Sex and age effect on shoulder condition

Increasing age raised the shoulder condition probability, with a higher rate for males than females (Fig. 3). The raise trend was similar for OA and CTA. Increasing age raised OA probability by 17 pp (89% PI [14, 20]) for females, and 21 pp (89% PI [16, 26]) for males, and raised CTA probability by 22 pp (89% PI [19, 26]) for females, and 26 pp (89% PI [21, 33]) for males.

3.2. Height effect on shoulder condition

Increasing height decreased OA probability by 1.5 pp (89% PI [0.0, 2.4]) for females, and 1.7 pp (89% PI [0.0, 2.2]) for males, and decreased CTA probability by 3 pp (89% PI [2, 4]) for females, and 9 pp (89% PI [7, 10]) for males (Fig. 4).

3.3. Weight effect on shoulder condition

Increasing weight raised OA probability with a higher rate for females (24 pp (89% PI [18, 30])) than males (8 pp (89% PI [5, 10])), while lowered CTA probability with a higher rate for males (20 pp (89% PI [15, 24])) than females (10 pp (89% PI [9, 11])) (Fig. 5).

3.4. Anatomical variables effect on shoulder condition

Increasing AA and GIA raised the CTA probability (Fig. 6, 6). AA effect was almost the same for females and males (36 pp (89% PI [27, 45]) females, 33 pp (89% PI [22, 43]) males), while the GIA effect was higher for females (45 pp (89% PI [34, 55]) females, 31 pp (89% PI [19, 42]) males). Increasing AA and GIA lowered the probability of OA, with a more pronounced effect observed in females compared to males. For females, the reduction in OA probability was 44 pp (89% PI [33, 52]) for AA and 55 pp (89% PI [43, 64]) for GIA. In contrast, for males, the reductions were 17 pp (89% PI [11, 24]) for AA and 23 pp (89% PI [14, 30]) for GIA.

Increasing GVA (more anterior) decreased the probability of OA, higher for males (63 pp (89% PI [46, 75])) than females (33 pp (89% PI [23, 41])), while it had a negligible effect on CTA probability (7 pp (89% PI [4, 11]) females, 0.005 pp (89% PI [0.0 0.01]) males) (Fig. 6).

Increasing ATA raised OA and CTA probability for females (15 pp (89% PI [10, 17]) for OA and 11 pp (89% PI [7, 14]) for CTA) and CTA probability for males (21 pp (89% PI [13, 28])), while it decreased OA probability for males (25 pp (89% PI [16, 33])) (Fig. 6).

Increasing APA increased both OA and CTA probability almost uniformly, and higher for females (15 pp (89% PI [11, 19]) for OA and 12 pp (89% PI [8, 14]) for CTA) than males (4 pp (89% PI [2, 6]) for OA and 4 pp (89% PI [2, 7]) for CTA) (Fig. 6).

3.5. Comparison

In this section, we assessed the impact of each variable on OA and CTA, organizing them from the highest to the lowest effect observed.

For OA, GVA had the most substantial effect, decreasing OA probability notably in males compared to females. GIA also reduced OA probability, with a greater reduction observed in females. AA further contributed to a decrease in OA probability, showing a larger effect in females than in males. Age increased OA probability, exhibiting a stronger effect in males than in females. Weight positively influenced OA probability, with a more pronounced effect in females. ATA was associated with an increase in OA probability for females while decreasing it for males. APA increased OA probability in both sexes, more prominently in females. Height had a minimal effect, leading to a slight reduction in OA probability, particularly in females.

For CTA, AA had the most considerable positive association, increasing CTA probability similarly across sexes, though slightly more in females. GIA also positively influenced CTA probability, with a stronger effect in females. Age was positively associated with CTA, showing a slightly stronger increase in males than in females. Weight negatively impacted CTA probability, decreasing it more substantially in males than in females. ATA had a positive association with CTA probability in both sexes, with a greater effect in males. APA increased CTA probability in both sexes, with a larger effect noted in females. GVA had a negligible effect on CTA probability, slightly reducing it in females and having almost no impact in males. Height had little influence on CTA probability, resulting in a slight reduction more pronounced in males than in females.

