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SCHOLARLY REVIEW

Why Are Masters Sprinters Slower Than Their Younger Counterparts? Physiological, Biomechanical, and Motor Control Related Implications for Training Program Design

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Elite sprint performances typically peak during an athlete's 20s and decline thereafter with age. The mechanisms underpinning this sprint performance decline are often reported to be strength-based in nature with reductions in strength capacities driving increases in ground contact time and decreases in stride lengths and frequency. However, an as-of-yet underexplored aspect of Masters sprint performance is that of age-related degradation in neuromuscular infrastructure, which manifests as a decline in both strength and movement coordination. Here, the authors explore reductions in sprint performance in Masters athletes in a holistic fashion, blending discussion of strength and power changes with neuromuscular alterations along with mechanical and technical age-related alterations. In doing so, the authors provide recommendations to Masters sprinters—and the aging population, in general—as to how best to support sprint ability and general function with age, identifying nutritional interventions that support performance and function and suggesting useful programming strategies and injury-reduction techniques.

Keywords: neuromuscular, resistance training, strength, type-II

Lifelong physical activity is an important determinant of health and well-being (Kannus, 1999), and this becomes increasingly important as we age with increased levels of muscle mass—and the maintenance of that mass—associated with better preservation of function and lower rates of all-cause mortality in older adults (Cooper, Kuh, & Hardy, 2010; Rantanen, 2003). As a result of increased awareness of the relationship between activity and health as we age, more older adults are turning to organized sports as a way to enhance their motivation to maintain their fitness, and one increasingly popular area is that of Masters athletics (Dionigi, Baker, & Horton 2011). Like its mainstream counterparts, the World Athletics Championships and Olympic Games, Masters athletics has a competitive arm, which includes World and European Championships, and competing in these championships drives the motivation of many Masters athletes to improve and progress (Young, 2013).

It is well-established that elite sprint performance—as quantified by race time—decreases with age (Aguiar et al., 2020; Arampatzis, Degens, Baltzopoulos, & Rittweger, 2011; Korhonen, Haverinen, & Degens, 2014) and that this decrease accelerates after approximately 70 years of age (Ganse, Ganse, Dahl, & Degens, 2018), detailed in Figure 1 as follows. A glance at the histories of the fastest male 100-m runners of all time (data not shown) further demonstrates the modifying effect of age; of the 24 athletes to have run 9.86 s or faster, the median age of personal best is 24.5 years with the oldest on that list, Justin Gatlin, the M35 100-m world record (WR) holder, having achieved his personal best time of 9.74 s at the age of 33 years. Finally, there is evidence of a "constituent age effect" in Masters sprinters with the younger athletes within each 5-year age group appearing to be both overrepresented at competitions (Medic, Lares, & Young, 2018; Medic, Starkes, Weir, Young, & Grove, 2009; Medic, Starkes, & Young, 2007) and more likely to be age group WR holders (detailed in Figure 2). However, there are exceptions to this trend; well-trained young adult sprint athletes are more likely to experience a greater decline with age, whereas Masters sprinters who did not take part in organized training until a relatively older age may experience either less of a decline with age or, in some cases, improvements (Korhonen et al., 2014). Kim Collins, the 2003 100-m World Champion (aged 27 years) ran his 100-m personal best (9.93) at the age of 40 years, and Merlene Ottey ran her personal best (10.74) and won an Olympic 100-m Silver medal in her 36th year. Nevertheless, the overwhelming trend is for a decrease in absolute sprint performance after approximately 20–30 years of age (Korhonen et al., 2014).

That sprint performance decreases with age, therefore, is clear and somewhat unsurprising; however, it is crucial to understand why this age-related degradation in elite performance occurs. An understanding of the underpinning reasons—be they physiological, biomechanical, or more holistic in origin—would better assist elite Masters athletes and their coaches in the design and development of training programs aimed at reducing this age-related decline. Furthermore, evidence of efficacy in elite older athletes may increase our understanding of maintaining function in all older adults, opening the door for improved population health initiatives aimed at improving health span as opposed to lifespan (Christensen, Doblhammer, Rau, & Vaupel, 2009). Accordingly, the aim of this article is to answer two related questions:

(a) Why are elite Masters sprinters slower than their younger counterparts? and

(b) How can we use this knowledge to enhance performance?

