RESEARCH ARTICLE SUMMARY

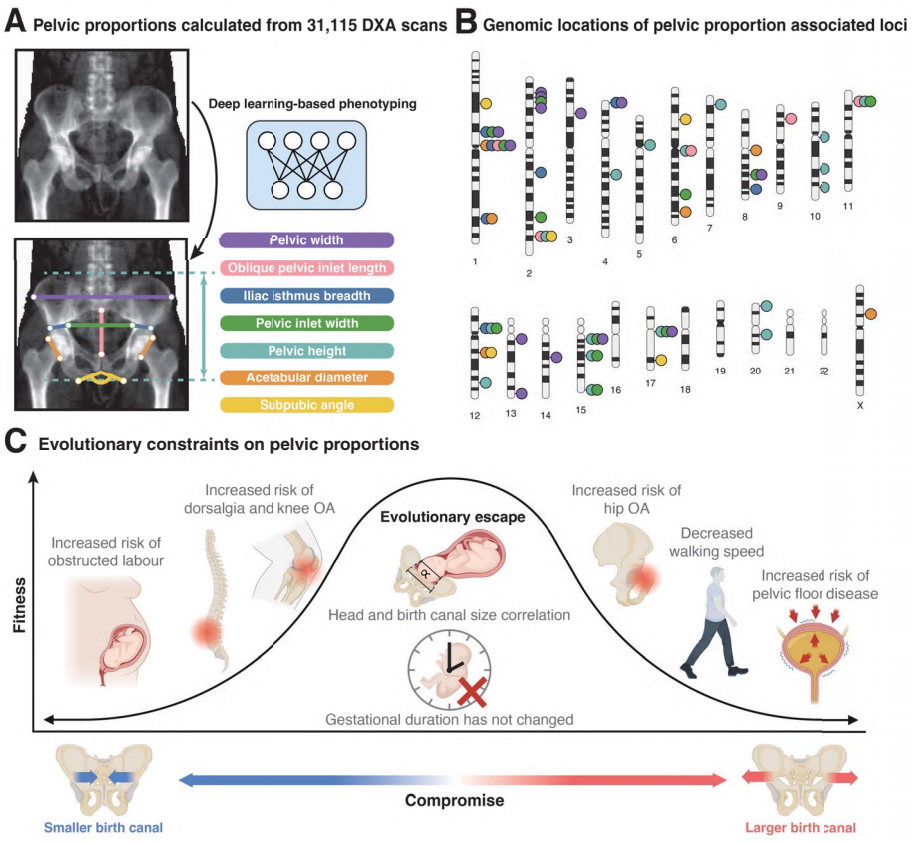
EVOLUTION

The genetic architecture of and evolutionary constraints on the human pelvic form

Liaoyi Xu, Eucharist Kun, Devansh Pandey, Joyce Y. Wang, Marianne F. Brasil*, Tarjinder Singh*, Vagheesh M. Narasimhan*

INTRODUCTION: Human pelvic shape has undergone significant evolutionary change since the divergence from the chimpanzee lineage. This transformation involved the reduction of pelvic canal dimensions to support bipedal locomotion. At the same time, human brain size also expanded significantly, which gave rise to the obstetrical dilemma, a hypothesis that highlights the mismatch between the large brain size of infants and the narrowed female birth canal. Initially proposed in the 1960s, empirical support for this classic hypothesis has been equivocal, largely owing to limitations in sample size and a lack of appropriate types of data.

RATIONALE: By using a deep learning model, we extracted a set of seven pelvic phenotypes, including three measures of the birth canal, from dual-energy x-ray absorptiometry (DXA) scans of 31,115 individuals from the UK Biobank. To elucidate the genetic underpinnings of pelvic morphology, we conducted a genome-wide association study (GWAS) on these seven pelvic traits and combined these results with multiple physiological and clinical outcomes related to childbirth, locomotion, and pelvic floor function. We also tested relevant evolutionary hypotheses to further explore potential evolutionary responses to alleviate the obstetrical dilemma.



Understanding the genetic basis of human pelvic proportions and how they impact locomotion, pelvic floor function, and childbirth. (**A**) Measurement of pelvic proportions using deep learning–based landmark estimation on 31,115 DXAs. (**B**) Location of loci that localize to a single protein-coding gene associated with various pelvic proportions colored according to the scheme in (A). (**C**) Multiple lines of evidence supporting and expanding on the obstetrical dilemma hypothesis. OA, osteoarthritis. [Figure created with BioRender.com]

RESULTS: The results revealed that all pelvic proportions are highly heritable (~32 to 48%), and a GWAS of these traits identified 180 independent loci. Unlike other skeletal proportions, such as long bone lengths, the subpubic angle, which determines the size of the birth canal, shows an estimated genetic correlation between sexes significantly less than 1, consistent with sex-specific reproductive functions. Additionally, although pelvic proportions display left-right asymmetry, this asymmetry is not heritable and is instead associated with handedness. We conducted phenotypic and genetic association analyses to link pelvic proportions to three facets of the obstetrical dilemma. For childbirth-related outcomes, narrower birth canal phenotypes were linked to an increased risk of emergency cesarean sections and obstructed labor owing to insufficient dilation but not obstruction due to fetal positioning, suggesting that childbirth imposes selective pressure to widen the birth canal. Conversely, larger birth canals were associated with reduced walking pace and a decreased risk of back pain but an increased risk of hip osteoarthritis, suggesting that lifelong effects of birth canal width reduction on locomotor efficiency are mixed. However, larger birth canal width was significantly associated with an increased risk of genital prolapse and incontinence, suggesting that pelvic floor disorders impose additional constraints. Lastly, we investigated whether the dilemma might have been alleviated through evolution. In line with recent evidence from great apes, we found no evidence for an association between pelvic proportions and gestational duration, suggesting that there has not been selection for earlier childbirth. Instead, we observed that infant and adult head widths were genetically correlated with maternal birth canal widths.

CONCLUSION: Our study provides fresh insights into a 60-year-old debate in human evolution. Beyond pressures imposed by childbirth, as initially proposed by the obstetrical dilemma hypothesis, our findings suggest that, rather than just locomotion, pelvic floor health may have played a significant role in reducing birth canal width in our transition to bipedalism. Our observed genetic correlation between birth canal width and infant and adult head width provides support for the coevolution of the human brain and pelvis. ■

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The genetic architecture of and evolutionary constraints on the human pelvic form

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Human pelvic evolution following the human-chimpanzee divergence is thought to result in an obstetrical dilemma, a mismatch between large infant brains and narrowed female birth canals, but empirical evidence has been equivocal. By using deep learning on 31,115 dual-energy x-ray absorptiometry scans from UK Biobank, we identified 180 loci associated with seven highly heritable pelvic phenotypes. Birth canal phenotypes showed sex-specific genetic architecture, aligning with reproductive function. Larger birth canals were linked to slower walking pace and reduced back pain but increased hip osteoarthritis risk, whereas narrower birth canals were associated with reduced pelvic floor disorder risk but increased obstructed labor risk. Lastly, genetic correlation between birth canal and head widths provides evidence of coevolution between the human pelvis and brain, partially mitigating the dilemma.

