The Role of Exercise as a Therapy for Children With Duchenne Muscular Dystrophy

Stephen P. Sayers

Duchenne muscular dystrophy (DMD) is a disease affecting muscle fiber integrity in boys that leads to progressive weakness in skeletal muscle and premature death. Currently, there is no known cure for the disease. Different interventions have been explored to delay the progression of the disease and improve the quality of life for the DMD patient. Physical activity is one treatment that has generated controversy due to the increased mechanical stress placed on the muscle during contraction. This review explores the literature in animal models and human DMD patients and evaluates the known theoretical risks and benefits of increased physical activity in DMD patients.

Duchenne muscular dystrophy (DMD) is a disease affecting approximately one in 3,000 male births (19). The disease is characterized by wasting of the muscles due to an aberration in muscle fiber structure that leads to severe atrophy and progressive weakness of the muscles as a child matures (6, 14, 36). Because of the degenerative nature of the disease, by age 12 most children are no longer ambulatory and must be confined to a wheelchair. By the late teens or early twenties, death occurs due to the weakened condition of the respiratory and cardiac muscles (19, 28). Diagnoses of DMD are usually not made until a child is 4 or 5 years old, when symptoms of poor coordination begin to appear as clumsiness or the inability to walk. Weakness in the hip girdle/proximal lower extremity and paraspinal muscles tends to make walking awkward and difficult, exemplified by a sagging of the pelvis as the foot is raised and a concomitant tilting of the body to one side, commonly called a waddling gait. An extreme anteroposterior curvature of the lumbar spine (lordosis) is also apparent, and frequent falls tend to occur when running is attempted (19). The clinical signs associated with the disease include pseudohypertrophy of some muscles (especially the calf muscles), quadriceps femoris atrophy, weakness in the anterior tibialis with associated heel cord tightness, and a reduction in cognitive ability (27).

Although the cause of the disease has been discovered, there is currently no cure. The hope for children with DMD is that the symptoms of the disease can be alleviated and progression of the disease delayed. Many different therapies have
been explored to decrease tissue wasting in dystrophic human and animal models. Some of these interventions include drug therapies such as the administration of glucocorticoids (9, 21), gene therapies such as myoblast transplantation (10, 20, 23), and treatment with β-adrenergic agonists (6, 7, 40). Other forms of therapy include increases in muscular activity using either low-frequency electrical stimulation (24, 30, 31, 41), endurance or resistance exercise (1–4, 6, 7, 13–17, 32, 35, 37, 39), or both (24).

Because of the lack of efficacy, short-term benefits only, and/or side effects of drug therapies, the impracticality of myoblast transfer (too many muscles need to be injected), the paucity of current data on gene therapies, and the lack of known efficacy of artificial forms of muscle contraction, many DMD patients are left with no proactive therapy but rather prevention of contractures and maintenance of activities of daily living. Exercise, on the other hand, may be a more easily applied intervention that could be considered as a potential alternative for the DMD patient. There is some controversy surrounding the recommendation of exercise for the DMD patient (35, 39) due to the mechanical stresses imposed on the weakened muscle, joints, tendons, and bones during the course of muscular contraction and weight bearing, as well as the lack of proven benefit. However, a well-designed exercise program that considers factors such as maturation, severity and location of the muscle weakness, rate of progression of the weakness, type of exercise (e.g., resistance or endurance), frequency, intensity, and duration of training (10) may contribute to a beneficial response to increased physical activity.

**Background**

The primary abnormality in DMD is the lack of the 427 kd protein dystrophin, found primarily in the subsarcolemmal region of skeletal, smooth, and cardiac muscle (27). Dystrophin plays an important role in maintaining the integrity of muscle fibers because of a transmembrane glycoprotein complex attached to the intracellular cytoskeleton (cytoplasmic actin) via dystrophin. The attachment of the glycoprotein complex to the extracellular matrix via laminin provides the link from intracellular to extracellular and provides mechanical stability to the membrane. Without dystrophin, the link between the intracellular cytoskeleton and the glycoprotein complex (which attaches to laminin in the extracellular matrix) is disrupted, resulting in the inability of the muscle to withstand mechanical stress (28).

