CONFERENCE REPORT:

Sixth National Scientific Workshop of the Muscular Dystrophy Association of Canada

BANFF, ALBERTA - FEBRUARY 5-7, 1982

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The sixth and by far the largest scientific workshop ever devoted to Muscular Dystrophy Research in Canada was held at the Banff Centre in Banff, Alberta from February 5 - 7, 1982. About 100 scientists registered from across Canada, the U.S. and the U.K. Over 80 scientific communications, poster demonstrations and guest lectures were presented on many aspects of dystrophy research, as well as related basic research. The informal atmosphere offered by the Banff Centre added considerably to the discussions which took place during and between the four scientific sessions. This was the first scientific workshop sponsored by the Muscular Dystrophy Association of Canada (MDAC) to be held in Western Canada, and it was organized by a committee from the University of Alberta in Edmonton, who also served as coauthors of this article summarizing the proceedings. The Alberta government offered its hospitality in the form of a banquet, which was enjoyed by the scientists and other participants. Because of the number of papers presented at each session, we can only mention briefly some of those presented in each session. However, a complete program is included here, as well as abstracts (in alphabetical order) from those authors who wanted a fuller account to be published.

SESSION I

The session opened with two papers related to the problems of carrier detection in Duchenne dystrophy. *Percy et al.* presented further data using logistic discrimination as a means of increasing the value of results from multiple tests in carrier detection. The data clearly bore out the previously reported enhancement by this statistical discriminatory method as applied to series of individual tests. It was pointed out by the authors that positive detection of normals (in the population at risk) was of equal importance. *Barber et al.* discussed a new approach to heterozygote detection using cloned fibroblasts. Although their initial results did not show obvious differences between heterozygotes and normals, the technique clearly has potential importance.

The use of cloned human cells also figured strongly in other presentations. For example, *Askanis* beautifully illustrated the use of tissue culture for Schwann cells in various human neuropathies. The possibility that *Wrogemann et al.* may have demonstrated a gene product in Duchenne dystrophy cultured fibroblasts by two-dimensional gel electrophoresis raised considerable interest. Although these authors were able to supply a rough molecular weight for the "disappearing" protein (i.e. not present in normal fibroblasts), this will require more detailed study before the importance of the discovery can be assessed.

Increased interest is being shown by researchers in myotonic dystrophy. This partly stems from the relative frequency with which this disease is seen and is now, in some parts of the country, the most common type of dystrophy. *Banerjee et al.* reported a study on the causes of the hypercatabolic reduction of IgG in this dystrophy, and proposed the hypothesis that the low affinity of the Fc protector receptors in monocytes could account for the consumption of IgG. *Belanger* and *McComas* described a meticulous study of the dorsiflexors and plantiflexors in dystrophic patients and showed how effective the combined clinical and electrophysiological observations can be in providing base data for follow-up purposes. This should provide a model for those concerned with detailed recording of clinical findings.

Myasthenia gravis still retains a number of problem areas, but perhaps the most cogent are those that relate to management. *Humphrey* and *St. George-Hyslop* were able to show convincingly the importance of persistent immunosuppression in the management of myasthenia and obtained a significant reduction in relapse rate and clinical severity in those patients treated with azathioprine. *Quastel* and *Pennefather* reported data which permits a more quantitative assessment of receptor dysfunction in this disease.

The first session concluded with a description of a spring-loaded knee joint orthosis for Duchenne muscular dystrophy patients. While this certainly provides for a more stable knee joint allowing knee flexion the mechanism is still far from perfect and clearly requires more work before it can be of general value.

At the conclusion of the platform presentations a series of poster demonstrations were held. Some of these represented elaborations of platform papers. For example, a poster was presented by *Belanger* and *Quinlan* on muscle testing. *St. George-Hyslop* and *Humphrey* presented anti-AChR antibody titre correlations with clinical status in myasthenia gravis indicating that over a long term this correlation was not close.

The remaining poster presentations covered a relatively wide field from tissue culture to clinical assessment of structural spinal deformities in Duchenne dystrophy. While all these poster presentations had a worthwhile message as is indicated in their description in the abstracts, particular men-

tion should perhaps be made of the ongoing studies of *Thompson et al.* and the contributions of *Yoneda et al.* The use of direct tissue isoelectric focusing demonstrated in the poster by *Thompson et al.* in studying diagnostic needle muscle biopsies appears to be a valuable contribution to the methodology that can be applied to the study of muscle in the clinic. While the results presented were preliminary, it is quite clear that a lot more is going to be heard of this technique. The method of assessment of degrees of spinal deformity established as an ongoing project at The Hospital for Sick Children by *Yoneda* and others is important in the management and prevention of the distressing spinal distortions that can occur not only in muscular dystrophy, but in other neuromuscular diseases in which paraspinal muscles are weak. This group now has enough experience in the application of the method to the assessment of spinal deformities that a nationwide application of the method through The Hospital for Sick Children is being encouraged.