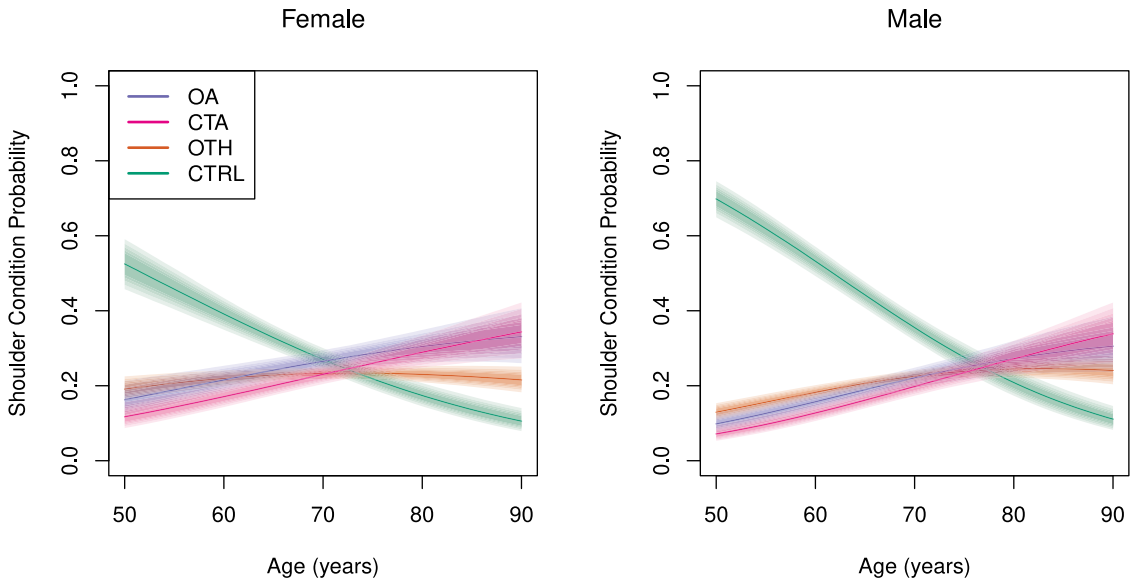


Fig. 3. Shoulder condition probability as a function of age for each sex.

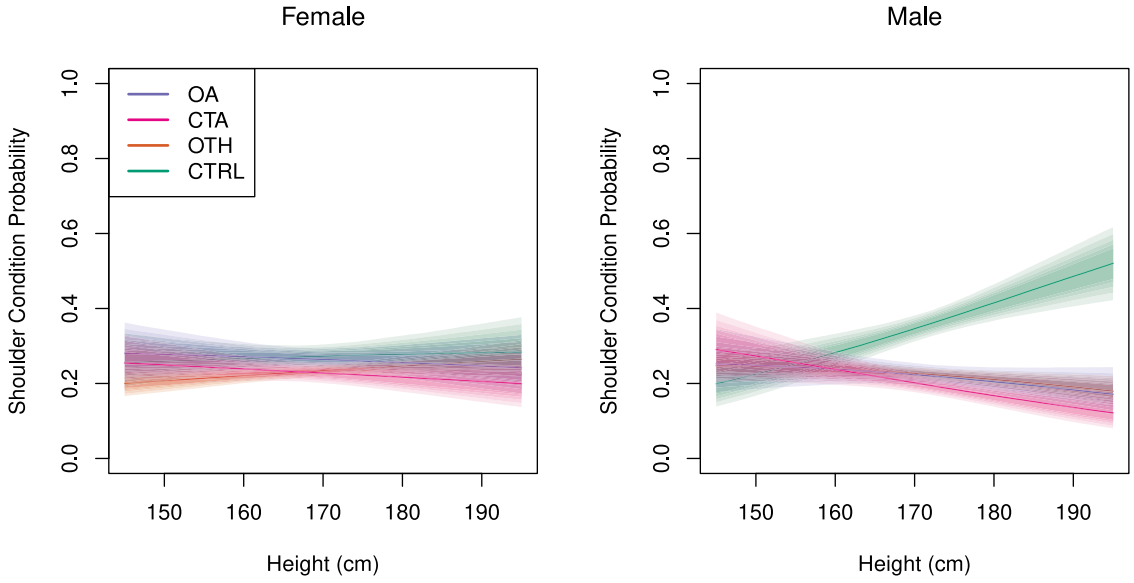


Fig. 4. Shoulder condition probability as a function of height for each sex at 70 years.