The hominin skeleton has undergone major morphological change associated with the transition to bipedalism. Some of the most substantial changes occurred in the pelvis, resulting in a superoinferiorly short and mediolaterally flaring pelvis relative to the modern great apes (*1*, *2*). These features are believed to have emerged early in hominin evolution, with these changes in pelvic anatomy allowing for the positioning of the upper body above the lower limb joints and facilitating the maintenance of an upright posture (*3*). Although debate continues about the details of gait mechanics in fossil hominins (*1*), the modern human pelvis is clearly adapted to habitual bipedality and undergoes a specific motion during walking that is thought to reduce energetic costs associated with bipedal locomotion (*4*).

The suite of adaptations for bipedality includes a reduction of the bi-acetabular distance, minimizing pelvic rotation during bipedal movement and consequently enhancing efficiency (*5*, *6*). This narrowing of the bi-acetabular distance results in a narrower birth canal and is thought to stand in direct opposition to the birthing of children with significantly larger brains than our evolutionary predecessors (*5*–*13*), the latter evolutionary shift occurring more recently than the former (*14*–*16*). In the 1960s, this functional and evolutionary conflict was coined the "obstetrical dilemma" by Washburn (*12*). In the six decades since then, the obstetrical dilemma has been a source of intense debate, and different studies have attempted to test the hypothesis by using various approaches and forms of empirical data (*6*, *13*, *17*–*19*). One area of contention centers on the relationship between pelvic shape and walking efficiency or walking speed. Some studies have found that there is an association between the two (*5*, *20*), whereas others have not (*21*–*24*). Another point of debate revolves around whether variation in birth canal proportions is associated with obstruction during delivery (*5*, *7*–*13*, *19*, *25*–*28*). Recently, appreciation has grown for the concept of a multifactorial pelvis, which proposes that the role of pelvic width reduction is not just to facilitate efficient bipedal locomotion but also to reduce the risk of pelvic floor disorders (*29*, *30*). Pelvic canal width reduction improves the pelvic floor's ability to support the fetus and the internal organs and to prevent incontinence (*5*, *31*, *32*).

In addition to debates about the association between pelvic morphology and locomotion, childbirth, and pelvic floor function, it has been suggested that, in modern humans, the obstetrical dilemma has been alleviated through evolution. Washburn (*12*) proposed that the obstetrical dilemma had been "solved by the delivery of the fetus at a much earlier stage of development" in humans relative to the other great apes. Today, this is generally taken to mean that human newborns are born at a relatively immature stage of development, thereby limiting the extent of brain growth before birth and ensuring that the newborn can successfully traverse the birth canal during delivery (*5*, *6*, *29*). However, Washburn did not specify what he meant by "earlier stage," leading to some ambiguity and some interpretations that suggest that humans have a shortened gestational length relative to other primates (*5*). This hypothesis has been challenged and updated in recent years, as human gestational length and newborn size have been found to align with or exceed expectations for primates of our size (*13*, *33*–*35*) [see (*5*, *6*, *36*) for alternate usages and historical perspectives on the term "obstetrical dilemma"].

Although different aspects of the dilemma have been tackled over the past few decades, these previous studies suffer from several shortcomings. One issue with many studies, particularly those involving clinical outcomes, is that measurements of pelvic dimensions were collected externally (*21*, *24*), which may not adequately reflect the skeletal constraints imposed, particularly with respect to the birth canal. Another issue is that some earlier studies lack complete information about individual lifetime health records and are unable to distinguish between fine-grained but important details, such as elective and emergency cesarean sections (C-sections). However, the major challenge contributing to the ongoing debate is the limited sample size in many of these studies, which often only have data on a few hundred individuals (sample sizes and references of previous papers are reported in table S1). In addition, data obtained for each study is often only capable of addressing one facet of the dilemma, as datasets examining childbirth outcomes and pelvic morphology often do not include data about pelvic floor function or walking speed or efficiency for the same individuals.

Although functional genomic datasets examining gene expression through development as well as comparative gene expression between the great apes and humans for the pelvis have yielded valuable insights (*37*–*39*), direct association studies between pelvic phenotypic and genetic variation have not yet been carried out. Thus, the genetic basis of pelvic morphology in humans or any other vertebrate is largely unknown.

In this study, we applied methods in computer vision to derive a comprehensive set of seven skeletal measurements of the human pelvis from full-body dual-energy x-ray absorptiometry (DXA) images at biobank scale. We performed genome-wide scans on these seven phenotypes to identify loci associated with variation in pelvic proportions. By using summary statistics from these image-derived phenotypes (IDPs), we linked human pelvic proportions through phenotypic and genetic correlation with other biobank phenotypes, with an emphasis on locomotor, pelvic floor, and childbirth-related outcomes.

Results

A deep-learning approach to measure pelvic morphology

To study the genetic basis of the human pelvis, we jointly analyzed DXA and genetic data from 42,284 individuals in the UK Biobank (UKB).

