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Education Posters

E-2000

The Effect of Beta Glucans on Ampicillin. C. CAPPIELLO Warwick High School, West Orange St., No 301. Email: christina.g.j.cappiello@gmail.com

It is of great importance to study the potentiating effects of Beta Glucans on Ampicillin. Beta Glucans could help those stricken with antibiotic resistance by improving the efficiency of the antibiotic without increasing the dosage. To measure the potentiating effects, Nutrient Broth innoculated with E.coli was prepared and divided into 7 flasks. To 6 of the flasks, a concentration of 5.0mg/L of Ampicillin was added, and to 5 of the flasks, varying concentrations of Beta Glucans were added. The concentrations of Beta Glucans were 105.0mg/L, 7.2mg/L, 5.1mg/L, 3.3mg/L, and 1.1mg/L. After inoculation, 70 agar plates with 10 1 microliter samples from each flask were incubated and average CFU's were counted. There were statistically significant differences between the group without Ampicillin or Beta Glucan and the test groups with both Beta Glucan and Ampicillin. The control group had 567,700 CFU/mL, while the test groups containing both Glucan and Ampicillin had anywhere from 1,200-3,600 CFU/mL. There was also a statistically significant difference between the group with just Ampicillin and the groups with both substances. The results show that the performance of Ampicillin was greatly increased when used in conjunction with Beta Glucans. Because of Ampicillin's increased efficiency, the amount of bacteria colonies was dramatically reduced.

E-2001

Nano Particles for Mitochondria-targeted Delivery of Inhibitory Deoxyribonucleotides (DNAs). JONATHAN CHEN, Alex Ditzel, and Michelle Liang. Northgate High School, 425 Castle Rock Road, Walnut Creek, CA 94598. Email: jjmaomi@gmail.com

Antisense oligonucleotides have long been demonstrated as specific and effective agents for replication, transcription and translation inhibition in vivo. In 1998, FDA approved the first antisense oligonucleotide therapeutics, Vitravene, for treating retinitis of AIDS patients (*Orr, R. M. Curr. Opin. Mol. Ther., 2001, 3*, 288 *–294; Roehr, B. J. Int. Assoc. Physicians AIDS Care, 1998, 4*, 14 *–16)*. However, oligonucleotides have been largely missing from the portfolio of therapies, because of their poor stability during circulation and low permeability across biological membranes. We'd like to develop a new delivery system for short nucleotides. Toward this goal, we synthesized a series of nanoparticles using poly(lactide-co-glycolide)-*b*-polyethyleneglycol (PLGA-PEG), a material that has been shown safe and biodegradable. Fluorescently labeled DNA oligonucleotides complementary to a unique DNA sequence in the mitochondria of breast cancer cells were packed into these nanoparticles. Particle size and DNA oligo encapsulation efficiency were optimized by the variation of mixing speed and solvent system. These positively charged PLGA nanoparticles are expected to enter cells through endosomes, and then penetrate the mitochondrial membrane because of a negative cross-membrane potential (Marrche, S. and Dhar, S. *Proc. Natl. Acad. Sci. U.S.A*. 2012, *109*, 16288-16293; Marrache, S. *et al. ACS Nano*, 2013, *7*, 7392–7402). The polymer capsules would shield oligonucleotides from endonuclease degradation and help them reach targeted cell compartment during this process. We will study the cell penetration efficiency of these fluorescent DNA-carrying nanoparitcles, and investigate their possible inhibitory effect on the growth of breast cancer cells.