Although this review will focus on the physiological, biomechanical, and motor control-related aspects of Masters sprint performance, it is important to acknowledge that there is considerable evidence that both psychological and social factors likely contribute to the slowing of sprint speed—or at least sprint performance—with

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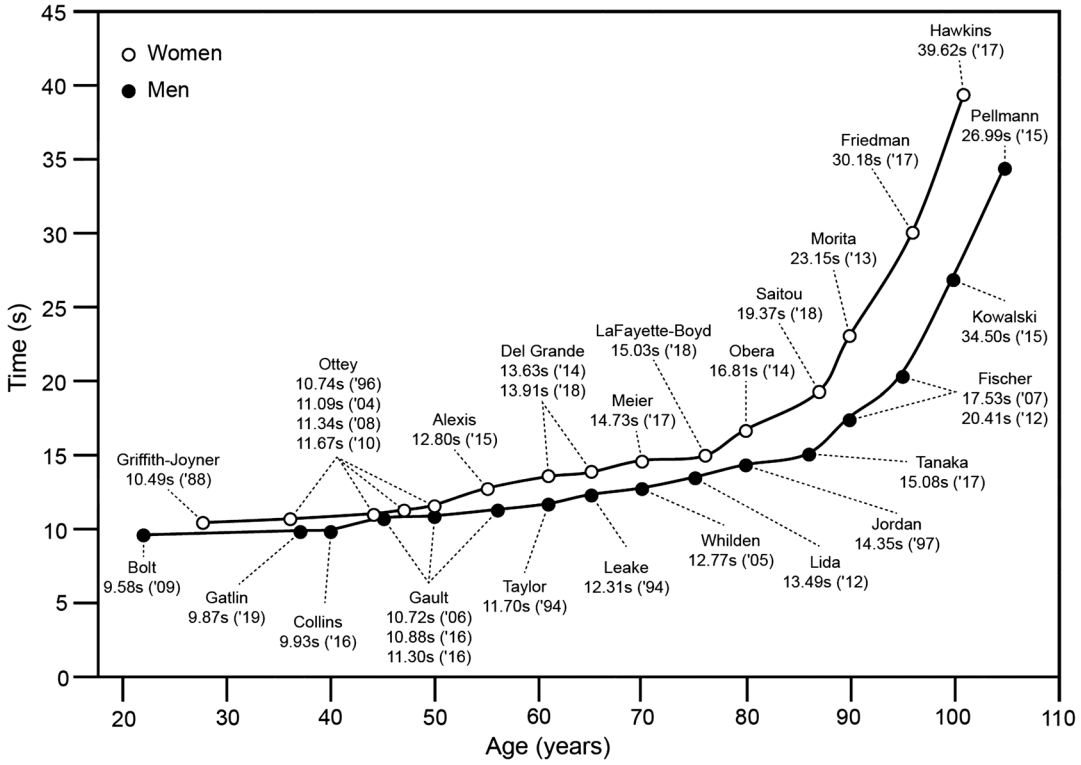

Figure 1 — 100-m World Record performance by Masters age group. (From World Masters Athletics, Records. Available at https://world-masters-athletics.com/records/, accessed 14 May 2020; IAAF Athletics, Senior Indoor Records. Available at http://www.iaaf.org/statistics/records, accessed 14 May 2020).

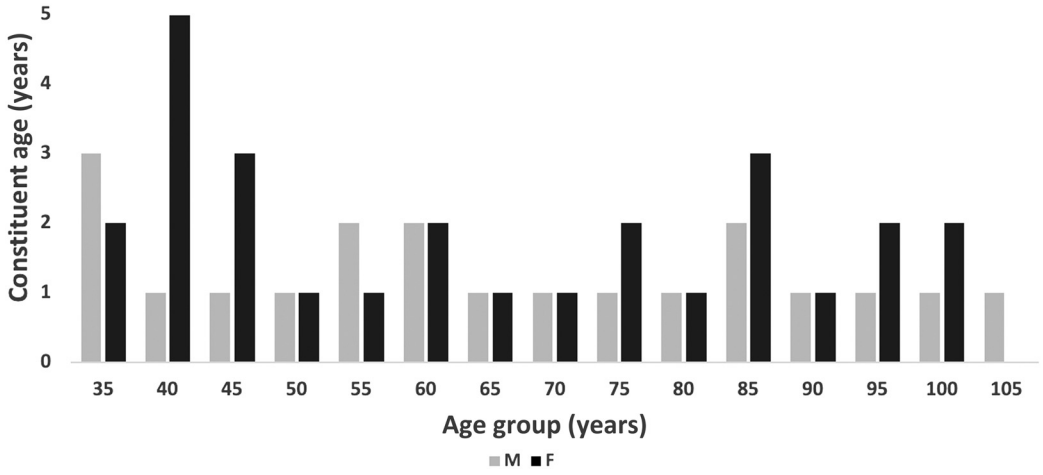

Figure 2 — Evidence for a "constituent age effect" in Masters sprinters with World Record times more likely to be achieved by relatively "younger" athletes in each age group (From World Masters Athletics, Records. Available at https://world-masters-athletics.com/records/, accessed 14 May 2020). Athletes in the first year of the age group are recorded as being in Year 1 on this graph, athletes in the second year in Year 2, and so on.

age. As mentioned previously, there is a constituent age effect in Masters athletics with first- and second-year athletes within each 5-year age group more likely to participate at National and International championships than relatively older athletes within each 5-year age group (Medic et al., 2007, 2009; Medic, Young, & Grove, 2013). Medic et al. (2013) reported that awareness of this constituent age effect affected athlete motivation with relatively older Masters swimmers within each age group, reporting lower overall training frequencies and durations along with avoidance of competitions wherein they perceived a disadvantage due to their age. Being relatively younger within each age group has also been associated with higher levels of intrinsic motivation (Hodge, Allen, & Smellie, 2008) along with an increase in perceived competence and expectation of success (Medic et al., 2013; Wilson & Stephens 2005), both of which may also influence competition performance. Finally, changes in physiological function in older athletes may not be due to aging

per se but, instead, reduced activity levels into older age (Borges, Reaburn, Driller, & Argus, 2016) or lower overall rates of sports participation (Tanaka & Seals, 2008) alongside the potentially lower levels of motivation as discussed previously. Accordingly, some of the age-associated changes in function and capacity may be—at least partially—driven by inactivity as opposed to aging directly.

