2006학년도 1학기

생명과학연구소보

生命科學研究所報

1호

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제 1호

Vol. 1
Institute of
Life Sciences

발행인 생명과학연구소
주소 강원도 춘천시 효자 1동 192-1번지,
강원대학교 미래관 7층 (200-160)
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강원대학교 생명과학연구소는 1980년 4월 본교에서 가장 처음으로 설립된 이과 계열의 연구소로써 본교는 물론 춘천 지역, 나아가 강원지역의 생명과학 발전에 오랫동안 기여하였습니다. 그 동안 많은 교수님들께서 연구소원으로 활동하신 덕에 연구소가 다양한 연구 및 학술활동을 직·간접적으로 지원할 수 있었습니다. 이제 연구소의 소식을 매학기 전하는 연구소보를 작성하여 당해 학기의 연구소 활동을 정리하고 개선할 점을 파악하여 연구소가 더욱 발전할 수 있도록 노력하고자 합니다. 이 연구소보에는 ① 연구소 운영위원회의 결정사항, ② 연구소의 학술활동 (세미나 및 심포지엄 현황), ③ 기타 연구소가 수행한 연구/학술활동에 대한 소식을 포함하였습니다. 연구소원으로 활동하시는 여러 교수님들의 지원과 관심에 감사합니다.

2006학년도 기간에 4회의 운영위원회의, 7회의 세미나, 그리고 2회의 심포지엄을 가졌습니다. 운영위원회의를 통하여 연구소의 활성을 위한 좋은 의견을 모았고, 내실이 있는 연구소로 발전하기 위하여 연구소원의 확인, 인센티브 제도의 정착 등을 결의하였습니다. 감사합니다.

운영위원회의 결과

주 ID

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보낸 날짜: 2006년 6월 20일 화요일 오후 1:51
제목: 운영위원회의 결과

선생님들께, 06. 6. 20.

오늘 총 일곱 분의 운영위원 선생님들을 모시고 운영위원회의를 하였으며, 그 회의에서 결정된 내용을 알려드립니다.

1. 운영위원 (가나다 순) : 김경훈 교수, 김근철 교수, 김병철 교수, 이한수 교수, 임창진 교수, 정두일 교수, 한상화 교수, 한장희 교수 (여덟 분)

2. 연구소 전용 사무실 문제 : 실사구시관 104호실의 연구소 전용구간은 2006학년도 2학기 학교에서 실시할 연구소 평가지침에 의하여 평가를 받은 후 공간의 활용 문제를 다시 논의한다.

오늘 회의에 참석하신 선생님들께 감사합니다.

최형태 드림

보낸 날짜: 2006년 8월 3일 목요일 오후 2:38
제목: 생명과학연구소 운영에 대한 안내 및 부탁 (최형태)

선생님들께, 06. 8. 3.

안녕하십니까? 지난 6월부터 생명과학연구소를 맡은 최형태 입니다. 지난 2004년 연구소 평가 때 보고한 자료의 연구소원 명단에 근거하여 이 메일을 보냅니다. 2006년 2학기 부터 연구소 운영에 대하여 최근 두 차례 운영위원회에서 결정된 것을 알려드리고 부탁말씀도 드리고자 합니다.

1. 2006년도 예산이 지난해에 비하여 귀속연구비가 많이 줄어 (1/2.5) 연구소 운영을 간소화 하고자 합니다.

2. 학기당 세미나 6회, 심포지엄 1회를 계획하고 있으며 세미나 연사료는 20/25/30 만원 (춘천권/강원 및 수도권/충남 및 기타의 연사)으로, **심포지엄은 1회 당 160만원 지원합니다. 단 심포지엄은 연구소가 주관기관이 되어야 합니다.**

3. 세미나 후 연사를 모시는 저녁은 연구소에서 (현행대로) 진행합니다. 그 이후는 자유시간입니다.

4. 이번 2학기 세미나는 9월부터 매 1, 3째 목요일로 계획하고 있습니다. 도중에 공휴일, 시험, 학회 등으로 겹칠 때에는 약간의 변동이 있습니다.
모든 연구소원들께서 세미나 연사를 추천해 주시기 바랍니다.

5. 내년에는 연구소 예산에 여유가 있도록, 즉 귀속연구비가 많이 오도록 도와 주시기 바랍니다.

* 올 해에는 심포지엄을 2학기에 2회 가능합니다. 또는 06년도 2학기에 1회, 07년도 1학기에 1회로 여는 것도 좋습니다.

