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EFFECTS OF EEG-BASED ACTIVE ASSISTED NEUROFEEDBACK THERAPY ON HEMIPLEGIC UPPER EXTREMITY MOTOR FUNCTION

Joo-Hee Park and **Hye-Seon, Jeon**
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The purpose of this thesis was to investigate the effect of EEG-based active assisted neurofeedback therapy (AANT) on stroke patients to improve both their upper extremity functionality and brain activity. Twenty chronic hemiplegic patients were included in this study. The subjects were assigned to two groups (10 per group), the control, which received only physical therapy and the experimental, which additionally received AANT. Subjects in both groups underwent routine physical rehabilitation, involving 30 minutes of exercise, 3 times/week for 4 weeks. Subjects in the experimental group performed an active assisted wrist extension exercise, which was combined with EEG neurofeedback. AANT was performed for 1 hour, 3 times/week for 4 weeks. Specifically, the subjects were asked to try extending their wrist and finger while looking at a monitor, which depicted the magnitude of real-time mu rhythm from the EEG. After an obvious voluntary suppression of the mu rhythm was achieved with the initiation of the wrist/finger extension, a physical therapist assisted the participant to attain full wrist and finger extension. The outcome variables of pre- and post- treatment evaluation included the EEG mu rhythm We found that the electromyogram (EMG) activity and upper extremity Fugl-Meyer Assessment (FMA) score were significantly increased in patients of the experimental than in those of the control group. In addition, there was a significant increase in brain activity of the affected (contralateral) sensorimotor area (SMA) in the experimental, but not in the control group. Spasticity, on the other hand, was significantly decreased in the experimental, but not in the control group. According to the results of this experiment, AANT improved brain activity in the affected SMA as well as upper extremity functionality in stroke patients. Therefore, we suggest neurofeedback therapy combined with proper physical guidance, as a promising treatment option for chronic stroke patients.

Note:

BIOGRAPHY

JooHee Park has completed her doctoral degree at the age of 31 years from Yonsei University and post-doctoral course from Yonsei University School of Physical Therapy. HyeSeon Jeon is professor of Yonsei University.

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EPITOPE FINGERPRINTING FOR RECOGNITION OF THE POLYCLONAL SERUM AUTOANTIBODIES OF ALZHEIMER'S DISEASE

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Autoantibodies (aAb) associated with Alzheimer's disease (AD) have not been sufficiently characterized and their exact involvement is undefined. The use of information technology and computerized analysis with phage display technology was used, in the present research, to map the epitope of putative self-antigens in AD patients. A 12-mer random peptide library, displayed on M13 phages, was screened using IgG from AD patients with two repetitions. Seventy-one peptides were isolated; however, only 10 were positive using the Elisa assay technique (Elisa Index >1). The results showed that the epitope regions of the immunoreactive peptides, identified by phage display analysis, were on the exposed surfaces of the proteins. The putative antigens MAST1, Enah, MAO-A, X11/MINT1, HGF, SNX14, ARHGAP 11A, APC, and CENTG3, which have been associated with AD or have functions in neural tissue, may indicate possible therapeutic targets.

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CLINICAL TRIALS OF CELL THERAPIES IN NEUROMUSCULAR DISEASES

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Cell therapy is an experimental strategy for a potential treatment of genetic and / or degenerative muscular disorders, among which the most important target is Duchenne muscular dystrophy (DMD). To be used in this approach, the cells must be myogenic, that is, they must have at least one of the following properties: (1) fuse with the patient's myofibers to induce the expression of therapeutic proteins in them, (2) form new myofibers, and / or (3) give rise to new muscle specific stem cells (satellite cells). Considering reports of experiments conducted on mice and dogs, the repertoire of cells exhibiting some of the myogenic capacities seems to have expanded in recent years. Among these cells we have: CD56+ muscle-derived cells, muscle-derived stem cells, CD133+ cells, mesoangioblasts / pericytes, myoendothelial cells, ALDH+ cells, PW1+/Pax7– interstitial cells, and ß-4-integrin+ cells. The clinical studies of cell therapy conducted so far showed that, of the four cell types transplanted in patients with DMD, namely CD56+ muscle-derived cells, bone marrow derived cells, CD133+ cells and mesoangioblasts, the only for which there were observed myogenic properties in the clinics were CD56+ muscle-derived cells, that is, satellite cell derived myoblasts. In a clinical trial, we allotransplanted CD56+ muscle-derived cells in 1 cm3 of muscle in 9 patients with DMD immunosuppressed with tacrolimus. Four weeks later, we observed restoration of the therapeutic protein (dystrophin) in 3.5% to 26% myofibers. Evidences of small myofiber neoformation and of potentially graft-derived satellite cells were also observed. A 26-years old DMD patient also received cell allotransplantations under tacrolimus immunosuppression in different muscles, restoring dytrophin in 27.5% of myofibers at 1 month and in 34.5% at 18 months. This patient evidenced that our protocol was feasible in large muscles of humans and that long-term expression of donor-derived dystrophin can be obtained under proper immunosuppression. Further improvements are desirable for efficient clinical applications of this strategy and we are currently working on it.