Why Are Masters Sprinters Slower?

The kinematic properties of elite sprint performance have been well studied (Haugen, McGhie, & Ettema, 2019; Hay, 1994; Hunter, Marshall, & McNair, 2004; Mann, 2013; Mero, Komi, & Gregor, 1992). Sprint performance occurs through an interaction of step length and step frequency, each of which are comprised of varying underlying factors (Hay, 1994; Hunter et al., 2004). In general, elite sprinters achieve greater step lengths than nonelite sprinters but similar step frequencies (Gajer, Thepaut-Mathieu, & Lehenaff, 1999; Haugen et al., 2019; Mann, 2013; Salo, Bezodis, Batterham, & Kerwin, 2011), although there is considerable variation within each given performance level as to whether an athlete is concerned about step frequency or length reliant (Salo et al., 2011). Other kinematic variables of interest include ground contact time (GCT), with elite male sprinters tending to spend <0.09 s on the ground during maximum velocity sprinting (Mann, 2013), and hip flexion angle, with elite sprinters achieving greater hip flexion values than their nonelite counterparts (Kunz & Kaufmann, 1981; Mann, 2013).

The biomechanics of sprint performance in elite Masters runners has also been reasonably well studied (Hamilton, 1993; Korhonen, Mero, & Suominen, 2003; Korhonen et al., 2009; Roberts, Cheung, Hafez, & Hong, 1997). After collecting data from national championships and the World Veterans Championships, Hamilton (1993) reported that stride length, flight time, swing time, and hip range of motion were all significantly lower in Masters compared with elite young adult sprinters, whereas GCT was significantly higher. Korhonen et al. (2003) examined the performances of 70 elite Masters athletes competing in the European Veterans Athletics Championships, concluding that step length demonstrated a significant reduction with increasing age, whereas step rate remained largely unchanged until very old age (>80 years). The GCT was also significantly increased with age with a concurrent decrease in flight time. This age-associated increase in GCT and reduction in step length (SL) have also been reported elsewhere (Korhonen et al., 2009; Roberts et al., 1997).

As a result of the aforementioned research, it appears that:

(a) Masters athletes demonstrate a reduction in maximal sprint performance with age (Korhonen et al., 2003, 2009).

(b) This reduction in sprint speed appears to be due to a decrease in SL (Hamilton 1993; Korhonen et al., 2003) and flight time (Korhonen et al., 2003) along with a concurrent increase in GCT (Korhonen et al., 2009; Roberts et al., 1997).

Elite sprinters achieve a relatively short GCT as they are able to minimize the breaking forces they experience during foot contact and produce the required amount of force to achieve the required horizontal and vertical velocities to deliver an optimized SL in a short period of time. As elite Masters sprinters demonstrate a reduction in GCT and SL, it is important to consider which of these variables drives the other. Korhonen et al. (2009) compared the sprint kinetics and kinematics of young adult (17–33 years) and elite Masters (40–82 years) male sprinters, reporting that GCT was significantly correlated with sprint velocity when adjusted for age and that ground reaction forces (GRF) were substantially lower in Masters when compared with young adult sprinters. Alongside this reduction in GRF and increases in GCT, Masters sprinters had significantly lower knee extensor and plantar flexor muscle thickness along with significantly lower half squat 1RM and countermovement jump (CMJ) test scores than younger sprinters. In addition, Masters sprinters had significantly lower Type II, but not Type I, muscle fiber area and a significantly lower Type II/Type I fiber area ratio (Korhonen et al., 2009). These results suggest that the age-related decline in maximal velocity running speed is primarily driven by decreases in stride length, which occur alongside an increase in GCT. These changes are potentially due to age-associated losses in muscle strength, which appears to be related to decreases in total muscle mass and, more specifically, a relative decrease in Type II muscle fiber mass (Korhonen et al., 2009). However, this relationship is somewhat complex with other aspects of muscle function, and not just a reduction in muscle strength, potentially driving this. For example, Goodpaster et al. (2006) reported that, although lean mass loss in a cohort of nonathlete older subjects was associated with a reduction in strength, this loss of strength occurred more rapidly than the loss of lean muscle mass. As a result, the authors suggested that muscle quality, defined as strength generated per unit of mass, may be more important than strength per se, with muscle quality being influenced by aspects such as total contractile protein content of the muscle along with less fat infiltration (Shaffer et al., 2017). In addition, changes in neuromuscular function, including slower rates of muscle activation, may, in turn, negatively affect muscle power and contraction speed (Reid & Fielding, 2012), harming sprint performance. Nevertheless, muscle strength, among additional factors such as muscle quality and power, appear important in driving sprint performance. Further evidence of the importance of strength-related measures on sprint performance comes from research in younger adult sprinters; for example, Young, Mc Lean, & Ardagna (1995) demonstrated how both maximum strength and measures of stretch-shortening cycle—important in reducing GCT—were strongly related to sprint performance. Similarly, Cronin and Hansen (2005) reported significant correlations between jump squat and performance in terms of CMJ height, along with 5-, 10-, and 30-m sprint times in rugby league players. Given this relationship between strength, power, and speed performance and given that Masters sprinters typically demonstrate reductions in muscle mass and strength, a primary strategy for enhancing Masters sprint performance would appear to be the amelioration of age-associated reductions in Type II muscle fiber and the concurrent decrease in muscle mass and strength.