* 세미나 연사 추천은 9월 연사는 8월 25일까지 그 후의 연사는 9월 22일까지 추천

하시면 고맙겠습니다.

* 심포지엄 개최 신청은 9월 말까지 저에게 해 주시면 취합하여 운영위원회를 통하여 결정하겠습니다.

기타 궁금한 내용은 저에게 문의하시기 바랍니다. (교내 8543)

최형태 드림

운영위원께, 06. 10. 10.

어제 운영위원회의 결과 (및 기타)를 정리하여 보냅니다.
- 11월 중순까지 작성할 연구소 평가를 위하여 연구소원들의 연구업적 update 부탁
- 연구원 및 운영위원 정리
- 연구소의 학술활동 활성화 (예: Biofestival 공동 참여 등 --> 바이오산업진흥원장에게 문의한 결과 11월 2,3일에 예정된 사업의 주관기관은 이미 결정되었다 함)
(그래서 별도의 공동사업을 추진하기로 구두 약속)
- 연구소 주관 심포지엄을 계획하시도록 권유
이상입니다.

최형태 드림

운영위원님들께, 06. 11. 30.

오늘 낮 운영위원회의에서 결정된 사항을 정리하여 보냅니다. 참고하시고, 연구소 활성화에 다양한 의견을 주시기 바랍니다.
- 연구소원 정리 및 확보: 연말에 소장이 기존 연구소원들에게 메일로 지속적인 활동 부탁, 동시에 생물공학과 교수 및 신임교수들을 연구소원으로 초빙 노력함
- 연구소 운영위원: 기존의 운영위원 외에 1-2명의 신임급 교수를 초빙하도록 노력
- 연구소 행정실 문제: 본부 및 자연대와 협의하여 행정실 확보 노력
- "생명공학연구소" 명칭 문제: 평의원실에 강력한 의견을 제시하기로 함
- 개교 60주년 기념 학술대회: 조만간 결정
- 학술심포지엄 개최 건: 분자생물학회/생화학회의 후원을 받아 연구소 주관으로 심포지엄 개최를 올해 안으로 최대한 노력하기로 함 (분야: 스트레스 적응, 또는 기타) 이상입니다.
최형태 드림

세미나 연사, 제목 및 초록

392회 (2006. 6. 1)

연사 : 김 은 희

School of Bioscience & Biotechnology, Chungnam National University, Daejeon

Abstract

Death promoting mechanism of Fas associated factor 1

This study presents death promoting mechanism of Fas associated factor 1 (FAF1). FAF1 induces cell death via extrinsic and intrinsic death pathways. FAF1 serves as a member of Fas-death-inducing signaling complex (Fas-DISC) and potentiates Fas-mediated apoptosis. FAF1 suppresses NF-B activation by IKK activity attenuation and cytoplasmic retention of NF-B-p65. Therefore, FAF1 promotes cell death in two aspects i.e. lowering cell's survival potential cells by supressing NF-B activity and potentiating apoptotic signal transduction as a member of Fas-DISC. FAF1 is also essential for the execution of ischemic cell death via caspase-independent cell death pathway (FAF1→ROS→PARP→AIF). Cell-based inhibitory compound screening identified a hit molecule (KR33196) targeting FAF1-mediated death pathway and its derivatives were synthesized and their efficacies were evaluated. KR-33196 demonstrated therapeutic potential as a novel anti-ischemic agent in MCAO model.

393회 (2006. 6.15)

연사 : 허 송 욱
Korea Basic Science Institute/Bio-imaging

Abstract

Molecular Approach - Stress (Psychotic Depression)

Adrenaline (epinephrine) and glucocorticoids are important neurohormone/ neurotransmitter released from the adrenal gland in response to physiological and environmental stress.It is known that stress, dysregulation of the HPA axis and activation of the stress hormones, epinephrine and glucocorticoids are critical contributors to disease entities, especially neuropsychiatric disorders. It is therefore important to understand these regulatory controls, in order to learn how aberrant gene function can lead to illness and disease.

The research strategy underlying these investigations involves four components. First, regulation of the PNMT gene will be studied. PNMT, phenylethanolamine N-methytransferase, is the epinephrine-synthesizing enzyme in the catecholamine synthesis. The second component of my research strategy involves characterizing the structure-function of glucocorticoid receptor (stress hormone receptor, GR). Special emphasis is placed on genetic alteration in the Guyanese squirrel monkey GR resulting in impaired transactivation and different RU486 response. Third, the pattern of gene expression is examined using the rat and further squirrel monkey as an animal model system for the human brain and psychiatric diseased human brain. In this study, GeneChip (Oligonucleotides array) is used to profile transcripts of stress and heterogeneity of regional brain. Based on the results of these last two lines of investigation, the fourth research strategy involves the generation of testable hypotheses regarding the gene that may be down/up-regulated by stress. These hypotheses are tested by investigating neurogenesis in hippocampal organotypic slice culture and animal model.