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DEMENTIA OF ALZHEIMER'S TYPE AMONG ARAB POPULATIONS: GENETICS AND EPIDEMIOLOGICAL STUDIES

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Introduction: Neurodegenerative disorders, Primarily, are multifactorial diseases characterized by chronic and progressive loss of neurons in discrete areas of the brain, causing debilitating symptoms and globally decreasing cognitive function such as dementia, loss of memory, loss of sensory or motor capability, decreased overall quality of life and well-being, disability, and eventually, premature death.

Objective: To study the genetic and environmental risk factors and the prevalence of dementia of the Alzheimer type (DAT) among the elderly in an Arab community in Israel.

Material and Methods: Epidemiological and genetic studies of dementia have rarely been reported in an Arab population. Alzheimer disease (AD [MIM #104300]) is a progressive, neurodegenerative disease characterized clinically by gradual loss of memory and pathologically by neurofibrillary tangles and amyloid plaques in the brain. We have observed an unusually high prevalence of dementia of the Alzheimer type (DAT) in Wadi Ara, an inbred Arab community in northern Israel comprising 850 persons over the age of 60 years. Apolipoprotein E (APOE- ε4), has been established as a strong susceptibility marker that accounts for nearly 30% of the risk in late-onset AD.

Results: Remarkably, in our study DAT is not associated with APOE because the frequency of the 4 allele is very low in both nondemented (2.4%) and demented elders (3.6%). We also map chromosomal loci contributing to DAT susceptibility; we conducted a 10 cM scan in a series of twenty cases and twenty controls selected from one hamula. Markers from 18 chromosomal regions showed significant allelic association with DAT ($P<0.05$). Locations on chromosomes 2, 9 and 10 remained significant after testing additional affected and non-demented individuals. Significant associations were also observed for markers on chromosome 12, which overlap with a locus implicated in previous genome scans. Additionally, several lines of evidence support for a role of angiotensin converting enzyme (ACE) in Alzheimer disease (AD). Most genetic studies have focused on an Alu insertion/deletion (I/D) polymorphism in the ACE gene (DCP1) and have yielded conflicting results. We evaluated the association between 15 (SNPs) in DCP1, including the I/D variant, and AD in a sample of 92 patients with AD and 166 non-demented controls from an inbred Israeli Arab community.Although there was no evidence for association between AD and I/D, we observed significant association with SNPs rs4343 ($P= .00001$) and rs4351 ($P=.01$).

Conclusion: In Wadi Ara the high prevalence may be due to a founder effect enhanced by consanguinity, which make this population attractive for investigating DAT susceptibility recessive genes; thus, a specific disease susceptibility allele may be overrepresented in cognitively impaired subjects compared with cognitively healthy residents. Other two main conclusions can be drawn from the genome-wide linkage and linkage disequilibrium (LD) studies. Firstly, multiple genes are involved in DAT. Secondly, there is a high level of consistency among linkage and association studies regarding the general location of putative AD genes. However, the general location of putative AD genes on a given chromosome covers a broad region, which may contain several genes.

