TUESDAY PROGRAM SCHEDULE

Registration Opens at Monona Terrace (Tu 6:00 - 10:00 P.M.)

PNIRS Scholars (Tu 7:00 -10:00 P.M.)
Supported by NIMH R13 MH59793, Cousins Center, UCLA Neuropsychiatric Institute
(Meeting Rooms K-R at Monona Terrace)

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WEDNESDAY PROGRAM SCHEDULE

Breakfast (Wed 8:00 - 9:00 A.M.)
(Grand Terrace at Monona Terrace)

Registration Open All Day (Wed 8:00 A.M. - 6:00 P.M.)

PNIRS Council (Wed 9:00 - 11:00 A.M.)
(Meeting Room R)

PNIRS Educational Short Course (Wed 9:00 - 11:00 A.M.)
CLINICAL CONDITIONS ON THE BORDERS OF PNI
(Main Lecture Hall, Monona Terrace)

9:00 - 9:30 CYTOKINES IN ALLERGY AND ASTHMA. William Busse
(PNI Rapporteur, Duck-Hee Kang)

9:30 - 10:00 CANCER IMMUNOTHERAPY. Paul Sondel
(PNI Rapporteur, Andy Miller)

10:00 - 10:30 THE EMOTIONAL BRAIN. Richard Davidson
(PNI Rapporteur, Margaret Kemeny)

10:30 - 11:00 SLEEP AND HEALTH. Ruth Benca
(PNI Rapporteur, Mark Opp)

Coffee Break (Wed 11:00 - 11:30 A.M.)
(Grand Terrace at Monona Terrace)

Presidential Welcoming Remarks (Wed 11:30 - 12:00)
(Main Lecture Hall at Monona Terrace)

Lunch (Wed 12:00 - 1:00 P.M.)
(Grand Terrace at Monona Terrace)

Symposium 1 (Wed 1:00 - 3:00 P.M.)
(Main Lecture Hall at Monona Terrace)

FETAL, NEONATAL AND PUBERTAL ORIGINS OF ADULT DISEASE
Organizer: Deborah Hodgson

1:00 - 1:25 PRENATAL UNDERNUTRITION, GLUCOCORTICOIDS AND THE EARLY ORIGINS OF HYPERTENSION Isabella Caroline McMillen

1:25 - 1:50 NEONATAL EXPOSURE TO BACTERIAL ENDOTOXIN IS ASSOCIATED WITH INCREASED ANXIETY-RELATED BEHAVIOUR AND ALTERED METABOLIC RESPONSIVITY IN ADULTHOOD Deborah M Hodgson

1:50 - 2:15 PUBERTY, SOCIAL ISOLATION AND INCREASED RISK FOR INFECTIOUS AND MALIGNANT DISEASE DURING AGING Martha K McClintock

2:15 - 2:40 DEVELOPMENT OF A MATURE NK SUPPRESSIVE RESPONSE TO THE β-ADRENERGIC AGONIST METAPROTERENOL Gayle G Page

2:40 - 3:00 DEVELOPMENTAL EFFECTS OF EARLY IMMUNE STRESS ON SOCIAL BEHAVIOR Douglas Granger

Refreshments (Wed 3:00 - 3:30 P.M.)
(Grand Terrace at Monona Terrace)

Oral Session 1 (Wed 3:30 - 5:00 P.M.)
(Main Lecture Hall at Monona Terrace)

Endocrine-Immune Interactions
Chairs: Andrew Miller & David Padgett

3:30 - 3:45 A59 DYSREGULATION OF THE HPA AXIS IN PATIENTS WITH RA DETERMINED BY THE DEXAMETHASONE-CRF TEST Michael S. Harbuz

3:45 - 4:00 A136 ANTI-INFLAMMATORY COOPERATIVITY OF CORTICOSTEROIDS AND NOREPINEPHRINE IN VIVO AND IN VITRO IN PATIENTS WITH RHEUMATOID ARTHRITIS Rainer H. Straub

4:00 - 4:15 A48 GLUCOCORTICOID-SENSITIVITY IN SUBJECTS WITH DIMINISHED FUNCTION OF THE HYPOTHALAMUS-PITUITARY-AXIS (HPA) Andrea Gierens

4:15 - 4:30 A128 GREATER IL-6 INDUCTION MAY COMPENSATE FOR CRH-DEFICIENCY IN VIRUS-INDUCED CORTICOSTERONE RELEASE Marni N. Silverman

4:30 - 4:45 A126 EXPRESSION OF STEROID RESISTANCE REQUIRES A SECOND SIGNAL: ROLE OF TOLL-LIKE RECEPTORS John Sheridan

4:45 - 5:00 A12 PROTECTING CELLULAR IMMUNITY FROM SUPPRESSION BY SURGERY AND STRESS HORMONES: AN APPROACH TO PREVENT SURGERY-INDUCED TUMOR PROGRESSION Shamgar Ben-Eliyahu

Norman Cousins Memorial Lecture (Wed 6:30 - 7:30 P.M.)
(Main Lecture Hall at Monona Terrace)

Steve Maier (University of Colorado)

IMPLICATIONS OF THE BIDIRECTIONAL COMMUNICATION BETWEEN THE BRAIN AND IMMUNE SYSTEM

Reception (Wed 7:30 - 9:30 P.M.)
(Grand Terrace at Monona Terrace)

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THURSDAY PROGRAM SCHEDULE

Breakfast (Th 7:30 - 9:00 A.M.)
(Grand Terrace at Monona Terrace)

Early Bird Workshop (Th 8:00 - 9:00 A.M.)
(Main Lecture Hall at Monona Terrace)

CONVERGING PERSPECTIVES: EAST MEETS WEST
Discussants: Duck-Hee Kang, Richard Kradin

8:00 - 8:25 INSIGHTS FROM TRADITIONAL CHINESE MEDICINE Cai Song

8:30 - 8:55 ENDOCRINE AND IMMUNE EFFECTS OF CHUNDOSUNBUP QI-TRAINING Hoon Ryu

Oral Session 2 (Th 9:00 - 10:15 A.M.)
(Main Lecture Hall at Monona Terrace)

Neuroimmune Interactions
Chairs: Robert Bonneau & Cobi Heijnen

9:00 - 9:15 A50 NEURAL-IMMUNE INTERFACE IN THE AREA POSTREMA: ULTRASTRUCTURAL EVIDENCE FOR FUNCTIONAL INTERACTIONS BETWEEN NEURONS AND DENDRITIC IMMUNE CELLS Lisa E. Goehler

9:15 - 9:30 A155 THE YIN AND YANG OF REGULATING GLUTAMATE EXCITOTOXICITY IN CEREBELLAR GRANULAR NEURONS Wen-Hong Shen

9:30 - 9:45 A38 THE ROLE OF NITRIC OXIDE SYNTHASE IN THE INDOLEAMINERGIC RESPONSES TO IL-1 AND LPS Adrian J. Dunn

9:45 - 10:00 A131 OMEGA-3, BUT NOT ANTIDEPRESSANT SERTRALINE, ATTENUATES IL-1-INDUCED RELEASE OF NEUROTRANSMITTERS IN THE NUCLEUS ACCUMBENS: AN INVO MICRODIALYSIS STUDY Cai Song

10:00 - 10:15 A77 REDUCED CELLULAR IMMUNITY IN ANIMALS DEFICIENT FOR THE DOPAMINE TRANSPORTER (DAT) Annemieke Kavelaars

Coffee Break (Th 10:15 - 10:45 A.M.)
(Grand Terrace at Monona Terrace)

Oral Session 3 (Th 10:45 - 12:00)
(Main Lecture Hall at Monona Terrace)

Human PNI Relations
Chairs: Suzanne Segerstrom & Susan Lutgendorf

10:45 - 11:00 A118 MARITAL DISCORD AND IMMUNITY: WIVES' VERBAL AGGRESSION PREDICTS DECLINES IN INTERFERON-GAMMA PRODUCTION Theodore F. Robles

11:00 - 11:15 A91 THE PSYCHOLOGICAL IMPACT OF BREAST CANCER DIAGNOSIS PRODUCES PROLONGED ALTERATIONS IN TH1/TH2 CYTOKINES AND NATURAL KILLER CELL ACTIVITY Herbert L. Mathews

11:15 - 11:30 A90 EXAMINATION STRESS ALTERS CYTOKINE AND CHEMOKINE EXPRESSION IN HUMAN ORAL WOUNDS Phillip T. Marucha

11:30 - 11: 55 A28 DEPRESSION AND OSTEOPOROSIS: ASSOCIATION OR CAUSAL LINK ? Giovanni Cizza

Lunch (Th 12:00 - 1:00 P.M.)
(Grand Terrace at Monona Terrace)

(also NCI Sponsored Luncheon, Organizer: Michael Stefanek
By Invitation, La Follette Room, Hilton Second Floor)

Symposium 2 (Th 1:00 - 3:00 P.M.)
(Main Lecture Hall at Monona Terrace)

MODULATION OF BRAIN AND PERIPHERAL HOST DEFENSE BY OPIOIDS. CANNABINOIDS, AND NICOTINE
Organizers: Burt Sharp & Richard Weber

1:00 - 1:30 CANNABINOID REGULATION OF T-CELL NFAT AND IL-2 Norbert E. Kaminski

1:30 - 2:00 OPIOID MODULATION OF CHEMOKINE RECEPTOR EXPRESSION AND FUNCTION Thomas E. Rogers

2:00 - 2:30 OPIOID MODULATION OF ACTIVATED GLIAL CELLS: KEEPING THE 'FRIENDLY' FIRE FRIENDLY Phillip K Peterson

2:30 - 3:00 ROLE OF INTERLEUKIN-1 BETA IN NICOTINE-INDUCED IMMUNOSUPPRESSION Mohan L. Sopori

Refreshments (Th 3:00 - 3:30 P.M.)
(Grand Terrace at Monona Terrace)

POSTER SESSION 1 (Th 3:00 - 5:00 P.M.)
(Meeting Rooms K-R at Monona Terrace)

(First Author Last Name A - L, for titles see end of program)

GEORGE F. SOLOMON MEMORIAL SESSION (Th 5:15 - 6:15 P.M.)