Further Underpinning Drivers of Declining Sprinting Speed

As outlined earlier, diminishing muscular strength, commonly attributed to decreasing muscle mass and/or unfavorable fiber-type transitions, is the most frequently cited cause of declining sprinting speed (Korhonen et al., 2009). Substantial evidence, however, demonstrates that both strength and movement coordination begin to diminish before changes in muscle mass are evident and, furthermore, after muscle loss reductions in strength are greater than can be explained by muscle atrophy alone (Goodpaster et al., 2006; Hepple & Rice, 2016; Ward et al, 2015). Current perspectives, emanating from neural, molecular, and biological domains, instead suggest that early functional impairments are driven by the gradual deterioration of multiple facets of neuromuscular infrastructure.

These deteriorations occur in both central and peripheral arms of the neuromuscular system and include motor neuron loss, declining nerve function (Hepple & Rice, 2016; Ward et al., 2015), axonal degeneration, increasing neuromuscular junction instability, attenuated tissue regeneration, and increasing fibrosis driven by immunologically mediated reductions in stem cell populations. Collectively, these inseparably entwined, mutually accelerating chains of interrelated deficits are suggested to detract from dynamic movement control (Kiely, Pickering, & Collins, 2019). As an example, the multiple components of sensorimotor control, including sensory acuity, ability to accurately activate the tissue, and the loading capacity of tissue, degrade over time; combined with the accumulation of muscular damage that occurs over time, the library of available coordinative responses to running-imposed mechanical challenges declines (Kiely, Pickering, & Collins, 2019). Nevertheless, it is important to note that the interactions between these progressively accumulating structural changes remain poorly understood.

In relation to declining sprint performance, an example of a structural change potentially contributing to diminishing motor performance occurs at the level of the motor unit (MU). Across the adult lifespan, MUs are subject to regular denervation and reinnervation events (Anagnostou & Hepple, 2020; Hepple, 2018). These denervation–reinnervation cycles involve a transient disconnect, and subsequent reconnect, of individual muscle fibers from, and to, the motor neuron. Inevitably, as the number of denervation–reinnervation events accrue over time, regeneration errors accumulate in tandem. Progressively, after each denervation–reinnervation event, a fraction of motor endplates lose an innervating motoneuron, resulting in an accumulation of denervated fibers that cannot contribute to duty cycles. These fibers subsequently atrophy (Hepple & Rice, 2016).

In young adults, muscle fibers belonging to distinct MUs are typically intermingled in complex mosaic-like distributions. As we age, however, repeating cycles of denervation–reinnervation result in fiber type grouping whereby fibers of the same type "clump" together. Through these mechanisms, the number of viable MUs declines. Surviving, still innervated MUs compensate by "sprouting" to recruit more muscle fibers (Anagnostou & Hepple, 2020). And so, although the number of MUs declines, the number of individual fibers activated by surviving MUs increases. A subsequent implication is that motor control becomes more coarse-grained, movement smoothness diminishes, motor precision and efficiency decline, and energy costs and injury risks increase (Kiely, Pickering, & Collins, 2019).

In summary, deteriorating neural infrastructures throughout the central nervous system (CNS) instigate a slow, progressive cascade of motor control deficits, which subsequently change how efficiently, and effectively, peripheral tissues can be deployed to accommodate the physical stress of dynamic movement. As a result, the timings and relative positionings of tissue structures regulating the subsequent allocation of mechanical loads between muscle and tendon, joints, MU collectives, and hard tissues all become slightly less precise, more clumsy, and less adaptive. This movement control deterioration inevitably exacerbates mechanical shock loadings, thereby exposing the athlete to increased microtrauma, increased sensitivity, and increased injury risk. Subsequently, neuromuscular degeneration appears the most likely driver of the muscle strength and power deficits associated with aging. In response to the major whole muscle decrements, multiple compensatory mechanisms are implemented within the contractile properties of surviving muscle fibers in the attempt to stall age-driven muscle power and function decline (Reid et al., 2014).