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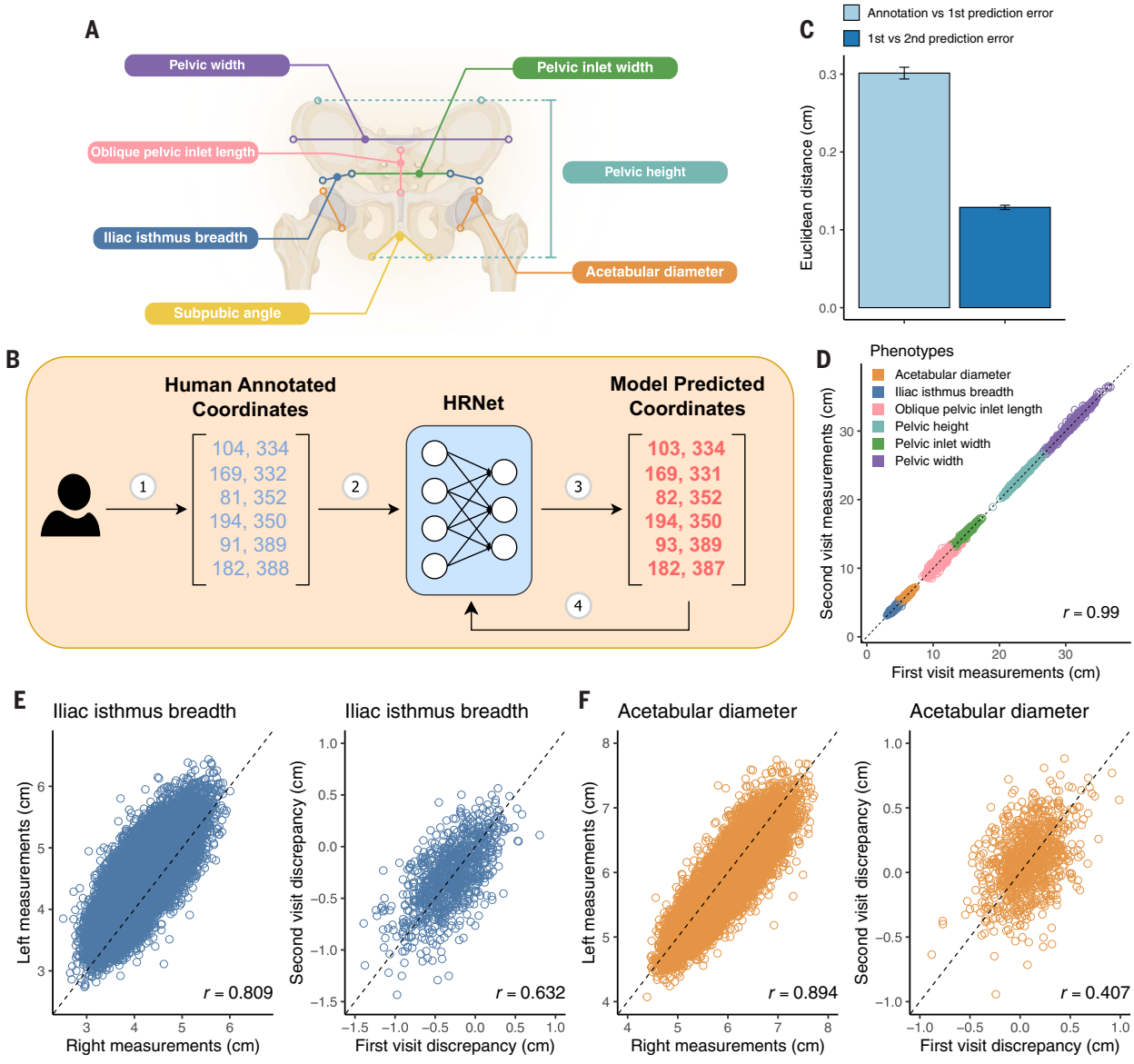

Fig. 1. Deep learning–based image quantification and validation. (**A**) Deep learning–based image landmark estimation using the HRNet architecture is shown. During this process, 293 training images manually annotated with specific landmarks were used to train the model, which was used to perform automatic annotation of landmarks on the rest of images in the dataset from which pelvic measurements were calculated. (**B**) Model-in-the-loop training data workflow. The coordinates from the 293 training images initially annotated by humans were used as a training set to train a model that was then redeployed on the training data. This helped to remove variation present in human labeling of the images and refined the training data itself. (**C**) Model-in-the-loop training reduces annotation variability. The light blue bar indicates the average Euclidean distances between human annotated landmarks and the model's first prediction on 58 validation set images. The dark blue bar indicates the average Euclidean distance between first and second model prediction on 58 validation set images. (**D**) Correlation of lengths measured from the first and second imaging visits for the same individual. (**E**) (Left) The correlation between the left- and right-side measurements of the iliac isthmus breadth. (Right) The correlation of the left-right discrepancy in the iliac isthmus breadth between the first and second imaging visit. (**F**) (Left) The correlation between the left- and right-side measurements of the acetabular diameter. (Right) The correlation of the left-right discrepancy in the acetabular diameter between the first and second imaging visit.

Individuals from this dataset are between 40 and 80 years old and reflect adult skeletal morphology. We report baseline information about our analyzed cohort in table S2. By using a previously published deep learning–based image quality control (QC) pipeline (*40*, *41*), we retained only DXA images for the full body, which included the entire pelvis, and removed images that contained image artifacts, atypical aspect ratios, and other abnormalities, retaining 39,469 images of high quality. These images were then uniformly cropped and padded to focus on the pelvis for subsequent analysis (*42*).

After performing image QC, we manually annotated 17 landmarks on 293 randomly selected pelvic images (fig. S2) to train our model. To assess the accuracy of our manual annotations, we reannotated 20 images from the initial set of 293 and refined this annotation through model-in-the-loop labeling (*42*) (Fig. 1, B and C). Our deep-learning model was based on a High-Resolution Network (HRNet) architecture chosen because it maintains a high-resolution representation throughout the model that improves

the performance of landmarking for this task on benchmarking tasks. These methods were robustly applied to a similar task of identifying joints on the overall skeleton (*40*) (see materials and methods).

Validation of human pelvic phenotype estimates

After training and validating the deep-learning model on the 293 manually annotated images, we applied this model to predict the 17 landmarks on the rest of the 39,469 full-body DXA images. We then calculated the pixel Euclidean distances between pairs of landmark coordinates to ascertain six length phenotypes, pelvic width, pelvic inlet width, oblique pelvic inlet width, iliac isthmus breadth, pelvic height, and acetabular diameter, as well as one angle phenotype, subpubic angle (Fig. 1A). To standardize images with varying aspect ratios, we rescaled pixels into centimeters for each image resolution. This was achieved by regressing the height we measured in pixels against standing height in centimeters, as measured in the UKB assessments (*42*) (fig. S11). For all seven pelvic measurements, we excluded individuals exceeding four standard deviations from the mean (*42*) (fig. S11).

Following outlier removal, we validated the accuracy of our measurements on the remaining samples in two ways. Firstly, we calculated the average error between labels in the validation data and model performance, which was 2 pixels across all 17 landmarks (fig. S3). Secondly, we analyzed 935 individuals with repeat imaging visits at least two years apart. The correlation of all pelvic length phenotypes between the first and second imaging visits was greater than 0.99 (Fig. 1D). This indicates that the phenotype estimations through our deep-learning model are both accurate and highly replicable.