Gail Ironson, Margaret Kemeny, and Robert Ader
(Main Lecture Hall at Monona Terrace)

Trainee Dinner (Essen House, Th 7:30 - 9:30 P.M.)

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FRIDAY PROGRAM SCHEDULE

Breakfast Buffet (Fri 8:00 - 9:00 A.M.)
(Grand Terrace at Monona Terrace)

Breakfast Roundtable Discussion:
NIH Funding of PNI & Related Areas: Program and Review Perspectives
Deborah Ader (NIAMS), Fred Altman (NIMH), Kathy Kopnisky (NIMH),
Michael Stefanek (NCI), Thomas Tatham (CSR)

BD Biosciences-Pharmingen Measurement of Cytokines and their Pathways
Speaker: Kevin Weller, Technical Application Specialist
(*Main Lecture Hall*)

Oral Session 4 (Fri 9:00 - 10:15 A.M.)
(Main Lecture Hall at Monona Terrace)

Opioid Influences on Immunity
Chairs: Toby K. Eisenstein & Donald Lysle

9:00 - 9:15 A40 NOCICEPTION IS IMMUNOSUPPRESSIVE. Toby K. Eisenstein

9:15 - 9:30 A1 DESENSITIZATION OF OPIOID RECEPTORS IN VIVO BY SELECTED CHEMOKINES Martin W. Adler

9:30 - 9:45 A121 CHRONIC MORPHINE TREATMENT REGULATES IL-4 SYNTHESIS THROUGH THE TRANSCRIPTION FACTOR GATA-3 Sabita Roy

9:45 - 10:00 A70 CHARACTERIZATION OF ENDOMORPHIN (EM)-1 AND EM-2 IN HUMAN LEUKOCYTES AND IN RAT IMMUNE TISSUES IN A MODEL OF INFLAMMATION David S. Jessop

10:00 - 10:15 A41 SEX DIFFERENCES IN OPIOID-INDUCED ENHANCEMENT OF CONTACT HYPERSENSITIVITY: CLINICAL OUTCOMES AND MOLECULAR MEDIATORS Jay C. Elliott

Coffee Break (Fri 10:15 - 10:45 A.M.)
(Grand Terrace at Monona Terrace)

Oral Session 5 (Fri 10:45 A.M.- 12:00)
(Main Lecture Hall at Monona Terrace)

Molecular Biology
Chairs: Virginia Sanders & Keith Kelley

10:45 - 11:00 A117 ACUTE STRESS ACTIVATES LEUKOCYTE TRANSCRIPTION FACTORS Vanessa Richlin

11:00 - 11:15 A68 MODULATION OF INFLUENZA A/PR8 INDUCED INNATE CYTOKINE GENE EXPRESSION AND NATURAL KILLER CELL ACTIVITY BY RESTRAINT STRESS John Hunzeker

11:15 - 11:30 A29 ATP INDUCES THE PROCESSING AND RELEASE OF INTERLEUKIN-1β BY SCHWANN CELLS IN RESPONSE TO LIPOPOLYSACCHARIDE VIA A P2X7 PURINERGIC RECEPTOR Aurore Colomar

11:30 - 11:55 LB2 INSULIN RESISTANCE: DOES IT CAUSE CYTOKINE RESISTANCE? Gregory Freund

Lunch (Fri 12:00 - 1:00 P.M.)
(Grand Terrace at Monona Terrace)

(also Mind & Immunity Roundtable Discussion, Organized by S. Segerstrom)

Symposium 3 (Fri 1:00 - 3:00 P.M.)
(Main Lecture Hall at Monona Terrace)

DEPRESSION AND IMMUNITY: NEW LIGHT ON A DARK SUBJECT
Organizer: Steven Schleifer

1:00 - 1:25 CYTOKINE–INDUCED DEPRESSION: IMPLICATIONS FOR THE ROLE OF THE IMMUNE SYSTEM IN MOOD DISORDERS Andrew H. Miller

1:50 - 2:15 BEHAVIORAL AND NEUROCHEMICAL EFFECTS OF CYTOKINES IN ANIMAL STUDIES Zul Merali

1:25 - 1:50 ACTIVATION OF THE INFLAMMATORY RESPONSE SYSTEM IN DEPRESSIVE CONDITIONS Michael Maes

2:15 - 2:40 SOLUBLE CELL ADHESION MOLECULES, INFLAMMATORY CYTOKINES, AND DEPRESSION IN ACUTE CORONARY SYNDROME PATIENTS Michael Irwin

2:40 - 3:00 INTEGRATIVE DISCUSSION Steven Schleifer

Refreshments (Fri 3:00 - 3:30 P.M.)
(Grand Terrace at Monona Terrace)

POSTER SESSION 2 (Fri 3:00 - 5:00 P.M.)
(Meeting Rooms K-R at Monona Terrace)

(First Author Last Names M - Z
And All Late-Breaking Abstracts, for listing see end of program)

Friday Night Dinner on Your Own

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SATURDAY PROGRAM SCHEDULE

Breakfast (Sat 8:30 - 10:00 A.M.)
(Grand Terrace at Monona Terrace)

Symposium 4 (Sat 10:00 - 12:00)
(Main Lecture Hall at Monona Terrace)

NEW INSIGHTS FROM MOLECULAR BIOLOGY

Organizer: Keith W. Kelley

10:00 - 10:30 MECHANISMS INVOLVED IN CEREBRAL INNATE IMMUNITY Serge Rivest

10:30 - 11:00 A NOVEL MECHANISM BY WHICH TNF RECEPTORS PROMOTE MUSCLE WASTING. Suzanne R. Broussard

11:00 - 11:30 RECRUITMENT AND FUNCTION OF T CELLS IN THE BRAIN Zsuzsanna Fabry

11:30 - 12:00 GENE EXPRESSION PROFILE OF THE AGING BRAIN Richard Weindruch

Lunch (Sat 12:00 - 1:00 P.M.)
(Grand Terrace at Monona Terrace)

BBI Editorial Board Meeting and Lunch
(LaFollette Room at the Hilton)

Oral Session 6 (Sat 1:00-3:00 P.M.)
Stress and Immunity
Chairs: Manfred Schedlowski & Linda Watkins

1:00 - 1:15 A106 EFFECTS OF PRENATAL STRESS ON STRESS-INDUCED BEHAVIORAL AND IMMUNOLOGICAL RESPONSES IN MICE Joao Palermo-Netto

1:15 - 1:30 A7 PRENATAL STRESS ALTERS THE DEVELOPMENT OF THE INTESTINAL MICROFLORA IN RHESUS MONKEYS Michael T. Bailey

1:30 - 1:45 A93 THE CYTOKINERGIC RESPONSE TO STRESS DEPENDS ON LATERALIZATION Pierre Neveu

1:45 - 2:00 A36 ENDOGENOUS STRESS HORMONES, CORTICOSTERONE AND EPINEPHRINE, ENHANCE MACROPHAGE INFILTRATION AND IL1-β GENE EXPRESSION IN SKIN DELAYED TYPE HYPERSENSITIVITY REACTIONS Firdaus S. Dhabhar

2:00 - 2:15 A46 PHYSICAL ACTIVITY REDUCES CIRCULATING AND TISSUE INFLAMMATORY CYTOKINE AND SYMPATHETIC RESPONSES TO STRESS Monika Fleshner

2:15 - 2:30 A146 ACUTE STRESS ADMINISTERED EITHER BEFORE PRIMARY IMMUNIZATION OR BEFORE CHALLENGE ENHANCES SKIN CELL MEDIATED IMMUNITY TO KEYHOLE LIMPET HEMOCYANIN (KLH) Kavitha Viswanathan

2:30 - 2:45 A6 GLUCOCORTICOID INHIBITION OF PRO-INFLAMMATORY CYTOKINES IN LOW AND HIGH CORTISOL PRODUCING INDIVIDUALS Jennie Axen

2:45 - 3:00 A98 EFFECTS OF STRESS IN IMMUNOCOMPROMISED HOSTS Jan A. Moynihan

Refreshments (Sat 3:00 - 3:30 P.M.)
(Grand Terrace at Monona Terrace)

Business Meeting (Sat 3:30 - 4:30 P.M.)
(Main Lecture Hall at Monona Terrace)

Banquet and Dance (Sat 7:30 - 11:30 P.M.)
(Grand Terrace & Hall of Ideas at Monona Terrace)

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TITLES AND AUTHORS FOR POSTER SESSION 1 (Th 3:00 - 5:00 P.M.)
(Meeting Rooms K-R at Monona Terrace)

A2 IS HAPPINESS RELATED TO IMMUNE FUNCTION?
Ahmad Alipour

A3 PSYCHONEUROIMMUNOLOGIC PROFILES OF WOMEN WITH FIBROMYALGIA.
Carmen Alonso, Daniel Muller, Barbara Loevinger, Julie Surbaugh, Chris Erickson, Christopher Coe

A4 FACTORS AFFECTING THE INFILTRATION OF LYMPHOCYTES INTO THE CENTRAL NERVOUS SYSTEM AND DEVELOPMENT OF ENCEPHALITIS DURING HERPES SIMPLEX VIRUS INFECTION.
Crystal S. Anglen, Aji Nair, Robert H. Bonneau