SESSION II

A number of abnormalities in muscle biochemistry, physiology and morphology were described for murine, avian and hamster models of dystrophy in the second session. Developmental changes and properties of limb muscles in adult mice *(Bressler et al; Parry)* and wing muscles of chickens *(Gandy et al; Hoekman et al.)* indicated that there is an apparent lack of nerve-induced differentiation of slow and fast skeletal muscles in dystrophy. The most striking physiological change in the affected muscles, besides a progressive weakness, is a decreased rate of relaxation after twitch or tetanic contractions, which may be due to a slower reabsorption of calcium ions by the sarcoplasmic reticulum *(Gordon* and *Stein)*. Histochemically, the fibre distribution appears to move selectively to a greater proportion of fast oxidative fibres *(Ovalle et al; Parry)*. Some of these changes, as well as changes in physiological properties may be associated with the severely altered patterns of activation of the limb muscles in dystrophic mice as a result of the lack of Schwann cell ensheathment in the lumbosacral roots *(Bray et al.)*.

Activity patterns in dystrophic animals were implicated also in the necrotic changes occurring in the muscles, since necrosis could be completely halted in young dystrophic hamsters by cord transection *(Karpati et al.)*. Abnormal thymic function was also implicated in the genesis of the dystrophic process and in the degenerative changes occurring in the muscles. *Potworowski & Lemieux* described preliminary findings of depressed immune reactivity in murine dystrophy due to abnormal thymic function. Dystrophic mice showed depressed reactivity to antibodies raised in culture to thymic epithelial antigens, either with or without exogenous thymic hormone. The thymus also showed abnormal levels of mast cells and contained histamine in murine and avian dystrophy. Levels of histamine and mast cells were significantly raised in muscle. The changes in these levels in muscle and thymus were progressive with age and were suggested to be important in the progressive degeneration in muscle *(Befus* and *Nielson)*.

Jasch described changes in proportions of myosin light chains but these did not appear to significantly alter the rate of rise of tension. The normal temperature dependence of dystrophic muscle suggested that defects in contractile proteins did not underly changes in contractile speed. Rather, alterations in calcium pumping, perhaps secondary to calcium loading, may account for the decreased rate of relaxation in dystrophic muscles *(Gordon* and *Stein)*.

Changes in protein synthesis in dystrophic muscles are progressive with age. Significant alterations in protein synthesis was evident from reduction in activity of polysomes in dystrophic hamsters *(Brakier-Gingras* and *Jolicoeur)*, alterations in acetylcholinesterase isozyme content and distribution *(Lindenbaum* and *Livett; Gisiger* and *Stephens)*, reduction in synthesis of alpha-actinin and other Z-line proteins and a later decline in synthesis of myofibrillar proteins *(Atkinson et al.)*. High voltage electron microscopy is currently being used by the group in London *(Shivers et al.)* to visualize the spatial arrangement of alpha-actinin, and contractile proteins to determine the anchoring of actin filaments at the Z-line in the normal and the dystrophic muscle. Alpha-actinin was also implicated in the decline in intramembrane particles distributed in plasma membranes *(Graham et al.)*.

The mouse model of dystrophy differs from other animal models and from the human dystrophy in the abnormal axonal ensheathment of the lumbosacral spinal roots. *Bray et al.* provided evidence that the location of the dystrophic abnormality in the axons was not the exclusive mechanism responsible for poor axonal ensheathment, because regenerating axons after crush injury became normally ensheathed. Abnormalities may therefore be a function of time rather than locality. Evidence was also presented to show that uptake and/or transport of nerve growth factor in sciatic nerves of dystrophic mice was significantly less than normal *(Rutherford* and *Boegman)*.

The second session also included a special guest lecture by *Dr. Stanley Appel. Appel* is Chairman of Neurology and Director of the Jerry Lewis Neuromuscular Research Center at Baylor University in Houston. He is well known for his outstanding biochemical work on a variety of nerve and muscle diseases. *Appel* spoke on growth factors which influence the increase in size of motor cell bodies in the spinal cord and the elongation of their nerve fibres to the periphery. Growth factors are proteins which can be isolated from muscles, sense organs and Schwann cells. Attention was focused on the role of growth factors in amyothrophic lateral sclerosis (A.L.S.).