This structural abnormality in the muscle membrane in the DMD child may result in an increased susceptibility of the muscle to sustain damage. Focal areas of discontinuity are evident in the membrane of the DMD muscle cell (26). Because of this there is an elevation of serum creatine kinase in the blood due the loss of membrane permeability. Elevation of serum creatine kinase has also been observed in fetal blood suggesting that membrane permeability is affected long before clinical signs of the disease are manifested. There may also be an increase in calcium content of dystrophic muscles due to increases in extracellular calcium influx through damaged membranes, or decreases in calcium efflux at the sarcoplasmic reticulum. Both scenarios result in an increase in calcium in the cell, leading to elevated proteolytic damage to the muscle. Finally, because of a lack of dystrophin there is less mechanical reinforcement to the sarcolemma, leaving muscle predisposed to contraction-induced injury (27).
Exercise Protocols as Therapy for DMD

During contraction the sarcolemma is exposed to different physical stresses, both longitudinal and radial. Concentric, isometric, and eccentric contractions generate longitudinal stress that is maximal at the myotendinous junction (33), where immunocytochemical dystrophin staining has revealed an abundance of dystrophin (29). Contraction also transmits force radially to the membrane via transverse filamentous structures at the M-line and I-bands on either side of the Z-line (28), and the intensity of dystrophin staining corresponds very strongly to these areas. Thus, the periodicity of dystrophin staining is particularly intense at sites where forces that are transmitted radially and longitudinally are greatest. It could be argued that the ability of dystrophic muscle to withstand mechanical forces during contraction may be compromised and that exercise may be harmful. However, the majority of the animal research suggests otherwise, and the limited human data may also support the use of exercise. The positive response to exercise observed in dystrophic human and animal models may be due to the transition of fiber types from type II toward a slower phenotype (type I) following exercise training (14, 15, 27), or a selective destruction of type II fibers that retards further muscle degeneration in DMD (28). It is thought that because type II fibers with larger diameters are less resistant to mechanical stress, they are more susceptible to damage during contraction and are preferentially degenerated during muscular dystrophy (11, 15, 18).

Studies using DMD patients and animal models have examined the effects of exercise on the dystrophic properties of muscle (1, 2, 3, 7, 13–17, 22, 35, 36, 39). The results of several of these studies are presented in Tables 1 and 2. Unfortunately, the mode of exercise differs in the literature between species, with animals using predominantly endurance-type exercises (swimming or running) and DMD patients using either resistance exercise or electrical stimulation of the muscle which mimics resistance exercise. Thus, comparisons between species may be tenuous. The animal model chosen is the mdx mouse, a genetic strain of mouse that is homologous to the DMD patient because of a gene defect that results in mouse muscle containing no dystrophin.

