Research Update: 2009: http://talk.news-medical.net/profile/AyushMuscularDystrophySociety

Muscular dystrophies are a group of more than 20 different genetic neuromuscular disorders, some more debilitating than others. The most common, Duchenne muscular dystrophy (DMD) affects 1 in 3500 boys. There is a problem in the chromosome that codes for a protein called dystrophin which maintains the integrity of the muscle cell wall. Eventually irreversible destruction of the muscle cells occurs. There is no cure for any form of muscular dystrophy some medicines, ayurvedic remedies and therapies can slow the course of the disease. Human trials of gene therapy with the dystrophin gene are on the near horizon. For instance, scientists are researching ways to insert a working dystrophin gene into the muscles of boys with Duchenne and Becker muscular dystrophies. Panch Karma procedures are also found useful in the life term management of muscular dystrophy. Ayurvedic researchers are investigating the potential of certain muscle-building Rasayana medicines to slow down or reverse the progression of muscular dystrophy. Other trials are looking into the effects of the dietary supplements Withania somnifera, Curcuma longa, Creatine and glutamine on muscle energy production and storage. **-AMDS India.**

Ayurvedic Management of Muscular Dystrophies : 55 Patients of DMD, BMD & LGMD

This recent paper by Dr Mukesh D Jain* deals with a main thrust clinical program of care through research on Neuro-muscular disability with a focus on complementary Ayurvedic and Yogic approach of treatment. Clinical evaluation involving Rasayana based Ayurvedic herbs along with Til-Mash Pinda swedana; Shat-Bala Prasarni Anuvasana Vasti and Yogic support yielded mixed results in **55** boys with 28 Duchenne Muscular dystrophy (DMD); 19 with Becker's Muscular Dystrophy (BMD) and 8 boys with Limb Girdle Muscular Dystrophy (LGMD). Although this Ayurvedic program did not have the hope for the genetic effect of restoring dystrophin production, it

did reduce serum CK levels. This could be a sign of decreased muscle damage. Since there was definite improvement in functional ability, it is possible that complementary Ayurvedic treatment allow longer survival with minimum disability. This carry a message to needy people and interested healthcare professionals that complementary Ayurvedic & Yogic help is available to patients afflicted by disabling condition with substantial loss of functional mobility of muscles, ligaments and joints.

Introduction: The word dystrophy comes from Latin and Greek roots meaning "faulty nutrition." When doctors first began describing muscle diseases in the 19th century, they had few tools other their own eyes. Muscles in many diseases appeared to be wasting away, and the doctors theorized that they somehow weren't being properly nourished. Today, we know that many muscle wasting diseases are caused by defects in genes for the muscle proteins. Most of these proteins appear to play a role in supporting the structure of muscle fibers, although some proteins may play a role in the biochemical processes that go on inside the muscle fibers. The term Muscular dystrophy refers to a group of genetic diseases marked by progressive weakness and degeneration of the skeletal or voluntary muscles, which control movement. The muscles of the heart and some other involuntary muscles are also affected in some forms of muscular dystrophy, and a few forms involve other organs as well. The major forms of muscular dystrophy are myotonic, Duchenne, Becker, limb-girdle, facioscapulo-humeral, congenital, oculo-pharangeal, distal and Emery-Dreifuss. Some of these names are based on the locations of affected muscles. For example, "facioscapulo-humeral" refers to the muscles that move the face, scapula (shoulder blade) and humerus (upper arm bone). Others are based on the type of muscle problem involved ("myotonic" means difficulty relaxing muscles), the age of onset of the disease (as in "congenital," or birth-onset, dystrophy), or the doctors who first described the disease (Duchenne, Becker, Emery and Dreifuss are doctors' names). Forms of muscular dystrophy differ in severity, age of onset, muscle first and most often affected, the rate at which symptoms progress, and the way the disorders are inherited. The muscular dystrophy is diagnosed by muscle biopsy, DNA testing, electromyogram (E M G) and nerve conduction velocity (N C V). Blood enzyme tests are helpful because degenerating muscle become "leaky". They leak enzymes, which can then be detected in the blood. Presence of these enzymes in the blood at higher than normal levels is a sign of muscular dystrophy. One such enzyme is Creatine kinase, or CK. The CK level is elevated in many forms of muscular dystrophy, some forms resulting in a higher level than others.