SESSION III

The third session dealt with the properties of normal and atrophic muscles. Muscles undergo atrophic changes when they are disconnected from their nerve fibres (denervated), as well as in various other disease states. When electrically stimulated, the time course of the contractile events is slowed *(Webster* and *Bressler)* and the reabsorption of calcium ions is reduced in denervated muscles *(Boegman* and *Wan)*, as in dystrophic muscles.

Davis demonstrated that an extract of nerve, when injected daily, could prevent some components of the atrophy in muscles subjected to denervation. Thus, nerves contain growth factors which are important for the maintenance of normal

muscle properties, in addition to the growth factors described by *Appel,* which can be isolated from muscle and affect nerve growth.

The interaction of nerve and muscle in normal growth and development, as well as in disease processes, is an exciting research area which should provide new possibilities for treatment of nerve and muscle diseases in the future. These processes can be studied in a wide variety of animals including lobsters *(Govind)* and chickens *(Bloom* and *Cosmos; Butler et al; Steele).* A particularly interesting method for studying these interactions is by producing chimaeras (organisms formed by grafting differing genetic information) from different species such as chick and quail *(Beresford et al.)* or genetically different strains of the same species such as the mouse *(Peterson* and *Cross).* In this type of experiment the influence of genetically normal nerves on diseased muscles can be studied and vice-versa.

Several papers in this session dealt with normal muscles and reported further evidence on the interactions of muscle proteins *(Cote et al.),* the limiting rate constants in muscular contraction *(Stein et al.),* the regulation of glucose transport *(Klip et al.),* the structure of muscle calsequestrin *(Reithmeier* and *Cozens)* and the function of the sarcoplasmic reticulum *(MacLennan et al.).* Interestingly, development of the sarcoplasmic reticulum, which absorbs calcium ions from the muscle cytoplasm, is inhibited in the absence of spontaneous contractions of cultured skeletal muscle cells *(Charuk* and *Holland).* In contrast, defects in dystrophic muscle membranes may lead to calcium loading, and *Carpenter* and *Karpati* have developed a micro-puncture technique to simulate such defects. Finally, several papers dealt with the effects on normal neuromuscular function of age *(Vandervoort et al.),* bodybuilding *(MacDougall et al.)* and fatigue *(Hunter et al.).*

SESSION IV

The session opened with a presentation in which *Szewczuk* described an abnormal immune response in dystrophic mice when injected with a thymic-dependent antigen. Since this change in thymic function was age-related, defective maturation of the thymus may be involved in the progression of muscular dystrophy. *Slonecker et al.* showed, using whole thymic transplants from dystrophic mice, that hormonal interactions with the thymus may influence the development of dystrophic traits.

Two papers from *Pena's* laboratory suggested that aggregation of intermediate filaments in cultured human skin fibroblasts caused by neurotoxins such as 2,5 hexanedione or acrylamide strongly resemble the aggregation observed in giant axonal neuropathy. This aggregation is not due to an alteration in vimentin, the major protein of intermediate filaments and suggests that a regulatory protein involved in the organization of intermediate filaments may be involved.

Lectins are useful in detecting alterations in cell surface carbohydrate. These probes in conjunction with a sensitive visualization technique and transfer of proteins from polyacrylamide gels to nitro-cellulose paper have been used by *Gordon et al.* to examine differences between the cell surface carbohydrate in normal and dystrophic cells. Lectins can also be used to visualize membrane oligosaccharides in thin sections of muscle. *Parfett et al.* illustrated the role of cell surface carbohydrate in muscle development and found that myoblasts resistant to the lectin Concanavalin A failed to fuse.

Poznansky et al. found that alterations in the membrane fluidity of squid axon, as a result of changes in cholesterol level, had no effect on sodium or potassium currents. Cholesterol in the plasma membrane of axons was found to move from one side of the bilayer to the other with a half time of about 10 minutes. *Logan et al.* showed that proline uptake into muscle cells in culture was stimulated by calcium as well as by sodium.

Creatine kinase is a useful marker enzyme for certain types of muscular dystrophy. *Guslits* and *Jacobs* using highly resolving gel systems showed that up to 21 active enzyme subspecies could be detected in extracts of human skeletal muscle. The ability to resolve the subforms of creatine kinase may provide a powerful diagnostic tool for muscle disorders. *Kreis et al.* showed that isolation of the defective gene of Duchenne Muscular Dystrophy is now possible using powerful recombinant DNA methodology.

