Extraordinary Fast-Twitch Fiber Abundance Elite Weightlifters

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ABSTRACT

Human skeletal muscle fibers exist across a continuum of slow → fast-twitch. The amount of each fiber *type* (FT) influences muscle performance but remains largely unexplored in elite athletes, particularly from strength/power sports. To address this nescience, v*astus lateralis* (*VL*) biopsies were performed on World/Olympic (female, n=6, "WCF") and National-caliber (female, n=9, "NCF"; and male, n=6, "NCM") American weightlifters. Participant accolades included 3 Olympic Games, 19 World Championships, 25 National records, and >170 National/International medals. Samples were analyzed for myosin heavy chain (MHC) content via SDS-PAGE using two distinct techniques: single fiber (SF) distribution (%) and homogenate (HG) composition. These athletes displayed the highest MHC IIa concentrations ever reported in healthy *VL* (23±9% I, 5±3% I/IIa, 67±13% IIa, and 6±10% IIa/IIx), with WCF expressing a notable 71±17% (NCF=67±8%, NCM=63±16%). The heavyweights accounted for 91% of the MHC IIa/IIx fibers. When compared to SF, HG overestimated MHC I (23±9 vs. 31±9%) and IIx (0±0 vs. 3±6%) by misclassifying I/IIa fibers as I and IIa/IIx fibers as IIx. These findings suggest athlete caliber (World vs. National), training experience, and body mass determine FT% more than sex and refutes the common pronouncement that women possess more slow and fewer fast-twitch fibers than men. Our results also show the abundance of pure MHC IIa and rarity of IIx in elite strength/power-trained athletes, indicate a potential link between MHC IIa/IIx frequency and body mass, and question the fidelity of HG as a measure of FT% distribution. The extreme fast-twitch abundance partially explains how elite weightlifters generate high forces in rapid time-frames. These data highlight the need for more cellular and molecular muscle research on elite anaerobic athletes.

50 **INTRODUCTION**

51 Legendary Italian physician Stefano Lorenzini made the first distinction of "red" and
52 "white" muscle fibers (myofibers) in 1678, and almost 200 years later (1873) French histologist
53 Louis-Antoine Ranvier confirmed the existence of two distinct myofiber *types* in vertebrate
54 skeletal muscle. Reintroduction of the skeletal muscle biopsy procedure in 1962 (1) allowed
55 scientists to begin exploring the topic in athletes and resulted in the discovery that each fiber type
56 (FT) is comprised of a unique myosin heavy chain (MHC) isoform signature. Human skeletal
57 muscle therefore contains three *pure* (MHC I, IIa, and IIx) and several *hybrid* (single myofibers
58 that co-expresses multiple MHC isoforms) FT (2). The pure and hybrid FT combine to form a
59 robust slow → fast continuum (MHC I → I/IIa → IIa → IIa/IIx → IIx) with each displaying
60 specific morphological, metabolic, and contractile properties (3-6). FT distribution (FT%), or the
61 relative quantity of each FT in a given muscle, influences whole muscle function (7) and is often
62 highly correlated with athletic performance (3, 7-13).

63 Extensive evidence indicates endurance athletes possess a slow-twitch myofiber majority
64 (9, 10, 12, 14, 15), yet relatively few investigations have explored FT in speed, power, or strength
65 athletes. Initial research in the 1970-80's found resistance-trained men expressed high quantities
66 (~60-65%) of fast-twitch fibers (11, 12, 15, 16), which was substantiated by later studies on elite
67 powerlifters (17) and national-caliber (*Olympic*) weightlifters (8). This trailblazing work provided
68 an important foundation, but used sub-elite participants (18) and/or laboratory methods that failed
69 to accurately resolve the highly prevalent hybrids (19-21) - which compromises measurement
70 fidelity and produces erroneous FT% conclusions (19, 22-25). More precise techniques were
71 developed in the early 1990's that allowed proper quantification of FT% by analyzing each single
72 myofiber (SF).

73 Since this time only 13 studies (Table 1) implemented SF in young speed, power, or
74 strength-trained individuals (5, 13, 19, 20, 22, 23, 25-30), and only 3 included females (n = 13,
75 total). Only 5/13 included athletes: unknown-caliber male sprinters (n = 6) (25), male soccer
76 players (n = 8) (24), elite female track and field runners (n = 6) (20), National-caliber male
77 bodybuilders (n = 8) (19), and a former World-champion male sprinter (n = 1) (13). Accurately
78 accounting for the full FT spectrum resulted in all five studies finding far lower MHC IIa
79 concentrations than expected (52%, 30%, 16%, 39%, and 34%, respectively). The extremely low
80 16% found by Parcell et al. (2003) (20) is possibly explained by sex as females are often purported
81 to possess more slow-twitch fibers than men (31, 32). Such sex-specific phenotypes are often the
82 case in murine models (31), but the topic remains unexplored in athletes. Moreover, these data are
83 difficult to interpret as the athletes sampled were from a combination of several dissimilar events
84 (i.e., pole vault, heptathlon, 400 m hurdles, etc.).
85

86 **Table 1:** Summary of literature reporting single muscle fiber myosin heavy chain (MHC)
87 fiber-type from the *vastus lateralis* in young speed, power, or strength-trained individuals.