Implications for Training

Resistance Training

Given the importance of maintaining skeletal muscle size and strength in supporting the performance of Masters sprinters, training strategies should be developed to support these two aims along with the selective targeting of Type II muscle fibers, which appear to be at increased risk of age-associated atrophy (Aagaard, Suetta, Caserotti, Magnusson, & Kjær, 2010). Although there is substantial research demonstrating the effectiveness of resistance training in enhancing strength, power, and hypertrophy in younger and older subjects (Jozsi, Campbell, Joseph, Davey, & Evans, 1999; Newton et al., 2002) and the effectiveness of this training type in improving sprint performance in younger athletes (Delecluse 1997; Moir, Sanders, Button, Glaister, 2007), there is surprisingly little research exploring the effectiveness of resistance training in enhancing sprint performance in Masters athletes (Delvecchio & Reaburn, 2013).

Cristea et al. (2008) randomly assigned 11 male Masters sprinters (aged 52–78 years) to a twice-weekly resistance training intervention or control group for a 20-week period. The resistance training program was delivered in a periodized manner, moving from a 4-week hypertrophy phase (three to four sets of eight to 12 repetitions at 50–70% 1RM) to mixed training weeks comprised of maximal strength training (two to three sets of four to six repetitions at 70–85% 1RM), explosive weight training (two to three sets of four to six repetitions at 35–60% 1RM), and plyometric exercises (two to three sets of three to 10 repetitions) carried out in an alternating fashion to support recovery. Strength training was primarily focused on the leg extensors and hamstrings (e.g., leg press, half squat, Romanian Deadlift) along with supplementary exercises (e.g., bench press, push press). The plyometric exercise component utilized both vertical and horizontal jumping exercises. Athletes from the resistance training arm demonstrated a 21% increase in knee extension strength, a 40% increase in knee flexion strength, and a 27% increase in back squat 1RM along with improvements in power (squat jump, 10% improvement; reactive jump test, 29% improvement). There were no significant improvements in the control group. In terms of muscle fiber type, there were no significant changes in overall percentage, but the cross-sectional area of Type II (all) and Type IIA fibers was significantly greater than at baseline. These improvements in strength and increases in Type II and Type IIA cross-sectional area also carried over to sprint performance; those in the intervention group demonstrated a 2% (approximately 0.17 s; $p < .01$) improvement in 60-m sprint performance and a 3% (approximately 0.06 m; $p < .05$) improvement in step length. Importantly, these subjects had previous strength training experience of approximately 1 hr per week, suggesting that performance improvements can still occur in trained Masters sprinters. Similar to Cristea et al. (2008), Reaburn and Mackinnon (1996) placed eight sprint-trained male Masters athletes through an 8-week resistance training program focused on hypertrophy comprising three sessions per week of 12, 10, and 8 repetitions at 80% 1RM across multiple exercises. Significant improvements in strength and 100-m performance were reported upon completion of the intervention.

Aside from the studies by Reaburn and Mackinnon (1996) and Cristea et al. (2008), there is a paucity of studies examining the effects of a resistance training program on the performance of Masters sprinters (Delvecchio & Reaburn, 2013; Korhonen et al., 2014). However, the early promising results in previously strength-trained (Cristea et al., 2008) and untrained (Reaburn & Mackinnon,

1996) Masters athletes along with the strong evidence of performance improvements in young and older participants following strength training (Newton et al., 2002) suggest that both resistance and plyometric training, along with sprint training, should, perhaps, form the cornerstone of the Masters sprinter's training program.

Neuromuscular Control and Coordination

Physical activity is perhaps the preeminent intervention demonstrated to slow age-related detriments at both neurological and muscle tissue levels (Cartee, Hepple, Bamman, & Zierath, 2016; Reid et al., 2014). Indeed, for a portion of the lifespan, exercise training may also be able to reverse some of these changes (Cartee et al., 2016; Hepple and Rice, 2016; Moro, Brightwell, Volpi, Rasmussen, & Fry, 2020; Piasecki et al., 2019). However, long-term athletic training does not prevent age-related decline of muscle size, and current findings illustrate that MU numbers decrease even in the athletic older adult (Piasecki et al., 2019). This ultimately inevitable decline is, however, substantially suppressed in the musculature of active adults compared with sedentary age-matched counterparts (Power et al., 2010).

As outlined earlier, the majority of research has focused on the positive effects of resistance training on age-related muscular and functional decline. Less effort, however, has focused on the potential benefits of exercise modalities that may more specifically target skill-dependent dimensions of athletic performance. For example, training modalities that target facets of coordination have previously proven to be particularly effective with prior investigations demonstrating the benefits of training interventions targeting balance (Wiesmeier et al., 2017), stability (Hamed, Bohm, Mersmann, & Arampatzis, 2018), cutaneous reflexes and enhanced proprioceptive awareness (Craig, Goble, & Doumas, 2016; Peters, McKeown, Carpenter, & Inglis, 2016), and postural stability training (Martínez-Amat et al, 2013).

