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Sex differences in the nine-point Beighton hypermobility test scores

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Abstract

Joint hypermobility syndromes are often misinterpreted and wrongly diagnosed. Widely accepted naming such as Heritable Disorder of Connective Tissue has been shown to manifest features that closely overlap with better known disorders such as Marfan and Ehlers-Danlos syndromes and Osteogenesis Imperfecta. Currently the widely accepted "all-inclusive" diagnosis for generalised hypermobility is known as Benign Joint Hypermobility Syndrome (BJHS). People that do not have pain in their joints but still have lax joints are just considered to be hypermobile. It is currently understood that these are genetic-based conditions where connective tissue proteins such as collagen are formed differently. This results in the joints, muscles, tendons and ligaments being laxer and more fragile than is the case in non-hypermobile individuals. The aim of this study was to investigate possible differences in the occurrence of BJHS between males and females from a representative sample of a general population (aged 18-25). Testing was done on 300 recruits and 180 university students. The sample consisted of 55% males and 45% females. All subjects were tested using the 9 - point Beighton criteria for diagnosis of BJHS, with scores of 4/9 and greater being indicative of BJHS. All data were pooled and differences were observed when comparing male and female Beighton test scores. Results showed 36.41% incidence for females compared to 13.96% for males ($p \leq 0.05$). These differences may be due to hormonal influence on the laxity of tendons and ligaments. Furthermore it can also question the efficiency with which the Beighton score distinguishes between BJHS and general joint flexibility.

Keywords: Hypermobility, sex differences, Benign Joint Hypermobility Syndrome.

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Introduction

Joint hypermobility syndromes are often misinterpreted and diagnosed incorrectly, as the different types joint disorders are so closely related. Joint hypermobility first appeared in rheumatology reports as an important entity in 1967 (Kirk, Ansell & Bywaters, 1967). Now, some 40 years on, it is better

understood and more widely recognised. Widely accepted nomenclature like HDCT (Heritable Disorder of Connective Tissue) has been shown to manifest features that closely overlap with better known disorders such as Marfan's and Ehlers-Danlos syndromes and Osteogenesis Imperfecta (Grahame, Bird & Child, 2000). Currently the widely accepted "all-inclusive" diagnosis for hypermobility disorders is known as Benign Joint Hypermobility Syndrome (BJHS). The occurrence of BJHS without chronic arthralgia should not be viewed as a disease. However, acute bouts of joint pain may be experienced, as well as localised signs such as frequent sprains and dislocations (Nef & Gerber, 1998).

For many years extensive research has been done on the physiological, pathological and genetic mechanisms that play a part in BJHS. Research currently points to the fact that these are genetically-based conditions, where connective tissue proteins such as collagen are formed differently (Beighton, Grahame & Bird, 1999). Type 1 collagen is the most common collagen that tends to be affected and is contained in tendons, ligaments, joint capsules, skin, demineralised bone and nerve receptors (Russek, 1999.). The exact mechanism at work in BJHS has not been established, although it can be linked to the same pathogenic process that causes hypermobility in patients suffering from osteogenesis imperfecta, where the deficiency arises from the substitution of the amino acid glycine to bulkier amino acids in the triple helix structure of collagen. This exchange causes the larger amino acid side-chains to form steric hindrances that create a "bulge" in the collagen complex, which compromises both the molecular nano-mechanics as well as the interaction between the molecules (Rauch & Glorieux, 2004). This results in the joints, muscles, tendons and ligaments being laxer and more fragile than is the case in the general population (Beighton *et al*., 1999). Current thinking suggests that the shape of bones and low muscle tone may also be attributed+ to hypermobility, especially in ball and socket joints that are shallow. This gives rise to a greater range of motion around the joint (Myerson & Badekas, 2000).

The question arises as to whether or not hypermobile subjects have a greater risk of injury? Scientists have shown that the hypermobile population tends to have weaker proprioceptive ability. This decreased sense of joint position, which in itself is a strong indicator to greater risk of injury, may also be a factor in accelerated degeneration of joints in these individuals (Hall, Ferrell, Sturrock, Hamblen & Baxendale, 1995). Also, it has been reported that those with Hypermobility Syndrome (HMS) are more susceptible to osteoarthritis (Beighton *et al*., 1999). Increased nerve compression disorders (El-Shahaly & El-Sherif, 1991), mitral valve prolapse (Grahame, Edwards & Pitcher, 1981), chondromalacia patellae, excessive anterior mandibular movement (Buckingham, Braun & Harinstein, 1991) uterine prolapse, and varicose veins are some conditions that are also more prevalent in hypermobile individuals (Nef & Gerber, 1998). Regarding the prevalence of physical injury in those with BJHS, studies on professional footballers have shown no difference in the

incidence of injury frequency of athletes with BJHS as compared with those without. It was however interesting to note that players with HMS took longer to recover from injuries (Collinge & Simmonds, 2009). Studies done on professional ballet dancers have confirmed prolonged recovery time for dancers with BJHS (McCormack, Biggs, Hakim & Grahame, 2004; Briggs, McCormack, Hakim & Grahame, 2009).