394회 (2006. 9 .7)

연사 : 이 정 형
강원대학교 생명과학부 생화학과

Abstract

Discovery and Mode of Action of NF-κB inhibitors from Natural Products.

NF-κB regulates the transcription of a large number of genes, particularly those involved in immune, inflammatory, and antiapoptotic responses. Inappropriate regulation of NF-κB is directly involved in a wide range of human disorders, including a variety of cancers, neurodegenerative diseases, ataxiatelangiectasia, arthritis, asthma, inflammatory bowel disease, and numerous other inflammatory conditions. Recently, NF-κB and the signaling pathways that regulate its activity have become a focal point for intense drug discovery and development efforts. Well-known antiinflammatory substances such as glucocorticoids or aspirin and salicylate exert, at least a part of their effects, by inhibiting NF-κB activity. Several antiinflammatory medicinal plants also contain structurally diverse compounds that inhibit NF-κB activation. In our search for inhibitors of NF-κB activation from natural products, we identified dozens of compounds from several medicinal plants such as *Celastrus orbiculatus. Isodon japonicus, Alpina cochingera, Artemisia sylvatica, and Inula britanica.* These inhibitors were categorized into three groups based on the mode of action. One group of inhibitors interferes with the induced degradation of IκB family members by inhibiting IκB kinase. Another group of inhibitors exerts their effects only in the cell nucleus by impairing the transcriptional activity of RelA subunit of NF-κB. Further group of inhibitors interferes with DNA-binding activity of NF-κB by directly targeting p50 subunit. In this seminar, it will be present and discussed an IκB kinase inhibitor and a transcriptional activity inhibitor of RelA(p65).

395회 (2006. 9.21)

연사 : 정 유 진
강원대학교 생명과학부 생물학과

Abstract

Immunity to tuberculosis: On the limited protective value of anti-tuberculosis immunity in a susceptible host.

Only 5-10% of immunocompetant humans are susceptible to tuberculosis (TB), and of those who become infected more than 85% develop the disease exclusively in the lungs. In immunocompromise humans, by contrast, the disease can be systemic, involve multiple organs and result in much earlier death. This indicates that most susceptible, immuunocompetent humans generate an anti-TB immune response that exerts a growth inhibitory influence on Mycobacterium tuberculosis (Mtb) and acts to confine infection-induced disease to the lungs. It is now established that humans with active TB generate Th1-mediated anti-Mtb immunity.

Mice are also susceptible to tuberculosis, and like most susceptible humans develop disease exclusively in the lungs. Mice also generate Th1-mediated anti-Mtb immunity that is capable of inhibiting Mtb growth in the lungs at about day 20 on infection, and of holding infection at a stationary level. However, stationary level infection induces progressive lung pathology that is eventually lethal. It can be argued, therefore, that immunity to mouse TB is like immunity to the disease in susceptible humans, and that the reason for failure of immunity to resolve infection in mice will prove to be the same as that for failure of immunity to resolve infection in humans.

The purpose of vaccination is to enable susceptible hosts to mount a successful Th1 immune response, and it seems reasonable to propose that the requirements for successful vaccination of mice will apply to susceptible humans. Research in many laboratories is aimed at developing vaccines that are more protective than the vaccine currently in use, the attenuated BCG strain of M.bovis that is considered of limited value in protecting humans. It is clear, however, that attempts to vaccinate mice against Mtb infection with a variety of recently developed

vaccines have proved no more successful than vaccinating with BCG. In all cases vaccination provided about 1 log of protection against an Mtb challenge infection.

The purpose of this presentation is to discuss the meaning of the 1 log of protection afforded by vaccination, and to argue that the protection afforded by any one vaccine will prove to be wholly attributable to its ability to enable the host to generate an

earlier Th1 immune response that serves to inhibit Mtb growth at an earlier stage of infection when the number of Mtb is about 1 log lower. It will be argued further that earlier expression of immunity does not enable mice to resolve the lower level of infection, but enables them, instead, to hold infection at a lower stationary level that is disease inducing. The inability of naive and vaccinated mice to resolve infection may not be the result of an inadequate number of Mtb-specific T cells, but a consequence of functional limitations of macrophages that are transcriptional activated by Th1 cytokines to express immunity. If inhibition of Mtb growth is part of a metabolic counter response on the part of pathogen that enables it to survive the antimicrobial defenses acquired by activated macrophages, then, the earlier that Th1 immunity mediates macrophage activation the earlier the pathogen will defend itself by entering a state of nonreplicating persistence. It is apparent that Mtb has evolved to survive in susceptible humans in a nonreplicating state that, with the aid of Th1 immunity, enables it to induce lung pathology of a chronic type that favors its long-term transmission to new hosts.