4. Discussion

Our objective was to estimate the effect of scapular anatomy, represented by five angles, on shoulder pathologies. We assumed a causal model for the effect of these angles on shoulder condition and we found the minimal adjustment set of variables by applying do-calculus. We developed a Bayesian model to estimate the effect while adjusting for sex, age, height, and weight. We also developed alternative causal models to evaluate the effect of sex, age, height, and weight on shoulder condition. We found the posterior distribution for all of the models with Hamiltonian Monte Carlo, and we used this distribution to simulate the effect of every exposure on each shoulder condition by varying it while keeping other variables constant.

Acromion angle (AA) and glenoid inclination angle (GIA) increased the cuff tear arthropathy (CTA) probability and decreased the osteoarthritis (OA) probability, and it was on average higher for females than males. Glenoid version angle (GVA) (more anterior) decreased OA probability, higher for males than females. Acromion tilt angle (ATA) and acromion posterior angle (APA) increased OA and CTA probability for females. ATA decreased OA probability and increased CTA probability for males. The effect of GVA on CTA probability, and APA on OA and CTA probability for males was negligible (on average below 10 pp) compared to other anatomical variables' effects. Age increased shoulder condition probability, with a higher rate for males. Height decreased shoulder condition probability for males. Weight increased OA probability with a higher rate for females than males. We observed sex differences for the anatomical variables' effect on shoulder condition (Fig. 6).

Age is reported as a significant risk factor for shoulder condition, with the condition being more common in females [38], which complied with our findings (Fig. 3). We could not find studies on the effect of height and weight on shoulder condition probability. Most of the studies evaluated body mass index (BMI) correlation with shoulder condition. Based on our assumed DAG, height affects weight, which means to find the total effect of height on shoulder condition, weight should not be considered, while for estimating the total effect of weight on shoulder condition, height should be adjusted. This was the rationale behind considering height and weight separately, not as BMI. It was less