Further benefit may be achieved via the maintenance or recovery of muscle tissue characteristics. Importantly, conventional strength training does not optimally stimulate tissue remodeling. Conventional isotonic strength training is highly energy consuming and typically only generates high muscle tensions over a short time interval. Isometric and/or eccentric exercises demand less energy investment and impose higher tensions across the working tissues, providing a greater remodeling stimulus. As an illustrative example, hamstring stiffness—a key contributor to running speed—was previously demonstrated to increase significantly with isometric, but not isotonic, training (Blackburn & Norcross, 2014).

Furthermore, increasingly precise and economical exercise prescription may enhance training efficacy in older sprinters. For example, implementing "repetitions to failure" did not enhance training benefits during concurrent training in healthy older men (da Silva et al., 2018); muscle power training using one or three sets resulted in equivalent power gains in older females (Radaelli et al., 2019), and higher volumes of power training did not increase training-derived power increases in older women (Radaelli et al., 2018). Similarly, the use of nonconventional loading schemes, such as "cluster sets" whereby sets of repetitions are interspersed with short rest intervals, has proven more effective in terms of strength and power improvements in healthy older men (Iacono, Martone, & Hayes, 2020). This logic suggests that training modalities that might not necessarily benefit adolescent or mature sprinters—such as, for example, single-leg balance activities—may benefit Masters sprinters or, at least, may (accepting age-related declines in these capacities) benefit Masters sprinters to a greater extent, with a further argument that Masters strength training programs could benefit from the strategic use of nontraditional training designs that may prove less effortful, yet more productive, for the older athlete (Aas et al., 2020; Chen et al., 2017; Hoppeler, 2016).

Technical Abilities

Applying force in a more horizontal direction during sprint acceleration is a major factor to performance, yet this orientation of force appears to be limited in Masters-aged athletes. Compared with their younger sprint athlete counterparts, Masters sprinters have been shown to demonstrate lower mechanical effectiveness of force application during sprint acceleration, which describes the technical ability to orient ground reaction force in a more horizontal direction as running velocity increases (Pantoja, De Villarreal, Brisswalter, Peyré-Tartaruga, & Morin, 2016). Mechanical effectiveness characterizes the ratio of forces applied to the ground, where the horizontal (anteroposterior) component of the GRF (F_H) vector is represented as a percentage of the total GRF (F_{TOT}) vector for one contact period (Morin, Edouard, & Samozino, 2011; Rabita et al., 2015; Samozino et al., 2016). The higher the ratio of force, the more horizontal orientation of the GRF has been achieved (Hicks, Schuster, Samozino, & Morin, 2020). Morin et al. (2011) previously demonstrated this to be a key determining factor to acceleration performance. Pantoja et al. (2016) also highlighted that the application of force at the start of the sprint effort, when the ratio of forces are maximized, was substantially hindered by athlete age with older athletes demonstrating less than half the mechanical effectiveness (33.4% on average) of elite sprint athletes (71.6%). From a technical perspective, lower mechanical effectiveness during acceleration will push the Masters athlete into an upright posture earlier in the race, changing or highlighting the importance of the acceleration phase in Masters athletes. Limb strength, balance, and limited hip extension have been provided as possible hindrances to horizontal force application during acceleration. Resistance training exercises that focus on horizontal force production have previously been suggested for sprint athletes attempting to improve mechanical effectiveness during sprint acceleration (Hicks et al., 2020).

Mechanical Qualities

Maximal horizontal power displayed during sprint performance has been identified as a declining mechanical quality in Masters-aged sprint athletes (Hamilton, 1993; Korhonen et al., 2003). Maximal power in the horizontal direction is the product of its contributing components from the linear force–velocity relationship, F_0 (theoretical horizontal force at null velocity), and V_0 (theoretical horizontal velocity at null force). Mechanical power has previously been positively correlated with mean 100 m speed (Morin et al., 2012). These mechanical qualities that characterize the neuromuscular system however also act as constraints on acceleration performance.

Pantoja et al. (2016) showed from a study of 27 male sprinters (aged 39–96 years) that the mechanical determinants of sprint acceleration, force, velocity, and power were found to decrease linearly with age at a rate of 1.60%, 1.10%, and 0.94% per year, respectively. Interestingly, when comparing the youngest with the oldest Masters-aged athlete in the study, the authors found drastically lower maximal power values with the oldest athlete, 22.1 W/kg to 3.57 W/kg, respectively, suggesting an inevitable decline in this

quality through aging. Arampatzis et al. (2011) have also put forward that a reduction in overall training load may be the cost of age-related changes along with changes to body composition, both of which will impact physiological, neuromuscular, and mechanical qualities. Furthermore, if maximal power during acceleration decreases with age, it is a direct result of a reduction in the ability to produce force (and transmit force) and/or a reduction in contraction velocity and, therefore, rate of force development plus tendon compliance, all of which are neuromuscular or musculoskeletal components influenced by specific types of training. Importantly, resistance training exercises focused on hypertrophy, force production, movement velocity, and optimizing power have been shown to positively affect the plasticity of the neuromuscular system (Suchomel, Nimphius, Bellon, & Stone, 2018), yet with Masters aged sprint athletes, it may be a process of attenuating the inevitable decline in physiological and neuromuscular characteristics associated with aging (Arampatzis et al., 2011; Korhonen et al., 2006).