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THE STORY OF GLATIRAMER ACETATE (COPAXONE) IN THE TREATMENT OF MULTIPLE SCLEROSIS - THE POTENTIAL FOR NEUROPROTECTION BY IMMUNOMODULATORY TREATMENT

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Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory autoimmune reactivity in the central nervous system (CNS) results in demyelination, axonal and neuronal pathology. Treatment strategies thus aim to reduce the detrimental inflammation and induce neuroprotective repair processes.The synthetic copolymer Copaxone (glatiramer acetate, GA), an approved drug for the treatment of MS, is the first and so far the only therapeutic agent to have a copolymer as its active ingredient. Using the animal model of MS - experimental autoimmune encephalomyelitis (EAE), the mechanism of action of GA was elucidated. These studies indicated that GA treatment generates immunomodulatory shift from the inflammatory towards the anti-inflammatory pathways, such as Th2-cells that cross the blood brain barrier (BBB) and secrete *in situ* anti-inflammatory cytokines, as well as T-regulatory cells (Tregs) that suppress the disease. The consequences of GA treatment on the CNS injury inflicted by the disease were studied using immunohistochemistry, electron microscopy, and magnetic resonance imaging. These analyses revealed reduced demyelination and neuro-axonal damages, as well as neuroprotective repair processes such as neurotrophic factors secretion, remyelination and neurogenesis.These combined findings indicate that immunomodulatory treatment can counteract the neurodegenerative disease course, supporting linkage between immunomodulation, neuroprotection and therapeutic activity in the CNS.

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PHASE I/IIA TRIALS IN GBM PATIENTS USING A NEW DRUG (CEREBRACA WAFER) TARGETING AXL RECEPTOR TYROSINE KINASE

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Malignant brain glioma is a highly invasive disease with a very high death rate. The effective treatment method for this disease is still an unmet medical need. In our previous reports, a pure compound EF-001 showed activities to arrest the growth and initiate apoptosis of malignant brain glioma. To overcome the limitation of the blood-brain barrier, a local-release system with EF-001 incorporated into a biodegradable polyanhydride material, p(CPP-SA), was developed and named "Cerebraca Wafer". Both the *in vitro* and the *in vivo* release kinetics of the Cerebraca Wafers have been characterized. The *in situ* therapeutic effects of the Cerebraca Wafer on brain gliomas were demonstrated by FGF-SV40 transgenic mice and orthotopic brain tumor F344 rat models. Significant effects on the inhibition of tumor growth and the increase of survival rate by Cerebraca Wafer implantations were observed, with no significant adverse effects on the rodents. Mechanisms involved in the antitumor effect of EF-001, downstream of AXL receptor tyrosine kinase, including the up-regulation of Nurr77 by PKC, the repression of human telomerase reverse transcriptase (hTERT) transcriptional activity via down-regulating Sp1 expression, and the down-regulation of the S-phase kinase-associated protein 2 (Skp2) which resulted in the brain tumor senescence, were also investigated in the study. The toxicity studies and the PIC/s GMP grade Cerebraca Wafer production for clinical trials were accomplished. IND (investigational New Drug) application for the Phase I/IIa clinical trials aiming to determine the safety and the efficacy of Cerebraca Wafer implantation on the recurrent GBM patients have been approved by USFDA and TFDA at 2016. The trial is now being performed at Hualien Tzu-Chi hospital and Tri-Service General hospital, with the first Wafer implantation surgery completed at the end of 2017. The preliminary results for the first three trial patients will be presented and discussed.

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PERSONALIZED AND PRECISION ONCOLOGY THROUGH THE VIEW OF TRANSLATIONAL APPLICATIONS AND INNOVATIVE TOOLS TO MANAGE CANCER PROGRESSION