Pollack et al. showed by indirect immunofluorescence that the components of the cytoskeleton of fibroblasts from dystrophic hamster fibroblasts had a normal cellular distribution. The tubulin and high molecular weight microtubule-associated proteins could be visualized using a sensitive Gold immunolabelling procedure *(Stearns* and *Ochs).*

Heat shock treatment of cells can result in alterations in the activity of specific genes. *J. Bag* found that heat shock treatment of muscle cells in culture resulted in the production of several new polypeptides with weights of 25,000, 65,000 and 81,000 daltons, while the synthesis of some normal cellular proteins including those of the cytoskeleton were decreased. A similar treatment of fibroblasts from normal and dystrophic hamsters also showed an increased synthesis of 25,000 and 64,000 dalton proteins *(Pollack et al.). Cates* and *Holland* showed that a 70,000 dalton cell surface protein could be detected on the cell surface of cultured chick skeletal muscle cells by the inability of antibody preabsorbed with intact cells to bind to this cell surface component.

Else and *Barth* have localized a Ca^{2+}-dependent protease to the plasma membrane of muscle cells using indrect immunofluorescence. Inhibition of these Ca^{2+}-dependent proteases by thiol reagents results in less damage to muscle cells *(Jasmin* and *Proschek).*

The finding that a protein kinase from muscle cells binds mRNA suggests that this protein may play a regulatory role in muscle development *(Sell et al.).* The level of protein initiation was decreased in all tissues (from dystrophic mice) examined by *Nicholls* and *Nickson.* This was due to a decrease in the activity of initiation factor e1F-2. A decrease in the snythetic activity of polysomes isolated from dystrophic heart muscle was suggested by *Bakier-Gingras* and *Jolicoeur* to be due to a defect in the ribosome itself.

A highlight of the final session was a guest lecture by *King Engel,* who is Professor of Neurology and Director of the Center for Neuromuscular Diseases at the University of Southern California in Los Angeles. *Engel* is well known for

his innovative treatments of various neuromuscular diseases, as well as his anatomical slides illustrating the nature of the disorders at the cellular level. Those present at the workshop were treated to a review of the pathogenesis and treatment of neuromuscular diseases, which was impressive in terms of the sheer beauty of the illustrations, as well as the scope of the knowledge they represented.

Also in the final session, John Connors (executive director of MDAC) showed a videotape that the Association has recently made with the help of Access television in Alberta on the treatment of muscular dystrophy. Groups interested in obtaining a copy of this tape should contact Mr. Connors. MDAC should also be congratulated on its continuing support of these scientific workshops. Ideas generated in a few minutes of discussion at these meetings can lead to projects that may take years to complete in the laboratory, but can have important implications for the treatment of neuromuscular diseases. Many people left the meeting feeling that real progress had been made since the last workshop in Hamilton two years ago, and that there is tremendous hope for the future.

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Sixth National Scientific Workshop of the Muscular Dystrophy Association of Canada

BANFF, ALBERTA
February 5 - 7, 1982

PROGRAM

SESSION I: Defects and treatments in human neuromuscular diseases.
Chairmen: *A. McComas, G. Monckton.*

Application of logistic discrimination to duchenne muscular dystrophy carrier detection. *M.E. Percy, M.W. Thompson, T. Stukel and D.F. Andrews. Hospital for Sick Children and Department of Statistics, University of Toronto.*

Cloning skin fibroblasts from carriers of duchenne muscular dystrophy. *B.H. Barber, A. Chin and D. Hoar, Department of Medical Genetics, University of Toronto.*

The use of tissue culture of Schwann cells in human neuropathies. *V. Askanas, University of Southern California, Los Angeles*

Progress in the search for mutant protein(s) in duchenne muscular dystrophy (DMD). *K. Wrogemann, E. Rosenmann, C. Kreis, M. Dobbs and R.G. Thompson. Departments of Biochemistry and Pediatrics (Genetics), University of Manitoba, Winnipeg.*

Monocyte IgG-Fc receptor concentration and affinity in myotonic dystrophy. *Diponkar Banerjee, Jeffrey McClintock, Meredith M. Silver and Arthur J. Hudson. Departments of Pathology and Clinical Neurological Sciences, University of Western Ontario, London.*

Comprehensive neuromuscular assessment in myotonic dystrophy. *A.Y. Belanger and A.J. McComas. McMaster University Medical Centre, Hamilton.*

Two functional nicotinic receptors of different affinity on human peripheral blood lymphocytes. *Marek Rola-Pleszczynski, Louis Ménard and Simon Lemaire, Immunology Division, Department of Pediatrics and Department of Pharmacology, Université de Sherbrooke.*