These technical and mechanical limitations to performance for Masters sprinters may provide insight into the state of the neuromuscular system with aging but, perhaps, also provide some areas of focus and training recommendations for Masters athletes.

Athlete Recovery

Although there is a general perception that older adults take longer to recover from exercise, there is a surprising paucity of data empirically exploring whether this is actually the case (Borges et al., 2016). This is especially true when it comes to speed–power exercise, such as sprint and resistance training; the authors of a 2016 review (Borges et al., 2016) identified only six studies comparing the postexercise recovery kinetics between young and older adults (Bieuzen, Hausswirth, Louis, & Brisswalter, 2010; Darr, Bassett, Morgan, & Thomas, 1988; Easthope et al., 2010; Fell, Haseler, Gaffney, Reaburn, & Harrison, 2006; Fell, Reaburn, & Harrison, 2008; Sultana et al., 2012) with only one of these (Bieuzen et al., 2010) doing so in the context of resistance training. Since that review, additional research (Borges, Reaburn, Doering, Argus, & Driller, 2018) suggests that, in general, there is no difference in the magnitude of biological markers of postexercise recovery between young and old adults and that older adults likely do not require increased periods of recovery postexercise (Borges et al., 2016, 2018). However, it does appear that Masters athletes *perceive* greater levels of fatigue 48 hr postexercise when compared with younger adults, report significantly greater feelings of muscle soreness, and have significantly lower levels of motivation (Borges et al., 2018). As a result, although there might be no physiological need for increased recovery durations, the blunted perceptual and psychological markers of recovery postexercise may necessitate longer recovery periods in Masters athletes. The use of subjective markers of perceived recovery, previously shown to be valid and reliable (Saw, Main, & Gastin, 2016), may, therefore, be useful in guiding athletes and coaches in their decisions.

Injury Prevention

Injuries are a reasonably common occurrence in high-level athletes both in and out of competition (Drew, Raysmith, & Charlton, 2017; Feddermann-Demont, Junge, Edouard, Branco, & Alonso, 2014; Raysmith & Drew, 2016). Masters runners appear to be at an increased risk of injury compared with their younger counterparts; for example, McKean, Manson, and Stanish (2006) reported that Masters runners were significantly more likely to get injured, and suffer more injuries, than younger adults. Although the studied population was primarily recreational endurance runners as opposed to sprinters, Masters athletes in that cohort were also significantly more likely to suffer soft tissue injuries than younger athletes with the calf, Achilles, and hamstrings all at increased risk (McKean et al., 2006). As the hamstring muscles are more susceptible to injury during sprinting (Chumanov, Schache, Heiderscheit, & Thelen, 2012) and age is a risk factor for hamstring strain injury (Gabbe, Bennell, & Finch, 2006), it is logical to suggest that Masters sprinters are at an increased risk of hamstring injury, although, to our knowledge, this has yet to be empirically quantified. Given the relationship between injury and decreased likelihood of achieving a performance goal (Drew et al., 2017; Raysmith & Drew, 2016) and that Masters athletes are more likely to experience such injuries (Gabbe et al., 2006; McKean et al., 2006), it appears logical to suggest that Masters athletes undertake preventative measures to mitigate their risk of injury. This should include eccentric loading activities for the hamstring (Bourne et al., 2018), regular exposure to high-speed running (Edouard et al., 2019), and calf strengthening exercises (Alfredson, Pietilä, Jonsson, & Lorentzon, 1998). Inclusion of these exercise types within the Masters sprinters program should assist in enhancing training and competition availability and, as a result, performance (Raysmith & Drew, 2016).

Nutritional Support

So far, we have demonstrated that a key training pillar in supporting the performance of elite Masters sprinters should be the maintenance, or amelioration of the loss, of muscle mass and strength, particularly focused on Type II muscle fibers. To that end, there are a number of different nutritional strategies that could be considered to support Masters sprinters' performance.

Dietary Protein

An important part of the recovery and adaptive process to a period of loading is an increase in muscle protein synthesis (MPS), which is a crucial driver of skeletal muscle hypertrophy (Phillips, 2014). When MPS rates exceed that of muscle protein breakdown, positive muscle protein balance is achieved and hypertrophy can take place (Phillips, 2014). Training, especially resistance training, stimulates MPS as does ingestion of a protein-rich meal. However, there is evidence that, in older adults, this posttraining and postprandial MPS is somewhat blunted when compared with younger adults with this blunted MPS termed *anabolic resistance* (Burd, Gorissen, & Van Loon, 2013; Doering et al., 2016). This anabolic resistance can be somewhat overcome by consuming sufficient overall total protein with the recommended amount being around 1.2–1.8 g/kg of body mass per day (Desbrow, Burd, Tarnopolsky, Moore, & Elliott-Sale, 2019; Morton, Traylor, Weijs, & Phillips, 2018). This practice can be further optimized by consuming approximately 0.4 g protein per kilogram of bodyweight posttraining and regularly spaced throughout the day (Desbrow et al., 2019; Morton et al., 2018).