Reference	Subjects	Condition	MHC Distribution (%)					
			I	I/IIa	IIa	IIa/IIx	IIx	I/IIa/IIx
Andersen (1994)	Sprinters; 6M (23y)	Post 12-week RE & Interval Training	41	1	52	5	0	0
Andersen (1994)	Soccer; 8M (23y)	National Players Post 12-week RE Training	59	3	30	9	0	<1
Williamson (2001)	Non Ath; 6M (25y) 6F (21 y)	Post 12-week RE Training	30 35	5 3	59 52	5 12	0 0	0 0
Parcell (2003)	Track & Field; 6F (23y)	Division I / Interntional - Caliber	57	9	16	14	1	1

Raue (2005)	Non Ath; 6M (24y) 6M (24y)	Post-Con RE Post-Ecc RE	38 25	1 7	34 39	27 25	0 2	<1 <1
Parcell (2005)	Non Ath; 10M (22y)	Post 8-week Sprint Cycle Training	34	8	44	12	0	2
Malisoux (2006)	Non Ath; 8M (23)	Post 8-week Plyometric Training	28	5	42	26	2	0
Kesidis (2008)	Bodybuilding; 8M (26 y)	National-Caliber	35	19	39	7	0	0
Trappe (2015)	Sprinter; 1M (? y)	Previously World Champion	24	5	34	9	24	0
Murach (2016)	Non Ath; 9M (25y)	Resistance Trained	17	10	60	11	<1	<1
Bagley (2017)	Non Ath; 15M (25y)	Resistance Trained	20	10	58	11	1	1
Arevalo (2017)	Non Ath; 13M (24y)	Resistance Trained	28	9	60	3	<1	<1
Tobias (2017)	Non Ath; 1F (32y)	Concurrently Trained	45	13	31	9	0	2

MHC = Myosin heavy chain; M = Male; F = Female; y = Year; RE = Resistance exercise; Non Ath = Not a competitive athlete; Con = Concentric, Ecc = Eccentric

Numerous other knowledge gaps persist because in over 50 years of human muscle FT research only two studies have utilized SF with elite (i.e., world or international) athletes (one male sprinter and six female track and field) and no research has done so with any strength or power athlete. Thus, the purpose of this study was to examine the FT% of elite weightlifters to provide novel insight into the phenotype of competitive female and male strength and power athletes.

100 **METHODS**

101 Experimental Approach to the Problem

102 Twenty-one elite (‘*Olympic’)* Weightlifters (15 female, 6 male) underwent resting *vastus*
103 *lateralis* biopsies between 2-96 hours after competing in either the International Weightlifting
104 Federation World Championships or the USA Weightlifting American Open Finals (2017). All
105 procedures and risks were explained to the athletes prior to obtaining written consent and
106 completing medical and exercise history questionnaires. Performance records in the Snatch and
107 Clean and Jerk (1RM), competition medals, and other accolades were gathered from personal
108 interviews and publically available records from these or other sanctioned meets. Each muscle
109 sample was analyzed for MHC content using two distinct FT techniques: single fiber (SF) and
110 homogenate (HG). The University Institutional Review Board approved all experimental
111 procedures prior to any testing.

112 Participants

113 Participants were subdivided into three categories:

114 1. Olympic or World-caliber (n = 6 female): WCF
115 2. National-caliber female (n = 9): NCF
116 3. National-caliber male (n = 6): NCM

117 Athletes were considered “World-caliber” if they were on the most recent Olympic or
118 World team and competed at the most recent National event. Athletes were considered “National-
119 caliber” if they were top 5 placers at the 2017 American Open Finals meet but had never been on
120 a World or Olympic team. Athletes spanned multiple weight categories, had a minimum of two
121 years of National competition experience, had competed exclusively for the United States of

America, and were otherwise eligible for all American National meets (Table 2). Athlete accolades at the time of data collection included participation in 3 Olympic Games, 19 World Championships, 11 Pan American Championships, 49 National Championships, 32 American Opens, 8 University National Championships, and 25 Junior World/Pan American/National Championships. Participants also held 25 National records and >170 National/International medals. One athlete had tested positive for substances prohibited by the World Anti-Doping Agency and was suspended from the sport for two years prior to participating in the study.

129 **Table 2.** Descriptive information of elite female and male American weightlifters.

	Age (y)	Body Mass (kg)	Height (cm)	Years Competing (y)	Snatch Relative 1RM	Clean & Jerk Relative 1RM
WCF	28.2 ± 3.6*	81.2 ± 36.0	164.0 ± 11.1	7.7 ± 4.7*ǂ	1.32 ± 0.31*	1.69 ± 0.40*
NCF	23.6 ± 3.9	66.6 ± 11.0	164.8 ± 7.0	3.8 ± 0.8	1.29 ± 0.18ǂ	1.68 ± 0.19ǂ
NCM	26.0 ± 2.4	85.3 ± 26.9	169.0 ± 9.0	3.3 ± .08	1.64 ± 0.25	2.04 ± 0.29
Average	25.6 ± 3.8	76.1 ± 25.0	165.8 ± 8.7	4.8 ± 3.1	1.40 ± 0.28	1.79 ± .032

130 Data are described as mean ± standard deviation. WCF = Olympic or World-caliber female (n = 6), NCF = National-caliber female (n
131 = 9), NCM = National-caliber male (n = 6). Relative 1RM = competition record one repetition maximum divided by body mass. Years
132 competing = number of years competing in USA Weightlifting sanctioned meets. * = significantly different than NCF. ǂ =
133 significantly different than NCM. Significant = $p < 0.05$.