A5 SOCIAL STRESS ALTERS SPLENOCYTE PHENOTYPE AND FUNCTION: ANALYSIS OF BEHAVIORAL INTERACTIONS.
Ronit Avitsur, Jennifer L. Stark, Firdaus S. Dhabhar, John F. Sheridan

A8 MORPHINE SULPHATE INHIBITS JURKAT T-LYMPHOCYTE CELL CYCLING THROUGH INDUCTION OF G2/M ARREST.
Sudha Balasubramanian, JingHua Wang, Jennifer Kelschenbach, Richard Charboneau, Roderick Barke, Sabita Roy

A9 INTERLEUKIN-1 BETA (IL-1β) IMPAIRS HIPPOCAMPAL-DEPENDENT MEMORY CONSOLIDATION.
Ruth M. Barrientos, Emily A. Higgins, David B. Sprunger, Linda R. Watkins, Jerry W. Rudy, Steven F. Maier

A10 GROUP HOUSING VS. INDIVIDUALLY HOUSING: WHAT IS STRESSFUL FOR MALE MICE?
Alessandro Bartolomucci, Alessio Chirieleison, Paola Palanza, Paola Sacerdote, Alberto E. Panerai, Graziano Ceresini, Marco D. Poli, Stefano Parmigiani

A11 CONCANAVALIN A (CONA)-INDUCED SPLEEN CELL PROLIFERATION AND CYTOKINE PRODUCTION IN MALE LEWIS RATS: AGE-RELATED DIFFERENCES AND EFFECTS OF β-ADRENERGIC RECEPTOR (βAR) STIMULATION.
Denise L. Bellinger, Cheri Lubahn, Jill Schaller, Dianne Lorton

A13 SHORT DAY LENGTHS AUGMENT STRESS-INDUCED LEUKOCYTE TRAFFICKING AND STRESS-INDUCED ENHANCEMENT OF SKIN IMMUNE FUNCTION.
Staci D. Bilbo, Firdaus S. Dhabhar, Kavitha Viswanathan, Alison Saul, Steven M. Yellon, Randy J. Nelson

A14 EFFECTS OF INTRAPERITONEAL LIPOPOLYSACCHARIDE ON MORRIS WATER MAZE LEARNING IN MALE C57BL/6J MICE.
Gary W. Boehm, Nathan L. Sparkman, Gail Pinto, Vincent J. Scott

A15 THE ROLE OF CORTICOSTEROID AND β-ADRENERGIC RECEPTORS IN STRESS-MEDIATED SUPPRESSION OF CYTOTOXIC T LYMPHOCYTE (CTL)-MEDIATED PROTECTION AGAINST MUCOSAL HERPES SIMPLEX VIRUS (HSV) INFECTION.
Robert H. Bonneau, Keith M. Wonnacott, Heidi D. Leob

A16 LEUKOCYTE REDISTRIBUTION IN RESPONSE TO ACUTE REAL-LIFE STRESS IS A STABLE INDIVIDUAL CHARACTERISTIC.
Jos A. Bosch, Firdaus S. Dhabhar, Alison Light, Phillip T. Marucha

A17 BIPHASIC CHANGE IN NK CELL NUMBERS FOLLOWING EXPERIMENTAL STRESSORS: CORTICOSTEROID MEDIATION.
Dana H. Bovbjerg

A18 RADIATION-INDUCED FATIGUE AND PROINFLAMMATORY CYTOKINES IN BREAST CANCER PATIENTS: PRELIMINARY RESULTS.
Julienne E. Bower, Patricia A. Ganz, May Lin Tao, John L. Fahey

A19 MODERATE EXERCISE ALTERS PSYCHOSOCIAL FACTORS AND IMMUNE RESPONSE TO INFLUENZA VACCINE IN OLDER ADULTS.
Barbara A. Breidenbach, Kayla Rozeboom, Crystal L. Patterson, Wanglok Lee, Joan E. Cunnick, Marian L. Kohut

A20 ANTIBODY STATUS FOLLOWING MENINGITIS C CONJUGATE VACCINATION IS PREDICTED BY PERCEIVED STRESS AND PSYCHOLOGICAL WELL-BEING.
Victoria E. Burns, Mark Drayson, Douglas Carroll, Christopher Ring

A21 PSYCHOLOGICAL STRESS, CORTISOL, AND ANTIBODY RESPONSE TO INFLUENZA VACCINATION IN A YOUNG, HEALTHY COHORT.
Victoria E. Burns, Douglas Carroll, Mark Drayson, Martin Whitham, Christopher Ring

A22 EXTRACELLULAR HSP72 CONTRIBUTES TO STRESS-INDUCED FACILITATION OF INNATE IMMUNITY.
Jay Campisi, Monika Fleshner

A23 ACUTE COLD/RESTRAINT STRESS INHIBITED HOST RESISTANCE TO *LISTERIA MONOCYTOGENES* VIA β-ADRENERGIC RECEPTORS.
Ling Cao, Chad A. Hudson, David A. Lawrence

A24 BUPRENORPHINE INDUCES NALTREXONE REVERSIBLE IMMUNE ALTERATIONS.
K.A. Carrigan, T.B. Saurer, S.G. Ijames, D.T. Lysle

A25 FECAL CORTICOIDS PROVIDE A MEASURE OF ACUTE AND CHRONIC ADRENAL FUNCTION.
Sonia A. Cavigelli, Tina K. Whitney, Martha K. McClintock

A26 LEUKEMIA INHIBITORY FACTOR IS REQUIRED FOR THE NEUROENDOCRINE CONTROL OF THE ACTH AXIS INFLAMMATORY RESPONSE.
Vera Chesnokova, Anastasia Kariagina, Shlomo Melmed

A27 EFFECTS OF PLANT INTERACTIONS ON CORTISOL, SIGA, AND EMOTIONAL RESPONSES OF COLLEGE STUDENTS.
Hyejin Cho, Richard H. Mattson, J. Ernest Minton

A30 METHYLENEDIOXYMETHAMPHETAMINE (MDMA; "ECSTASY") PROMOTES AN ANTI-INFLAMMATORY CYTOKINE PHENOTYPE: A ROLE FOR CATECHOLAMINES.
Thomas J. Connor , John P. Kelly

A31 MOOD AND THE CYTOKINE RESPONSE TO INFLUENZA VIRUS IN THE ELDERLY.
Erin S. Costanzo, Susan K. Lutgendorf, Marian L. Kohut, Nicole Nisly, Shawn Spooner, Kayla Rozeboom, Janet E. McElhaney

A32 STRESS INDUCED LONG TERM EFFECTS OF COLONY HOUSING ON NUMBERS AND ACTIVITY OF NK CELLS IN F344 RATS.
Lutz Dawils, Volker Stefanski

A33 EFFECTS OF AGGRESSIVE CONFRONTATIONS AND WOUNDING ON CYTOKINE PRODUCTION IN FATTENING PIGS.
Johanna de Groot, Jan Willem Scholten, Leo Kruijt, Wim Boersma

A35 REGULATION OF B7.2 (CD86) EXPRESSION BY IL-4.
Eric L. Deszo, Danett K. Brake, Keith W. Kelley, Gregory G. Freund

A37 PREDICTORS OF CORTISOL REACTIVITY TO ACUTE PSYCHOLOGICAL STRESSORS: A META-ANALYTIC REVIEW.
Sally S. Dickerson, Margaret E. Kemeny

A39 RELATIONSHIP BETWEEN THE DIURNAL PATTERN OF SALIVARY S-IGA AND CORTISOL: ASSOCIATIONS WITH PSYCHOSOCIAL VARIABLES AND HEALTH.
Susan Edwards, Angela Clow, Philip Evans, Frank Hucklebridge

A42 EVIDENCE THAT B1 PRODUCTION OF NIGM IS ELEVATED IN THE PERITONEAL CAVITY AFTER VOLUNTARY FREEWHEEL RUNNING.
Gwendolyn F. Elphick, Benjamin N. Greenwood, Jay Campisi, Monika Fleshner

A43 NEUROENDOCRINE-IMMUNE RESPONSES TO NUTRIENT CHALLENGE IN PATIENTS WITH IRRITABLE BOWEL SYNDROME (IBS) AND CONTROLS.
Sigrid Elsenbruch, Andreas Lysson, Deniz Oezcan, Gerald Holtmann, Marion U. Goebel, Manfred Schedlowski

A44 ENHANCEMENT OF IgG2 RESPONSE ASSOCIATED WITH INTERLEUKIN-1β-INDUCED CONDITIONED TASTE AVERSION.
Enrique Espinosa, Oscar Flores-Muciño , Georgina Perez-GarcÌa , Ana C. Vazquez-Camacho , Federico Bermudez-Rattoni

A45 ANDROSTENEDIOL REGULATION OF NF-KB TRANSCRIPTIONAL ACTIVITY: A POTENTIAL MECHANISM FOR ANTI-GLUCOCORTICOID ACTIVITY.
Michael Farrow, David A. Padgett

A47 NITRIC OXIDE: A POSSIBLE CAUSE FOR STRESS INDUCED DYSREGULATION OF WOUND HEALING.
Praveenkumar Gajendrareddy, Michael P. Horan, Phillip T. Marucha

A49 ACUTE EFFECTS OF INTERFERON-BETA THERAPY ON HPA-AXIS ACTIVITY, LEUKOCYTE DISTRIBUTION AND MOOD STATES IN MULTIPLE SCLEROSIS PATIENTS AND HEALTHY SUBJECTS.
Marion U. Goebel, Frank Czolbe, Holger Becker, Michael S. Exton, Bernhard Saller, Manfred Schedlowski, Volker Limmroth