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A new systems approach to diseased states and wellness result in a new branch in the healthcare services, namely, personalized and precision medicine (PPM) to stimulate the development in a variety of clinical disciplines including Personalized and Precision Oncology (PPO). To achieve the implementation of PPO concept into the daily practice, it is necessary to create a fundamentally new strategy based upon the subclinical recognition of cancer-associated bioindicators (biopredictors and biomarkers) of pre-cancer abnormalities long before the disease clinically manifests itself. Each decision-maker values the impact of their decision to use PPO on their own budget and well-being, which may not necessarily be optimal for society as a whole. It would be extremely useful to integrate data harvesting from different databanks for applications such as prediction and personalization of further treatment to thus provide more tailored measures for the cancer patients and persons-at-risk resulting in improved outcomes whilst securing the healthy state and wellness, reduced adverse events, and more cost effective use of health care resources. Rapidly improving understanding of PPO, emerging novel therapeutics, and increasingly available and affordable next-generation sequencing have created an opportunity for delivering genomically informed personalized cancer therapy. Alterations that are targetable either directly or indirectly with approved or investigational therapies are potentially "actionable". At this time, evidence linking predictive biomarkers to therapies is strong for only a few genomic markers in the context of specific cancer types. Deciding what therapy options to pursue can also be daunting, especially when tumors harbor more than one potentially actionable aberration. Further, different mutations/variants in a single gene may have different functional consequences, and response to targeted agents may be context dependent. However, early clinical trials with new molecular entities are increasingly conducted in a biomarker-selected fashion, and even when trials are not biomarker-selected, much effort is placed on enrolling patients onto clinical trials where they have the highest probability of response. Implementation of PPO requires a lot before the current model "oncologist-cancer patient" could be gradually displaced by a new model "medical advisor-healthy person-at-risk". This is the reason for developing global scientific, clinical, social, and educational projects in the area of PPO to elicit the content of the new branch. Recognizing the need to define the policies required for sustained innovation in cancer research and care in an era of cost containment, the stakeholder community must engage in an ongoing dialogue and identify areas for collaboration.

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QUALITY OF LIFE, MOOD, SOCIAL SUPPORT, AND SPIRITUALITY AMONG BREAST CANCER SURVIVORS FROM DIFFERENT ETHNIC GROUPS

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While there is a large body of literature on psychological aspects of breast cancer few studies have been focused on differences between ethnic groups.

Method: A sample of 178 breast cancer survivors (45 African Americans, 52 Asian/Pacific Islanders, 54 Caucasian, and 25 Latinas) who were on average 2 years post-treatment were interviewed and given surveys to complete every six months for two years.

Results: Latinas had significantly lower appraisal support and depression than Caucasians. More African Americans and Latinas engaged in spiritual activities such as prayer. African Americans had more spiritual support than Caucasians or Latinas. Overall QOL at 4 years was predicted by previous physical and functional well-being, breast cancer-specific items, vigor, and current levels of social support. Physical QOL was predicted by previous levels of physical and functional well-being and current levels of functional and social/family well-being. Functional well-being was predicted by prior levels of physical, functional, social/family well-being, and current levels of physical well-being and vigor. Emotional well-being was predicted by previous levels of emotional well-being and current physical well-being, breast cancer-specific items, and anxiety. Social/family well-being was predicted by previous levels of social/family well-being, social support, and confusion. The breast cancer-specific items were predicted by age, previous levels of breast cancer-specific items, confusion, current levels of emotional and functional well-being, and spirituality.

Conclusions: Two years after the end of treatment difference were seen between ethnic groups on depression, appraisal, and spiritual support. Therefore, spirituality and spiritual support as well as social support be assessed at the beginning of cancer treatment. Quality of life should also be assessed over time, even after treatment.

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SKIN PRION AND ITS IMPLICATIONS IN PRION DISEASES AND OTHER NEURODEGENERATIVE DISEASES

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Prions (or PrP^{Sc}) are associated with a group of fatal transmissible prion diseases including sporadic Creutzfeldt-Jakob disease (sCJD, the most common human prion disease) in humans as well as scrapie, mad cow disease, and chronic wasting disease in animals. The currently incurable sCJD is transmissible, due to the contamination of abundant infectious prions in the brain through medical or surgical procedures. Some epidemiological studies have also associated sCJD risk with non-neurosurgeries, suggesting that prions may be present in other tissues such as skin. In addition, once disease onset has occurred, the brain becomes inevitably damaged. So, preclinical detection is key to providing the critical window for early treatments before irreversible brain damages occur once cures become available. Our recent study using the highly sensitive real-time quaking-induced conversion (RT-QuIC) assay and humanized transgenic (Tg) mice-based bioassay revealed that the skin of sCJD patients harbors infectious prions (Orrú et al., 2017). Moreover, our new preliminary results further indicate that skin PrPSc is detectable by RT-QuIC and serial protein misfolding cyclic amplification assays far ahead of neuropathological changes in prion-infected animal models. Our findings not only raise concerns about the potential for iatrogenic sCJD transmission via skin but also provide a basis for establishing alternative premortem and postmortem diagnostic assays for prion diseases. Moreover, they may improve our understanding of the role of other skin misfolded proteins in the diagnosis and pathogenesis of neurodegenerative diseases such as Alzheimer's and Parkinson's diseases in which disease-specific misfolded proteins have been detected in the skin of patients with these diseases. [Supported by the CJD Foundation, NIH (NS062787, NS087588 and NS096626), and CDC].