Human pelvic asymmetry is associated with handedness and is not heritable

Next, we wanted to examine the genetic basis of pelvic asymmetry. To do this, we examined the correlation between measurements on the left and the right side of the pelvis. The two phenotypes with measurements on each side were iliac isthmus breath and acetabular diameter. The left-right correlations for iliac isthmus breadth and acetabular diameter were 0.809 and 0.894, respectively (Fig. 1, E and F). The average difference between the measurements in the iliac isthmus breadth between the left and right sides was 0.287 cm [$P < 2 \times 10^{-16}$, 95% confidence interval (CI) = 0.280 to 0.294], and for acetabular diameter, it was 0.101 cm ($P < 2 \times 10^{-16}$, 95% CI = 0.093 to 0.108). Though these differences were small, we found that they were replicable: left- and right-side discrepancies in individuals across two imaging visits had Pearson correlations of 0.632 and 0.407 for iliac isthmus breadth and acetabular diameter, respectively (Fig. 1, E and F). This suggests that we can capture a measure of pelvic asymmetry beyond measurement error. On estimating the heritability of this trait by using genome-wide complex trait analysis (GCTA; *43*), we found that it was consistent with zero [heritability (h_g^2) for acetabular diameter discrepancy = 0.0131, SE = 0.0149; h_g^2 for iliac isthmus breadth discrepancy = 0.0275, SE = 0.0158]. However, we observed a significant association between pelvic asymmetry and handedness, another trait that is also not significantly heritable (left-handed h_g^2 = 0.0064, right-handed h_g^2 = 0.0058 in 360,913 individuals). We regressed the left and right pelvic phenotype ratio against handedness while controlling for age and sex. Right-handed individuals tended to have significantly larger right acetabular diameters (regression $P = 8.31 \times 10^{-6}$) and marginally larger left iliac isthmus breadth than left-handed individuals (regression $P = 0.0665$). This suggests that left-right pelvic asymmetry might be driven by left- or right-side dominance, which is largely environmentally determined, but left-right dominance affects movement patterns and consequently skeletal development.

Genome-wide association study of human pelvic proportions

We performed genome-wide association studies (GWASs) with imputed genotype data in the UKB to identify variants associated with each pelvic phenotype. We applied standard variant and sample QC and focused our analyses on 31,115 individuals of "white British ancestry," as defined by the UKB genetic assessment, and 7.4 million common biallelic single-nucleotide polymorphisms (SNPs) with minor allele frequency >1%. We used BOLT-LMM (*44*) to regress each skeletal measure on variants by using a LMM association framework. We included height as a covariate to directly adjust for differences in body size between individuals and focus on skeletal proportions instead of overall length. We also adjusted for body size differences in two other ways: by dividing each phenotype by height to generate a skeletal proportion and by including a "leave-one-chromosome-out" polygenic risk score (PRS) for height as a covariate in the GWAS (*45*). Variant effect sizes with either height or height combined with the one-chromosome-out PRS as a covariate were highly correlated [Pearson correlation coefficient (r) = 0.99] (*42*). For downstream analyses, we focused on the results from the GWAS that included height as a covariate. Notably, we show that the genetic correlation between pelvic proportion phenotypes and height are 0, and PRS estimated from pelvic proportion GWAS summary statistics can not explain any variance of height (figs. S13 and S14).

After generating summary statistics for each skeletal measure, we estimated SNP heritability with linkage disequilibrium score regression (LDSC) (*46*) and GCTA–restricted maximum likelihood (REML) (*43*). All traits were highly heritable, with SNP heritability between 32 and 48% for GCTA-REML and between 28 and 45% for LDSC (*42*) (Fig. 2B and fig. S16). Across the six pelvic phenotypes adjusted by height (pelvic width, pelvic height, iliac isthmus breath, acetabular diameter, pelvic inlet width, oblique pelvic inlet length) and subpubic angle, we identified 342 loci at $P < 5 \times 10^{-8}$ and 241 loci at $P < 7.14 \times 10^{-9}$ (Bonferroni correction for seven traits). Of these loci, 180 are independently significant at $P < 5 \times 10^{-8}$ [LD (r^2) < 0.1] across all seven phenotypes (119 after Bonferroni correction for seven traits at $P < 7.14 \times 10^{-9}$) (Fig. 2A).

Biological insights from pelvic associations

Out of the 180 independent loci identified across GWASs (table S11), 51 loci overlapped a single protein-coding gene within each clumped region (Fig. 2B). Of these 51 genes, 22 (43%) result in abnormal skeletal phenotypes when disrupted in mice according to the Human-Mouse Disease Connection database (*40*). Eight genes (*COL11A1*, *NPR3*, *CDC5L*, *TNFRSF11B*, *TBX5*, *FBN1*, *SMAD3*, and *TBX4*) are associated with rare skeletal diseases in humans (table S11). In some cases, genes associated with specific pelvic proportions in our GWAS contribute to human pelvic abnormalities. We found that *TBX15* and *TBX4*, two T-box transcription factors, have been associated with differences in pelvic inlet width and pelvic height in model organisms, and, in humans, large effect rare mutations in *TBX15* and *TBX4* genes lead to pelvic abnormalities such as hypoplasia of the pelvis and small patella syndrome (*47*, *48*). Thus, our GWAS of pelvic proportions identified genes that were previously associated with skeletal developmental biology and Mendelian skeletal phenotypes and demonstrates the potential for future functional and knockout studies.

Sexual dimorphism in the genetic basis of pelvic proportions

The human pelvis plays a critical role in childbirth and is one of the most phenotypically dimorphic skeletal regions between males and females (*49*–*51*). Given the distinct reproductive functionalities between the male and female pelvis, we examined whether the genetic basis of our seven pelvic phenotypes differed between males and females. To do so, we carried out genetic correlation analysis between a GWAS carried out in males versus females. This analysis serves as a way to measure genetic dimorphism. Functionally similar pelvic phenotypes, such as pelvic height, exhibit similar genetic architectures between males and females. By contrast, birth canal–related phenotypes, such as the subpubic angle, showed estimated genetic correlations significantly divergent from 1. This difference in estimated genetic correlation

