DOCUMENT NUMBER

Q-94-109A

STATUS

THIS DOCUMENT IS CLASSIFIED COSMIC-**MAGIC** UNDER AUTHORITY OF TOP SECRET EXECUTIVE DECISION 91-1812-4A

CLEARANCE

MAGIC

AUTHORITY

Written under authorization from the Government of the UNITED STATES OF AMERICA, DEPARTMENT OF DEFENSE, DEPARTMENT OF THE NAVY, Naval Intelligence Command/Naval Space Command.

RELEVANT SCOPE

This document is routed to the appropriate **MAGIC**-level directorate authorities of the Naval Space Command, pursuant to UNOST (adopted 19 December 1966; enforced 10 October 1967). The final routing has been approved to Razor Back, by direction of 92-NSC-117. The contents of this document are to be regarded as a final report (spec. K-24) of the Principal Investigator, Working Group Leader (R-4800, Occupant Papoose Site 4), as determined by his Commanding Officer, Cmd. John Anthony McGuinness, M.D. (U.S.N., N.S.A.), under final routing to R. Adm. J. McConnell. (U.S.N., N.S.A., MJ-Cosmic) for disbursement.

ORDER

Directed by the N.R.L.: "Determine, to scientific certainty, the reasons for *in vivo* neuronal repair failure, at dendritic terminal ends, from a set of cellular samples, *in vitro*. Classify such reasons, functionally, to ascertain the mechanisms of such failure, then isolate the most probable pre-existing cellular conditions giving allowance to proper regeneration."

SPECIFIC SCOPE

This results of *in vitro* experimentation under NSA/NSC directed Project Aquarius (Subintegument Neuronal Aspirative Avulsion Sampling Subsection King-24 {K-24}, Extraterrestrial Biological Entity {EBE} A.K. "AQ-J-Rod" {JR}) are herein related.

COURSE OF ACTION

GENERAL: As cellular reclassification was necessary, dated by previous testing of culture material from an "...unknown origin...", the following methodology was employed. From Tuesday, 19 July 1994 (22.00 hrs. U.T.) to Wednesday, 25 September 1996 (00:00 hrs. U.T.) 275 individual aspirative (16mm x 4mm, 0.042 μPsc pressurized stick) samples were removed (at C/Sphere S.T.P.) from the right upper-appendage of JR, 6.500 cm to 6.850 cm dorsocentral to the medial supinator longus-analog musculature, located 1.610mm into (adducted) the sinuous musculo-spiral-to-posterior interosseous-analog neuronal supraflexure, and 1.500cm (abducted) along the median supinator brevis-analog. Such methodology required the introduction of the Principal Investigator into the Pressurized Clean Sphere, which constituted an I.G.A.-declared "Extraterrestrial Close Encounter (E.C.E.), Class IV.c.". Protocols for the debriefing of the Principal Investigator were followed (Document Number unknown to the writer). Resultant samples were imaged, labeled, and transferred to C/Sphere S.T.P. elation tubes for analysis.

INITIAL SAMPLE STABILIZATION/PROPAGATION: Cell-Tissue-Culture (C.T.C.) Teams were deployed to plate and propagate biopsy material, under direction of Cpt. Jonathan Fisher, M.D., Ph.D. (U.S.N., N.R.L.). The subdivided samples (1-100 per each stick) were designated as K-24-1 through K-24-100, respectively. Each fraction, specialized analysis, were given subsplit labels K-24-1-a,b,c,..."n" through K-24-100-a,b,c,..."n", respectively. The plating activities were entirely successful at C/Sphere S.T.P., save K-24-16, K-24-36, and K-24-81. Heightened osmotic pressure from excess colloidal suspension was interpreted as the reason for cellular stasis and death. Equal Combinations of pressurized dictostelium-desoxycholate agar media, racemic to 10.000/100.000 (%) glucose-acetate medium produced the highest cellular, fibrilar, growth rates (1.000 generations per 26.302 days). Nominal contamination-restriction protocols were followed (see: Cross contamination protocol 6, N.R.L., Document Number Q-93-016B., for procedural guidelines).