Other factors for which correlations have been reported with hypermobility are race and gender. The evidence in these demographics are however conflicting. In a study which queried a military medical database in the United States, it was found that the prevalent race and gender associated with hypermobility was white females compared to black and "other" (Scher, Owens, Sturdivant & Wolf, 2010). These findings substantially contradict a study done in India, where a sample group of 829 Indian children between ages 3-19 were tested according to the Beighton 9-point protocol. They reported a 58.7% incidence of joint hypermobility, and a near equal gender incidence (Hasija, Khubchandani & Shenoi, 2008). Thus it can be noted that there are still questions to be answered regarding gender, race and incidence of BJHS in general populations.

The aim of this study was to identify the gender discrepancies concerning the incidence of BJHS in a general population. It is generally excepted that hypermobility is more prevalent among women, although many of the available studies were not done in recent years, had seemingly small sample groups and were not representative of the general population, as the samples consisted of children, individuals diagnosed with hypermobility related syndromes and sufferers of rheumatism in the older age group (Hudson, Hudson, Fitzcharles, Cohen, Starr & Esdaile, 1998).

Methods

Sample

The total sample for this study consisted of 480 individuals. The sample was randomly selected from four geographical areas of South Africa. The four groups that participated consisted of three training samples (total of 300 recruits) that spend at least two hours a day engaged in vigorous activity (> 6 MET's ; 1 MET = $3.5 ml.kg^{-1}.min^{-1}$) according to the American College of Sports Medicine classification of physical activity (Thompson, Gordon, & Pescatello, 2010). These subjects represented three different law enforcement academies (LEA 1, LEA 2, LEA 3), and the last group consisted of 180 2^{nd}-year university students. The inclusion criteria for all groups required the participants to be between the ages 18-25, written and signed informed consent to voluntary participation in the study and completion of a Physical Activity Readiness Questionnaire (PAR-Q) form. The exclusion criteria included any history of osteologic or rheumatologic diseases, as to exclude the occurrence of hypermobility as a symptom of these

syndromes. The purpose of the study was to only identify benign joint hypermobility syndrome. The total sample comprised 45% males (age 20.7 +- 1.16) and 55% females (age 20.4 +- 1.33).

HMS test

Several methods and criteria for testing HMS have been developed and re-evaluated over the years of study in this field. In this study subjects were tested according to the Beighton 9 -point scoring system. This is a modification of the Carter and Wilkinson scoring system and has been in use for many years to identify widespread hypermobility (Beighton *et al*., 1999). During the testing procedure, subjects were asked to extend (and hyper-extend if possible) 9 specific joints. For each joint a set point limit for range of motion is prescribed by the testing criteria as set out in Table 1. In every instance where the set point limit is exceeded by the subject, one score is awarded per joint. In order for the subjects to perform the movements correctly, the researcher illustrates the required action and then judges the range of motion achieved by the subject and awards scores accordingly. Table 1 and the illustrations in Figure 1 indicate the applicable joints and actions prescribed by the test criteria.

Table 1: Beighton 9-point hypermobility test

Joint	Setpoint limit criteria	Points
left little (fifth) finger	passive dorsiflexion beyond 90°	**1**
right little (fifth) finger	passive dorsiflexion beyond 90°	**1**
left thumb	passive dorsiflexion to the flexor aspect of the forearm	**1**
right thumb	passive dorsiflexion to the flexor aspect of the forearm	**1**
left elbow	hyperextend beyond 10°	**1**
right elbow	hyperextend beyond 10°	**1**
left knee	hyperextend beyond 10°	**1**
right knee	hyperextend beyond 10°	**1**
forward flexion of trunk with knees full extended	palms and hands can rest flat on the floor	**1**

Source: Grahame *et al.* (2000).

Figure 1: Illustrated maneuvers of the Beighton 9-point hypermobility test (Grahame *et al*., 2000)

For diagnostic purposes, the Beighton criteria state that a score of 4/9 or greater indicates the probability of BJHS (Beighton *et al*., 1999). The method is accurate and concise, and it is ideal for processing data from large samples. This method

is also one of the major criteria used in the revised Beighton diagnostic criteria for BJHS, which consists of a system combining major and minor indicators like the occurrence of artrhalgia, varicose veins, soft tissue rheumatism, abnormal skin and re-occurring dislocation and sub-location of joints (Grahame *et al*., 2000). Anthropometric measurements, body mass index (BMI) and body fat % data were also measured by means of height (m), weight (kg) and skinfold measurement. BMI was calculated by dividing the participants' weight in kilograms by their height in meters squared ($kg.m^{-2}$). Skinfold measures were recorded by using Body Logic© calibrated skinfold calipers to measure sub-cutaneous fat at 6 anthropometric sites (triceps, sub-scapular, abdominal, supra-iliac, thigh and calf).