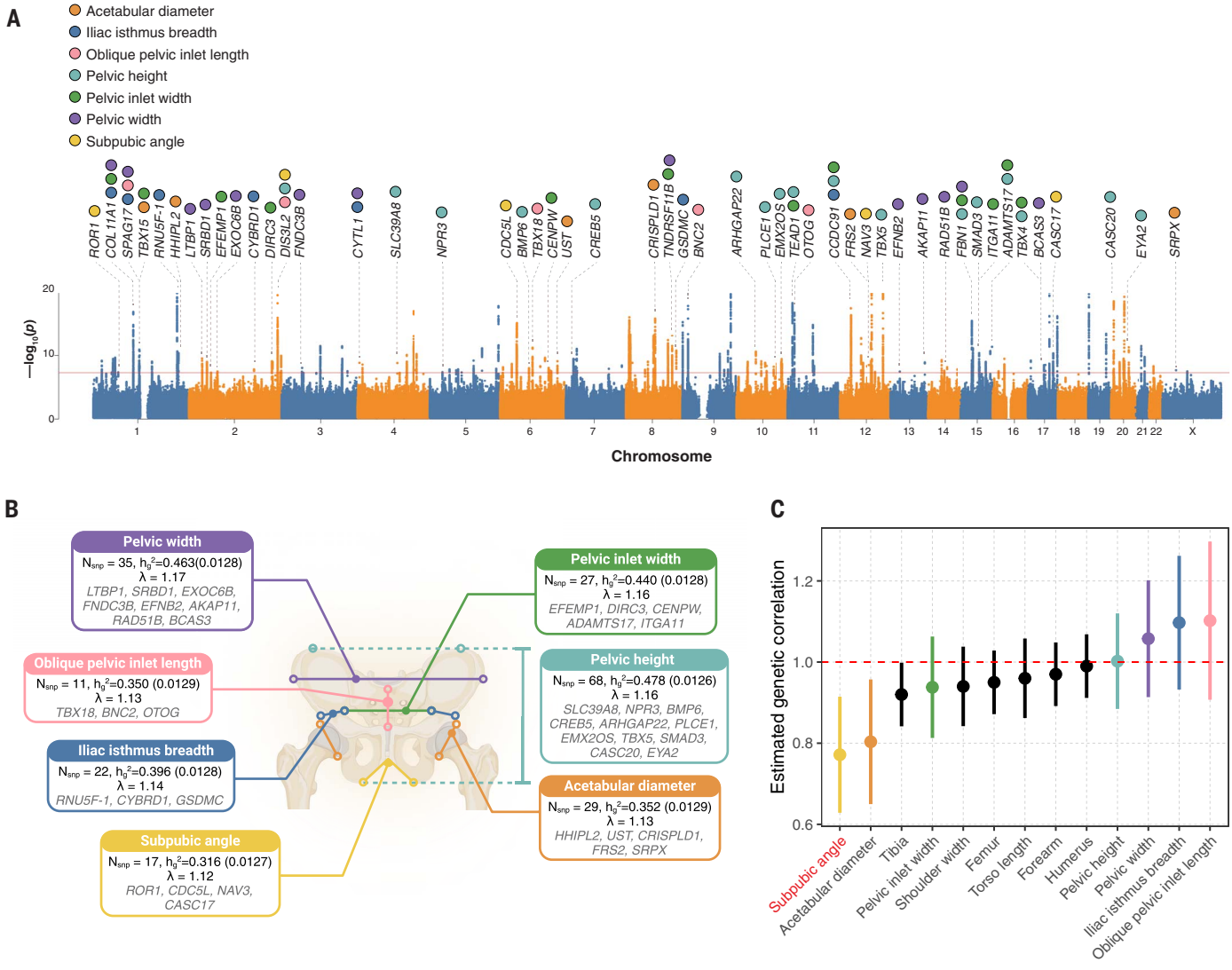

Fig. 2. Genome-wide association results. (**A**) Manhattan plot of a GWAS performed across six pelvic proportions and the subpubic angle; the lowest *P* value for any trait at each SNP is annotated. Loci over the genome-wide significance threshold that are close to only a single gene are annotated. (**B**) Shown are the total number of genome-wide significant loci per trait, heritability (GCTA-REML), estimate of genomic inflation λ (from LDSC), and associated genes of loci that are specific to each skeletal trait (again annotating only loci that map to a region with a protein-coding gene within 1 Mb of each clumped region). (**C**) Estimated genetic correlation between female and male pelvic phenotypes and other skeletal traits, including lengths of the tibia, femur, torso, forearm, and humerus. Error bars represent the 95% confidence intervals, with traits significantly different from one after FDR correction highlighted in red. Heritability greater than 1 is due to small sample size. Subpubic angle, shown in red on the *x* axis, is the only trait that is significantly different from 1 after FDR correction.

is in contrast to virtually all other skeletal traits previously examined, such as arm, leg, torso, and shoulder dimensions. These other traits all showed estimated genetic correlations not significantly different from 1 in the same cohort (Fig. 2C), suggesting that sex-specific reproductive requirements of the human birth canal are driving differences in genetic architecture between sexes for these pelvic traits.

Genetic and phenotypic association of pelvic proportions with locomotor phenotypes

We examined how pelvic proportions were associated with walking pace and musculoskeletal disorders, such as knee, hip, and back osteoarthritis (OA), which are degenerative conditions that arise from lifetime cumulative effects of gait and motion. Firstly, we used logistic regression to examine phenotypic associations between pelvic proportions and these phenotypes (Fig. 3A) while controlling for age, sex, weight, height, and other major risk factors for OA (*42*). After correcting for multiple testing at a false discovery rate (FDR) < 5% across all associations, we found that two birth canal–related phenotypes were associated with increased self-reported walking pace [oblique pelvic inlet length, $P = 5.3 \times 10^{-3}$, odds ratio (OR) = 0.96; subpubic angle, $P = 4.4 \times 10^{-4}$, OR = 0.92) (table S15). As a positive control, we examined another skeletal trait, leg-to-torso length, which we found to be significantly positively associated with walking speed ($P = 2.97 \times 10^{-8}$, OR = 1.08), in line with previous results and with mechanical modeling (*5*, *6*). These results provide empirical evidence that narrower birth canal proportions in humans are associated with increased walking speed (phenotypic association: between oblique pelvic inlet length and walking pace and between subpubic angle and walking pace. However, examining the associations with OA-related phenotypes, we found that having smaller birth canal–related phenotypes also increased the risk of back pain or dorsalgia (phenotypic association:

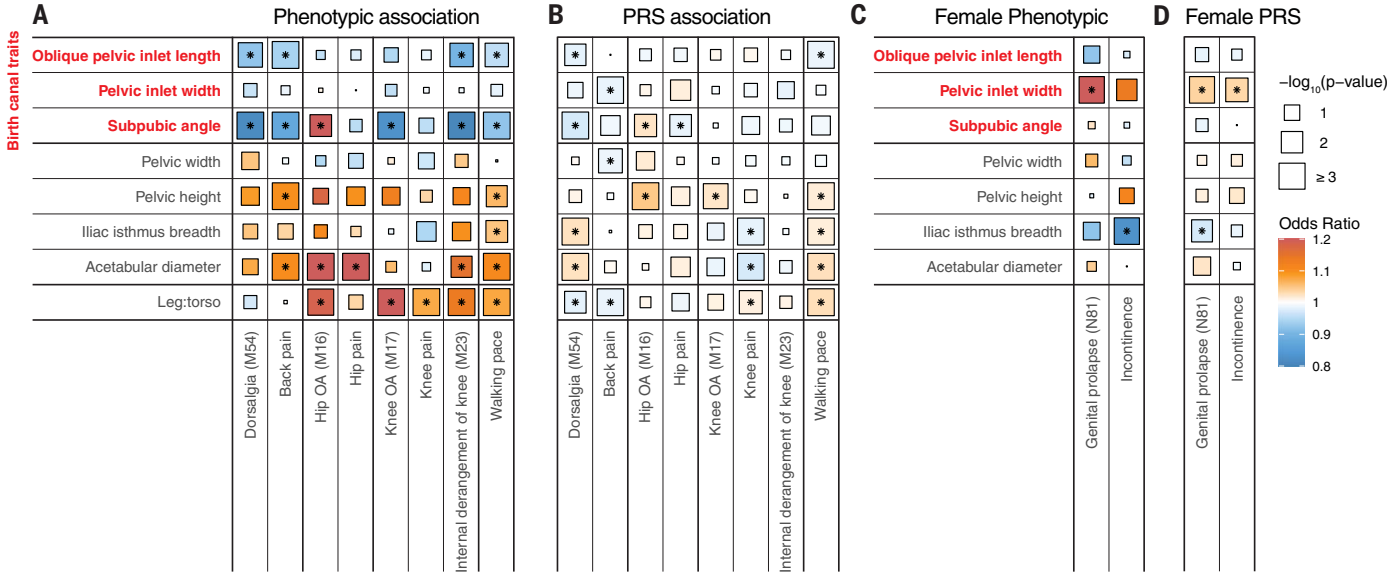

Fig. 3. Association between pelvic traits, pain phenotypes, musculoskeletal diseases, and walking pace. (**A**) Phenotypic associations from logistic regression analyses of musculoskeletal disease traits, self-reported pain, and walking pace on pelvic proportions. (**B**) PRS associations between musculoskeletal disease traits, walking pace, and pelvic proportions. (**C**) Female phenotypic associations between pelvic floor disorders and pelvic proportions. (**D**) Female PRS associations between pelvic floor disorders and pelvic proportions. For all panels, associations that are significant after FDR correction are annotated with an asterisk. The color and size keys to the right of (D) represent ORs for the phenotypic and PRS associations and *P* values, respectively. The number notations in parentheses are the ICD-10 codes associated with each disease: M54, dorsalgia; M16, coxarthrosis (arthrosis of hip); M17, gonarthrosis (arthrosis of knee); M23, internal derangement of knee; and N81, female genital prolapse.

between oblique pelvic inlet length and dorsalgia, $P = 3.45 \times 10^{-3}$, OR = 0.93; between subpubic angle and dorsalgia, $P = 3.28 \times 10^{-7}$, OR = 0.82; between subpubic angle and back pain, $P = 5.16 \times 10^{-7}$, OR = 0.87) (Fig. 3A and table S15). Additionally, individuals with smaller birth canal phenotypes were at decreased risk of hip OA (phenotypic association: between subpubic angle and hip OA, $P = 1.18 \times 10^{-2}$, OR = 1.27) but increased risk of knee OA (phenotypic association: between subpubic angle and knee OA, $P = 9.97 \times 10^{-4}$, OR = 0.83; between subpubic angle and internal derangement of knee, $P = 9.71 \times 10^{-5}$, OR = 0.81) (Fig. 3A and table S15).

To complement these phenotypic associations, we also analyzed 361,140 UKB participants who had not undergone DXA imaging and were of "white British ancestry" for predictive risk based on PRS derived from our GWAS on pelvic proportions for the imaged set of individuals (Fig. 3B and table S18). We generated PRS with Bayesian regression and continuous shrinkage priors (*52*) using HapMap3 SNPs and ran a logistic regression of the generated risk scores and traits, adjusting for the first 20 principal components of ancestry, and sex as well as age, weight, and other major risk factors of OA (*42*). Our genetic association analysis mirrored our phenotype association analysis and suggests that individuals with smaller birth canal proportions have on average a faster walking pace and are at the same time more susceptible to back pain and strain, common consequences of bipedal locomotion due to the distribution of weight on just two limbs (genetic association between leg to torso ratio and walking pace, $P = 1.00 \times 10^{-13}$, OR = 1.03; genetic association between oblique pelvic inlet length and walking pace, $P = 8.09 \times 10^{-4}$, OR = 0.98; genetic association between oblique pelvic inlet length and dorsalgia, $P = 1.31 \times 10^{-2}$, OR = 0.98; genetic association between pelvic inlet width and back pain, $P = 1.25 \times 10^{-3}$, OR = 0.98; genetic association between subpubic angle and dorsalgia, $P = 1.02 \times 10^{-4}$, OR = 0.97) (Fig. 3B and table S15).

Genetic and phenotypic association of pelvic proportions with pelvic floor function

Next, we combined all incontinence-related phenotypes from the ICD-10 record, including stress incontinence (N39.3), other specified urinary incontinence (N39.4), fecal incontinence (R15), and unspecified urinary incontinence (R32), into a single binary phenotype. We first carried out logistic regression analysis to examine the association between the binary incontinence-related phenotypes and female pelvic phenotypes. In this regression, we controlled for the same set of covariates that we used in the locomotion association analysis but also included the number of live births as an additional covariate. We observed a significant association between the female pelvic inlet width phenotype and genital prolapse ($P = 1.31 \times 10^{-5}$, OR = 1.21). There was a marginal association between the pelvic inlet width phenotype and incontinence ($P = 1.12 \times 10^{-2}$, OR = 1.14; after FDR correction, $P = 0.052$) (Fig. 3C and table S16).