Neuromuscular transmission in myasthenia gravis. *D.M.J. Quastel and P. Pennefather, Department of Pharmacology, The University of British Columbia, Vancouver.*

The efficacy of azathioprine in myasthenia gravis. *John G. Humphrey, and P. St. George-Hyslop. Division of Neurology, Toronto General Hospital and University of Toronto.*

A spring-loaded knee joint orthosis for duchenne muscular dystrophy patients. *P. Allard, M. Duhaime and P.S. Thiry, Pediatric Research Center, Ste-Justine Hospital, Montréal.*

POSTERS

In vitro study of growth and differentiation of normal and dystrophic human skeletal muscle. *G. Jasmin, C. Tautu and L. Proscheck, Département de pathologie, Faculté de médecine, Université de Montréal.*

Segmental myofibre necrosis in myotonic dystrophy. *Meredith M. Silver, Diponkar Banerjee and Arthur J. Hudson. Department of Pathology, St. Joseph's Hospital, London, Ontario.*

Muscle function studies in human plantar-flexor and dorsi-flexor muscles. *A. Belanger and J. Quinlan. McMaster University Medical Centre, Hamilton, Ontario.*

Correlations between serum anti-AChR Ab titre and clinical parameters of M.G. *P. St.-George-Hyslop, D.A.G. Mickle and John G. Humphrey.*

The respiratory muscles in myotonic dystrophy. *R. Bégin, M.A. Bureau, L. Lupien, J.P. Bernier and M. Geoffroy. Unité de Recherche Pulmonaire, CHUS, Université de Sherbrooke, Sherbrooke, Québec.*

Ultrastructural studies in duchenne muscular dystrophy. *H. Stephens, Department of Anatomy, Université de Montréal.*

A new method for assessing spinal stability in muscular dystrophy patients. *Bruce Yoneda, Junichi Monji, Jan Koreska, Colin Moseley, The Hospital for Sick Children, Toronto, Ontario.*

Carriers of duchenne muscular dystrophy: effects of exercise. *H. Stephens, L. Reynolds, M. Vanasse, J. Patterson, G. Hall, S. Bloom, K. Whittaker, D. Moss and V. Dubowitz, Jerry Lewis Muscular Research Centre, London, England.*

The MDAC Clinic Data Base at the Hospital for Sick Children. *Jan Koreska, Department of Medical Engineering, The Hospital for Sick Children, Toronto, Ontario.*

The application of direct tissue isoelectric focusing to the study of diagnostic needle muscle biopsies. *B.J. Thompson, A.H.M. Burghes, M.J. Dunn and V. Dubowitz. Jerry Lewis Muscle Research Centre, Department of Paediatrics and Neonatal Medicine, Hammersmith Hospital, DuCane Road, London, England.*

SESSION II: Studies on animal models of dystrophy.

Chairmen: *B. Livett, T. Gordon.*

Contractile properties of dystrophic skeletal muscle. *B.H. Bressler, W.K. Ovalle, L.G. Jasch, C.E. Slonecker. Department of Anatomy, University of British Columbia, Vancouver.*

A comparison of fore-limb and hind-limb muscles in dystrophic mouse. *D.J. Parry, University of Ottawa, Ottawa, Ontario.*

Temperature dependence of contractile properties in normal and dystrophic mouse muscles. *T. Gordon and R.B. Stein, Departments of Pharmacology and Physiology, University of Alberta, Edmonton, Alberta.*

Changes in the distribution of five proteins in dystrophic skeletal muscle: identification of the proteins, a study of their developmental significance and nerve-dependence in normal animals. *L.G. Jasch, Department of Anatomy, University of British Columbia, Vancouver.*

The synthesis of Z-line proteins in skeletal muscle of normal and dystrophic mice. *B.G. Atkinson, R.R. Shivers and M. Pollock. Cell Science Laboratories, Department of Zoology, University of Western Ontario, London, Ontario.*

Chronic electrical stimulation fails to induce necrosis in hind leg muscles of cordotomized dystrophic hamsters (UM-X 7.1). *G. Karpati, S. Carpenter and S. Prescott, Montreal Neurological Institute - McGill University, Montreal, P.Q.*

Histamine and mast cell abnormalities in avian and murine muscular dystrophy. *A. Dean Befus and Laurie Nielsen. McMaster University, Hamilton, Ontario.*