Duchenne muscular dystrophy (DMD) is the most common lethal X-linked recessive disorder, affecting 1 in 3,500 live male births (1). DMD children show early symptoms of muscle degeneration, frequently develop contractures, and lose the ability to walk between 6 and 12 years of age. With progressive disease, most patients succumb to death

from respiratory failure and cardiac dysfunction in their twenties (2). The primary cause of this disease stems from mutations in the dystrophin gene, which is essential for the structural and functional integrity of muscles (3). Mutations in dystrophin result in membrane damage, allowing massive infiltration of immune cells, chronic inflammation, necrosis, and severe muscle degeneration (2). Normally, muscle cells possess the capacity to regenerate in response to injury signals. However, this ability is lost in DMD, presumably due to an exhaustion of satellite cells during ongoing degeneration and regeneration cycles (1). Although dystrophin mutations represent the primary cause of DMD, it is the secondary processes involving persistent inflammation and impaired regeneration that likely exacerbate disease progression. DMD is characterized by (i) Onset of muscle weakness usually before 4 years of age, (ii) Selective muscle involvement of pelvic and pectoral girdles, (iii) Hypertrophy of the calves muscle, (iv) grossly elevated serum C K levels and (v) Relentlessly progressive weakness of muscle, leading to inability to work within 10 years of onset and later to contractures and thoracic deformity. There is no specific cure in any system of medicine and the death usually occurs before the age of 20 years caused by respiratory failure or less frequently by cardiac involvement.(4)

Becker muscular dystrophy (BMD) was initially described by Becker and Kiener in 1955.(5, 6). The signs, symptoms and the course of Becker muscular dystrophy (BMD) are similar to those of Duchenne but generally appear later and progress more slowly. BMD is generally milder than DMD. The clinical distinction between the 2 conditions is relatively easy because (i) less severe muscle weakness is observed in patients with BMD and (ii) affected maternal uncles with BMD continue to be ambulatory after age 15-20 years. Accuracy of diagnosis has been refined with the recognition of the dystrophin gene defects and with dystrophin staining of muscle biopsy specimens.(3, 4, 5). The Becker dystrophy can first appear much later than Duchenne, even as late as age 25. The progression is typically slower, with the ability to walk usually preserved in to the 30s. The severity of the disease varies, and boys and men with Becker dystrophy have a longer life expectancy than those with Duchenne. The progression of weakness depends on how much dystrophin is made and how well it functions in the muscles.

Limb-girdle muscular dystrophies (L G M D) are neuromuscular disorders characterized by proximal muscular weakness of the pelvic and shoulder girdles and a variable progression with symptoms, ranging from very severe to mild (7), (4). The onset of Limb Girdle Muscular dystrophy (L G M D) is generally in adolescence or early adulthood. In most common forms, L G M D causes progressive weakness that starts in the hips and moves to the shoulders. The weakness progresses to include the arms and legs. Within 20 years of onset, walking is difficult. Researchers have found that autosomal recessive limb-girdle dystrophy can result from gene defect on chromosomes 2, 13, 15, and 17, and that an autosomal dominant form can result from gene defects on

chromosome 5. A gene on chromosome 15 that codes for the enzyme calpain 3 may also play a role in some cases of L G M D.

Pathogenesis: Dystrophy is a genetic defect and is caused by lack of a single muscle protein Dystrophin (1 of 3000 muscle proteins). DMD and BMD are due to different changes in the dystrophin gene, which contains information for a protein that is important for muscle cells to work properly. This gene is located on the X chromosome. Dystrophin is localized to the sarcolemma in normal skeletal muscle, but is completely absent in muscle from DMD patients (8). Usually disease is inherited but is also caused by spontaneous mutation more than 30% of the time. Each child of the carrier mother has a 50% chance of inheritance of Muscular dystrophy. Though girls can be carriers, more than 80% show no muscular dystrophy related symptoms. At present a hypothesis postulates a defect in the sarcolemma membrane which allows a substance (or substances), as yet unknown but which could possibly be calcium, to enter the muscle fiber too freely, and there to activate neutral proteases which, in turn, maintain an excessive degree of muscle catabolism and lead to muscle fiber necrosis. (9, 10, 11, 12).