134 Procedures

135 *Vastus Lateralis Muscle Biopsies*

136 Following 30 minutes of supine rest, athletes underwent a mid-muscle belly
137 (approximately halfway between the greater trochanter and patella) biopsy of the *vastus lateralis*.
138 A detailed description of the biopsy procedure has been previously described by our lab (9, 22, 23,
139 33). Briefly, a small area of the thigh was numbed by injection of a local anesthetic
140 (Xylocaine/Lidocaine without epinephrine). An approximately ¼ inch incision was made in the
141 superficial cutaneous tissues. Muscle samples were obtained using the Bergström technique with
142 suction (1), immediately cleansed of excess blood and connective tissue, divided into
143 approximately 10-15 mg strips, placed into cold skinning solution (125 mM K propionate, 2.0 mM
144 EGTA, 4.0 mM ATP, 1.0 mM $MgCl_2$, 20.0 mM imidazole [pH 7.0], and 50% [vol ml/vol ml]
145 glycerol), and stored at -20° C for at least one week. Each sample was split such that a portion (~5
146 mg) could be used for single fiber isolation or homogenization. The incision site was cleaned,
147 pulled closed with a sterile Band-Aid, and covered with sterile gauze and cohesive bandage tape.

148

149 *Myosin Heavy Chain Fiber Type Identification*

150 All biopsy samples were analyzed for MHC via SDS-PAGE using two distinct techniques:
151 single fiber *distribution* (SF) and homogenate *composition* (HG). For SF, individual fibers (N =
152 2,147; 102 ± 3 fibers per athlete) were mechanically isolated with fine tweezers under a light
153 microscope and placed in 80 µl of sodium dodecyl sulfate (SDS) buffer (1% SDS, 23 mM EDTA,
154 0.008% bromophenol blue, 15% glycerol, and 715 mM b-mercaptoethanol [pH 6.8]). HG samples
155 (~5 mg) were hand homogenized and then diluted between 1:10 to 1:50 based on sample amount

156 and protein quantity. As described in detail elsewhere (5, 9, 22, 23, 27), 1-2 µl aliquots of both SF
157 or HG (run separately) were then loaded into individual wells in a 3.5% loading and 5% separating
158 gel (SDS-PAGE), run at 5°C for 15.5 hours (SE 600 Series; Hoefer, San Francisco, CA, USA),
159 and silver stained for MHC identification. The SF approach used known molecular weights and
160 standards to identify the MHC isoform (MHC I, I/IIa, IIa, IIa/IIx, and IIx) of each individual
161 myofiber. This enabled the most accurate calculation of the distribution/percent frequency (FT%)
162 of each FT contained within the muscle sample (21). For example, if 100 fibers were analyzed and
163 30 were identified as MHC I, 60 as MHC IIa, and 10 as MHC IIa/IIx, the FT% would be calculated
164 as 30% MHC I, 60% MHC IIa, and 10% MHC IIa/IIx. HG utilized densitometry (ImageJ, National
165 Institutes of Health, Bethesda, MD) to quantify the relative MHC protein composition (i.e., percent
166 area occupied by each pure isoform; MHC I, IIa, and IIx) of each sample, which is correlated
167 highly with FT area (34). Thus, SF indicates how frequently each isoform exists but cannot address
168 how much area each FT occupies within the muscle. HG addresses the latter, but cannot delineate
169 hybrids, therefore inaccurately quantifying FT% (9, 21-25, 27).

170 **Statistical Analysis**

171 Potential differences between groups in descriptive information were examined via
172 ANOVA. For SF, potential differences in FT% between groups were assessed via a 3 (group:
173 WCF, NCF, NCM) x 4 (fiber type: MHC I, I/IIa, IIa, IIa/IIx) ANOVA. For HG, potential
174 differences in FT composition between groups were examined via a 3 (group: WCF, NCF, NCM)
175 x 3 (fiber type: MHC I, IIa, IIx) ANOVA. Comparison of SF vs. HG was accomplished by a 2
176 (group: SF, HG) x 3 (fiber type: MHC I, IIa, IIx) ANOVA. Effect size was calculated with Cohen's
177 D (0.2 = small difference, 0.5 = medium difference, and 0.8 = large difference) to identify the
178 magnitude of difference between two groups. Pearson Product Moment Correlations (*r*) were

179 assessed for WCF, NCF, and NCM between 1RM, body mass, and SF FT%. All individual FT
180 data are reported in Table 3. Data are reported as mean ± standard deviation (SD), unless otherwise
181 noted. Significance was established *a priori* at an alpha level of $p < 0.05$. All analyses were
182 performed with SPSS (SPSS Statistics Version 24, IBM).
183

184 **RESULTS**

185 *Descriptive*

186 WCF were significantly older than NCF, but not NCM (Table 2). WCF also had
187 significantly more years of sport competition experience than NCF and NCM. Yet, NCM exceeded
188 both WCF and NCF in relative strength in both the Snatch 1RM and Clean and Jerk 1RM.

189

190 **Table 3.** Individual single muscle fiber type distribution (SF) and homogenate composition (HG)
191 of elite female and male American weightlifters**.** Data are reported as a percentage.