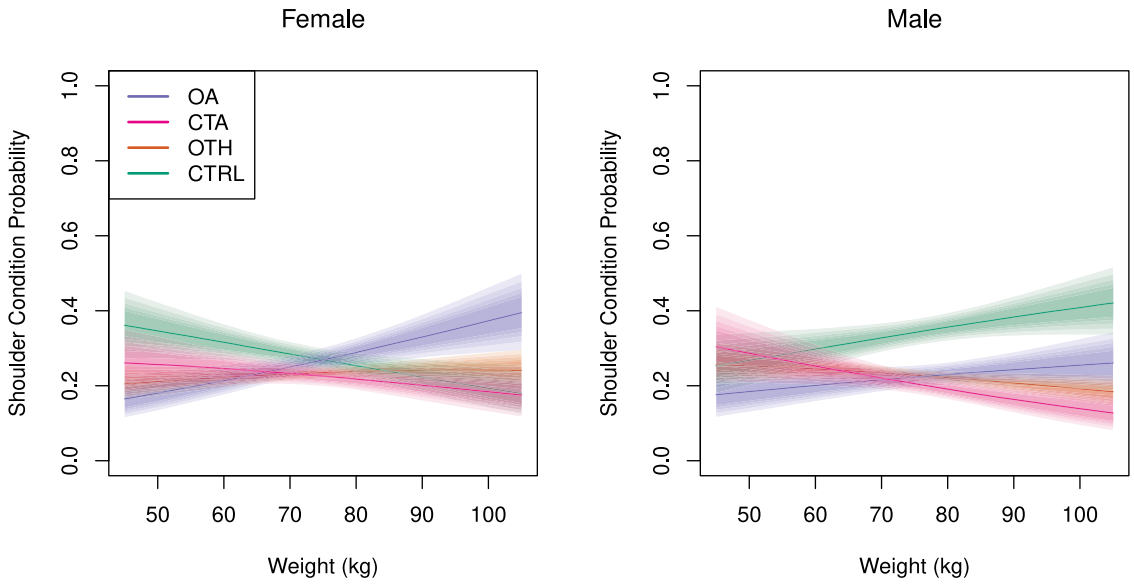


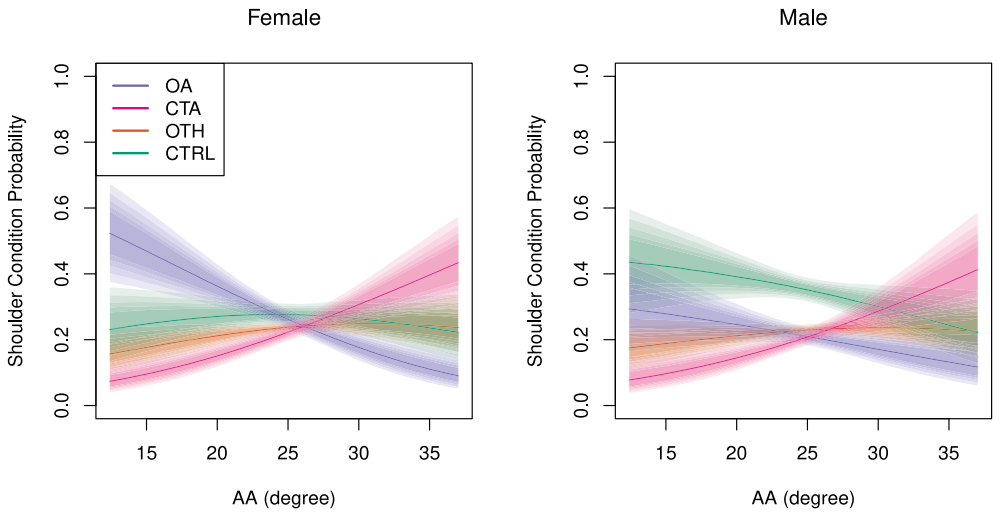
Fig. 5. Shoulder condition probability as a function of weight for each sex at 70 years and 170 cm height.

probable for tall males to have shoulder condition (Fig. 4). This effect was negligible for females. Our findings confirmed previous reports on the impact of weight on OA (Fig. 5) [39]. The acromion shape in the sagittal and coronal plane was described by multiple metrics and their association with different shoulder disorders was analyzed. A direct comparison of the results of these studies with ours was not possible as none of them built a model to see the effect of anatomical variables on shoulder condition probability. Researchers mainly reported the null hypothesis significance testing results. Nyffeler et al. reported statistically significant differences in AI between patients with a full-thickness rotator cuff tear and the two groups of individuals with intact rotator cuffs [9]. More recently, Verhaegen et al. reported that a more laterally extended acromion to be associated with CTA [40]. Although we did not use the same metric for lateral extension of the acromion, AA was related to AI by definition, and to the definition used in [40]. We also reported the increase in CTA probability by increasing AA. APA has been suggested by Terrier et al. [20] as a potential predictor for OA, however, we found its effect on shoulder condition to be very small for females and negligible for males. Recent studies have not shown significant differences in ATA between rotator cuff tears and OA [41]. We found that ATA increased the OA and CTA probability of females, while it decreased the OA probability and increased the CTA probability for males.

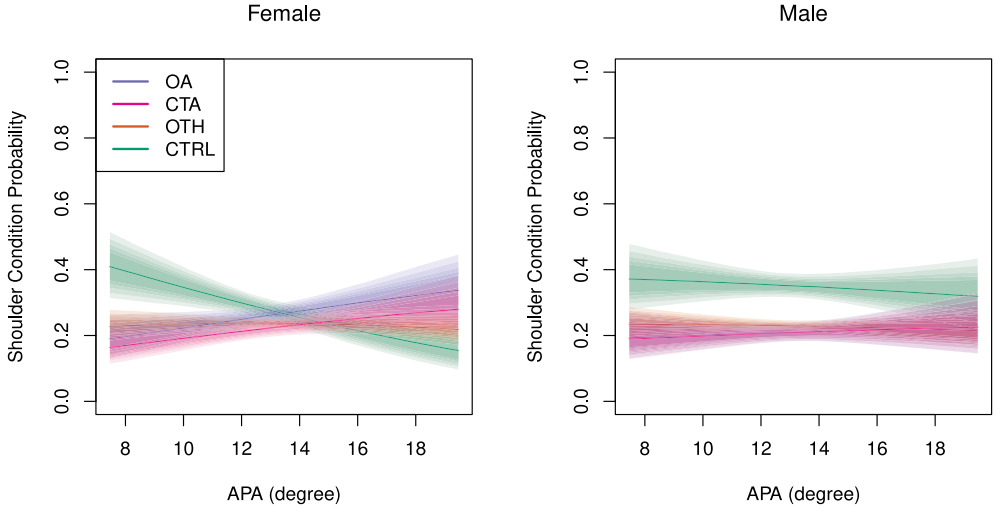
Regarding GIA, mixed trends have been reported. Hughes et al. analyzed eight cadaver shoulders using AP radiographs, noting higher GIA in shoulders with full-thickness rotator cuff tears compared to intact ones [11]. Conversely, Kandemir et al. found no significant difference in GIA between the intact cuff and tear shoulders in their study using a 3D digitizing system on 24 cadaveric shoulders [12]. We reported a decrease in OA probability and an increase in CTA probability as a function of GIA increase, an effect that was higher for females compared to males. The observed effect could be influenced by the sample size imbalance between males and females. However, research has shown that notable sex differences exist in musculoskeletal structures and conditions. Anatomical and biomechanical differences in shoulder morphology, coupled with hormonal fluctuations and variations in muscle strength, can significantly affect joint degeneration and injury risk [42]. These factors likely contributed to the variations observed in OA and CTA probabilities as GIA changes. A well-known metric in shoulder anatomy is CSA [1], and it was reported that degenerative rotator cuff tears were associated with significantly larger CSA, while primary glenohumeral osteoarthritis was associated with significantly smaller CSA compared to asymptomatic shoulders without these pathologies. We did not include CSA in our model, as by definition CSA was the addition of AA and GIA, and including it would block the information from AA and GIA to shoulder condition. While our study confirmed the usefulness of CSA in predicting CTA, as increasing both AA and GIA increased CTA probability, we argue that it was better to not include it in our causal model to see the isolated effect of AA and GIA on the shoulder condition. It was reported that posteriorly inclined glenoids (negative GVA) are associated with OA [1,15,43]. This complied with what we reported here (Fig. 6).