The β-Hydroxy-β-methylbutyrate (HMB) is a leucine metabolite that has, in some studies at least, been shown to support increases in muscle mass following training (Nissen & Abumrad, 1997). A recent meta-analysis (Holland, Roberts, Krieger, & Schoenfeld, 2019) suggested that the positive effects of HMB on muscle mass were small, but positive, and most pronounced in athletes with suboptimal intakes of protein. In older nonathletes, HMB was found to have no added effect on improvements in

muscle strength above exercise alone (Courel-Ibáñez, Vetrovsky, Dadova, Pallarés, & Steffl, 2019). However, there is tentative evidence to suggest that HMB may positively influence postexercise recovery (Silva et al., 2017; Wilson et al., 2013b), which, as discussed previously, may support increased training volumes and intensities in Masters athletes. If Masters athletes do wish to supplement with HMB, then regular consumption of approximately 3 g/day appears appropriate (Wilson et al., 2013a).

Creatine

Creatine is a naturally occurring nonprotein amino acid that is primarily found in red meat and seafood (Kreider et al., 2017). Creatine is predominately found in skeletal muscle (Kreider & Jung, 2011), playing an important role in energy production via the adenosine triphosphate-phosphocreatine (ATP-PC) system. During daily life, small amounts of intramuscular creatine are degraded and excreted; as a result, adults need to consume 1–3 g/day to maintain normal stores (Kreider et al., 2017). Creatine has been shown to be efficacious in enhancing lean mass improvements following training (Chilibeck, Magnus, & Anderson, 2007; Kreider et al., 1998) and increasing high-intensity exercise capacity (Juhász, Györe, Csende, Racz, & Tihanyi, 2009; Kreider et al., 2017) and is reasonably widely utilized by athletes (LaBotz & Smith, 1999). The efficacy of creatine supplementation on improvements in muscle mass and strength in the older adult has also been demonstrated (Chilibeck, Kaviani, Candow, & Zello, 2017; Devries & Phillips, 2014). Regular creatine use at doses of approximately 5 g/day is generally considered safe (Kreider et al., 2017) and so, for Masters athletes looking to increase or maintain muscle mass, its use is recommended.

Other Nutrients

Fish oil-derived omega-3 fatty acids have shown some initial promise as a treatment agent against age-related sarcopenia (Breen & Phillips, 2011; Di Girolamo et al., 2014) and so may assist Masters athletes in the development and maintenance of muscle mass and strength. Despite concerns that their use may dampen postexercise adaptations in younger adults (Cornu et al., 2020), nonsteroidal anti-inflammatory drugs do not impair MPS in older adults (Dideriksen et al., 2016) and when utilized along with resistance training, may promote both skeletal muscle hypertrophy and improvements in strength in this population (Trappe et al., 2011, 2016), although these findings are equivocal (Clifford, 2019).

From an acute workload perspective, caffeine is a well-established ergogenic aid that can enhance training performance (Grgic et al., 2020). Caffeine enhances performance in older adults (Duncan, Clarke, Tallis, Guimaraes-Ferreira, & Wright, 2014; Norager, Jensen, Madsen, & Laurberg, 2005); however, research on isolated mouse muscle suggests that the magnitude of ergogenic effect may be lower in older compared with younger adults (Tallis, James, Cox, & Duncan, 2017). A second ergogenic aid, β-alanine, has also been shown to acutely increase work capacity in younger adults (Trexler et al., 2015) and older adults (Stout et al., 2018; McCormack et al., 2013), alike, and so potentially represents a worthwhile avenue for exploration.

Summary and Practical Recommendations

For Masters sprinters, key and direct drivers of their slowing with increasing age are a loss of total muscle volume, maximal strength, power, and Type II muscle fibers. As a result, based on the research summarized within this article, we recommend that the training program of a Masters sprinter should aim to minimize any age-associated losses in these variables along with appropriate nutritional support. Regular resistance training (approximately 2–3 days/week) incorporating set and repetition schemes associated with skeletal muscle hypertrophy (e.g., three sets of 10 repetitions at 70% 1RM), maximal strength (e.g., two to four sets of four to six repetitions at 85% 1RM), and power (three sets of three to 10 repetitions at 35–60% 1RM) appear to be effective in enhancing the muscle size and strength of Masters sprinters along with their sprint performance. Nutritional support in the form of regular bolus doses of protein potentially coingested with creatine and HMB may also assist in supporting strength adaptations. However, resistance training is not sufficient to effectively reduce age-related losses in sprinting ability; as outlined earlier, the utilization of training aimed at enhancing neuromuscular control and coordination also appears crucial, and its inclusion in the training of Masters sprinters is also recommended.

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