T-cell differentiation in murine muscular dystrophy: preliminary results. *E.F. Potworowski and Suzanne Lemieux, Institut Armand-Frappier, Laval, Quebec.*

Enhanced axonal ensheathment in regenerated spinal roots of dystrophic mice. *G.M. Bray, T. Carlstedt, S. David, W. Wilcox and A.J. Aguayo. Neurosciences Unit, The Montreal General Hospital and McGill University, Montreal.*

GUEST LECTURE: Growth Factors in motoneuron development: an approach to the etiology of A.L.S. *S. Appel, Baylor University, Houston, TX, U.S.A. Chairman: D. MacLennan.*

POSTERS

Properties of motor units in dystrophic mouse EDL. *D.S. Bateson and D.J. Parry, University of Ottawa, Ottawa, Ontario.*

Abnormal distribution of fibre types in a slow-twitch muscle of the dystrophic mouse during postnatal development. *W.K. Ovalle, B.H. Bressler, L.G. Jasch, C.E. Slonecker, Department of Anatomy, University of British Columbia, Vancouver.*

Axonal transport of ^{125}I-NGF in dystrophic and normal mice. *Philip S. Rutherford and Roland J. Boegman, Department of Pharmacology, Queen's University, Kingston, Ontario.*

Comparison of *In Vivo* contractile and pharmacologic responses of a fast twitch skeletal muscle in normal (Line 412 New Hampshire and White Leghorn) and dystrophic (Line 413 New Hampshire and Storrs White Leghorn Blackcross) chickens. *T.B. Hoekman, S.E. Howlett, V. Umanee, and P. Redfern Facultz of Medicine, Memorial University of Newfoundland, St. John's, Newfoundland.*

Dystrophic murine skeletal muscle Z-discs: high voltage electron microscopy. *R.R. Shivers, D.I. Rodenhiser and B.G. Atkinson. Cell Science Laboratories, Department of Zoology, University of Western Ontario, London, Ontario.*

Quantitative analysis of intramembrane particle distribution in plasma membranes of dystrophic hamster cardiomyopathy. *K.A. Graham, R.R. Shivers and B.G. Atkinson. Cell Science Laboratories, Department of Zoology, University of Western Ontario, London, Ontario.*

Phenotypic alterations of tonic muscles innervated by nerve of twitch muscles in dystrophic chickens. *A.C. Gandy, E. Cosmos, and J. Butler. McMaster University, Health Sciences Center, Hamilton, Ontario.*

Perturbations in the protein synthesis activity of polysomes from dystrophic hamsters. *Léa Brakier-Gingras and Christine Jolicoeur, Department de Biochimie, Université de Montréal.*

Acetylcholinesterase (AChE) isoenzymes in murine muscular dystrophy. *Michael L. Lindenbaum and Bruce G. Livett, The Montreal General Hospital and McGill University, Montreal, Quebec.*

Molecular forms and cytochemical distribution of acetylcholinesterase (AChE) in skeletal muscles of the 129/ReJ dystrophic mouse. *V. Gisiger and H. Stephens, Department of Anatomy, Université de Montréal.*

The transport of 3-0-methyl-D-glucose (3-0-MG) and the accumulation of 2-deoxyglucose (2-DG) in normal and dystrophic hamster muscle. *J. Elbrink and B.A. Phipps, Department of Pharmacology, University of Alberta, Edmonton.*

SESSION III: Properties of normal and atrophic muscles.

Chairmen: *E. Cosmos, R. Stein.*

What limits the rise and fall of mammalian muscle contractions? *R.B. Stein, T. Gordon and J. Shriver. Departments of Physiology, Pharmacology and Biochemistry, University of Alberta, Edmonton, Alberta.*

The interactions of skeletal muscle, smooth muscle and non-muscle tropomyosins with F-actin and skeletal muscle troponin and its components. *G.P. Côté, A.S. Mak, J.R. Pearlstone, C. Sanders and L.B. Smillie. MRC Group in Protein Structure and Function, Department of Biochemistry, University of Alberta, Edmonton, Alberta.*

Calcium uptake by denervated muscle. *Roland Boegman and Kee Wan, Department of Pharmacology, Queen's University, Kingston, Ontario.*

Effect of the inhibition of spontaneous contractions of cultured skeletal muscle cells on the development of sarcoplasmic reticulum. *J.H.M. Charuk and P.C. Holland, Montreal Neurological Institute, McGill University, Montreal, Quebec.*

Contractile properties of denervated skeletal muscle. *D.M.S. Webster and B.H. Bressler, Department of Anatomy, University of British Columbia, Vancouver.*