	MHC I		**MHC I/IIa**	**MHC IIa**		**MHC IIa/IIx**	**MHC IIx**	
Athlete	**SF%**	**HG%**	**SF%**	**SF%**	**HG%**	**SF%**	**SF%**	**HG%**
World-Caliber Female Weightlifters								
1	13	31	7	74	69	7	0	0
2	39	34	13	48	66	0	0	0
3	18	21	2	79	79	1	0	0
4	9	19	2	89	81	0	0	0
5	12	22	4	85	78	0	0	0
6*	10	17	9	52	70	28	0	13

National-Caliber Female Weightlifters								
7	23	38	1	76	62	0	0	0
8*	14	18	1	63	68	22	0	15
9	32	43	6	62	57	0	0	0
10	29	39	8	58	61	4	0	0
11	20	26	2	78	74	0	0	0
12	29	46	5	66	54	0	0	0
13	25	30	2	73	70	0	0	0
14	19	25	6	74	75	0	0	0
15	34	35	6	56	65	4	0	0
National-Caliber Male Weightlifters								
16	29	40	8	63	60	0	0	0
17	26	37	1	73	63	0	0	0
18	32	44	3	65	56	0	0	0
19	7	18	3	84	82	6	0	0
20*	26	36	3	54	57	17	0	7
21*	29	33	2	37	49	32	0	18

MHC = myosin heavy chain. * Denotes athlete in the heavyweight (or super) (>90 kg for women and >105 kg for men) category.

198 *Single Fiber Analysis (SF)*

199 FT% for all lifters combined was 23 ± 9% I, 5 ± 3% I/IIa, 67 ± 13% IIa, and 6 ± 10%
200 IIa/IIx. No MHC IIx or I/IIa/IIx fibers were identified. No significant differences existed between
201 groups, despite WCF possessing 8% (absolute, not percent difference) less MHC I than NCF (d =
202 0.88) and NCM (d = 0.78) (Figure 1). The difference in MHC IIa between WCF and NCM (also
203 8%) was also not statistically significant, but had a moderate effect size (d = 0.50). The vast
204 majority of the MHC IIa/IIx fibers (91%) belonged to just five lifters, all of whom competed in
205 the heavyweight or super heavyweight categories (≥ 90 kg for women and ≥105 kg for men). Thus,
206 significant correlations were present between body mass and MHC IIa/IIx frequency for WCF (*r*
207 = 0.919, p = 0.010) and NCF (*r* = 0.826, p = 0.006) while a trend existed for NCM (*r* = 0.757, p =
208 0.080).

209

210 **Figure 1. Myosin heavy chain (MHC) fiber type distribution of elite American weightlifters.**
211 WCF = Olympic or World-caliber female (n = 6), NCF = National-caliber female (n = 9), NCM =
212 National-caliber male (n = 6). Data are reported as a percentage ± standard deviation.

213

214 *Homogenate Analysis (HG)*

215 FT composition for all lifters combined was 31 ± 9% I, 67 ± 9% IIa, and 3 ± 6% IIx. MHC
216 I tended (p = 0.08) to be lower in WCF (24 ± 7%) than NCF (33 ± 9%, p = 0.125, d = 1.33) and
217 NCM (35 ± 9%, p = 0.106, d = 1.14), yet MHC IIa was significantly higher (p = 0.046) in WCF
218 (74 ± 6%) than NCF (65 ± 7%, p = 0.145, d = 1.28) and NCM (61 ± 11%, p = 0.043, d = 1.39). FT
219 was significantly different (p < 0.001) between SF and HG for MHC I (p = 0.005) and MHC IIx

220 (p = 0.046), but not MHC IIa. SF MHC IIa/IIx and HG MHC IIx were highly correlated (r = 0.96,
221 p < 0. 001). No correlations existed for SF or HG between FT% and Snatch or Clean and Jerk
222 relative 1RM.

223

224 **DISCUSSION**

225 The current study resulted in the most detailed investigation of muscle phenotype in
226 Olympic and World-caliber anaerobic athletes published to date. Additionally, the data enabled
227 the first comparison and differentiation of World vs. National-caliber athletes at the single fiber
228 level. The current study was also the most precise description of FT% in strength or power sport
229 competitors, and the first ever in females. The MHC IIa abundance was the highest in healthy
230 muscle (VL) ever reported, especially for females. These data suggest athlete caliber and/or
231 training history influences FT% more than sex *per se* and also questions the pronouncement that
232 male athletes possess more fast-twitch myofibers than females. Our utilization of two different
233 typing methods confirmed the limitations of HG for FT% (inappropriately categorizes MHC I/IIa
234 as MHC I and MHC IIa/IIx as MHC IIx) and also allowed identification of a previously
235 undocumented relationship between body mass and MHC IIa/IIx concentrations. The unique
236 morphology and phenotypes in our participants highlight the need to further study elite anaerobic
237 athletes, particularly females.