No treatment is at present known in any system of medicine which has any definite influence upon muscular dystrophy. The absence of specific treatment for muscular dystrophy makes it all the more important to consider complementary and alternative approaches of treatment. In India, the Dystrophy boys always seek Ayurvedic help in the hope for some relief. The Ayurvedic treatment involving Rasayana group of herbo-mineral or gold based medicine, yogic support and specific Panch karma procedures have shown definite protective influence and longer survival upon muscular dystrophy. Ayurvedic Acharyas carefully consider this condition as adibala-pravrit Mamsa-vata-kshaya due to srotorodha. There is depletion of Mamsagni paving the way of Ama formation. It is followed by vitiation of Kapha dosha. (13, 14, 15). While srotorodha produces hypertrophy in particular region, it also manifests as first prokopa and then depletion of Vata element. This complex pathogenesis is responsible for progressive wasting and necrosis of the affected muscle fibers.

Ayurveda visualizes 13 major types of Agnis (enzyme complex) which are responsible for the process of metabolism. Each of seven dhatus has individual dhatvagnis. The increase or decrease of a particular dhatus depends upon the increase or decrease of respective dhatvagnis. According to Charak, Mamsa-kshaya may be present when there is prolonged majjagata kupita Vata. This is always followed by depletion of Vata element. It is genetic predisposition (Beeja dosha) that convert physiological Vata element in to pathological morbidity. The srotodushti (? Sarcolemma membrane defect) is responsible for the mamsa dhatu kshaya.

The concept of Dosha-Dhatu-Mala (D. D. M) is unique in Ayurveda. The dhatus are those substances which are retained by the body and always rejuvenated or replenished. Ras-Rakta-Mamsa-Meda-Asthi-Majja and Sukra are seven dhatus which develops in human body in a fixed, sequential manner one from the other. Each succeeding dhatu is a metabolic refinement of the previous dhatu and get nourished by it. The first dhatu, Rasa (nutrient fluid) is the metabolic end product of the digestion that takes place within gastro-intestinal tract. The Rasa dhatu has to be metabolized in to Rakta dhatu. The Mamsa dhatu comes from Rakta dhatu and in turn, give rise to Meda dhatu. The Asthi dhatu is the product of Meda dhatupaka that contains Majja dhatu which is the prime seat of Vata element. (13, 15)

We know that Vata (Prana) and Rakta dhatu are two major life sustaining elements in the body. The Vata has been attributed like genetic material that carries life information essential for different activities. The Rakta dhatu is the basis of biological force that provides nutrition at cellular level and paves the way of excretion of metabolic toxins. The driving force beyond Rakta dhatu is Vata element which circulates itself to cellular level along with Rakta. The conjoint circulation of both Rakta and Vata is manifestation of life (Prana). This Prana is responsible for the contraction and relaxation of muscle fibers or muscular activity. It means we have to focus our attention on the dhatvagnis paka of Rasa-Rakta-Mamsa and Meda dhatus besides Asthi and Majja dhatus. In this context Ayurvedic Rasayana therapy has significant role to play. (16, 17) Ancient Ayurvedic physicians had developed certain dietary and therapeutic measures to arrest/delay degeneration process and rejuvenating whole functional dynamics of the body system. This revitalization and rejuvenation is known as the 'Rasayana Chikitsa' (rejuvenation therapy). Rasayana are special ayurvedic resources that increase enzymatic essence of each dhatu starting from Rasa dhatu. Ayurveda uses herbal mineral and metallic source for this purpose. Traditionally, Rasayana drugs are used against a plethora of seemingly diverse disorders with no patho-physiological connections according to modern medicine. Though, this group of plants generally possesses strong antioxidant activity, only a few have been investigated in detail. Neurodegenerative diseases have been reported as reactive oxygen species mediated and several Rasayana plants with potent antioxidant activity have been documented by many investigators (18). The pure gold bhasma in low dose has been used successfully in the management of degenerative diseases of mamsa and Majja dhatu. (19, 20, 21). Certain Ayurvedic herbs known for their Rasayana effects are being scientifically verified for their possible effect in the management of Muscular dystrophy. The well known herb ***Curcuma longa*** *has* been widely investigated for the immuno-localization and activation of nuclear factor-