Animal Models

Several studies have examined the effects of endurance exercise on very young mdx mice (less than 1 year old); however, there may be a question of whether the dystrophic condition of the muscles of these younger animals is similar to the condition of the muscles in DMD patients (16). Hayes and Williams (14) examined voluntary wheel running on the contractile, fatigue, and histochemical properties of mdx and control muscles in mice. Results revealed that the soleus (SOL) muscles of exercised mdx mice were able to produce more force than the SOL of the control muscles. Hayes et al. (13) observed that weighted endurance swimming in mdx mice increased oxidative capacity in fast and slow twitch hindlimb skeletal muscle without compromising force generation. Both studies observed that there was a greater fatigue resistance of the SOL of the mdx mice, although this had not been observed in other studies (7, 39). Carter et al. (2), however, observed that voluntary running for one month had no effect on the force generation of the extensor digitorum longus (EDL) of young mdx mice but increased force generation in the SOL. These incongruities may have been due to the shorter duration of this study (1 month) compared to others (10 weeks to 1 year). In another
<table>
<thead>
<tr>
<th>Study</th>
<th>Mode of exercise</th>
<th>N</th>
<th>Age</th>
<th>Duration</th>
<th>Results of exercise training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carter et al. (2)</td>
<td>Wheel running</td>
<td>10 <em>mdx</em>-exercised</td>
<td>4 weeks</td>
<td>1 month</td>
<td>SOL: increased CSA; no change in strength (adult); increased CSA, increased strength (young)</td>
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<tr>
<td></td>
<td></td>
<td>10 <em>mdx</em>-exercised</td>
<td>6 months</td>
<td>1 month</td>
<td>EDL: increased CSA, decreased strength (adult); no increase in CSA, no change in strength (young)</td>
</tr>
<tr>
<td>Dupont-Versteegden et al. (6)</td>
<td>Wheel running</td>
<td>10 <em>mdx</em>-exercised</td>
<td>3 weeks</td>
<td>12 months</td>
<td>30% increase in active tension of DIA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10 <em>mdx</em>-exercised</td>
<td>3 weeks</td>
<td>10–13 months</td>
<td>No change in fatigue profile of SOL or DIA</td>
</tr>
<tr>
<td>Dupont-Versteegden et al. (7)</td>
<td>Wheel running</td>
<td>10 <em>mdx</em>-exercised</td>
<td>3 weeks</td>
<td>15 weeks</td>
<td>Increased % type I fibers in SOL and EDL</td>
</tr>
<tr>
<td></td>
<td>Wheel running</td>
<td>10 <em>mdx</em>-sedentary</td>
<td>5 weeks</td>
<td>15 weeks</td>
<td>Increased fatigue resistance in SOL and EDL</td>
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<tr>
<td></td>
<td>Endurance swimming</td>
<td>9 <em>mdx</em>-sedentary</td>
<td>4 weeks</td>
<td>16 weeks</td>
<td>Increase % type I fibers in EDL and SOL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 <em>mdx</em>-exercised</td>
<td></td>
<td></td>
<td>EDL: greater resistance to fatigue</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9 <em>mdx</em>-sedentary</td>
<td></td>
<td></td>
<td>SOL: greater absolute and relative force production</td>
</tr>
<tr>
<td>Hayes and Williams (13)</td>
<td>Wheel running</td>
<td>7 <em>mdx</em>-exercised</td>
<td>24 months</td>
<td>10 weeks</td>
<td>Increased relative force production in SOL and EDL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 <em>mdx</em>-sedentary</td>
<td>24 months</td>
<td></td>
<td>EDL: increased fatigue resistance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 <em>mdx</em></td>
<td>18 months</td>
<td>11 months</td>
<td>SOL: increased specific tension, but no change in fatigue characteristics</td>
</tr>
<tr>
<td></td>
<td></td>
<td>8 control</td>
<td></td>
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</tbody>
</table>

*Note.* DIA (diaphragm); SOL (soleus); EDL (extensor digitorum longus); CSA (cross-sectional area).
Table 2  Results of Selected Resistance Exercise Protocols Using DMD Patients

<table>
<thead>
<tr>
<th>Study</th>
<th>Mode of exercise</th>
<th>N</th>
<th>Age</th>
<th>Duration</th>
<th>Results of exercise training</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abraham and Rogoff (1)</td>
<td>Resistance exercise</td>
<td>27 DMD</td>
<td>6–20 years</td>
<td>7 months</td>
<td>Small improvements in some subjects</td>
</tr>
<tr>
<td>de Lateur and Giaconi (3)</td>
<td>Resistance exercise</td>
<td>4 DMD</td>
<td>4–11 years</td>
<td>6 months</td>
<td>Improved muscle strength at 5 and 9 months</td>
</tr>
<tr>
<td>DiMarco et al. (4)</td>
<td>Resistance breathing</td>
<td>5 DMD</td>
<td>7–21 years</td>
<td>6 or 12 weeks</td>
<td>Improved respiratory muscle endurance after 6 and 12 weeks</td>
</tr>
<tr>
<td>Dubowitz et al. (5)</td>
<td>HFES</td>
<td>6 DMD</td>
<td>4–7 years</td>
<td>7 weeks</td>
<td>Decreased MVC in TA muscle</td>
</tr>
<tr>
<td>Hoberman (7)</td>
<td>Resistance exercise, breathing exercise</td>
<td>10 DMD</td>
<td>9–13 years</td>
<td>4 months</td>
<td>Additional LFES in 3 subjects for 8 additional weeks improved MVC</td>
</tr>
<tr>
<td>Milner-Brown and Miller (24)</td>
<td>LFES and resistance exercise</td>
<td>10&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17–62 years</td>
<td>14 months</td>
<td>Rate of muscle strength loss declined</td>
</tr>
<tr>
<td>Scott et al. (29)</td>
<td>LFES</td>
<td>16 DMD</td>
<td>5–12 years</td>
<td>7–10 weeks</td>
<td>Improved vital capacity and endurance</td>
</tr>
<tr>
<td>Scott et al. (30)</td>
<td>LFES</td>
<td>15 DMD</td>
<td>2–13 years</td>
<td>7–11 weeks</td>
<td>LFES and WT increased muscle strength</td>
</tr>
<tr>
<td>Smith et al. (31)</td>
<td>Resistance breathing</td>
<td>8 DMD</td>
<td>8–16 years</td>
<td>5 weeks</td>
<td>WT alone increased muscle strength</td>
</tr>
<tr>
<td>Vignos and Watkins (34)</td>
<td>Resistance exercise</td>
<td>14 DMD</td>
<td>5–10 years</td>
<td>12 months</td>
<td>LFES alone was ineffective</td>
</tr>
<tr>
<td>Wanke et al. (36)</td>
<td>Resistance breathing</td>
<td>15 DMD</td>
<td>10–24 years</td>
<td>6 months</td>
<td>47% increase in MVC in young subjects</td>
</tr>
<tr>
<td>Zupan (40)</td>
<td>LFES</td>
<td>7 DMD</td>
<td>6–9 years</td>
<td>3 or 9 months</td>
<td>No change in MVC of older subjects</td>
</tr>
</tbody>
</table>