We also conducted a GWAS restricted to female individuals who were imaged and computed a PRS for approximately 200,000 females of "white British ancestry" who were independent from the GWAS set. 18,020 individuals out of the 200,000 individuals had one of these incontinence phenotypes. We then regressed binary incidence of genital prolapse and incontinence against PRS for all female pelvic traits, controlling for the number of live births and age (*42*). Out of the pelvic proportions, the only significant positive association we observed was with the width of the birth canal (between pelvic inlet width and genital prolapse, $P = 4.3 \times 10^{-4}$, OR = 1.04; between pelvic inlet width and incontinence, $P = 4.2 \times 10^{-3}$, OR = 1.03) (Fig. 3D and table S19). Similar to the locomotor phenotype association analysis, the results from the pelvic floor function phenotypic association and the PRS association were well aligned. These results offer support for the multifactorial pelvic hypothesis, suggesting that a narrower birth canal improves pelvic floor function. Pelvic floor function is critical in assisting bladder and bowel control and evacuation as well as in supporting the internal organs and the fetus during pregnancy, a function thought to be more critical in upright humans than in quadrupeds (*5*).

Genetic association of pelvic proportions with childbirth-related outcomes

Obstructed labor is thought to be more common in humans than any other modern primate species (*9*) and affects around 16% of deliveries today. It also has been a major cause of maternal and fetal death throughout human history, which suggests that it might play a major role in human evolution through the effects of natural selection (*53*, *54*). Here, we examined the impact of pelvic proportions on obstructed labor. Firstly, we focused on C-sections reported in the UKB. To avoid confounding effects due to elective C-sections, we focused on emergency C-sections, which are routinely performed in cases of obstruction. We conducted PRS association analysis and found that narrower birth canals were associated with increased risk of emergency C-sections (P = 0.0108, OR = 0.92). As childbirth-related outcomes were available only for a small portion of individuals in the UKB (<10% of all individuals) we also examined outcomes in the FinnGen's dataset for delivery-related traits (*55*). We identified a significant genetic correlation between birth canal traits and labor obstructions due to maternal pelvic abnormalities [genetic correlation (r_g) = −0.4, $P = 3.92 \times 10^{-6}$], of which a major component is dilation width (*42*) (Fig. 4B and table S21). However, we saw no association between obstructed labor owing to malpresentation of the fetus and pelvic traits (Fig. 4B and table S21). Because the position of the fetus can vary independently of the skeletal structure of the pelvis, this childbirth outcome serves as a negative control for this analysis. Combining both types of analysis, our results suggest significant associations between the size of the birth canal and the chance of obstruction during labor.

Evolutionary escape

Lastly, we investigated associations that might help explain how the obstetrical dilemma may have been alleviated throughout recent human evolution. We examined whether gestation length in humans is shorter than in other primates of comparable body size to assess whether gestational duration plays a role in circumventing the pelvic constraints on delivering relatively large-brained human infants (*12*, *56*, *57*). However, we found no association between gestational duration and any pelvic proportion, including those associated with the birth canal (Fig. 4C and table S19). This result is in line with more recent data on a fairly large sample of great apes suggesting that human children are not born significantly earlier than those of the other apes (*13*, *33*–*35*). However, we observed a significant genetic correlation between adult head width and pelvic inlet width (r_g = 0.22, $P = 2.3 \times 10^{-3}$) as well as a significant correlation between the width of the birth canal and neonatal birth weight, a proxy for neonatal head size [Pearson correlation, r ~ 0.7 (*58*)] (regression slope = 0.01, $P = 2.4 \times 10^{-4}$) (Fig. 4C and table S19). This suggests that natural selection might have led to genetic correlation between pelvic and head proportions, potentially reducing the risk of labor obstruction (*51*, *59*).

Discussion

In this study, we used deep learning to understand the genetic basis of skeletal elements that make up human pelvic proportions using DXA imaging data in a large population-based biobank. We found sex-specific differences in genetic architecture as well as differences in pelvic symmetry that were associated with handedness. We identified 180 independent genetic loci associated with pelvic proportions. We then examined different facets of the obstetrical dilemma, namely the relationship between pelvic proportions and locomotor-, pelvic floor–, and childbirth-related outcomes. Lastly, we analyzed possible ways in which evolution and natural selection might have alleviated the dilemma by looking at genetic correlations between gestation length, the birthweight of the child, and pelvic proportions.

In previous work on the obstetrical dilemma, studies have examined locomotor outcomes that

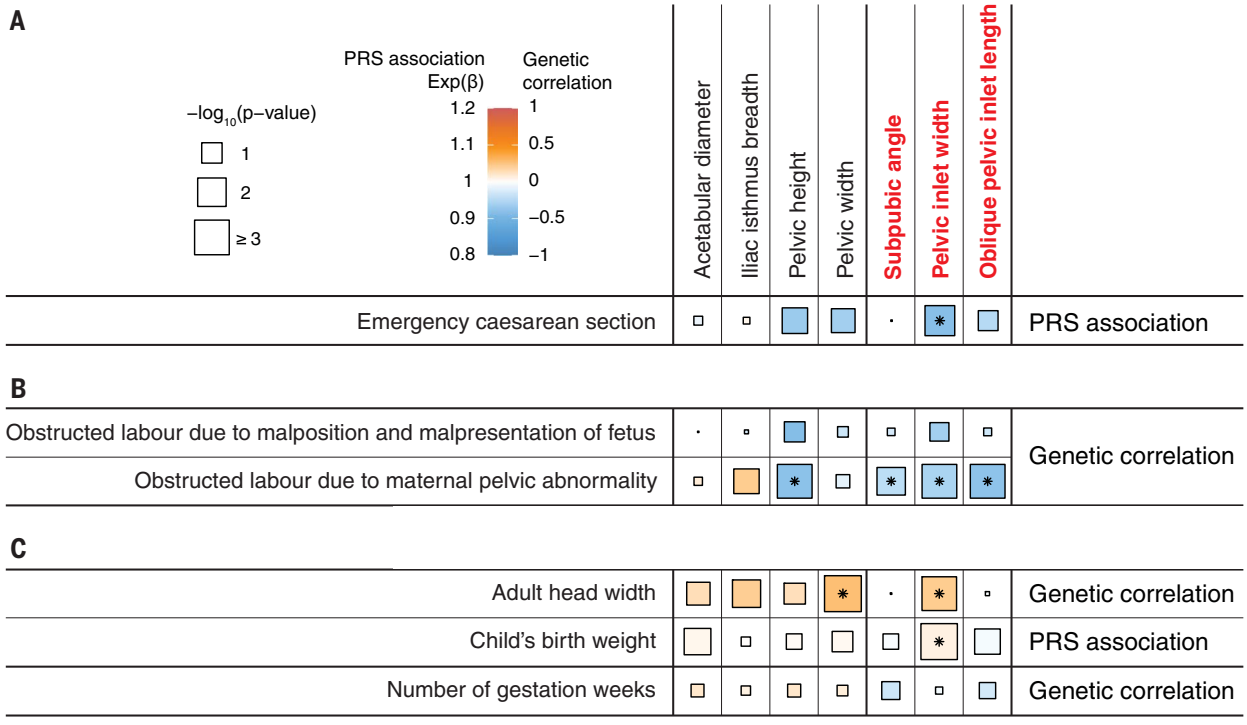

Fig. 4. Association between pelvic traits and childbirth-related outcomes. (**A**) PRS associations between pelvic traits and emergency C-section. (**B**) Genetic correlations between pelvic traits and obstructed labor, including obstructed labor due to malposition and malpresentation of fetus and obstructed labor due to maternal pelvic abnormality. (**C**) PRS associations or genetic correlation between pelvic traits and evolutionary escape variables, including adult head width, child birth weight, and gestation duration. For all panels, associations that are significant after FDR correction are indicated with an asterisk. Effect size for the PRS associations and genetic correlations are presented in various colors, and the P values are indicated by size.