396회 (2006.11. 2)

연사 : 이 동 석 교수
강원대학교 동물생명과학대학 동물생명공학과

Abstract

Impaired Synaptic Plasticity and Memory by Neuron specific Peroxiredoxin II KO.

It has been known well that Reactive oxygen species (ROS) generation by oxidative stress is implicated in various pathological conditions of the brain, such as epilepsy, ischemia/reperfusion, metal toxicity , trauma, neurodegenerative diseases including Parkinson's disease, and Alzheimer's disease, amyotrophic lateral sclerosis, and immunologic inflammatory disorders such as multiple sclerosis. Oxidative stress can result in damage to the cell through the oxidation of cellular components such as membrane lipids, proteins and DNA. Relationship between Reactive oxygen species (ROS) and synaptic plasticity (long-term potentiation; LTP) as an underline of mammalian learning and memory is concerned by Neurophysiology study. Although these studies were reported about relationship between ROS and LTP, the role of ROS in LTP has been not known well yet.

Peroxiredoxins (Prxs), previously referred to as NKEF (for natural killer enhancing factor) , TSA (for thiol-specific antioxidant) , Tpx (for thioredoxin peroxidase) , and PRP (for protective protein) are a novel family of antioxidant enzymes that have recently been characterized and control cytokine-induced peroxide levels which mediate signal transduction in mammalian cells has also been reported. The Prxs, consisted of six different members in mammalian, are widely distributed in most of tissues, but few informative results were reported in brain tissues.

Recently, in our study, the mapping of the distribution of the all Prx subtype proteins in the mammalian brain by immunohistochemistry study results may be indicative of the specific roles in their preferential neural cell types (Neuroscience letters (2005) Vol.381 pp252-257). In particular, Prx II immunoreactivity was mainly found in neurons such as hippocampus, cerebral cortex, etc. This indicates that Prx II may be associated with a regulatory role in signal transduction in specific neurons in addition to antioxidant activity. To determine the role of Prx2 in the brain, in this study, we investigated the functions of Prx2 about memory and learning using Prx II KO mice. We examined the ability of learning and memory in the PrxII KO mice. First, we performed recordings of long-term potentiation (LTP) on CA1 of the mice hippocampal slices for the ability of learning and memory in the PrxII KO mice. Maximal population spike amplitude was significantly different in old mice (from 6 months old) between wild type and PrxII KO

mice. And then, in the Morris water maze as spatial learning test, 12 months old PrxII KO mice showed significantly decrease compare to wild type mice. These results lead us to the possibility that Prx II is related to loss of LTP, learning and memory by aging.

397회 (2006.11.23)

연사 : 김 해 영
Department of Food Science and Biotechnology, Kyung Hee University

Abstract

Microarray Detection of Food-Borne Pathogens Using Specific Probes Prepared by Comparative Genomics

Food-borne pathogens are a major global healthproblem, and the rapid detection of food-borne pathogens is needed in epidemiology, the food industry, public health maintenance and bioterrorism prevention. To detect these pathogens, specific 70-mer oligonucleotide probes of 11 major food-borne pathogens were prepared by comparative genomics using the Basic Local Alignment Search Tool (BLAST) program between genomic DNA sequences of food-borne pathogens. Among the prepared probes, 10 probes from each food-borne pathogen were selected and a DNA microarray chip named "FBP-1"was constructed with 224 spots of 70-mer oligonucleotides for the detection of 11 food-borne pathogens. Various genomic DNAs of food-borne pathogens and non-pathogens were labeled with Cy3 dye and hybridized with the microarray chip. The constructed microarray chip showed a high specificity with the genomic DNA of each food-borne pathogen. Microarray data of various bacteria were analyzed and clustered. A phylogenetic tree showed a high discriminative power such that each food-borne pathogen was clustered and non-pathogens were discriminated from food-borne pathogens. These results can be applied as a rapid and convenient method to detect and identify food-borne pathogens in the food industry. In addition, this study demonstrated that genome sequence comparison and DNA microarray have a potential application in epidemiologic and taxonomic studies including food safety and biodefense analysis of microbial pathogens.