Note. LFES (low frequency electrical stimulation); HFES (high frequency electrical stimulation); WT (weight training); MVC (maximal voluntary contraction); TA (tibialis anterior).

*No DMD patients were used in this study.
study, long-term voluntary running increased the force generation capacity of the diaphragm in \( mdx \) mice (7). This improvement in diaphragm force generation has not been observed in healthy rats (22) or hamsters (8), possibly because the diaphragm is activated so frequently and is fully adapted to increased use (22). It is suggested that perhaps a training effect from running can be observed in the \( mdx \) mice because of the initial weakness in the diaphragm muscles and a greater potential for adaptation (7).

The diaphragm of the \( mdx \) mouse is preferentially damaged during the course of the disease due to a combination of factors. Forced lengthening or eccentric contractions occur during breathing, which increases the stress on the muscle (28, 36), the diaphragm is composed of predominantly type II fibers (80% fast-oxidative), and there is lifelong sustained use (11). Because death from DMD in boys is often the result of failure of the respiratory muscles (7, 19), studies showing an improvement in the force generation capacity of the respiratory muscles with exercise are encouraging. In another study, the diaphragm of \( mdx \) mice after 1 year showed a 30% increase in active tension following a voluntary running program (6).

Another important finding from exercise studies is that there is a greater percentage of type I fibers in \( mdx \) mouse skeletal muscle observed after low-intensity, long-term exercise (14, 16). This may not only be due to the type of training that the mice underwent in these studies, but the progression of muscular dystrophy results in a preferential degradation of type II fibers. Sedentary \( mdx \) mice were reported to have higher percentages of type I fibers in both the SOL and EDL after 16 weeks than non-\( mdx \) sedentary control mice (14). There is also a marked increase in the expression of type I myosin heavy chain (MHC) isoform during the course of the disease (28). Studies have shown that strength improvements using low-intensity exercise have not resulted in hypertrophy of the dystrophic muscle or an increase in type II fibers. This would prove deleterious to the DMD patient because of the increased susceptibility of a larger-type muscle to degeneration. However, Carter et al. (2) observed hypertrophy of the SOL muscle in adult \( mdx \) mice during voluntary running for 1 month without an increase in strength or fatiguability.

Problems may also exist with the aforementioned exercise research in the young \( mdx \) mice. Although both the \( mdx \) mouse under 1 year of age and the DMD patient suffer muscular degeneration, the \( mdx \) mouse has a period of regeneration that may compensate for the degeneration (16). The muscles of these younger \( mdx \) mice show little weakness during the first year of life, unlike the muscles of DMD patients throughout the course of the disease. Because of this resistance to further degeneration during the first year of life, exercise models using the \( mdx \) mouse less than 1 year of age may confound extrapolation of these results to DMD patients.

Two recent studies examined older \( mdx \) mice exposed to long-term voluntary exercise protocols (16, 39). Wineinger et al. (39) exposed \( mdx \) mice to 11 months of voluntary running. Although the researchers found no difference in the fatiguability of the SOL, the EDL muscle of exercised \( mdx \) mice was significantly more fatigue resistant than in sedentary \( mdx \) mice. There was no aggravation of \( mdx \) muscle disease, nor did running weaken the SOL or EDL muscles of \( mdx \) mice compared to \( mdx \) controls. Hayes and Williams (16) observed that old \( mdx \) mice exposed to low-intensity swimming for 10 weeks had a significantly greater
resistance to fatigue in SOL and EDL muscles. There was also observed a prefer-
ential atrophy of type II fibers and an increase in the percentage of type I fibers in
both the SOL and EDL. Both studies showed that older mdx mice responded fa-
vorably to low-intensity, long-term, nonweight-bearing exercise, which could prove
to be beneficial to the DMD patient. In contrast to these studies, Carter et al. (2)
observed hypertrophy in the EDL of adult mdx mice and a decrease in strength
after voluntary wheel running for 1 month. Again, differences in duration of train-
ing may have contributed to differences in results. No studies have examined the
effects of exercise on the diaphragm muscles in older mdx mice.