238 WCF contained the highest concentration of MHC IIa (71%) ever reported in the literature
239 to our knowledge. NCF (67%) and NCM (63%) also possessed more MHC IIa than previous
240 research in competitive bodybuilders (40%) (16, 19) as well as power/weightlifters (8, 11, 12, 15,
241 16, 18), elite track and field athletes (20, 35), and resistance-trained men (18, 19, 22, 23, 27, 29,

242 36), which all ranged from 50-60%. Only six previous studies using SF have found pure MHC IIa
243 concentrations of >50%, with just two reporting 60% (Table 1). The resulting minimal MHC I
244 (~17-25%) in our athletes was strikingly lower than elite female track and field athletes (57%) (20)
245 and National-caliber bodybuilders (35%) (19). These pronounced differences are likely explained
246 by the substantial dissimilarities in training styles (e.g., external loading strategies, contraction
247 type and velocity, training frequency, etc.) between the various sports. More research is therefore
248 needed to continue delineating the subtle but significant differences in FT% between top-
249 performing athletes in various anaerobic sports and the specific role each training approach might
250 play on altering MHC I and IIa distribution. Although it did not reach statistical significance, large
251 ES were evident and MHC IIa frequencies of 74%-89% occurred in 66% of WCF but only in 44%
252 and 33% of NCF and NCM, respectively. Thus, scientists should continue to examine what
253 separates World from National-level athletes.

254 WCF differed from NCF and NCM in both sex and years competing in the sport (~8 vs. 3
255 y). Sex comparisons in athletes remains tenuous (31, 37) because nearly all investigations utilize
256 non-gold standard FT% methods (21) and sedentary (38, 39) or "recreationally active" individuals
257 (32). Not only do our findings contradict the claim that women possess more slow-twitch
258 myofibers than men (40), they illustrate the opposite when accounting for talent level (WCF <
259 NCF = NCM). The current cross-sectional study-design precludes direct analysis, but extensive
260 research affords strong support for training history as a critical determinate of FT% (2, 9, 26, 28,
261 30, 41-44). Chronic exercise generally decreases hybrids (30, 42) and induces style-specific shifts
262 in FT% such as increases in MHC I with endurance (9, 43) or MHC IIa with sprint (28), plyometric
263 (26), or strength training (36, 43-45). For example, MHC I concentrations in an individual with
264 extensive endurance exercise history were nearly double that of his non-exercising monozygous

265 twin (9). Another study reported an increase in MHC IIa from 46% to 60% following 19 weeks of
266 resistance training (36). MHC IIa/IIx fibers appear particularly responsible for exercise-induced
267 increases in MHC IIa and are thus uncommon in exercise-trained individuals (9, 20, 22-25, 29,
268 43). A reduction of MHC IIx in favor of IIa following chronic resistance exercise is also purported
269 extensively in the literature (28, 34), yet the overwhelming majority of this evidence comes from
270 experiments with methodologies directly shown here and elsewhere (9, 24, 25) to produce
271 erroneous FT% conclusions.

272 Most research from the 1970's – 2000's utilized either ATPase histochemistry or HG SDS-
273 PAGE to determine FT% (8, 14-17, 24, 25, 34, 36). Similar to SF, histochemistry allows
274 assessment of individual fibers for calculation of percent distribution, yet it does not enable
275 simultaneous delineate of hybrids (36). HG suffers the same drawback and actually indicates FT
276 area/composition (34) more so than distribution making it greatly influenced by the size of each
277 fiber; which is not uniform across all FT (particularly in resistance trained individuals) (46). All
278 three approaches hold strong merit and are often correlated to each other (34, 47) and performance
279 (8), but are clearly not interchangeable for maximally precise FT% assessment. In the current
280 study, HG accurately quantified MHC IIa (within 0-4%), but not I or IIx. MHC I was overestimated
281 by 8% percent (23 vs. 31%), which is largely explained by the non-differentiated MHC I/IIa fibers
282 (5%). HG also greatly exaggerated MHC IIx, particularly in individuals with >4% MHC IIa/IIx.
283 The inability of HG to account for MHC IIa/IIx explains why MHC IIx appear common in some
284 studies (48) even though their actual abundance in healthy human skeletal muscle is extraordinarily
285 rare; typically <0.1% (9, 22-25, 27) and 0 of the >2,100 isolated fibers from the current sample.
286 Thus, the seeming conversion of MHC IIx to IIa with exercise is more precisely IIa/IIx changing
287 to IIa.

For these reasons MHC IIa/IIx are typically inversely associated with muscle health and physical activity (9, 43). Yet, the heavyweights (male and female) expressed irregularly high concentrations (24%) and accounted for 91% of all MHC IIa/IIx myofibers. Terzis and colleagues (2010) noted a similar abnormal abundance of MHC IIx (typed via HG, so likely IIa/IIx) in six large (116 kg, body fat composition >22%), but presumably well strength-trained throwers (35). Body composition was not assessed in the current study and little research exists on well-trained, but obese individuals. Thus, additional studies across a broader spectrum of physical size are required to truly interpret the correlations between body mass and MHC IIa/IIx prevalence.

Another juxtaposition was that of FT% and performance. Previous work in 94 kg male competitive weightlifters found strong correlations between FT composition (HG) and percent FT area to both snatch 1RM and vertical jump height (8), but not clean and jerk 1RM. We failed to identify any such correlations, but also utilized multiple sexes and weight classes. Thus, while FT% differed between our groups, that factor alone did not predict performance among our lifters. Several possible explanations exist for this discrepancy. First, FT area may determine whole muscle strength more than FT%. Second, neither studies found correlations to the clean and jerk, which is heavier and slower than the snatch or vertical jump. This compliments previous isokinetic research (23) and indicates FT% does not predict performance on strength tasks among strength-trained individuals. FT% probably determines movement speed more than force production (7). Further speculation on this point is unwarranted as limitations prohibited the ability to assess FT-specific size or contractile properties, which likely differed significantly across our groups (49) and are known to changes with training (3, 46).