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AUTONOMIC FUNCTION ASSESSMENT IN PARKINSON'S PATIENTS WITH YOGA PRACTICING USING KERNEL METHOD AND ENTRAINMENT TECHNIQUES

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The experimental procedure of lowering and raising a leg Figure 1, while the subject in supine position is considered to stimulate and entrain the autonomic nervous system of fifteen patients with Parkinson's disease practicing Yoga and fifteen age and sex matched control Parkinson's disease non Yoga practicing patients . The assessment of autonomic function for each group is achieved using an algorithm based on Volterra kernel estimation. By applying this algorithm and considering the process of lowering and raising a leg as stimulus input and the Heart Rate Variability signal (HRV) as output for system identification, a mathematical model is expressed as integral equations. The integral equations are considered and fixed for Parkinson's Patients without Yoga practicing and Parkinson's Yoga practicing patients so that the identification method reduced to the determination of the values within the integral called kernels, resulting in an integral equations whose input-output behavior is nearly identical to that of the system in both Control Parkinson's without yoga practicing patients and Parkinson's Yoga practicing patients. The model for each group contains the linear part (first order kernel) and quadratic part (second order kernel). A difference equation model was employed to represent the system for both control Parkinson's patients without Yoga practicing and Parkinson's Yoga practicing patients. The results show significant difference in first order kernel (impulse response) and second order kernel (mesh diagram) for each group. Using first order kernel and second order kernel, it is possible to assess autonomic function qualitatively and quantitatively in both groups.

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NEURAL CORRELATES OF CARE SETTING IN A SAMPLE OF CHINESE CHILDREN DOUBLE ORPHANED BY AIDS

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Dr. Behen will present data from a set of studies focusing on the functional and structural neural correlates of differential care settings (orphanage, kinship care, community group homes) (NIH: 5R21HD087108-02) in children double orphaned by AIDS. The talk will highlight the behavioral and neural phenotypes associated with such early adversity and across care settings, and also predictors of such outcomes, especially focusing on timing and care setting parameters.

Objective: Studies investigating the effects of early social deprivation associated with institutional rearing reveal increased incidence of cognitive/behavioral problems and altered neural structure/function, raising concerns about the use of institutional settings (i.e., orphanages) in the care of orphaned children, and prompting study of alternative programs (i.e., foster care) for the care of such children. However, empirical scrutiny of neurodevelopmental outcomes across care settings (and timing/care setting parameters associated with outcomes) is critical before a global push to foster care is undertaken.

Method: We applied neurocognitive/behavioral assessments, and structural/functional MR imaging in 124 Chinese children double-orphaned by HIV/AIDS (mean age=14.7+SD=1.5 years), across three care settings (orphanage, community group home, kinship care), and two age groups (onset of adversity <3years, >8 years of age). Data analyses included between-group comparisons on cognitive/behavioral outcomes and structural/functional neural connectivities. Regression analyses were used to identify/determine relationships between duration in care and outcomes across settings, and whether relationships are moderated by age of onset of adversity.

Results: Analyses revealed increased incidence of cognitive/behavioral problems in children raised in orphanages and kinship care compared to those in community group homes. Further, orphanage rearing was associated with altered neural connectivities, especially involving frontal and temporal regions, compared to community group home. Outcomes were associated with duration in care (longer duration in orphanage was associated with poorer outcomes over time; care in group homes was associated with improved outcomes over time); findings were accenuated in children with onset of adversity before 3 years.

Conclusions: Community group care was associated with improved neurodevelopmental outcomes compared to orphanage care. Such outcomes appear to be strengthened over time in such settings, particularly in children with early onset of adversity. Such data may have important implications for policy for how growing numbers of children, worldwide, can be best cared for following early adversity.

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