**Human Models**

There is a paucity of resistance exercise research and even fewer endurance exercise
studies in the DMD patient, most likely due to the controversy surrounding
the therapeutic value of increased activity on dystrophic muscle. However, some
very promising data has been reported in studies examining increases in muscular
activity either through resistance exercise (3, 35), very controlled, simulated low-
intensity exercise such as electrical stimulation (24, 30, 31, 41), and resistance
breathing (4, 32, 37).

One of the few studies to use resistance exercise in the treatment of DMD in
humans was conducted by Vignos and Watkins (35). This study was unique in that
the subjects involved were in the early stages of DMD before the disease had
progressed to the point where subjects were no longer ambulatory. Two previous
studies had examined resistance exercise in subjects already confined to wheel-
chairs (1, 17) and found either very small improvements of muscle strength in only
some of the subjects, or no significant improvement at all. Vignos and Watkins
(35) found that a 12-month resistance exercise program for 14 DMD children rang-
ing from 6–10 years old resulted in improved muscular strength in the first 4 months.
DMD patients were unable to sustain improvement over the entire year; however,
DMD patients using resistance exercise overall were significantly stronger than
control DMD patients at the end of the 1-year program. This was the first study to
establish the positive results of resistance training in DMD patients, and the re-
searchers emphasized the importance of establishing a training program early in
the disease process when there is a maximal amount of functioning tissue. A sub-
sequent study by de Lateur and Giaconi (3) using submaximal isokinetic leg ex-
tensions for 6 months found an improvement in the maximal torque of exercised
versus nonexercised quadriceps muscles both during the training period and up to
18 months after the study. The researchers concluded that submaximal exercise
exhibits no negative effects and may have some value in the strength improvement
of DMD patients. Milner-Brown and Miller (25) have also reported that high-re-
sistance weight training in patients with various forms of neuromuscular disorders
(limb-girdle, facioscapulohumeral, and Becker muscular dystrophies) resulted in
improvements in maximal force and reductions in the fatigue index.

Other muscle strengthening programs using human models have used simu-
lated muscular contractions through electrical stimulation protocols (5, 24, 30, 31,
41). Milner-Brown and Miller (24) examined the effects of chronic low-frequency
electrical stimulation alone and combined with weight training in 10 subjects with
varying forms of muscular dystrophy. Although no DMD patients were used in the
study, the subjects were described as having markedly weak to severely weak
muscles. The researchers found that electrical stimulation combined with low-resistance weight training resulted in significant increases in muscle strength in subjects with good initial strength. There was no improvement in strength in subjects with severely weak muscles. Electrical stimulation alone was generally shown to be ineffective, and in one subject with moderately weak muscles, maximal force actually worsened.

Several low-frequency electrical stimulation protocols using a slightly longer daily stimulation period have reported more success (30, 31, 41). One 9-month study by Zupan (41) examining low-frequency electrical stimulation alone on muscle strength and fatigue in 7 DMD patients ages 6–9 years old, found that in all subjects greater torques were measured on the electrically stimulated limb compared to the control limb at the end of the program. In addition, the stimulated muscles of DMD patients showed a greater resistance to fatigue. However, the mean torques of the stimulated limb began to decrease after the 5th month of the program, following the torque profile of the control limb. A study using an electrical stimulation protocol simulating high-intensity exercise, however, reported that high-frequency electrical stimulation resulted in decrements of muscle function in children with DMD (5).