Effect of nerve extract on atrophy of denervated muscles. *H.L. Davis, Department of Clinical Neurological Sciences, The University of Western Ontario, London, Ontario.*

Conversion of fast and slow fibers mediated by active muscle tension in developing lobsters. *C.K. Govind, Scarborough College, University of Toronto, West Hill, Ontario.*

Chick-quail chimaeras as models for *ex ovo* muscle differentiation. *B. Beresford, J. Butler, E. Cosmos and J. Bienenstock. Departments of Pathology and Neurosciences, McMaster University, H.S.C., Hamilton, Ontario.*

Expression of disease in mdg ↔ + chimaeras. *Alan Peterson and David Cross, The Montreal General Hospital Research Institute, McGill University, Montreal, Quebec.*

Fibrillations in myopathies. *S. Kereshi, Alexandra, Stuart, A.J. McComas, McMaster University Medical Centre, Hamilton, Ontario.*

POSTERS

A calmodulin dependent protein kinase system from skeletal muscle sarcoplasmic reticulum. *David H. MacLennan, Kevin P. Campbell and Haruhiko Takisawa. Banting and Best Department of Medical Research, University of Toronto, C.H. Best Institute, Toronto, Ontario.*

The relation between axon, myelin and conduction velocity during atrophy of mammalian peripheral nerves. *J. Gillespie, R.B. Stein and T. Milner. Departments of Occupational Therapy and Physiology, University of Alberta, Edmonton, Alberta.*

Effects of ageing on neuromuscular function in man. *A.A. Vandervoort, A.J. McComas. McMaster University Medical Centre, Hamilton, Ontario.*

Experimental micropuncture injury of skeletal muscles. *Stirling Carpenter, George Karpati, Department of Neurology-Neurosurgery, McGill University, Montreal Neurological Institute, Montreal, P.Q.*

Post-transcriptional regulation of ribosomal protein synthesis in myogenesis. *F.A. Jacobs, K. Maundrell and B. Sells, Laboratories of Molecular Biology, Faculty of Medicine, Memorial University of Newfoundland.*

The effect of denervation on skeletal muscle mitochondria. *George Carlin and Roland Boegman, Department of Pharmacology, Queen's University, Kingston, Ontario.*

Passive electrical properties of embryonic skeletal muscle fibers of the chick. *J.A. Steele, Department of Physiology, University of Alberta, Edmonton, Alberta.*

Experimental approaches to the study of nerve influences on fast and slow muscle during early embryonogenesis in the chicken. *J.W. Bloom and E. Cosmos. McMaster University Health Sciences Centre, Hamilton, Ontario.*

Response of brachial muscles to heterotopic innervation during embryogenesis. *J. Butler, E. Cosmos and J. Brierley. McMaster University, Health Sciences Center, Hamilton, Ontario.*

Regulation of glucose transport in L6 muscle cells in culture. *A. Klip, W.J. Logan and G. Li. Department of Neurology, Research Institute, The Hospital for Sick Children, Toronto, Ontario.*

Structural comparison of two forms of rabbit muscle calsequestrin. *R.A.F. Reithmeier and B. Cozens, Department of Biochemistry, University of Alberta, Edmonton, Alberta.*

An investigation of fibre hyperplasia in biceps brachii in bodybuilders: a preliminary report. *D. MacDougall, D. Sale, J. Clifford and S. Alway. McMaster University, Hamilton, Ontario.*

Human ankle stiffness dynamics: effect of muscle fatigue. *I.W. Hunter, P.L. Weiss and R.E. Kearney. Biomedical Engineering Unit, Faculty of Medicine, McGill University, Montreal, Quebec.*

SESSION IV: Further studies on normal and diseased cells.

Chairmen: *P. Holland, R. Reithmeier.*

Abnormal immune response in murine muscular dystrophy. *Myron Szewczuk, Department of Microbiology and Immunology, Queen's University, Kingston, Ontario.*

Giant axonal neuropathy: an inborn error of organization of intermediate filaments. *S.D.J. Pena, Department of Neurology and Neurosurgery, McGill University and Montreal Neurological Institute, Montreal, Quebec.*

The neurotoxin 2,5-hexanedione induces aggregation of intermediate filaments in cultured human skin fibroblasts. *H.D. Durham and S.D.J. Pena, Department of Neurology and Neurosurgery, McGill University and Montreal Neurological Institute, Montreal, P.Q.*

Lectins as probes for the study of cell surface glycoproteins of normal and dystrophic fibroblasts. *B.B. Gordon, C. Guerin and S.D.J. Pena, Department of Neurology and Neurosurgery, McGill University and Montreal Neurological Institute, Montreal, P.Q.*