The statistics were calculated using NCSS software. The descriptive statistics indicated ($\mu = 2.27 \pm 2.21$) for females and ($\mu = 1.14 \pm 1.66$). In the two-sample test report for independence, equal variances were rejected by the modified Levene test. Normal distribution was rejected at $\alpha = 0.05$. Figure 2 shows a box plot distribution of the average scores for males and females. A p-value of 0.0001 was reported for the correlation between these two values.

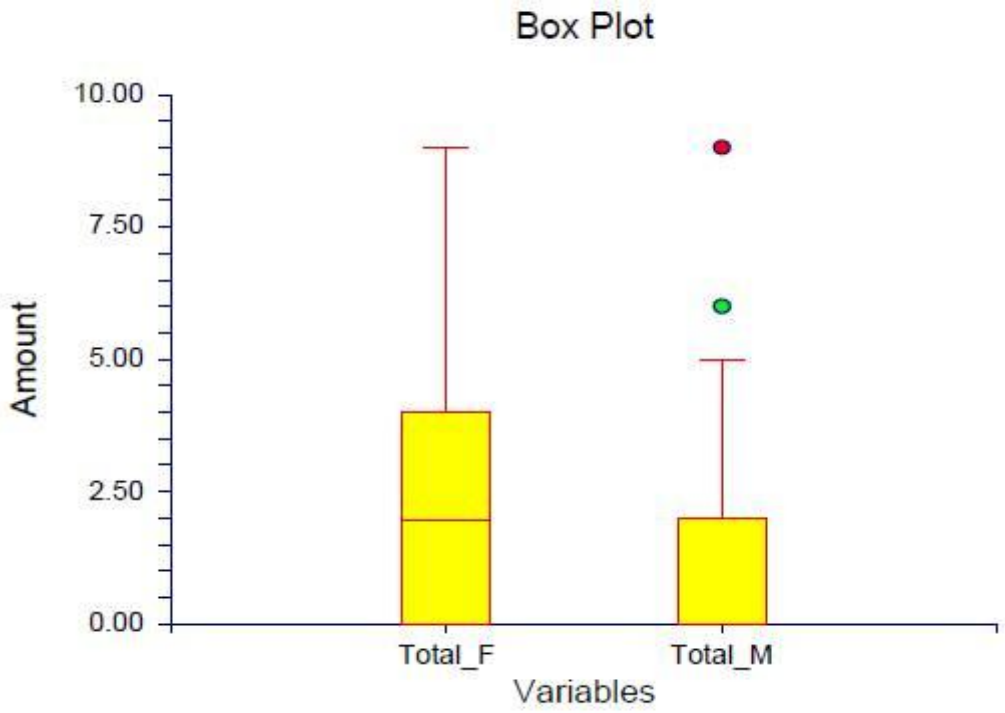

Figure 2: Box plot of gender specific results

Results

The results of the study indicated that 26.19% of all subjects tested were hypermobile (Table 2). When the incidence of BJHS in males (♂) and females (♀) was compared (Table 3), a higher prevalence was found in females than males. Gender specific totals showed a 36.41% prevalence for females compared to 13.96% for males.

Table 2: Incidence of hypermobility

Sample	2nd Years	LEA 1	LEA 2	LEA 3	Total
% HM > = 4	36.42%	17.31%	6.25%	14.35%	18.79%
% HM > = 6	16.05%	5.13%	1.56%	3.04%	6.36%
% HM = 9	3.09%	0.00%	0.00%	0.00%	1.04%
% Total population hypermobility > = 4					26.19%

Table 3: Gender-specific incidence of hypermobility

Sample	2nd Years		LEA 1		LEA 2		LEA 3		Total population	
	♂	♀	♂	♀	♂	♀	♂	♀	♂	♀
% HM > = 4	27.27	38.76	10.96	22.89	2.77	10.71	11.54	18.00	11.04	25.27
% HM > = 6	9.09	17.83	2.74	7.23	0.00	3.23	1.54	5.00	2.27	9.78
% HM = 9	3.03	3.10	0.00	0.00	0.00	0.00	0.77	1.00	0.65	1.36
% Gender specific totals									13.96	36.41

After statistical analysis no significant correlations between the occurrence of hypermobility and BMI or hypermobility and body fat percentages were found in the participants.