398회 (2006.12. 6)

연사 : 최 선 심
강원대학교 분자의생명공학전공

Abstract

Bioinformatics: Toward revealing secrets of the human genome.

Completion of the human genome sequencing induced a paradigm shift in biological research. Since the draft human genome sequence was published in 2001, many other genomes from lower organisms to higher organisms including several primates have been sequenced. The next step should be exploring the secrets of genomes. Here, I will briefly demonstrate how computer skills can be applied to the genome analyses. Further, I will introduce some of my studies of the human genome analyses through bioinformatics. First, using noncoding conservation as a proxy for the complexity of cis-regulatory DNA, it is shown that different classes of genes tend to have different levels of cis-regulatory complexity and that greater complexity can be found in genes involved in tissue-specific transcriptional regulation. Next, through a novel methodology called phylogeny-aided structural analysis, I will demonstrate that there are robust signals of interacting residue coevolution in mammalian proteomesathogens.

고등균류 분자생리학 심포지엄

심포지엄 진행 (2006년 11월 17일 오후 1시 - 6시 30분)

1. 오후 1:00 - 1:10 연구소장 인사 및 일정 소개

2. 1차 Session : 1:15 - 2:50 (좌장 : 강원대 송 홍규 교수)

   1:15 - 2:00 영지버섯 laccase 유전자의 분자생물학적 연구 (강원대 김경훈 교수)

   2:05 - 2:50 Regulation of transcription regulators, Tup1 & Tup2, by LAMMER kinase in fission yeast (충남대 박희문 교수)

3. 휴식 2:50 - 3:00

4. 2차 Session : 3:00 - 4:35 (좌장 : 우석대 김 종화 교수)

   3:00 - 3:45 Transcriptional down-regulation of hypoviral specific fungal hydrophobin, Cryparin, gene (전북대 김대혁 교수)

   3:50 - 4:35 A novel positive regulator of sexual development carrying $C_2H_2C_2H_2C_2HC$ type zinc finger domain in *Aspergillus nidulans* (원광대 한동민 교수)

5. 휴식 4:35 - 4:40

6. 3차 Session : 4:40 - 6:05 (좌장 : 배재대 채 순기 교수)

   4:40 - 5:15 느타리버섯의 발생단계별 유전자 발현양상의 분석 (건국대 이창수 교수)

   5:20 - 6:05 (특강) Thioredoxin reductase is required for growth and regulates entry into culmination of *Dictyostelium discoideum* (서울대 강사욱 교수)

7. 휴식 6:05 - 6:10

8. 종합 토론 6:10 - 6:30 (좌장: 강원대 최 형태 교수)

2006 강원 생명과학 심포지엄

심포지엄 진행 (2006년 12월 28일 오후 1시 - 6시 30분)

1. 오후 1:00 - 1:10 연구소장 인사 및 일정 소개

2. 1차 Session 1:15 - 2:45 (좌장 : 강원대 김 영명 교수)

   1:15 - 2:00 (특강) 해양 극한 생물자원의 확보 및 활용
   (국립 해양연구원 미생물 연구단장 김 상진 박사)

   2:05 - 2:45 Prostagladin E_2 stimulates angiogenesis and tumor growth by activating the nitric oxide/cGMP pathway
   (강원대 혈관연구센터 남궁 승 연구교수)

3. 휴식 2:45 - 3:00

4. 2차 Session 3:00 - 4:25 (좌장 : 한림대 송 동근 교수)

   3:00 - 3:40 Regulatory function of Snf1 kinase in yeast
   (강원대 식품생명공학과 김 명동 교수)

   3:45 - 4:25 Exploring a Plant Ran and Ran-binding proteins: Their involvement in the Regulation of Root Growth and Development
   (연세대 생명과학과 김 수환 교수)

5. 휴식 4:25 - 4:40

6. 3차 Session 4:40 - 6:05 (좌장 : 강원대 최 형태 교수)

   4:40 - 5:20 Cristin/R-spondin family proteins are novel ligands for the Frizzled8 and LRP6 receptors and activate beta-catenin signaling
   (한림대 MRC 감염성질환제어 연구센터 남 주석 교수)

   5:25 - 6:05 Development of therapeutic CpG-DNA for regulation of immune diseases (한림대 의대 미생물학교실 권 형주 교수)

7. 휴식 6:05 - 6:10

8. 종합토론 6:10 - 6:30 (좌장 : 강원대 김 경훈 교수)