E-2002

Preventing Cardiac Cell Death During Ischemia-reperfusion. M. GRABOWSKA[1] and J. J. Saucerman[2]. [1]Albemarle High School, Charlottesville, VA and [2]Dept. of Biomedical Engineering, University of Virginia, Charlottesville, VA. Email: monika.e.grabowska@gmail.com

Objectives: Ischemia-reperfusion (I/R) injury during heart attack may result in significant heart damage. As existing therapies to treat I/R injury are not effective, new treatments are needed. The goal of this study was to determine if antioxidants such as ascorbic acid protect cardiac cells from death in an in vitro model of hypoxia-reoxygenation, simulating the conditions of I/R in vivo. **Methods:** Cultured neonatal rat cardiac

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myocytes were exposed to 40 min mineral oil-induced hypoxia followed by two hours reperfusion with oxygenated media. Cell death, formation of reactive oxygen species (ROS), and spontaneous intracellular calcium (Ca^{2+}) oscillations were monitored using fluorescence microscopy. **Results:** Hypoxia impeded cell contractile function and Ca^{2+} oscillations, and generated low levels of ROS. After 40 min of oil-induced hypoxia, cell viability was reduced by 44 percent. Early reoxygenation was associated with a twofold increase in ROS formation and arrhythmic Ca^{2+} oscillations. After two hours of reoxygenation, over 80 percent of cells surviving hypoxia were dead. Antioxidants were tested to see if they could prevent cell death during hypoxia-reoxygenation. Adding ascorbic acid to the reoxygenation buffer significantly reduced ROS present in the system, increased cell survival to 60 percent, and restored cell contractility. Pre-incubation of cells with ascorbic acid also reduced cell death during hypoxia-reoxygenation. Treatment with enzymatic antioxidants glutathione (GSH) and N-acetyl cysteine (NAC), however, did not have any effect on cell survival in the system. **Conclusions:** This study suggests that both immediate application of and pre-incubation with ascorbic acid have protective effects on cardiac cells during hypoxia-reoxygenation. Ascorbic acid blocked ROS formation, substantially reduced cardiac cell death, and allowed the recovery of contractile function. These findings indicate that ROS contribute to I/R injury and that ascorbic acid protects cardiac cells by ROS scavenging.

E-2003

The Effect of Brain-derived Neurotrophic Factor on Retinoic Acid Differentiated SH-SY5Y Cells: A Model for Striatal-enriched Protein Tyrosine Phosphatase in Parkinson's Disease. ARCHETA RAJAGOPALAN. Choate Rosemary Hall, 333 Christian Street, Wallingford, CT 06492 and Lombroso Lab of Molecular Neuroscience, Yale Child Study Center, Yale University, New Haven, CT 06510. Email: arajagopalan15@choate.edu

Parkinson's disease (PD) is the second most common neurodegenerative disease, affecting four to six million of people worldwide. Characterized by the degeneration of dopaminergic neurons in the substantia nigra, PD commonly results in motor and cognitive deficits such as slurred speech and tremors. Striatal-enriched protein tyrosine phosphatase (STEP) is a tyrosine phosphatase enriched in the dopaminergic neurons of the substantia nigra. Recent studies indicate that STEP levels are up-regulated in PD, but its exact function in dopaminergic neurons is not known. To study the role of STEP in dopaminergic neurons in an in vitro model, a neuroblastoma (SH-SY5Y) cell line was differentiated to dopaminergic cells using retinoic acid (RA) and the expression of STEP and its substrates was examined. The results show that RA differentiated SH-SY5Y cells express STEP and its substrates pERK and pPyk2. This establishes the feasibility of using RA differentiated SH-SY5Y to study the effects of dopaminergic toxins that are related to PD on STEP and to find potential PD therapeutic molecules that modulate STEP activity. Further, because brain-derived neurotrophic factor (BDNF) has shown to reverse the neuronal death exhibited in PD, this research seeks to establish whether BDNF inhibits STEP to achieve such neuronal regrowth. To study this, STEP and STEP substrate levels were examined before and after BDNF treatment. The results reveal that BDNF affects STEP and STEP substrate levels, indicating a relationship between BDNF and STEP pathways. In conclusion, this research proves that RA differentiated SH-SY5Y cells can be used to further research on STEP in PD, and that BDNF potentially inhibits STEP and could be used as a component of future treatment of PD.