The results of the Zupan (41) electrical stimulation study were similar to the results observed in Vignos and Watkins (35) using weight training. The dystrophic muscles improved in strength in both studies but were unable to maintain that improvement over an extended period of time. In the Vignos and Watkins (35) and Zupan (41) studies, the improved strength measures began to decline after the 4th and 5th month of the study, respectively. Both studies suggested that even though strength levels may improve and delay the inevitable degeneration of DMD with muscular training, the progression of the disease is inevitable. The Milner-Brown and Miller (24) study and other low-frequency electrical stimulation research (30) suggest that electrical stimulation may act to develop or maintain muscles with more slow characteristics (type I fibers), as has been observed in mdx mouse studies using low-intensity exercise (6, 7, 14–16).

Similar to results from animal models, it has also been observed that the type IIb fibers in the muscles of DMD patients are subject to greater degeneration, while the type I fibers are mostly spared (38). Thus, exercise programs that cause hypertrophy of the muscle would not be beneficial to the DMD patient. A low-intensity, nonweight-bearing exercise that results in a shift in the phenotype of type II fibers to more fatigue-resistant type I fibers may delay the degeneration of muscle observed during DMD and decrease susceptibility to muscle damage through activity.

As in the animal model, the diaphragm and respiratory muscles of the DMD patient have been studied. Although there is research examining the effects of training on ventilatory strength and endurance, the results are equivocal. Studies have reported both an increase in ventilatory strength and endurance in DMD patients following training of the respiratory muscles by breathing against resistance (4, 37). Dimarco et al. (3) found significant improvements in maximal voluntary ventilation (MVV) tests in 5 DMD patients after inspiratory muscle endurance exercises. The improvements were observed after 6 weeks of training, and there was a further increase in MVV observed in 2 of the 5 DMD patients that continued in the study for an additional 6 weeks. These results contrast the results observed in resistance exercise and electrical stimulation studies in skeletal muscles of DMD
patients, where improvements appear to be limited after 4–5 weeks (35, 41). It is also interesting to note that 4 of the 5 DMD patients in this study were between 13–21 years of age and confined to wheelchairs due to the progression of the disease. Although respiratory muscle function deterioration is believed to parallel that of the limb musculature (4), Dimarco et al. (4) showed that significant improvements in ventilatory endurance could occur late in the disease progression. The researchers also reported that the greatest improvements occurred in those subjects with the greatest baseline level of respiratory muscle function, similar to other resistance exercise and electrical stimulation protocols (24, 35).

Another study using inspiratory resistance exercises, however, showed no significant effects on ventilatory strength or endurance (32). Using a similar protocol as Dimarco et al. (4), a twice daily 10–15-min training session, Smith et al. (32) observed no significant change in the total expired volume following MVV maneuvers after 5 weeks of training. Smith et al. (32) inferred that the increases observed in the Dimarco et al. (4) study may have been due to questionable baseline measurements or a learning effect and that recommendation of inspiratory training could be dangerous to the already weak respiratory muscles in the DMD patient. More research is needed to confirm whether resistance breathing is beneficial or potentially harmful to the DMD patient.

**Conclusions**

The muscles of DMD children are subject to greater degeneration than muscles of healthy children due to a lack of dystrophin, which is essential to maintaining muscle fiber integrity. Because muscles of DMD children are vulnerable to damage from mechanical stress, there is controversy in the literature regarding therapeutic intervention involving an increase in muscular activity. However, a majority of research using animal models and a small number of human studies suggest that increasing activity may actually delay muscle degeneration in dystrophic muscle. Those utilizing exercise programs in these vulnerable patients must employ caution. Only certain forms of exercise should be considered, such as those that use long-term, low-intensity, preferably no-load (but possibly low-load) weight-bearing activity. This type of exercise reduces mechanical stress on the muscle, does not promote hypertrophy, and may cause an increased expression of “slow” MHC isoforms found in type I fibers which are less vulnerable to degeneration. It seems important that exercise programs be incorporated when there is still an abundance of functioning muscle for benefits to be observed, and all exercise programs should be administered under the supervision of a knowledgeable physician. Because of the relatively small number of human studies that have been performed on the benefits of exercise for DMD patients, more research is warranted. There is especially a need for the development of specific exercise protocols at different stages of the disease process to maximize and preserve the functionally useful muscle as degeneration progresses. More research is also needed to determine the effects of training on ventilatory strength and endurance on the diaphragm and respiratory muscles of the DMD patient. It is also important to consider that exercise is not a cure and will only delay the inevitable degeneration of dystrophic muscle. However, exercise has been shown to improve muscular strength in limb girdle, facioscapulohumeral, and Becker muscular dystrophy and thus may act to improve the quality of life for the DMD patient.
References

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