VIEWABLE PREPARATIONS: Cellular preparations were presented by the "E.B.E.-Cross-Contamination and Viewing Protocols (Sections 1-4)", as approved, 2 April 1993 (N.R.L., Papoose Site 4, Document Number Q-93-016C.).

CELLULAR SUBFRACTION ANALYSIS: See Document Number Q-94-109B.

AMBIENT BIOCHEMISTRY: See Document Number Q-94-109C.

SUBFRACTION BIOCHEMISTRY: See Document Number Q-94-109D.

CONCLUSIONS/INTERPRETATIONS

As the conclusions, herein given, are directly interrelated with Document Numbers Q-94-109B to Q-94-109D, inclusive, the writer suggests that each be viewed *in toto*. The previous analysis (k{lower-case}-24), of then "...cells of an unknown origin..." yielded presumptive declarations that such specimens were operating under different methodologies than known terrestrial counterparts. That study also attempted the same process of "back-engineering" the cellular constituents, but yielded little more information than the basic cellular morphology, organelle analogs, membrane communication patterns, and pathways of cellular constituent biosynthesis. Study K{upper-case}-24 was deemed necessary, by the authority structure, for the purpose of handling aspirations of a fresher nature, whose *in situ* value would be known, and wherein a clear purpose to the investigation would be open to the operating group under the writer's control. The order was interpreted, clearly, as a study in the classification of neuronal dendritic repair failure, the establishment of the probable mechanisms of such failure, and the ascertaining of possible previous conditions (conditions that would necessarily have been present for acceptable evolution) when such repairs would have been successful. Additionally, the operating group took the opportunity to "back-engineer", together with theoretical applications, a potential route of ultimate repair of the dysfunctional system.

This report declares that the present operating group fulfilled the aforementioned order, and was also successful in positing the most likely route of genotype repair, a pathway completely reparative to the present phenotypic status. While the writer must present the findings, it must also be stated that the operating group members have made a unanimous declaration that certain methods to alter the present genotype should not be carried out. This position of conviction is held for moral and bio-ethical considerations, as such actions may deter from the normal process of natural selection in the human species.

Initial observations of extraterrestrial microglial-like-analog neuroblasts {?}(MENB's) revealed an approximated morphology to all such extraterrestrial cells, that being hypotrophic perikaryons (as opposed to terrestrial counterparts), with a mutipolar-type generalization. The presence of an external and internal cytoplasm, specifically herein defined as the neuroplasm (as previously described in k{lower case}-24), was again presented, with the organelle fusion being also demonstrated. The neurofibrils were present, with a 1-to-1 terrestrial relationship of

neuroprotofibrils (c. 0.009 mm). It occurred to the investigator that the neuroprotofibrils were selectively anastomosed to nissl-like bodies, extending afferently from the nuclear material, through the internal cytoplasm, then further cementing to the exterior of the internal cytoplasm (the "ground substance" of the external cytoplasm); however, such processes continued along primary axon units, but terminated at the primary-axonic-dendritic-process juncture (where the axoplasm sufficiently thinned allowing branching dendritc processes). This finding led the investigation toward its ultimate conclusions.

At the point of neuroprotofibril excision, the analog to the Incisure of Schmidt-Lanterman, Neurokeratin-like networks, and solid Endoneurium, cease. Cross culturing revealed that selective culture necrosis was not the origin. Further, such early terminations were found at highest rates (38 hits per 50 units at 25,000 diameters magnification), when adjacent to higher numbers of fibroblast-analogs, within the endoneurium. This correlation extended to myroneural junction regions, with a near 1-to-1 correspondence. Histologically, each myoneural junction viewed, demonstrated excessive filament depletion at the nominal axolemmal ridges, with high concentrations of mitochondrial-golgi-analog(s) (MG) at/near each synaptic trough's Basal Lamina, along the sides of each subneural cleft. This demonstration drew a conclusion of a pathological process that may associate myocyte physiology, fibroblast-analog response and/or mediation, membrane interractions, and axoplasm response.