Discussion

This study presents clear and concise results that are comparable to previous studies due to the standardised method of testing and diagnostic protocol that were used. Statistically, a significant difference was reported in the occurrence of BJHS between males and females.

The present findings are consistent with results of previous studies. A study that examined the link between glenohumeral joint instability and hypermobility also reported a prevalence of female hypermobility. It is interesting to note that a general prevalence of only 1.5% was reported by the researchers, even though

the tests were also conducted using the Beighton 9-point scale scoring system for scores of 4 and greater (Cameron, Duffey, De Berardino, Stoneman, Jones & Owens, 2010). Another study comprising 660 subjects from a music school that were tested for the frequency of incidence and nature of hypermobility, also reported a high prevalence among women (Larsson, Baum & Mudholkar, 1987). What was striking when comparing the most current results were the discrepancies regarding the reported rates and magnitudes of incidence.

If current trends suggest that BJHS is caused by genetic predisposition (Beighton *et al*., 1999), what causes this genetic defect to favour females if it is generally expected that there is no significant difference in the structural composition of connective tissue proteins between males and females? Figure 3 shows the triple-helix structure of collagen with the central glycine proteins. As previously stated the genetic deficiency that is the likely cause of hypermobility disrupts the inclusion of these proteins in the collagen structure (Rauch & Glorieux, 2004).

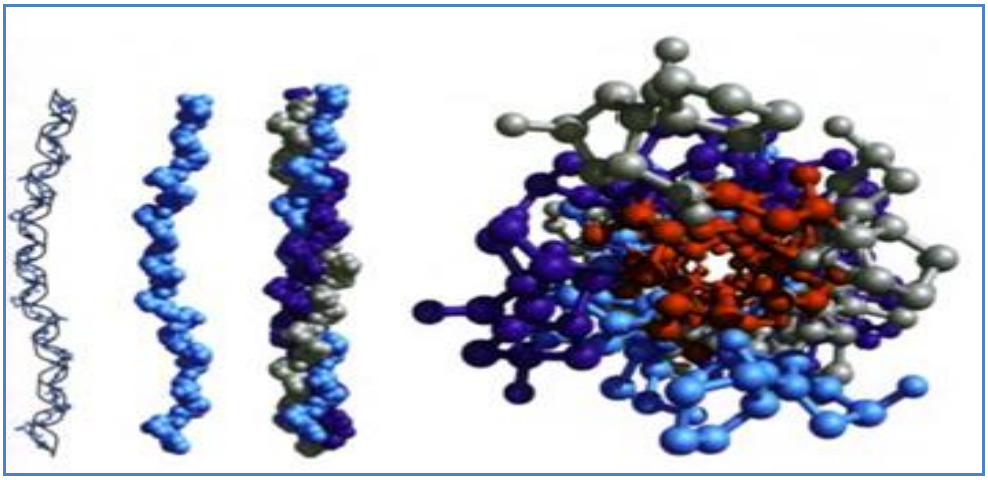

Figure 3: Structure of collagen with central glycine proteins (red)
(Nelson & Cox, 2003)

A more valid explanation can be found when looking at hormonal differences between men and women. The hormone relaxin is well known for its function of increasing laxity of the pubic symphysis and cervix prior to labour. The hormone is secreted by pregnant and non-pregnant women and its mechanism is believed to be the inhibition of collagen synthesis. Researchers found that the hormone increases laxity in the anterior cruciate ligament, and they confirmed the presence of relaxin receptors in this ligament (Dragoo, Lee, Benheim, Finerman & Hame, 2003). In a similar study the presence of estrogen and progesterone receptors in the anterior cruciate ligament were validated, when knee laxity was measured during various stages of the menstrual cycles of participants. Laxity of the knee increased at menstrual stages with varied secretion of these hormones (Park, Stefanyshyn, Hart, Loitz-Ramage & Ronsky, 2007). Thus, there is

substantial evidence to suggest that general joint laxity due to hormonal influence may play a major role in the prevalence of hypermobility among females.

Conclusions and Recommendations

Genetic disposition is the generally accepted cause of hypermobility, whether BJHS or those linked to malignant syndromes (Grahame *et al*., 2000). This study reported a significant difference in the prevalence of BJHS between males and females that could be attributed to factors other than genetic variance, such as the influence of female hormones on the laxity of joints. Future research should develop gender-based criteria for the diagnosis of BJHS. The purpose of such criteria is to distinguish between genetically influenced hypermobility and general laxity of ligaments that is caused by hormonal influence, joint anatomy, muscular tone and exercise induced flexibility, especially in females. This would ultimately lead to a clearer understanding and more accurate evaluation of hypermobility, as an indicative factor in various rheumatologic conditions.

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