A51 IN VITRO IMMUNOPOTENTIATING PROPERTIES AND TUMOR CELL TOXICITY INDUCED BY LOPHOPHORA WILLIAMSII (PEYOTE) CACTUS METHANOLIC EXTRACT.
Ricardo Gomez-Flores, Moises Franco-Molina, Richard J. Weber, Patricia Tamez-Guerra, Cristina Rodriguez-Padilla, Reyes Tamez-Guerra

A52 THE ROLE OF INTERLEUKIN-1 IN HIPPOCAMPAL-DEPENDENT MEMORY PROCESSES AND NEURAL PLASTICITY.
Inbal Goshen, Avi Avital, Ariel Kamsler, Gordon Winocur, Kerstin Iverfeldt, Menachem Segal, Gal Richter-Levin, Raz Yirmiya

A53 SEPSIS-INDUCED SICKNESS BEHAVIOR IN MICE.
Jill I. Granger, Luca Ratti, Subhash C. Datta, Mark R. Opp

A54 THE ROLE OF NOREPINEPHRINE IN ESCHERICHIA COLI O157:H7 ADHERENCE TO THE COLONIC EPITHELIUM.
Ben T. Green, Chunsheng Chen, David R. Brown, Mark Lyte

A55 UTILIZATION OF A MOUSE MODEL TO ASSESS THE EFFECTS OF MATERNAL STRESS ON THE DEVELOPMENT OF NEONATAL IMMUNITY AND SUSCEPTIBILITY TO HERPES SIMPLEX VIRUS (HSV) INFECTION.
Jodi L. Greenfield, Robert H. Bonneau

A56 VOLUNTARY FREEWHEEL RUNNING MODULATES STRESS-INDUCED ACTIVITY OF THE CENTRAL SYMPATHETIC NETWORK.
Benjamin N. Greenwood, Monika Fleshner

A57 STRESS AND IMMUNITY IN LACTATING AND BOTTLE-FEEDING POSTPARTUM MOTHERS.
Maureen Groer

A58 SERUM LEVELS OF IL-1 AND IL-4 CORRELATED WITH ALEXITHYMIA IN HEALTHY SUBJECTS.
Olivier Guilbaud, Maurice Corcos, Sabrina Paterniti, Linnea Hjarmasson, Marlene Moussa, Gwenole Loas, Gerard Chaouat, Philippe Jeammet

A60 THE EFFECT OF ANDROSTENEDIOL AND ANDOSTENETRIOL ON CUTANEOUS WOUND HEALING IN CD1 MICE.
Cynthia C. Head , Phillip T. Marucha, John F. Sheridan, David A. Padgett

A61 SOCIAL SUPPORT SATISFACTION RELATES TO HUSBANDS' AND WIVES' CORTISOL AND INTERFERON PRODUCTION.
Kathi L. Heffner, Janice K. Kiecolt-Glaser, William B. Malarkey, Ronald Glaser

A62 PATTERNS OF MORBIDITY AND MORTALITY, REPRODUCTIVE AGING AND CELL-MEDIATED IMMUNITY IN PSYCHOSOCIAL CONTEXT.
Gretchen L. Hermes, Judith LeFevre, Martha K. McClintock

A63 INTRACEREBRAL ADMINISTRATION OF IL-1B SUPPRESSES PERIPHERAL IL-6 RESPONSES IN THE RAT.
Deborah M. Hodgson , Lachlan Cornford

A64 EXPOSURE TO BACTERIAL ENDOTOXIN IN EARLY LIFE ALTERS IMMUNOLOGICAL RESPONSIVITY IN ADULTHOOD.
Deborah M. Hodgson, Amanda Brown, Frederick Walker

A66 HIV-1 GP120-INDUCED SPINAL PROINFLAMMATORY CYTOKINE PRODUCTION AND LOW THRESHOLD MECHANICAL ALLODYNIA ARE MEDIATED BY NITRIC OXIDE.
Adelina Holguin, Kevin A. O'Connor, Mike K. Hansen, Erin D. Milligan, Poonlarp Cheepsunthorn, Dave Martin, Steven F. Maier, Linda R. Watkins

A67 DIFFERENTIAL EXPRESSION OF VEGF SPLICE VARIANTS IN A MURINE MODEL OF STRESS IMPAIRED WOUND HEALING.
Michael P. Horan, Praveen Gajendrareddy, John F. Sheridan, Phillip T. Marucha

A69 EPINEPHRINE PROMOTES IMMUNE ANGIITIS IN VIVO: POTENTIAL ROLE OF CD4+CD25+ LYMPHOCYTES.
Felipe A. Jain, Long-hai Zhao, Carol Leary, Richard L. Kradin

A71 PRIOR ADMINISTRATION OF CENTRAL IL-1β SENSITIZES THE CYTOKINE AND HPA RESPONSE.
John D. Johnson, Kevin A. O'Connor, Joe C. Biedenkapp, Linda R. Watkins, Steve F. Maier

A72 THE TIMING OF SOCIAL STRESS ALTERS THE COURSE OF THEILER'S VIRUS INFECTION.
Robin R. Johnson, Tom H. Welsh, Larissa A. Husband, C. J. R. Welsh, Mary W. Meagher

A73 FATIGUE, OPTIMISM, PSYCHOLOGICAL DISTRESS, AND THEIR ASSOCIATIONS WITH NATURAL KILLER CELL ACTIVITY IN BREAST CANCER: PRELIMINARY FINDINGS.
Duck-Hee Kang, Teri Mobley

A74 CHRONIC IMIPRAMINE TREATMENT IMPAIRS CENTRAL PRO-INFLAMMATORY CYTOKINE RESPONSE TO ENDOTOXIN.
Anastasia Y. Kariagina, Robert N. Pechnick, Russell E. Poland, Shlomo Melmed, Vera Chesnokova

A75 MODULATION OF NOREPINEPHRINE BY CYCLOPHOSPHAMIDE: IMPLICATIONS FOR NEURAL-IMMUNE INTERACTIONS.
Jonathan Karp, Jennifer Szczytkowski

A76 OBSESSIVE-COMPULSIVE DISORDER CAUSED BY NORMAL PRESSURE HYDROCEPHALUS RESPONDING TO CSF DRAINAGE.
Yakir Kaufman, Yehoshua, Omer Bonneh, Tamir Ben-Hur

A78 SPLENIC NOREPINEPHRINE DEPLETION FOLLOWING EXPOSURE TO STRESS IS NOT DUE TO AN ALTERATION IN PLASMA CATECHOLAMINE CONCENTRATION.
Sarah L. Kennedy, Benjamin N. Greenwood, Taro P. Smith, Monika Fleshner

A80 RELATIONSHIPS BETWEEN THE INNATE IMMUNE SYSTEM (NKCA) AND PSYCHOLOGICAL PARAMETERS RELATED TO DEPRESSION.
A.I. Kook, I. Gold, O. Klein, A. Mizruchin, B. Shifron

A81 SECRETION OF S-IGA AND CORTISOL IN HUMAN SUBJECTS CAN BE MODULATED BY STIMULATION OF THE CEREBRAL CORTEX USING TRANSCRANIAL MAGNETIC STIMULATION.
Shirley Lambert, Phil Evans, Frank Hucklebridge, Angela Clow

A82 HEROIN-INDUCED ALTERATION OF INTERLEUKIN-1β, TUMOR NECROSIS FACTOR-α, AND NITRIC OXIDE PRODUCTION: COMPARISON OF ACUTE- AND SELF-ADMINISTRATION PROCEDURES.
Ryan K. Lanier, Stephanie G. Ijames, Kelly A. Carrigan, Regina M. Carelli, Donald T. Lysle

A83 PERCEPTION OF SUBJECTIVE HEALTH IS ASSOCIATED WITH LEVELS OF CIRCULATING CYTOKINES IN PRIMARY HEALTH CARE PATIENTS.
Mats Lekander, Maria Gustafsson, Stig Elofsson, Ing-Marie Neve, Anna-Lena Unden

A84 THE EFFECTS OF RELAXATION GUIDED IMAGERY ON PSYCHOLOGICAL VARIABLES AND NATURAL KILLER CELL AND CYTOKINE(IL-2) INDUCED IN BREAST CANCER PATIENTS.
Cecile A. Lengacher, L. Gonzalez, M. Bennett

A85 A NEW ANIMAL MODEL OF EMOTIONAL STRESS: BEHAVIORAL, NEUROENDOCRINE AND IMMUNOLOGICAL CONSEQUENCES.
Wenjuan Lin , Feng Shao

A86 INCREASING SYMPATHETIC TONE DURING THE INITIATION PHASE OF DISEASE DEVELOPMENT EXACERBATES ADJUVANT ARTHRITIS.
Dianne Lorton, Jill Schaller, Denise L. Bellinger, Cheri Lubahn

A87 WHO'S THE BOSS?: RELATIONSHIPS BETWEEN POWER WITHIN MARRIAGE, STRESS HORMONES, AND IMMUNE FUNCTION.
Timothy J. Loving, Janice K. Kiecolt-Glaser, Ron Glaser, William B. Malarkey

A88 EFFECTS OF THE GANGLIONIC BLOCKADE, OR β-ADRENERGIC RECEPTOR (β-AR) ANTAGONISTS AND AGONISTS ON SEVERITY OF ADJUVANT ARTHRITIS (AA) DURING DIFFERENT DISEASE PHASES.
Cheri Lubahn, Denise Bellinger, Jill Schaller, Dianne Lorton

A89 RELIGIOUS PARTICIPATION, INTERLEUKIN-6, AND MORTALITY IN OLDER ADULTS.
Susan K. Lutgendorf, Daniel Russell, Philip Ullrich, Tamara Harris, Robert Wallace