A detailed analysis of membrane activity was conducted, via freeze fractures (STM), separative biochemistry (UCFG/MOA/MP), and selective histopathological supravital staining (LM). [*See attached coding for machinery references.*] The results indicated that the hydrogen mediated phosphorylation, as was presented in a Danielli model in previous reference k{lower case}-24, occurred at higher rates of successful energy budgeting (5.000%, average, power) at those areas where subneural clefts were shortest. Further, the highest concentrations of mitochondrial-golgi-analogs were found at the shortest of such clefts. This necessary proximity was found at each analysis, and was therefore determined as part of the pathological process. External membrane structure, showed ion channeling at less concentrations, where the myoneural junctions met the above criteria for pathology. Present channel varieties were bordered by long chain (via MP/HPLC/GC/MS) glycoproteins [IgA equivalent at IUPAC tertiary top chain representation- NeuAc(a(2-6)...], later coded (via gel electrophoresis and PCR) specifically to expressions from the Major Histocompatability Complex (MCH) at locus HLA-Cw3(a), and seemingly selective to those ion channels that disfavor membrane disequilibria, thus lack of net polarity, and where protodesmosomes from the fibroblast-analogs communicated to the sides of the subneural cleft areas. That fibroblast-protodesmosomal-analog association has not been entirely explained.

It appears that the protonated phosphorylation complex, within the MG-analog, operating (as best known) is rather more interwoven with voltage gated membrane channels than previously thought. A classification analysis was competed (via MP) in order to verify the mode of regulation, the results of which demonstrated that the cristae-compartmentalized electron transport system (bioregulatory parallel capacitors), operated in a triad of spherical cristae, generating and temporarily preserving at 7.100 X 10exp(-12)pJ per cycle. This was accomplished by reflux of hydrogen, via the MG-associated Phosphoenylpyruvate Phosphotransferase-analog (PEP-analog) active pump, and using glucose-6-phosphate as a carrier, also found preserving energy within the system as X=2W/lexp(2); therefore inductance (a bioregulatory solenoid). The minimal output of this system (passive exothermic emission), within the cellular matrix under study, enabled G-proteins to modulate voltage-gated calcium channels, and simultaneously, internal ligand and kinase modulated varieties to act in antagonism. It was the localized effect of this antagonism that interrupted the potential sufficiently to begin collateral elimination of synapses (following complete disruption of the excitatory potential, at -75mV, and with K+ efflux), through progressive acidosis, secondary to the PEP-analog's output. This was the agent of neuropathogenicity. Correlating to increasing age, from interview with JR (Sigma authorized), higher rates of neuropathy are found. Additionally, gene mapping has postulated a correlation in the age-dependent expression of the IgA equivalent to the organism-wide

efficiency of receptor tyrosine kinases (Q-94-109C/D). From that line of evidence, repair processes were found altered, via translational control inhibition at pp90exp(rsk)-analog. Simply put, repair was faulted, via increasing age, by insufficient specific protein kinase levels.

Attempts to rectify the problem, via allogenic recombination, resulted in an allomeric response. The neuropathy continued. Human Subject #58-001 (refer to autopsy Document Q-96-029) supplied bone marrow for sequential plasmid recombinations via electroporation. Sequential addition of expression loci for pp44superscript(mapk/erk2) yielded a theorized alternate pathway, via pp70superscript(S6K) kinase, to translational control through S6 phosphorylation. Transplantation of such cell matrix inocula resulted in attenuation of the neuropathy, not localized, but over a considerably wide area (2 cu. mm inoculum to 100 sq. mm resolution). Under order from the investigator's Commanding Officer, transgenic inocula, resulting from liposomal fusions, were attempted using secondary spermatocyte stock, with the same degree of success, however; the mechanisms of that result remain unknown. Such lines of investigation, with a clear "cross breeding" intent, should be followed with the greatest concern and suggested "hesitation", as the leakage of such success could promote a 'wild' contaminant species to further the experimentation in an unabated fashion. The ultimate results of such a possible genetic introduction into the human population could be catastrophic.

This report is respectfully presented for consideration.

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Working Goup Leader, Project Aquarius, R-4800, Papoose Site 4

Dictated to:

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