CONCLUSION

This study provides novel insight into the muscle phenotype of elite competitive strength and power athletes. Our data indicate athlete caliber, training history, and body mass dictate FT% more than sex *per se*, but more work is needed to draw firm conclusions. The extreme fast-twitch abundance partially explains how elite weightlifters are able to generate high forces in short time-frames. Most athletes contained few hybrids and no MHC IIx or I/IIa/IIx, except the heavyweights who possessed large quantities of IIa/IIx. Future research should use high fidelity techniques to explore FT-specific distribution, size, and contractile properties in female and male athletes of various caliber, sports, and body size; ideally across several years of competition. The resulting knowledge could have practical significance if it enabled experimentation of differing training volumes or recovery protocols based on athlete-specific FT properties (50). More detailed fiber type profiles of elite strength and power athletes may eventually enable strength and conditioning professionals to create more individualized and effective programs.

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334 **REFERENCES**

335 1. Bergstrom J. Muscle electrolytes in man determined by neutron activation analysis on
336 needle biopsy specimens. The Scandinavian Journal of Clinical and Laboratory Investigations.
337 1962;14 (Suppl. 68).
338 2. Pette D, Staron RS. Myosin isoforms, muscle fiber types, and transitions. Microsc Res
339 Tech. 2000;50(6):500-9.
340 3. Methenitis S, Karandreas N, Spengos K, Zaras N, Stasinaki AN, Terzis G. Muscle Fiber
341 Conduction Velocity, Muscle Fiber Composition, and Power Performance. Med Sci Sports
342 Exerc. 2016;48(9):1761-71.
343 4. Galpin AJ, Raue U, Jemiolo B, Trappe TA, Harber MP, Minchev K, et al. Human
344 skeletal muscle fiber type specific protein content. Anal Biochem. 2012;425(2):175-82.
345 5. Tobias IS, Lazauskas KK, Arevalo JA, Bagley JR, Brown LE, Galpin AJ. Fiber type-
346 specific analysis of AMPK isoforms in human skeletal muscle: advancement in methods via
347 capillary nano-immunoassay. J Appl Physiol (1985). 2017.
348 6. Bagley JR. Fibre type-specific hypertrophy mechanisms in human skeletal muscle:
349 potential role of myonuclear addition. J Physiol. 2014;592(23):5147-8.
350 7. Bottinelli R, Pellegrino MA, Canepari M, Rossi R, Reggiani C. Specific contributions of
351 various muscle fibre types to human muscle performance: an in vitro study. J Electromyogr
352 Kinesiol. 1999;9(2):87-95.
353 8. Fry AC, Schilling BK, Staron RS, Hagerman FC, Hikida RS, Thrush JT. Muscle fiber
354 characteristics and performance correlates of male Olympic-style weightlifters. J Strength Cond
355 Res. 2003;17(4):746-54.
356 9. Bathgate KE, Bagley JR, Jo E, Talmadge RJ, Tobias IS, Brown LE, et al. Muscle health
357 and performance in monozygotic twins with 30 years of discordant exercise habits. Eur J Appl
358 Physiol. 2018.
359 10. Costill DL, Daniels J, Evans W, Fink W, Krahenbuhl G, Saltin B. Skeletal muscle
360 enzymes and fiber composition in male and female track athletes. J Appl Physiol.
361 1976;40(2):149-54.
362 11. Tesch PA, Karlsson J. Muscle fiber types and size in trained and untrained muscles of
363 elite athletes. J Appl Physiol (1985). 1985;59(6):1716-20.
364 12. Prince FP, Hikida RS, Hagerman FC. Human muscle fiber types in power lifters, distance
365 runners and untrained subjects. Pflugers Arch. 1976;363(1):19-26.
366 13. Trappe S, Luden N, Minchev K, Raue U, Jemiolo B, Trappe TA. Skeletal muscle
367 signature of a champion sprint runner. J Appl Physiol (1985). 2015;118(12):1460-6.
368 14. Gollnick PD, Armstrong RB, Saubert CWt, Piehl K, Saltin B. Enzyme activity and fiber
369 composition in skeletal muscle of untrained and trained men. J Appl Physiol. 1972;33(3):312-9.
370 15. Tesch PA, Thorsson A, Kaiser P. Muscle capillary supply and fiber type characteristics in
371 weight and power lifters. J Appl Physiol Respir Environ Exerc Physiol. 1984;56(1):35-8.
372 16. Tesch PA, Larsson L. Muscle hypertrophy in bodybuilders. Eur J Appl Physiol Occup
373 Physiol. 1982;49(3):301-6.
374 17. Fry AC, Webber JM, Weiss LW, Harber MP, Vaczi M, Pattison NA. Muscle fiber
375 characteristics of competitive power lifters. J Strength Cond Res. 2003;17(2):402-10.
376 18. Galpin AJ, Fry AC, Chiu LZ, Thomason DB, Schilling BK. High-power resistance
377 exercise induces MAPK phosphorylation in weightlifting trained men. Appl Physiol Nutr Metab.
378 2012;37(1):80-7.