The strength of this study lies in its unique approach to evaluating the effect of scapular anatomy on shoulder condition within a causal framework, setting it apart from numerous previous studies that focused on correlation. By considering possible DAGs, analyzing their implications, and identifying the minimal adjustment set of variables, we aimed to minimize confounding bias and accurately isolate the causal effect of scapular anatomy on shoulder condition. This approach enhances the validity of our findings by ensuring we control only for relevant variables and improving transparency in our assumptions about the underlying causal relationships [17,44,45]. Various estimation methods are available, such as regression, propensity score matching [46], g-estimation [47], inverse probability weighting [48], and Bayesian regression [49]. We chose Bayesian regression because of its ability to provide uncertainty estimates, incorporate prior information, and update beliefs based on observed data, offering a flexible framework for inferences [49,50]. To address the potential sensitivity of the results to the choice of prior distributions [51], we performed the recommended prior predictive simulations to ensure that our prior choices were robust and appropriate for our analysis [52]. This causal framework offers several advantages over traditional correlation analyses, including better control for confounders, handling of missing values, balancing of groups, separation of direct and indirect effects, and more accurate quantification of uncertainties, thereby addressing common limitations associated with retrospective observational data [17,53,54]. By employing this approach, we can derive more reliable and actionable insights for clinical practice.

The first limitation of our study concerned the DAG. Our DAG was possibly incomplete, nonetheless, we believe a model based on incomplete assumptions is better than a model based on no assumptions as it cannot be scientifically justified [17]. There could be other common causes (such as genetic information or developmental environment) of scapular anatomy and shoulder condition that we missed in the DAG



(a)