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A92 POSTNATAL MATERNAL SEPARATION DISRUPTS VIRAL CLEARANCE DURING THE ACUTE PHASE OF THEILER'S VIRUS INFECTION IN ADULT MICE.
Mary W. Meagher, Michail Belyavskyi, Amy N. Seive, Robin R. Johnson, Jane C. Welsh

A94 CYTOKINE CHANGES FOLLOWING RESTRAINT STRESS AND THEILER'S VIRUS INFECTION.
Wentao Mi, Michail Belyavskyi, Robin R. Johnson, Amy N. Seive, Mary M. Meagher, Jane C. Welsh

A95 SPINAL CORD FRACTALKINE, A CHEMOKINE, IS INVOLVED IN EXAGGERATED PAIN STATES.
Erin D. Milligan, Carin M. Twining, Gayle A. Chapman, Kevin A. O'Connor, Steven Maier, Linda R. Watkins

A96 THE EFFECTS OF SPACEFLIGHT ON PERIPHERAL LEUKOCYTE CELLULAR ADHESION MOLECULE EXPRESSION.
Paul J. Mills, Christy Perez, Nhu Thuy Nguyen, Brian P. Kennedy, Michael G Ziegler

A97 NOREPINEPHRINE BOTH INCREASES NITRIC OXIDE (NO) AND SUPPRESSES TH CYTOKINES.
Albert Moraska, Jay Campisi, Monika Fleshner

A99 EFFECTS OF INESCAPABLE SHOCK ON CENTRAL AND PERIPHERAL PROINFLAMMATORY CYTOKINE MRNA.
Kevin A. O'Connor, Michael K. Hansen, Laura M. Brooks, John D. Johnson, Joseph C. Biedenkapp, Linda R. Watkins, Steven F. Maier

A100 CYTOKINE INVOLVEMENT IN NARCOLEPSY: A LOOK AT TNF-α AND IL-6.
Michele L. Okun, Emmanuel Mignot, Scott Giese, Lin Ling, Mary E. Coussons-Read

A101 MORPHINE MODULATES $\gamma\delta$ T CELL FUNCTIONS IN SWINE.
Michael Raymond Olin, Jinhee Lee Lee, K. Hwa Choi, Thomas W. Molitor

A102 INTERLEUKIN-6 ALTERS SLEEP IN THE RAT.
Mark R. Opp, Jonathan D. Morrow, Dale Hogan, Eric M. Smith

A103 CENTRAL NOREPINEPHRINE DEPLETION REDUCES THE PROLIFERATION OF LYMPHOCYTES IN THE SPLEEN.
Gustavo Pacheco-Lopez, Maj-Britt Niemi, Manfred Schedlowski

A104 INCREASED IGG 3:4 RATIOS IN ADOLESCENT ANTISOCIAL FEMALES: EVIDENCE OF TH1/TH2 IMBALANCE?
Kathleen A. Pajer, Bruce Rabin, William P. Gardner

A105 EFFECTS OF PHYSICAL AND PSYCHOLOGICAL STRESSORS ON STRESS/ANXIETY LEVELS, MACROPHAGE ACTIVITY AND EHRLICH TUMOR GROWTH.
Joao Palermo-Neto, Cristina Oliveira Massoco

A107 EXERCISE TRAINING DIFFERENTIALLY ALTERS ANTIVIRAL AND MITOGEN-INDUCED RESPONSES IN AGED MICE.
Crystal L. Patterson, Renwei Wang, Joan E. Cunnick, Marian L. Kohut

A108 FLARES IN PATIENTS WITH SYSTEMIC LUPUS ERYTHEMATOSUS ARE ASSOCIATED WITH DAILY PSYCHOSOCICAL STRESS.
Cornelius R. Pawlak, Torsten Witte, Matthias Hundt, Hans Heiken, Birgitt Wiese, Cobi J. Heijnen, Reinhold E. Schmidt, Manfred Schedlowski

A109 ORAL ADMINISTRATION OF SUB-LETHAL DOSES OF MERCURY INDUCES MORPHOLOGICAL AND IMMUNOLOGICAL MODIFICATIONS IN TESTIS WITH ALTERATIONS IN TESTOSTERONE PRODUCTION IN RATS.

Marisol Pocino, Salvador Penna, Maria de Marval, Josep Lloret, Luis Gallardo

A110 INFLAMMATORY MEDIATORS IN EAE- AND LPS-ASSOCIATED BEHAVIORAL ALTERATIONS.
Yehuda Pollak, Einav Orion, Hayim Ovadia, Joseph Weidenfeld, Raz Yirmiya

A111 STRESS AND ANTIBODY RESPONSE TO IMMUNIZATION IN COLLEGE FRESHMAN.
Sarah Pressman, Sheldon Cohen, Greg Miller, Bruce Rabin

A112 MOLECULAR MECHANISMS FOR THE GLUCOCORTICOID RESISTANCE INDUCED BY SOCIAL STRESS.
Ning Quan, Ronit Avitsur, Jennifer Stark, Lingli He, Wenmin Lai, Firdaus Dhadhar, John F. Sheridan

A113 MACROPHAGES MEDIATE IMMUNOSUPPRESSION FOLLOWING WITHDRAWAL FROM MORPHINE IN MICE.
Rahil T. Rahim, Joseph J. Meissler Jr., Thomas J. Rogers, Ellen B. Geller, Martin W. Adler, Toby K. Eisenstein

A114 ICV INJECTIONS OF CORTICOTROPIN-RELEASING FACTOR ALTERS LYMPHOCYTE AND MONOCYTE CELLULAR ADHESION MOLECULE EXPRESSION.
Laura S. Redwine, Scott Southerland, Karen Britton

A115 PLASMA IL-6 AS A MEDIATOR OF SYMPTOMS OF DEPRESSION AND PHYSICAL FUNCTION IN CONGESTIVE HEART FAILURE PATIENTS.
Laura S. Redwine, Merna Sada, Alan Maisel, Michael Irwin

A116 LYMPHOCYTE CHEMOTAXIS IN RESPONSE TO A SPEECH TASK IN ALZHEIMER'S CAREGIVERS AND NON-CAREGIVER CONTROLS.
Laura S. Redwine, Paul J. Mills, Joel Dimsdale, Alan Maisel, Tom Patterson, Igor Grant

A119 IMPACT OF GENDER AND ORAL CONTRACEPTIVE USE ON GLUCOCORTICOID SENSITIVITY OF PRO-INFLAMMATORY CYTOKINE PRODUCTION AFTER PSYCHOSOCIAL STRESS.
Nicolas Rohleder, Jutta M. Wolf, Marcel Piel, Clemens Kirschbaum

A120 CONTEXT INAPPROPRIATE NEGATIVE AFFECT, EMOTION-MODULATED STARTLE AND ACTIVATED CORTISOL IN ELDERLY WOMEN.
Melissa A. Rosenkranz, Heather L. Urry, Marchell E. Thurow, Carol D. Ryff, Burton H. Singer, Ned H. Kalin, Daniel Muller, Richard J. Davidson

A122 NEUROENDOCRINE AND IMMUNE SYSTEM INTERACTIONS: GLUCOCORTICOIDS AND PROTECTION AGAINST T CELL-DEPENDENT DISEASE.
Rachelle Salomon, Melanie C. Ruzek, Bradley D. Pearce, Andrew H. Miller, Christine A. Biron

A123 CENTRAL ADMINISTRATION OF THE D_2-LIKE AGONIST 7-OH-DPAT ANTAGONIZES THE EFFECT OF MORPHINE ON IMMUNE STATUS.
Timothy B. Saurer, Kelly A. Carrigan, Donald T. Lysle

A124 CYTOSTATIC PROPERTY OF PROINFLAMMATORY CYTOKINES IN BREAST CANCER CELLS IS MEDIATED BY IMPAIRING DOWNSTREAM SIGNALING OF A GROWTH FACTOR RECEPTOR.
Wen-Hong Shen, Jian-Hua Zhou, Suzanne R. Broussard, Gregory G. Freund, Robert Dantzer, Keith W. Kelley

A125 ALTERATIONS IN CD8 T CELL POPULATIONS IN HSV-1 INFECTED MICE FOLLOWING ACUTE ADMINISTRATION OF MORPHINE.
Patricia A. Sheridan, Jan A. Moynihan

A127 CHRONIC RESTRAINT STRESS DURING EARLY INFECTION EXACERBATES THE SUBSEQUENT DEMYELINATING PHASE OF THEILER'S VIRUS.
Amy N. Sieve, Patrick Bridegam, Ralph Storts, Jane C. Welsh, Mary W. Meagher

A129 NFκB-RELATED GENE EXPRESSION IN ACTH TREATED LYMPHOCYTES.
Eric M. Smith, William Dalmeida, Jr., Thomas K. Hughes, Jr.