are associated with efficiency and energy use rather than speed. In this work, we did not have access to energetics data, but we did have access to outcomes associated with walking speed and OA, which relate to gait efficiency accumulated over a lifetime. Although self-reported walking pace may not seem an ideal measure several lines of evidence suggest that it is a reliable measure of actual walking pace. Firstly, self-reported walking pace is highly heritable (*60*). Secondly, it is associated with muscle strength and declines with body mass index and age in line with expectations (*61*, *62*). It is also associated with several disorders that are known to hinder locomotion, including hip OA, the leading cause of adult disability in the United States (*63*–*65*). Lastly, self-reported walking pace has been directly correlated with measured walking pace in a study with a reasonably large sample size and within the biobank (*66*, *67*). It has also been correlated with mean accelerometer assessed activity (*68*).

Our results on locomotion were mixed, with larger birth canal phenotypes related to lower walking speed, reduced risk of back pain and knee OA, but increased risk of hip OA. However, our results provide significant evidence for other facets of the dilemma associated with pelvic floor function and childbirth. Specifically, we show that larger birth canal phenotypes are associated with increased risk of pelvic floor disorders but also reduced risk of obstruction during labor, two phenotypes that have direct impacts on human evolution owing to intense natural selection acting on them. Pelvic floor disorders may not have immediate impacts, given that they typically occur after the birth of the first child, but they could impact the probability of the individual having a second child and thereby have an effect on evolutionary fitness (*6*, *29*, *69*–*72*). We also investigated several leading hypotheses about how the dilemma could have been alleviated over evolutionary time. Our data do not provide support for the idea that gestational duration has decreased to accommodate birthing large-brained infants; we observed no correlation between any pelvic proportions and gestational duration. However, our results indicate that there is a genetic correlation between pelvic proportions related to birth canal width and head size [which we obtained by using adult head width and child birth weight as a proxy (*58*)]. Across all the skeletal traits we examined, the significantly reduced genetic correlation observed between males and females exclusively for birth canal phenotypes also suggests that sexual dimorphism in these traits may have arisen through natural selection in response to different functional constraints. Although the association between our birth canal phenotypes and childbirth and pelvic floor disorder phenotypes is only modest (OR > 1.5), over evolutionary timescales, this slight but significant increase in risk could have led to small differences in overall fitness that compounded over hundreds or thousands of generations.

A limitation of our study is that we only had individuals aged between 40 and 80 years old. It has been suggested that age is a source of variation in pelvic proportions and that changes in functional constraints throughout parts of the reproductive life span are another means by which the dilemma could be alleviated (*73*). However, we did not have access to data from individuals from earlier ages to examine this hypothesis.

Taken together, our work combines imaging, genetic, health record, and survey data at biobank scale to reexamine a central 65-year-old hypothesis in human evolution that is standardly presented in textbooks. Our results support established hypotheses about the obstetrical dilemma, including those related to constraints posed by childbirth and locomotion, and also highlight the notable role of pelvic floor health.

Materials and methods summary

All patient data, including electronic health records, DXA images, and genotype data, were obtained from the UKB (*74*). To perform phenotyping on 31,115 full-body DXA images from the UKB, we modified existing deep-learning models (*75*, *76*) used for landmark estimation by adding final additional training layers with manual annotation on 293 images. We used the landmark estimation model to estimate the coordinates of 17 landmarks, and based on these landmarks, we extracted seven different pelvic IDPs, including three birth canal–related phenotypes: oblique pelvic inlet length, pelvic inlet width, and subpubic angle.

After filtering UKB participants and genotype data for QC, we ran GWASs using BOLT-LMM (*44*) for each phenotype while controlling for height. We further estimated the heritability of these traits using GCTA (*43*). We performed linkage disequilibrium–based SNP pruning with PLINK (*77*, *78*) to find independent loci across our pelvic phenotypes with $r^2 < 0.1$ and mapped these lead SNPs to corresponding genes using PLINK (*77*, *78*). Next, we queried the Human-Mouse Disease Connection (*79*) database to determine which mouse phenotypes and human diseases were associated with these pelvic-associated genes. We also performed MAGMA gene property analysis to assess enrichment between genes expressed in different pelvic subelements across various developmental stages (*37*) and genes identified from our pelvic phenotype GWAS (*80*). Furthermore, we carried out genetic correlation analysis between male and female pelvic phenotypes using LDSC (*81*).

To examine the obstetrical dilemma hypothesis from multiple angles, we first conducted both phenotypic and PRS analyses. For phenotypic analysis, we regressed the binary outcome of disease or reported pain in the hip, knee, back, and walking pace against pelvic phenotypes while controlling for clinically relevant covariates known to affect OA (*82*), including age, sex, weight, and other factors.

For PRS analysis, we generated PRSs for each pelvic phenotype using Bayesian regression with continuous shrinkage priors (*52*). We ran a logistic regression of the PRS on traits across all individuals, adjusting for the first 20 principal components of ancestry and other covariates used in the phenotypic association analysis. Additionally, we carried out female-specific PRS analysis to assess the association between pelvic traits and two pelvic floor disorder–related phenotypes (genital prolapse and incontinence), emergency C-section, number of gestation weeks, and child birth weight. We controlled for the same covariates used in the all-individuals PRS analysis, except for sex, and we also included the number of live births as an additional covariate.

To assess the correlation between the genetic architecture of pelvic phenotypes and obstetrical labor, we downloaded the GWAS summary statistics of obstetrical labor from FinnGen (*55*) and performed genetic correlation analysis using LDSC (*81*).

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SUPPLEMENTARY MATERIALS

science.org/doi/10.1126/science.adq1521
Materials and Methods
Figs. S1 to S19
Tables S1 to S24
References (*85*–*90*)

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