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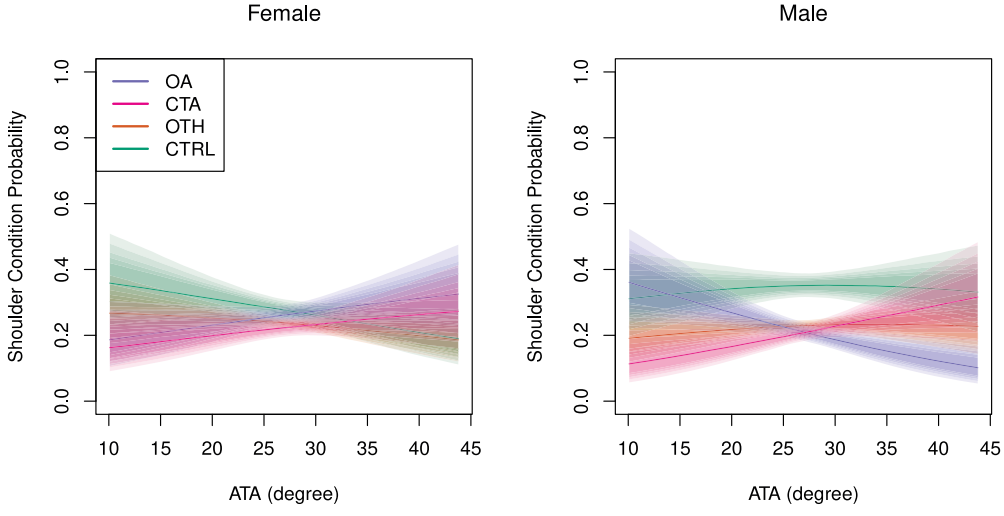
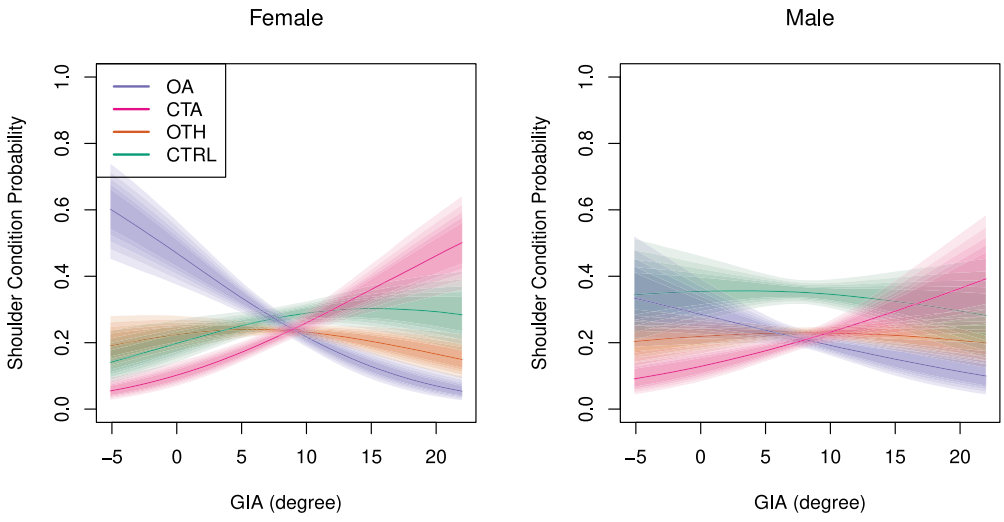
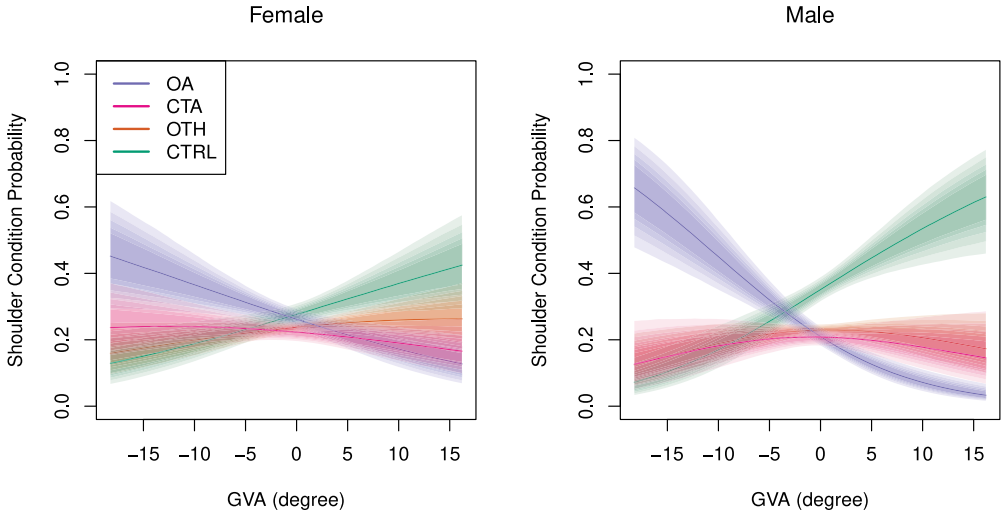


(c)

Fig. 6. Shoulder condition probability as a function of changing one exposure (a) AA (b) APA (c) ATA (d) GIA (e) GVA, while keeping other variables constant.