379 19. Kesidis N, Metaxas TI, Vrabas IS, Stefanidis P, Vamvakoudis E, Christoulas K, et al.
380 Myosin heavy chain isoform distribution in single fibres of bodybuilders. Eur J Appl Physiol.
381 2008;103(5):579-83.
382 20. Parcell AC, Sawyer RD, Craig Poole R. Single muscle fiber myosin heavy chain
383 distribution in elite female track athletes. Med Sci Sports Exerc. 2003;35(3):434-8.
384 21. Pandorf CE, Caiozzo VJ, Haddad F, Baldwin KM. A rationale for SDS-PAGE of MHC
385 isoforms as a gold standard for determining contractile phenotype. J Appl Physiol (1985).
386 2010;108(1):222-2; author reply 6.
387 22. Arevalo JA, Lynn SK, Bagley JR, Brown LE, Costa PB, Galpin AJ. Lower-Limb
388 Dominance, Performance, and Fiber Type in Resistance-trained Men. Med Sci Sports Exerc.
389 2017.
390 23. Bagley JR, McLeland KA, Arevalo JA, Brown LE, Coburn JW, Galpin AJ. Skeletal
391 Muscle Fatigability and Myosin Heavy Chain Fiber Type in Resistance Trained Men. J Strength
392 Cond Res. 2017;31(3):602-7.
393 24. Andersen JL, Klitgaard H, Bangsbo J, Saltin B. Myosin heavy chain isoforms in single
394 fibres from m. vastus lateralis of soccer players: effects of strength-training. Acta Physiol Scand.
395 1994;150(1):21-6.
396 25. Andersen JL, Klitgaard H, Saltin B. Myosin heavy chain isoforms in single fibres from
397 m. vastus lateralis of sprinters: influence of training. Acta Physiol Scand. 1994;151(2):135-42.
398 26. Malisoux L, Francaux M, Nielens H, Renard P, Lebacq J, Theisen D. Calcium sensitivity
399 of human single muscle fibers following plyometric training. Med Sci Sports Exerc.
400 2006;38(11):1901-8.
401 27. Murach KA, Bagley JR, McLeland KA, Arevalo JA, Ciccone AB, Malyszek KK, et al.
402 Improving human skeletal muscle myosin heavy chain fiber typing efficiency. J Muscle Res Cell
403 Motil. 2016;37(1-2):1-5.
404 28. Parcell AC, Sawyer RD, Drummond MJ, O'Neil B, Miller N, Woolstenhulme MT.
405 Single-fiber MHC polymorphic expression is unaffected by sprint cycle training. Med Sci Sports
406 Exerc. 2005;37(7):1133-7.
407 29. Raue U, Terpstra B, Williamson DL, Gallagher PM, Trappe SW. Effects of short-term
408 concentric vs. eccentric resistance training on single muscle fiber MHC distribution in humans.
409 Int J Sports Med. 2005;26(5):339-43.
410 30. Williamson DL, Gallagher PM, Carroll CC, Raue U, Trappe SW. Reduction in hybrid
411 single muscle fiber proportions with resistance training in humans. J Appl Physiol (1985).
412 2001;91(5):1955-61.
413 31. Haizlip KM, Harrison BC, Leinwand LA. Sex-based differences in skeletal muscle
414 kinetics and fiber-type composition. Physiology (Bethesda). 2015;30(1):30-9.
415 32. Norman B, Esbjornsson M, Rundqvist H, Osterlund T, von Walden F, Tesch PA.
416 Strength, power, fiber types, and mRNA expression in trained men and women with different
417 ACTN3 R577X genotypes. J Appl Physiol (1985). 2009;106(3):959-65.
418 33. Bagley JR, Galpin AJ. Three-dimensional printing of human skeletal muscle cells: An
419 interdisciplinary approach for studying biological systems. Biochem Mol Biol Educ.
420 2015;43(6):403-7.
421 34. Fry AC, Allemeier CA, Staron RS. Correlation between percentage fiber type area and
422 myosin heavy chain content in human skeletal muscle. Eur J Appl Physiol Occup Physiol.
423 1994;68(3):246-51.

35. Terzis G, Spengos K, Kavouras S, Manta P, Georgiadis G. Muscle fibre type composition and body composition in hammer throwers. J Sports Sci Med. 2010;9(1):104-9.
36. Adams GR, Hather BM, Baldwin KM, Dudley GA. Skeletal muscle myosin heavy chain composition and resistance training. J Appl Physiol (1985). 1993;74(2):911-5.
37. Prince FP, Hikida RS, Hagerman FC. Muscle fiber types in women athletes and non-athletes. Pflugers Arch. 1977;371(1-2):161-5.
38. Miller MS, Callahan DM, Tourville TW, Slauterbeck JR, Kaplan A, Fiske BR, et al. Moderate-intensity resistance exercise alters skeletal muscle molecular and cellular structure and function in inactive older adults with knee osteoarthritis. J Appl Physiol (1985). 2017;122(4):775-87.
39. Simoneau JA, Bouchard C. Human variation in skeletal muscle fiber-type proportion and enzyme activities. Am J Physiol. 1989;257(4 Pt 1):E567-72.
40. Welle S, Tawil R, Thornton CA. Sex-related differences in gene expression in human skeletal muscle. PLoS One. 2008;3(1):e1385.
41. Pette D, Staron RS. Mammalian skeletal muscle fiber type transitions. Int Rev Cytol. 1997;170:143-223.
42. Pette D. The adaptive potential of skeletal muscle fibers. Can J Appl Physiol. 2002;27(4):423-48.
43. Bagley JR, Murach KA, Trappe S. Microgravity-Induced Fiber Type Shift in Human Skeletal Muscle Gravitational and Space Biology. 2012;26(1):34-40.
44. Liu Y, Schlumberger A, Wirth K, Schmidtbleicher D, Steinacker JM. Different effects on human skeletal myosin heavy chain isoform expression: strength vs. combination training. J Appl Physiol (1985). 2003;94(6):2282-8.
45. Kryger AI, Andersen JL. Resistance training in the oldest old: consequences for muscle strength, fiber types, fiber size, and MHC isoforms. Scand J Med Sci Sports. 2007;17(4):422-30.
46. Grgic J, Schoenfeld BJ. Are the Hypertrophic Adaptations to High and Low-Load Resistance Training Muscle Fiber Type Specific? Front Physiol. 2018;9:402.
47. Staron RS. Correlation between myofibrillar ATPase activity and myosin heavy chain composition in single human muscle fibers. Histochemistry. 1991;96(1):21-4.
48. Aagaard P, Magnusson PS, Larsson B, Kjaer M, Krustrup P. Mechanical muscle function, morphology, and fiber type in lifelong trained elderly. Med Sci Sports Exerc. 2007;39(11):1989-96.
49. Staron RS, Hagerman FC, Hikida RS, Murray TF, Hostler DP, Crill MT, et al. Fiber type composition of the vastus lateralis muscle of young men and women. J Histochem Cytochem. 2000;48(5):623-9.
50. Jones N, Kiely J, Suraci B, Collins DJ, de Lorenzo D, Pickering C, et al. A genetic-based algorithm for personalized resistance training. Biol Sport. 2016;33(2):117-26.