A130 IL-1 BETA INDUCES IMPAIRMENTS IN COGNITIVE BEHAVIOURS IN RATS.
Cai Song

A132 EXERCISE CHALLENGE INDUCES SYMPTOMS OF CHRONIC FATIGUE SYNDROME (CFS) AND ELEVATES C4A ONLY IN CFS PATIENTS.
Bristol Sorensen, James F. Jones, Monika Fleshner

A133 COPING STYLE AND CYTOKINE PRODUCTION IN INDIVIDUALS WITH MULTIPLE SCLEROSIS AND NORMATIVE CONTROLS: AN UNEXPECTED RELATIONSHIP.
Matthew R. Sorenson, Linda Witek-Janusek, Herbert L. Mathews

A134 PSYCHOLOGICAL STRESS AND CYTOKINE PRODUCTION IN MULTIPLE SCLEROSIS: RELATIONSHIP WITH DISEASE SYMPTOMATOLOGY.
Matthew R. Sorenson, Linda Witek-Janusek, Herbert L. Mathews

A135 MIGRATION OF BLOOD T CELLS IN SOCIALLY STRESSED F344 RATS.
Volker Stefanski, Stefan Reber, Andre Peschel

A137 IMMUNOREGULATION OF IL-6 SECRETION BY ENDOGENOUS AND EXOGENOUS ADENOSINE AND BY EXOGENOUS PURINERGIC AGONISTS IN SPLENIC TISSUE SLICES.
Rainer H. Straub, Georg Pongratz, Christian Genzler, Andreas Michna, Simone Baier, Frieder Kees, Werner Falk, Jergen Schelmerich

A138 NATURALLY OCCURRING DIFFERENCES IN STRESS RESPONSE PREDICT ANGIOGENIC CAPACITY, TUMOR GROWTH AND METASTASIS FORMATION.
Marc A.T. Teunis, Annemieke Kavelaars, Joost M. Bakker, Bart Ellenbroek, Alexander R. Cools, Cobi J. Heijnen

A139 INTERLEUKIN-1β DISRUPTS IN VIVO LONG-TERM-POTENTIATION(LTP) BUT NOT PERFORMANCE IN SPATIAL LEARNING AND RETENTION TASKS.
Lisa M. Thomson , Robert J. Sutherland

A140 MU OPIOID RECEPTOR ANTAGONISM MODULATES NK CELL CYTOTOXICITY BUT NOT CELLULARITY DURING RESTRAINT STRESS AND INFLUENZA A VIRAL INFECTION.
Raymond J. Tseng, John Hunzeker, Firdaus S. Dhabhar, David Padgett, John Sheridan

A141 LATERALIZED BRAIN ELECTRICAL ACTIVITY PREDICTS DIURNAL SALIVARY CORTISOL RHYTHM AND REPORTED AFFECT.
Heather L. Urry, Melissa Rosenkranz, Marchell Thurow, Ned H. Kalin, Daniel Muller, Gayle D. Love, Carol Ryff, Burton Singer, Richard J. Davidson

A142 PLACEBO RESPONSE: POTENTIAL NEURO-PSYCHOLINGUISTIC MECHANISMS.
Ned Vankevich

A143 INFLAMMATORY VANILLOID (VR1) RECEPTORS ARE ACTIVATED BY PARTICLE SURFACE CHARGE.
Bellina Veronesi, Guangwei Wei

A144 THE DIFFERENTIAL EFFECTS OF METHADONE AND MORPHINE UPON THE IMMUNE SYSTEM OF RATS.
Kimberly R.N. Vietti, Thomas J. Martin, Susy A. Kim, Richard J. Weber

A145 STUDIES OF THE MECHANISM OF ANDROGEN-INDUCED THYMOCYTE APOPTOSIS.
Susan M. Viselli, Ann M. Szajkovics, Habib Shaikh

A147 STRESS, SOCIAL SUPPORT, AND IMMUNE RESPONSE IN WOMEN WITH BREAST CANCER.
Diane Von Ah, Duck-Hee Kang

A148 MU-OPIOID RECEPTOR IS INVOLVED IN RESTRAINT STRESS INDUCED IMMUNOSUPPRESSION.
Jinghua Wang, Richard Charboneau, Roderick A. Barke, Horace H. Loh, Sabita Roy

A149 JAK/STAT SIGNALING PATHWAY PROTECTS AGAINST NEUROPATHOGENIC ACTIONS OF INTERFERON (IFN)-α.
Jianping Wang, Iain L. Campbell

A150 HYPOCORTISOLISM AND INCREASED GLUCOCORTICOID SENSITIVITY OF PRO-INFLAMMATORY CYTOKINE PRODUCTION IN BOSNIAN WAR PTSD VICTIMS.
Jutta M. Wolf, Lijlana Joksimovic, Nicolas Rohleder, Clemens Kirschbaum

A151 GENETIC AND DEVELOPMENTAL EFFECTS OF INTERLEUKIN-1 SIGNALING ON PAIN.
Gilly Wolf, Yehuda Shavit, Inbal Goshen, Kiyoshi Takeda, Kersten Iverfeldt, Raz Yirmiya

A152 SEX DIFFERENCES AND PREDICTORS OF RAPID PROGRESSION IN SIV_{SME660} INFECTED MACAQUES.
Julie M. Worlein, Jennifer Leigh, Mark Laudenslager

A153 STRESS-RELATED MODULATION OF MATRIX METALLOPROTEINASE EXPRESSION.
Eric V. Yang, Cynthia M. Bane, Robert C. MacCallum, Janice K. Kiecolt-Glaser, William B. Malarkey, Ronald Glaser

A154 INTERLEUKIN-2 POTENTIATES STIMULANT-INDUCED INCREASES IN STEREOTYPED BEHAVIOR IN RODENTS.
Steven S. Zalcman, Michelle Wallenstein

A156 EFFECTS OF DAILY EXHAUSTIVE EXERCISE ON ALLOGENEIC TUMOR GROWTH IN MICE.
Mark R. Zielinski, Peggy Horn, K. Todd Keylock, Jeffrey A. Woods

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- all late-breaking abstracts are also included in Poster Session 2.

LB#1
CORRELATION OF IL-1RA/IL-1β BALANCE WITH COGNITIVE IMPAIRMENT DURING BRAIN DELAYED-TYPE HYPERSENSITIVITY RESPONSE TO BACILLUS CALMETTE-GUERIN IN RATS

Rose-Marie Bluthé[1], Karine Palin[1], Danièle Verrier[1], Viviane Tridon[1], Adrian Bristow[2], Robert Dantzer[1], & Jacques Lestage[1]
[1]Institut François Magendie, INSERM U394, France [2]NIBSC, Potters Bar, UK

Abstract
Multiple sclerosis (MS) is characterized by a chronic inflammatory reaction within the central nervous system. Cytokines play a critical role in this peripheral immune cell mediated process. The activity of a main inflammatory cytokine, interleukin-1β (IL-1β), is regulated by a specific endogenous antagonist, interleukin-1 receptor antagonist (IL-1Ra). The balance between IL-1Ra and IL-1β is thought to influence the initiation of inflammatory diseases. IL-1β and IL-1Ra concentrations were measured in the course of an experimental model of MS, developed by the group of Pr. Perry (UK), based on delayed-type hypersensitivity (DTH) response to bacillus Calmette-Guerin (BCG) in the hippocampus of Lewis rats. We show that the immune cell-mediated initiation of the early DTH response was correlated with the significant decrease of brain IL-1Ra expression and the maintainance of elevated IL-1β level. The IL-1Ra/IL-1β balance was strongly diminished in brain compartment and could explain at least partially the initiation of chronic brain inflammation after BCG challenge. In addition, impairment of spatial memory was also shown during the early phase of DTH response whereas no decrease of the number of hippocampic neurons was not yet detectable. Consequently, variation of IL-1Ra/IL-1β balance in favor of IL-1β correlates simultaneously with the initiation of immune cell-mediated brain lesions and impairment of spatial memory. The present work points out a crucial role of cytokine balance in cognitive function during inflammatory process. This research was supported by the French Association for Multiple Sclerosis Research (ARSEP).

LB#2
INSULIN RESISTANCE: DOES IT CAUSE CYTOKINE RESISTANCE?

G. F. Freund

University of Illinois, College of Medicine at Urbana, Champaign, USA

Insulin receptors and cytokine receptors share a number of intracellular signaling pathways. Therefore, conditions that induce insulin resistance, like stress, obesity and type 2 diabetes mellitus, can potentially cause cytokine resistance. It has been known for some time, that the cytokine TNFα and the growth factor PDGF induce insulin resistance. Their ability to inhibit insulin signaling relies on activation of serine kinases which serine phosphorylate insulin receptor substrates (IRSs). The impact of serine phosphorylated IRSs is twofold: they serve as poorer targets for the insulin receptor and they are degraded by the proteasome. Importantly, IFN-α, -β, -γ and IL-2, -4, -7, -9, -13, -15 also use the IRS family members in their signal transduction. These cytokines propagate their intracellular signals by activating the Janus kinases (Jaks) and the Jaks, like the insulin receptor, utilize serine phosphorylated IRSs poorly as substrates. Thus, through IRS serine phosphorylation and IRS proteasomal loss, cytokine signaling can be diminished in the insulin resistant state. This disruption in cytokine signaling may help explain why subacute chronic inflammation is seen with insulin resistance.

LB#3
EPHEDRINE AND GENETIC SUSCEPTIBILITY TO SLE: A RECIPE FOR TROUBLE

Chad A Hudson , Jane Kasten-Jolly , Victor C Huber , David A Lawrence
Wadsworth Center for Laboratories & Research, USA

Abstract

An overactivation of the sympathetic nervous system (SNS) has been shown in Systemic Lupus Erythematosus (SLE) patients, and pharmacological overstimulations of the SNS have induced SLE. A possible mechanism for SNS-induced SLE is inhibition of Th1 T cells via β2 adrenergic receptors, thus yielding a shift towards Th2. Ephedrine is a non-specific adrenergic receptor agonist found in metabolic enhancer supplements. To determine if ephedrine could exacerbate/induce SLE, (-)-ephedrine was pipet-fed in 5% sucrose water (10µl) to 6-wk old male and female BALB/c and lupus-prone New Zealand Mixed 391 (NZM391) mice. NZM391 mice are genetically predisposed to lupus manifestations such as glomerulonephritis and anti-nuclear autoantibody production. Daily doses of 100µg, 50µg or 0µg were given for three months, treatments equivalent to advised human usage. Starting one month after initial administration, sera were collected monthly and assessed for anti-DNA, urea nitrogen (SUN), creatine kinase activity (CKA) and IgG2a/IgG1. Compared to sucrose controls, NZM391 treatment groups had significant increases in SUN (50µg: Months 1-4, 100µg: Months 2-4) and anti-DNA (50µg: Months 3-4, 100µg: Months 2-4). Both treatments showed a significant decrease in average CKA over the 4 months. BALB/c mice showed none of these significant differences. NZM391 treatment groups also showed a decreased IgG2a/IgG1 (50µg: Month3, 100µg: Months 1-4 (Month3 significant)). These results indicate ephedrine can exacerbate SLE in lupus-prone mice at doses comparable to the recommended doses of ephedrine-containing supplements and suggest SNS activation plays a causal role in SLE via modulation of T cell activities.