(d)



(e)

Fig. 6. (*continued*).

and thus biased our estimate. Nonetheless, this study could be viewed as a relatively acceptable causal model for scapular anatomy's effect on shoulder condition, which could be tested by larger datasets and improved in the case of wrong predictions. Another concern regarding the DAG could be the direction of causality between GVA, GIA, and shoulder condition. Using cross-sectional observational data for causal inference has inherent limitations, as it captures information at a single time point rather than over an extended period. This lack of temporal sequencing restricts our ability to establish whether anatomical variations, such as GIA or GVA, precede or result from shoulder condition, as both could influence each other bidirectionally. Longitudinal studies, which capture data across time points, would allow for a clearer determination of causation, yet such data are rare in this field. Given that most available datasets are cross-sectional, researchers must often rely on these data to address pressing questions, and we have applied best practices in causal analysis to manage these constraints effectively. In this study, we excluded cases with extreme glenoid wear to ensure the validity of our causal inference model regarding the effects of GIA and GVA on shoulder condition. By doing so, we prevented pathology-induced changes in glenoid variations from biasing our estimates of how GIA and GVA affect the shoulder condition. This approach enables a clearer assessment of the true impact of anatomical variations on shoulder health. However, by removing cases with clear wear, we might have introduced selection bias, making the remaining sample less representative of the general population with shoulder pathologies and potentially affecting the generalizability of our findings. It is crucial to establish the temporal relationship between glenoid orientation and shoulder condition, ideally measuring glenoid orientation before the onset of pathology to ascertain causality. In an ideal scenario where temporal data exists, we could construct a dynamic causal model, represented by a DAG that unfolds over time [55]. This would allow for a more nuanced understanding of how initial anatomical variations influence the development and progression of shoulder pathology. This approach would further reduce confounding and provide a stronger basis for causal inference, enhancing the robustness of our findings and improving the model's applicability to real-world clinical settings. The second limitation of the study was the dataset size and its limitation to only one center in Switzerland. Indeed, having a larger dataset could be helpful to have more precise estimates, however, we used Bayesian statistics and reported the uncertainty of the estimates which was affected by the dataset size. Furthermore, our dataset was not balanced. While having an equal sample size across groups may seem ideal, it is not always the best approach for statistical analysis. A more informative sample would align with real-world proportions, such as

the prevalence ratios of specific pathologies and sex distributions. This approach minimizes sampling bias and improves the representativeness of study results. Although exact real-world distributions can be difficult to determine, prior studies suggest a prevalence of approximately 25% for OA in adults over 65, with a higher prevalence among females than males [56]. Similarly, studies report a prevalence of around 20% for CTA in females, also higher than in males [57,58]. In our study, the proportions of participants with OA were 32% for females and 13% for males, while for CTA, the rates were 21% for females and 6% for males. As a result, a limitation of our study was the lower representation of males in the pathological groups compared to their overall population ratios, which may affect the generalizability of our findings. Regarding the limitation to Switzerland, although ethnicity might affect the scapular anatomy, we do not believe that it might affect shoulder condition directly. A third limitation of our study was the reliance on automated measurements of anatomical variables. However, previous research has demonstrated that the deep learning models employed for landmark predictions achieve a level of accuracy that is considered clinically acceptable [19].

This causal model can answer interventional questions, such as estimating the change in shoulder condition probability for an individual patient with specific characteristics like sex, age, height, and weight if scapular anatomy is altered. This capability could be especially useful in planning correction surgery to achieve the best patient-specific scapular anatomy, minimizing the probability of developing pathology [59]. The model could also identify shoulder pathology at an early stage, years before the first symptoms, which might improve the management of the treatments.

CRediT authorship contribution statement

Pezhman Eghbali: Writing – review & editing, Writing – original draft, Visualization, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Osman Berk Satir:** Methodology, Data curation. **Fabio Becce:** Writing – review & editing, Funding acquisition, Data curation, Conceptualization. **Patrick Goetti:** Writing – review & editing, Supervision, Data curation, Conceptualization. **Philippe Büchler:** Writing – review & editing, Funding acquisition. **Dominique P. Pioletti:** Writing – review & editing. **Alexandre Terrier:** Writing – review & editing, Supervision, Project administration, Methodology, Funding acquisition, Data curation, Conceptualization.

Code and data availability

The data, R, and Stan code that support the findings of this study are available on gitlab.epfl.ch.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Alexandre Terrier reports financial support was provided by Swiss National Science Foundation. Alexandre Terrier reports financial support was provided by Lausanne Orthopedic Research Foundation. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary material related to this article can be found online at https://doi.org/10.1016/j.cmpb.2025.108666.

Data availability

The data, R, and Stan code that support the findings of this study are available on gitlab.epfl.ch.

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