{Kappa}B in polymyositis, dermatomyositis, and Duchenne muscular dystrophy (DMD). Data support the hypothesis that NF-?B contributes to the perpetuation of the dystrophic damage and show that its blockade produces beneficial effects on functional, biochemical, and morphological parameters. These new findings may have clinical

implications for the pharmacological treatment of patients with Muscular dystrophy. (22, 23, 24, 25, 26). ***Withania somnifera*** is wonderful ayurvedic herb that is widely used for stress, stamina and brain functions. It contains Withanolide which is anti-inflammatory; induces significant regeneration of axons, dendrites, pre-synapses and post-synapses in the neurons. It suppresses free radical generation; It also ameliorates neuronal dysfunction. (27), (28) (29). The cardiac problems associated with some forms of muscular dystrophy sometimes need treatment. ***Terminalia Arjuna*** has remarkable cardio protective, heart muscle strengthening properties. Current scientific research has proved that plant contains specific medically active constituents namely triterpine glycosides like arjunetosides I, II, III, IV, arjunine and arjunetein, phytosterols, rich in minerals like calcium, magnesium, zinc and copper. Regular use of Arjuna improves pumping activity of heart, improves cardiac muscle strength, decrease in LDL cholesterol levels. It has been reported to possess protective cardiovascular and hypolipidemic properties. (30). Similarly ***Tinospora cordifolia*** have been used in general debility, digestive disturbances, loss of appetite and fever in children. It is also an effective immunostimulant. Its principal constituents are tinosporine, tinosporide, cordifolide, and cordifol. The plant is used in Ayurvedic Rasayana to improve the immune system and the body's resistance to infections (31). ***Praval*** being a natural source of rich calcium, calcined Coral is widely used in ayurvedic medicine as a supplement in the treatment of variety of bone metabolic disorders associated with calcium deficiency. Praval bhasma is effective in the prevention of calcium deficient spinal contractures and bone deformities (32).

Usually Rasayana molecule does not work without purificatory procedures. The deepana, pachana process must be strengthened, the dosha must be balanced and metabolic toxins must be eliminated from dhatus through Panch karma procedures in order for Rasayana molecule to work (33), (35). Yogic support is always found useful in the management of Muscular dystrophy. Pawanamuktasa series of Yoga Asanas and Bhastrica Pranayama are important, especially when the dystrophy has progressed for several years. Deep breathing and laughing is often recommended to optimize respiratory care (19, 20).

Material & Methods: Keeping in view the complex pathogenesis involved in Muscular dystrophy, a special Ayurvedic Rasayana supplement was developed using deepan-pachan-srotorodhahara and mamsa dhatu vardhak Ayurvedic resources. The supplement developed and named as **Mamsagni Rasayana**. It combines the proven beneficial effects of Curcuma longa, Withania somnifera, Tinospora cordifolia, Terminalia Arjuna, calcined Coral (Praval) and Gold bhasma, all processed in fresh juice of Aloe Vera barbendensis. The dose of the Mamsagni Rasayana was fixed 20 mg per kilo body weight. The drug was administered orally in two equal doses after meal with 100 ml of milk for a period of 6 months.

Over 100 cases of Muscular dystrophy were enrolled at Panch Karma Clinic of Central Medical Institute, Bhilai and treated during March 1999 and September 2004. We have selected total 28 DMD boys, 19 BMD boys and 8 LMGD boys for inclusion in our clinical study. All the Dystrophy boys were subjected to 2 weeks Panch karma procedures before administration of oral Mamsagni Rasayana along with Yogic support for 6 months. The treatment agenda consisting of (i) Modified Til-Mash Pinda Swedana using fresh leaves of Tejapatra (Cinnamomum tamala), Nirgundi (Vitex negundo) and sprouted Methi (Trigonella foenum) seeds as additional ingredients to Til, Masha, unpolished rice and wheat bran, all cooked in the decoction of Bala (Sida cordifolia), Ashwagandha (Withania somnifera) and milk. (ii) Anuvasana Vasti with Shatbala-Prasarni oil. A blend of Sesame Oil 80%, Soya oil 10% and Castor oil 10% was used as base oil which was medicated by standard method. The principal herbs used in the oil preparation are Shatavari (Asparagus racemosus), Bala (Sida cordifolia), and Ashwagandha (W. somnifera), and Nirgundi (Vitex negundo), and Haridra (Curcuma domestica), and Daru Haridra (Berberis aristata), and Mustaka (Cyperus rotundus), and Barbreng (Embelia ribes), and Mamsa Rohini: (Soymida febrifuda). These herbs are used as Neuro-muscular tonic because of their Vata balancing properties.