LB#4
ACUTE PSYCHOLOGICAL STRESS ACCELERATES CUTANEOUS WOUND HEALING IN MALE SIBERIAN HAMSTERS (PHODOPUS SUNGORUS) EXPOSED TO SHORT DAY LENGTHS

Steven G Kinsey, Brian J Prendergast, & Randy J Nelson
Ohio State University, USA

Abstract
Animals inhabiting high latitudes have evolved mechanisms to contend with seasonal environmental changes. Siberian hamsters inhabit a region characterized by seasonal fluctuations in temperature and food availability. Consequently, they exhibit adaptations that serve to minimize energy expenditure during winter. Reproduction, for example, occurs exclusively during summer, and gonadal function exhibits marked seasonal variation that can be mimicked by manipulating day length. Changes in day length also alter several indices of immune function. This experiment tested the hypothesis that photoperiodic changes in immune function are integrated at an organismal level as reflected by the ability to heal a cutaneous wound. Given the well-documented effects of psychological stressors on immune function, we also tested the hypothesis that photoperiod modulates the effects of acute stress on wound healing. Male hamsters were housed in long (16L:8D; LD) or short (8L:16D; SD) day lengths for 8±1 weeks. SD-treatment was sufficient to induce winter reproductive status. Hamsters then received a dermal punch wound. For 3 days prior to, and 5 days after, wounding, hamsters were subjected to either 2 h daily restraint stress or a control treatment. Wounds were measured daily to quantify healing. Wounds of LD-hamsters healed significantly faster than did those of SD-hamsters. Restraint stress significantly accelerated healing but did so only in SD-hamsters. The results suggest that the enhancing effects of psychological stressors on immune function are apparent only when reproductive function is suppressed. In nature, enhanced wound healing coincident with the breeding season and territorial defense may be adaptive.

LB#5

RELATIONSHIP QUALITY AND IMMUNE RESPONSE IN MID-LIFE ADULTS.

Gayle Love[1], Cathlyn Leitzke[1], Carol Ryff[1], Burt Singer[1,2], & Daniel Muller[1]
[1]University of Wisconsin-Madison, [2]Princeton University, USA

Abstract
A growing literature links quality of social relationships and immune response. The purpose of the study was to focus on cumulative relational experience, with the specific hypothesis that persons with high spousal intimacy would have better responses to influenza vaccination. A subsample of subjects (n=89, 49 men and 40 women) from the Wisconsin Longitudinal Survey were given flu shots prior to the 1998-1999 flu season. Blood samples were obtained at vaccination and at 2 weeks, 4 weeks, and 6 months post vaccination. Antibody levels were determined via Hemagglutination Inhibition Assay. Intimacy was assessed via the Personal Assessment of Intimacy in Relationships (PAIR). Scale scores for 4, out of 6, dimensions of intimacy (emotional, sexual, intellectual, recreational) are converted to low/high dichotomous variables using gender specific median cut points. We find that women with high sexual, emotional, or intellectual intimacy are more likely to have a good antibody response. Among men, however, the predicted relationship is observed only among those with high emotional intimacy. Consistent with prior observations among those with high cumulative intimacy, women were significantly more likely to have a good antibody response, across time, than men. Furthermore, among those with high sexual intimacy, women are significantly more likely than men to have a good antibody response shortly after vaccination. In summary, persons scoring high on multiple dimensions of intimacy are more likely to make a good antibody response to influenza vaccination.

LB#6

ACUTE EFFECT OF QI-TRAINING ON CYTOTOXIC ACTIVITY OF NATURAL KILLER CELL WITHOUT SUBSETS CHANGE

Nam Ill Mo[1] , Myung Soo Lee[2], Seong Min Jeong[1], Sun-Rock Moon[3], Junghee Lee[4], & Hoon Ryu[4]
[1]ChunDoSunBup Institute, Kangwon Province, [2]Professional Graduate School of Oriental Medicine, [3]Department of Radiation Oncology, Wonkwang University Hospital, Iksan, South Korea, [4]Department of Neurology, Harvard Medical School, Boston, USA

Abstract
The present study investigated the effect of traditional psychosomatic intervention (Chun Do Sun Bup (CDSB) Qi training) on cellular immunity. The acute change of natural killer cell activity was determined at three time points, before (-10 min), immediately after, and two hours after the CDSB Qi training. Assays for cytotoxicity were analyzed by using a non-isotopic method which measures the LDH released from the tumor target cell. NK cell subsets (CD57) were examined by flow cytometry. An increase in NK cell cytotoxicity was observed immediately after CDSB Qi training. NK activity was significantly increased more than 1.6 times compared to the basal value before Qi training ($P<0.01$). The increased level of NK activity recovered to basal level within two hours. The resting (sedentary) subjects did not show any change of specific lysis for the resting 3 hours. There was no change in the number of NK cell subset after the CDSB Qi training. No correlation between the NK cytotoxicity and the NK cell number was found. These data suggest that the CDSB Qi training has an acute stimulatory effect on NK cell activity, exerting a beneficial immunological effect, but has no effect on the fluctuations of phenotypical changes of NK cell subset in men.

LB#7
NEUROIMMUNE INTERACTIONS IN THE PROGRESSION OF ORAL LICHEN PLANUS

Paolo Prolo[1], Elaina Cajulis[1], Marco Carrozzo[2], Sergio Gandolfo[2], Russell Christensen[1], Andrea Dovio[3], & Francesco Chiappelli[1]
[1]UCLA School of Dentistry, [2]Oral Medicine Section, University of Turin, Italy, [3]Internal Medicine, University of Turin, Italy

Abstract
Oral lichen planus (OLP) is a T cell-mediated inflammatory disease. Lesions characteristically lack B cells, plasma cells, immunoglobulin or complement. It is recognized as a model for neuroimmunology research in oral biology and medicine. Progression from the reticular to the atrophic, erosive and bullous forms may derive from psychoemotional turmoil. OLP lesions may be invaded by activated T cells, show a dense subepithelial lymphocytic infiltrate, and extensive epithelial basal cell destruction. The purpose of this study was to test whether or not OLP is associated with significant neuro-immune outcomes by testing the association between systemic markers of cellular immunity and mood states, with clinical stages of OLP (i.e., atrophic vs. erosive vs. bulloue lesions), and testing neuroimmune characteristics of histologic biopsies obtained from OLP lesions. We recruited patients with OLP at different stages of disease. We obtained lesion biopsies and normal soft mucosa tissue from every subject. We tested by immunohistochemistry the relative distribution of CD3+, CD25+, CD69+, CD26+ cells, and cell populations undergoing apoptosis. We evidenced perivascular apoptosis, parenchymal T cell invasion (CD3 staining), activated leukocytes in the parenchyma (CD69 staining), leukocytes expressing exopeptidase, CD26 in the parenchyma, which might metabolize endogenous opioids at the leukocyte plasma membrane. This psychoimmune study revealed alterations in circulating lymphocytes and in POMS scores at different stages of disease. It also revealed significant correlations between circulating CD4+ and memory (CD45Ra-) CD4+ cells and with POMS scores. In conclusion, our data emphasize the need to study neuroendocrine-immune inter-relationships in OLP in greater detail.

LB#8
NEURODEGENERATIVE AND IMMUNE CHANGES IN HUNTINGTON'S DISEASE

Sandra L Rogers, Robert A Bornstein, & Sandra K Kostyk
The Ohio State University-COM & PH, USA

Abstract
We investigated the prospective relationship between neurological and neuropsychological deterioration and changes in immune function in Huntington's Disease (HD). We predicted that a unique pattern of immune alterations would be apparent in HD, that these alterations would be present in the early stages of HD, and neuropsychiatric evaluations would correlate with immune changes. We recruited 6 individuals in early HD and 6 controls and followed them over 6 months. Individuals were evaluated on the Unified Huntington's Disease Rating Scale (UHDRS), which includes neurological staging and neuropsychiatric tests. Neuropsychiatric tests and immune measures were stable within groups for both time points, except the word sub-score on the Stroop Test which showed a significant decrease in the HD subjects from baseline to 6 months. The NK assay in the HD group demonstrated significantly higher cytotoxicity. Absolute numbers and percentages of NK cells did not differ, however, the HD population had a significantly higher percentage of CD56 DIM cells. Other leukocyte differences, included significantly higher percentages of CD3+, CD8+, and significantly lower CD4+, and CD4+/CD8+ ratios. sIL-2R and IL-6 were significantly elevated in HD. On neuropsychological testing, Symbol Digit Modality, Stroop Color and Word scores showed a significant negative correlation with NK cell cytotoxicity and IL-6 levels. These data indicate that HD individuals show altered immune functioning early in the disease process and that these differences are unique compared to other neurodegenerative disorders. Neuropsychological tests correlated with immune functioning and may facilitate prediction of neurodegenerative changes.