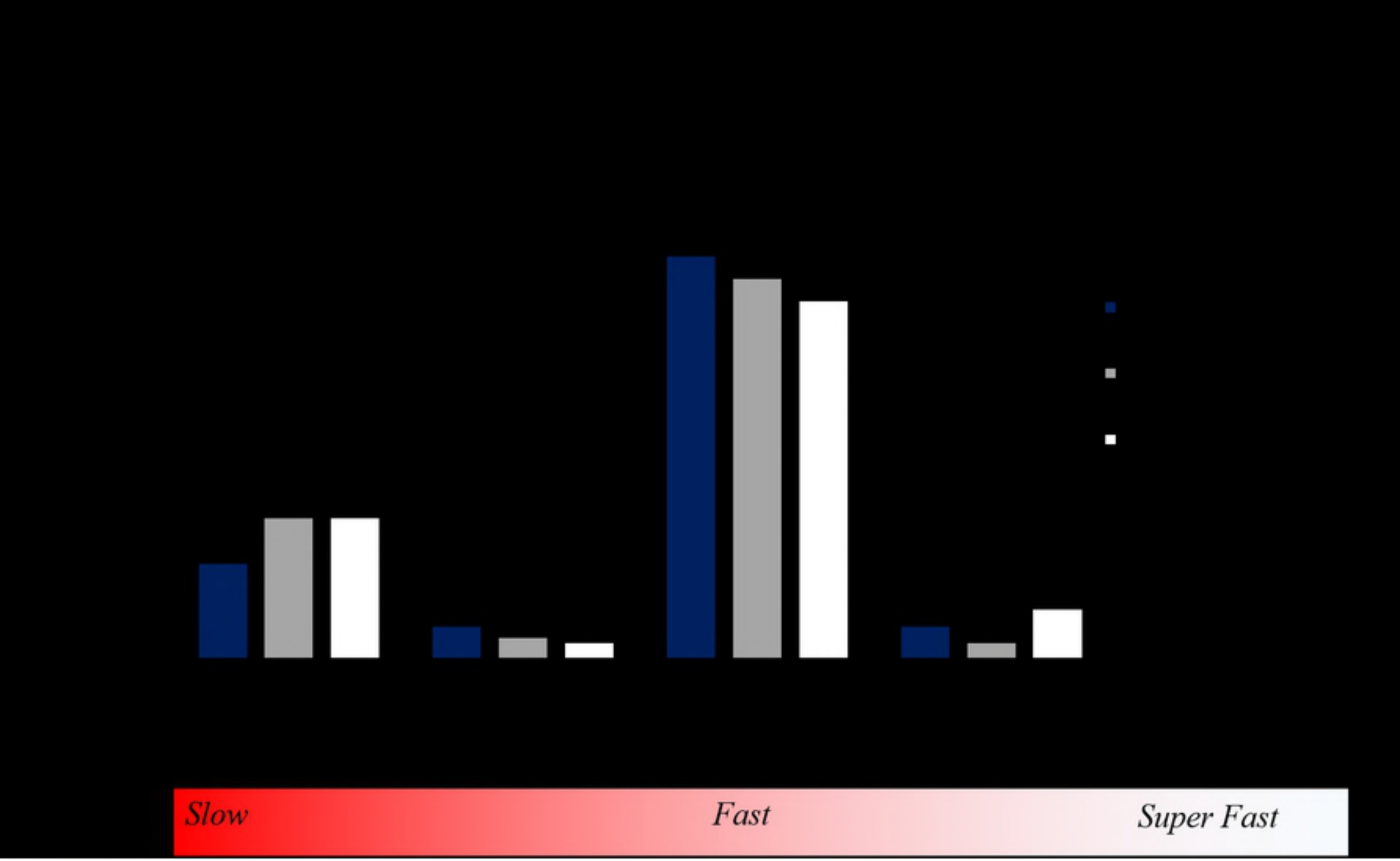

Figure 1