Observation & Results: In our study, the motor functions were evaluated using total motor score, upper and lower extremity function grades and timed function tests. Disability was quantitated with Barthel index. Children were found to have disabilities in multiple spheres of life, which were significantly influenced by the motor power. Barthel index was useful in identifying and quantifying specific areas of disabilities in these children.

It was observed that degeneration of muscle fibers was arrested after 6 weeks of administration of Mamsagni Rasayana. This was presumed on the basis of reduced CK level in blood, improved functional ability and quality of life. All the Dystrophy boys who completed clinical Study showed definite sign of improvement such as: (i) Weight loss, (ii) Decrease in walking difficulty and (iii) Decrease in the severity of contractures and scoliosis. However DMD boys have shown very slow progress.

Discussion: Today we know that muscle degenerative wasting conditions in young patients are caused by defect in genes for the muscle proteins. Most of these proteins appear to play a role in supporting the structure of muscle fibers, although some play a role of biochemical enzymes. DMD and BMD are caused by lack of dystrophin protein. The Ayurvedic Rasayana drugs are well known for their effect to delay / slow or reverse the progressive muscular degeneration. (14), (18), (20) (22) (34) (38). Some of the ingredients of Mamsagni Rasayana have been scientifically verified for their possible protective influence in muscular dystrophy (21-29). The Mamsagni Rasayana is supposed to boost Mamsagni at muscle tissue level. It balances the Vata derangement due to Ama and thus retards the muscular degeneration due to Ama (fat deposition). The Til-Masha Pinda Swedana stabilizes and improves the membrane defect removes the extra fat from the

tissue. The Anuvasana Vasti balances the Vata element. The Yogic support of Pawan Muktasana series minimize the contractures, a condition often associated with muscular dystrophy in which shortened muscles around joints cause abnormal and sometimes painful positioning of the joints. In addition, Pawan Muktasana along with certain other Asanas such as Bhujangasana may prevent or delay scoliosis, or curvature of the spine. The Bhastrika Pranayama may support Cardio- Respiratory system and may improve process of beta-oxidation at cellular level. Since we have noticed improved functional ability along with a fall in serum Creatine kinase (CK) level it means there is check on further muscle destruction.

Summary:

Muscular dystrophies are genetic disorders with no satisfactory treatment in any system of medicine. It is a progressive muscle-wasting disease due to a mutation in the dystrophin gene and the consequential protein deficiency in muscle. It results in chronic inflammation and severe skeletal muscle degeneration. How the lack of the sarcolemma protein dystrophin gives rise to the final disease status is still not clear. Several evidences suggest a role of deregulation of NF-kappa in muscular dystrophy. Nuclear factor kappa-B blockade reduces skeletal muscle degeneration and enhances muscle function. Regulation & control of NF?B is thus important. Ayurvedic Til-Mash Pinda Swedana treatment along with Rasayana herbo-mineral resources should be investigated in the light of possible influence on Sarcolemma membrane and NF?B: blockade. In this context targeted research is needed to identify safe ayurvedic herbs, Yogic techniques and Panch karma procedures to further improve complementary approach of Ayurveda. The Ayurvedic program is useful in the long term management of muscular dystrophies. There is further need of controlled studies and multi center clinical trials on a large scale with improved study design and assessment techniques.

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Following graphic images are not displayed :

1. Result Analysis on improvement in Functional ability (Barthel Scale)
2. Clinical improvement after 6 months.
3. Quality of Life improvement.

Review & Comments

This research update is submitted to the National Center for Complementary and Alternative Medicine

nccam.nih.gov/research.

It is quite interesting and fairly well done for ayurvedic science. The paper is recommended to the NAMA conference group.
Dr. Marc Halpern
President California College of Ayurveda, USA

Interesting study and well done with it. Forwarded to the chairman

of the scientific committee of the Ayurveda practitioners association, Dr Eduardo Cardona-Sanclemente.

Dr. Donn Brennan
President, Ayurveda Practitioners Association, UK

Nicely described approach from CAM point of view. There is possibility of applying for research grant to NCCAM that will facilitate to make a large scale project.
Prof. M. S. Rao, Sc.D.
Howard Univ. Col. Of Medicine
Washington, DC

Congratulation on work in this field. I´m open to discuss different topics for research.
Dr. Jorge Luis Berra
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