Lipid vesicle-mediated alteration of membrane cholesterol levels: effects on Na^+ and K^+ currents in squid axon. *Mark Poznansky[1], Joy Steele[1], Doug Eaton[2] and Malcolm Brodwick[2], Marine Biological Laboratory, Woods Hole, MA. and [1]Department of Physiology, University of Alberta, Edmonton, [2]Department of Physiology and Biophysics, University of Texas Medical Branch, Galveston.*

The influence of calcium and other ions on muscle amino acid transport. *W.J. Logan, A. Klip and E. Gagalang, Division of Neurology, Department of Paediatrics, University of Toronto and Hospital for Sick Children, Toronto, Ontario.*

Study of the further heterogeneity of the MM isozyme of creatine kinase. *Benjamin G. Guslits and Hans K. Jacobs, University of Manitoba, Faculty of Medicine, Department of Biochemistry, Winnipeg, Manitoba.*

Study of duchenne muscular dystrophy (DMD) at the nucleic acid level. *C. Kreis, J.L. Hamerton and K. Wrogemann. Departments of Biochemistry and Pediatrics (Genetics), University of Manitoba, Winnipeg, Manitoba.*

Cytoskeletal elements in normal and dystrophic hamster fibroblasts. *M. Pollock, R.R. Shivers and B.G. Atkinson. Cell Science Laboratories, Dept. of Zoology, University of Western Ontario, London, Ontario.*

GUEST LECTURE: New aspects of the pathogenesis and treatments of neuromuscular diseases. *Dr. K. Engel, University of Southern California, Los Angeles, CA. U.S.A. Chairman: G. Karpati.*

POSTERS

Activation of specific set of genes in muscle cells following physiological stress using heat shock treatment. *J. Bag, Faculty of Medicine, Memorial University of Newfoundland, St. John's, Newfoundland.*

Immunochemical studies of plasma membrane proteins in cultured chick skeletal muscles cells. *G.A. Cates and P.C. Holland, Department of Biochemistry, University of Western Ontario and Montreal Neurological Institute, McGill University, Montreal, P.Q.*

Two calcium currents in egg cells. *A.P. Fox, Department of Physiology, UCLA, CA. U.S.A.*

Calcium-dependent neutral proteinase in hamster cardiac and skeletal muscle. *John S. Elce and Renate Barth. Department of Biochemistry, Queen's University, Kingston, Ontario.*

Characterization of a cyclic AMP-independent protein kinase of embryonic chicken muscle that binds to messenger ribonucleic acids. *B.H. Sells, A. Hudson and J. Bag, Faculty of Medicine, Memorial University of Newfoundland, St. John's, Newfoundland.*

Thymic alterations in genetically dystrophic mice. *C.E. Slonecker, W.K. Ovalle, B.H. Bressler, L.G. Jasch, Department of Anatomy, University of British Columbia, Vancouver.*

The effect of heat shock on gene expression in fibroblasts from normal, carrier and dystrophic mice and hamsters. *M. Pollock, B.G. Atkinson and R.R. Shivers. Cell Science Laboratories, Department of Zoology, University of Western Ontario, London.*

Sexual dimorphism in the cellular immune response of dystrophic mice. *S. Colby-Germinario, W. Bindon and B.G. Livett, The Montreal General Hospital and McGill University, Montreal, Quebec.*

Effect of two new thiol protease inhibitors on the development of heart necrotic changes in polymyopathic hamsters. *G. Jasmin and L. Proschek, Department de pathologie, Faculté de médecine, Université de Montréal, Montréal.*

Initiation of translation in control and dystrophic mice. *D.M. Nicholls and K. Nickson, Department of Biology, York University, Downsview, Ontario.*

Lectins as cytochemical probes for visualization of membrane oligosaccharides in human muscle. *S.D.J. Pena, B.B. Gordon, G. Karpati and S. Carpenter, Department of Neurology and Neurosurgery, McGill University and Montreal Neurological Institute, Montreal, Quebec.*

Myogenesis is defective in concavavalin A-resistant myoblasts. *C.L.J. Parfett, J.C. Jamieson, J.A. Wright, Department of Microbiology, University of Manitoba, Winnipeg, Manitoba.*

Colloidal gold immunolabeling of whole-mount cytoskeletons: tubulin and HMW-MAPS. *Mark E. Stearns and Robert L. Ochs, Department of Molecular, Cellular and Developmental Biology, University of Colorado, Boulder, Colorado.*