LB#9
STRESS AND HUMAN IMMUNITY: META-ANALYTIC RESULTS AND HISTORICAL TRENDS

Suzanne C. Segerstrom[1] , Gregory E. Miller[2]
[1]University of Kentucky, [2]Washington University, St. Louis

Abstract
Almost a decade ago, Herbert and Cohen (1993) published a seminal meta-analytic review of 44 studies that explored the circumstances under which psychological stress associated with parameters of the human immune system. Since that time, this area of inquiry has grown tremendously, both in terms of the volume of studies that have been published and the sophistication of the questions motivating these studies. In this presentation we will offer an updated meta-analysis of research on stress and human immunity. The presentation will focus on historical changes in study design and immune outcomes studied, as well as the effects of the maturation of this literature on reported effect sizes.

LB#10
EFFECTS OF AUTOGENIC AND BIOFEEDBACK TRAINING ON ANXIETY AND SIDE EFFECTS OF CHEMOTHERAPY IN LEUKEMIC CHILDREN

Somporn Kantaradussa Triamchaisri[1], Chatsiri Mekwiwattanawong[2] Suree Kanjanawong[3], Thitawan Thamapirot[4], & Terdsuk DetKhong[5]

Abstract

This study aimed to assess the effects of relaxation technique, autogenic and biofeedback training for relieving the suffering from side effects of chemotherapy and general anxiety among leukemic children. A quasi experimental pretest-posttest design was used in the study. Twenty leukemic patients were recruited for this study. Ten subjects were trained in autogenic and biofeedback for 9 days (3 times/day). Ten others subjects underwent the routine care procedure. Data collection was done by using the interview technique and recording physical data. The difference between the two groups was calculated by using "the Mann-Whitney U Test" and the change within group between pretest and post test was calculated by using " The Wilcoxon Matched Pairs Signed Rank Test". Results of the study showed that the experimental group had significantly decreased anxiety, pulse, respiration, blood pressure and increase skin temperature after treatment. The control group experienced significantly increased side effects of chemotherapy between the pretest and the posttest, but anxiety, pulse, respiration, blood pressure and finger temperature did not change. The comparison between the two groups showed the experimental group felt significantly more relaxed, experienced less nausea/vomiting, worry and anxiety, and better sleep and appetite than the control group. Anxiety, side effects of chemotherapy, pulse respiration, and blood pressure in the experimental group were lower than those of the control group. The leukemic children experienced decreased anxiety and side effects of chemotherapy when they accepted the autogenic and biofeedback technique.

Symposium 1

FETAL, NEONATAL AND PUBERTAL ORIGINS OF ADULT DISEASE

Organizer: Deborah Hodgson[1]

Speakers: Deborah Hodgson[1], Caroline McMillen[2], Martha McClintock[3], Gayle Page[4] & Douglas Granger[5]

[1]Laboratory of Neuroimmunology, University of Newcastle, Australia, [2]Department of Physiology, Adelaide University, Adelaide, Australia, [3]Department of Psychology, The University of Chicago, USA, [4]Faculty of Nursing, Johns Hopkins University, USA, [5]Penn State, USA.

Abstract

Glucocorticoids (GCs) are essential for normal brain development. However, there is growing evidence that exposure of the developing brain to excess GC can affect behaviour, neuroendocrine and immune functions, and promote the onset of age-related disease. In this symposium Caroline McMillen will present data focusing on the responses of the fetal cardiovascular, sympatho-adrenal, HPA, and renin–angiotensin systems to experimental restriction of placental function in sheep, and discuss the consequences of these adaptations for fetal, neonatal, and adult health. Deborah Hodgson will present findings on the impact of neonatal stress. Her studies indicate that neonatal exposure to endotoxin is associated with impaired tumor immunity, hypersecretion of corticosterone, altered glucose metabolism and increased anxiety-related behavior in adulthood. Martha McClintock will discuss her research on the effects of social interactions and isolation during puberty on susceptibility to mammary tumors and infectious pneumonia in later adulthood. Her results indicate that the early social environment affects not only the HPA axis, but also humoral and cellular immune function. Gayle Page will present results indicating the age at which aspects of natural immunity become sensitive to sympathetic modulation provides a unique index of the impact of neonatal stress upon the immune system. Finally, Douglas Granger will discuss his research on the developmental origins of individual differences. His findings demonstrate that immune stimuli represent a special feature of

the physical environment with the potential to drive social development. This symposium brings together provocative evidence indicating that early life events are critical predictors of long-term health and immune outcomes later in life.

NEONATAL EXPOSURE TO BACTERIAL ENDOTOXIN IS ASSOCIATED WITH INCREASED ANXIETY-RELATED BEHAVIOUR AND ALTERED METABOLIC RESPONSIVITY IN ADULTHOOD.

Deborah M. Hodgson , James March, & Frederick Walker
University of Newcastle, Laboratory of Neuroimmunology, Australia

Abstract
Exposure to neonatal stress, in the form of bacterial endotoxin, can result in permanent alterations to the functioning of the hypothalamic-pituitary-adrenal (HPA) axis. This study investigated the long term implications of neonatal bacterial endotoxin exposure in rats on a) neuroendocrine responsivity, as measured by serum corticosterone levels, b) metabolic responsivity, as measured by the ability of insulin to clear a standardized glucose load and, c) behavioural anxiety, measured using the elevated plus maze. Neonatal Fischer 344 rats were injected intraperitoneally with Salmonella enteriditis (0.05mg/kg), or the equivalent volume of vehicle (PBS) on postnatal day 3, 5, and 7. In adulthood neonatal endotoxin-treated animals displayed significantly higher anxiety levels compared to saline-treated animals in the elevated plus maze ($p<0.001$). To assess insulin responsivity, half of the animals from each condition were placed in restraint for 12 hours. Immediately following the period of restraint, all animals were subjected to an intraperitoneal glucose tolerance test (IP-GTT) (glucose 2g/kg, ip). Blood samples were taken at 30, 60 and 120 minutes post IP-GTT. Endotoxin-treated animals were found to exhibit an insulin response similar to that found in insulin resistance. It was also determined that endotoxin treated animals had a potentiated serum corticosterone response to stress ($p<0.05$) and larger adrenal glands ($p<0.05$) compared to saline treated animals. These findings demonstrate that neonatal exposure to bacterial endotoxin produces long term alterations in neuroendocrine, metabolic and behavioural responsivity in later life, and that this may have long term consequences for physical and mental well-being.

PRENATAL UNDERNUTRITION, GLUCOCORTICOIDS AND THE EARLY ORIGINS OF HYPERTENSION

Isabella Caroline McMillen & Lisa Jane Edwards
Department of Physiology, Adelaide University, Australia

Abstract
A world wide series of epidemiological studies has shown that poor intrauterine growth is associated with an increased prevalence of cardiovascular disease, non insulin dependent diabetes mellitus and the Metabolic Syndrome in adult life. It has been postulated that a reduced intrauterine nutrient supply perturbs fetal growth and concomitantly alters or programs the structure and function of developing systems. One outcome of either a suboptimal placental or maternal nutrient supply, is exposure of the fetus to excess glucocorticoids which act to restrict fetal growth and to program permanent changes in the cardiovascular, endocrine and metabolic systems. There have been relatively few studies, however, in species, such as the human or sheep in which the fetal hypothalamo-pituitary-adrenal (HPA) axis has the capacity to respond to intrauterine stressors during late gestation. Recent work in the sheep has shown that poor placental function or inadequate maternal nutrition each result in an altered fetal substrate supply, an increased exposure of fetal tissues to glucocorticoids and specific changes in the regulation of arterial blood pressure through different mechanistic pathways. Furthermore it has been demonstrated that the timing, type and duration of maternal nutrient restriction are each important in determining the fetal adaptive responses and their pathophysiological sequelae in later life. These experimental studies provide a mechanistic basis upon which to identify the key elements of the causal pathways that underpin the early origins of adult disease

and the associated opportunities for therapeutic intervention.

PUBERTY, SOCIAL ISOLATION AND INCREASED RISK FOR INFECTIOUS AND MALIGNANT DISEASE DURING AGING

Martha K. McClintock
Department of Psychology, Institute for Mind and Biology, Univ. of Chicago, USA

Abstract
The aim of our research is to use an animal model to identify mechanisms and pathways by which social isolation in adolescence and middle age increases risk for infectious and malignant disease late in the life span. To date, our research has linked pathological outcomes with life-long individual housing of female Sprague-Dawley rats. Isolated female rats were more likely to develop spontaneous tumors or to die at an earlier age of opportunistic infectious pulmonary disease. At 18 months of age, 61% of socially isolated females had palpable mammary tumors whereas only 25%of group-housed females did. Moreover, among rats that had tumors by the end of their lives, isolated females developed them at three times the rate of group housed females. Remarkably, animals who are not isolated until the end of puberty, beginning in adulthood, had aging patterns similar to group housed animals, suggesting that puberty may be a sensitive period when social conditions affect the developing immune system. We have also found that patterns of morbidity and mortality are accompanied by differences in reproductive aging across these two conditions. Average ages for entering the acyclic phase of reproductive occurs 22% earlier in individually housed rats than in group housed rats. Currently, our working hypothesis is that social conditions that enhance immune and reproductive function early in the life span, result in accelerated aging. This acceleration may be manifest as a more rapid shift towards Th2 type responses in the isolated animal, resulting in greater risk to infectious and inflammatory disease.

DEVELOPMENT OF A MATURE NK SUPPRESSIVE RESPONSE TO THE β-ADRENERGIC AGONIST METAPROTERENOL

Gayle G. Page
Johns Hopkins University School of Nursing, USA

Abstract
Both acute stress and β-adrenergic stimulation have been shown to suppress natural killer (NK) cell function in mature but not prepubescent rats. This study was undertaken to ascertain the age at which inbred Fischer 344 rats exhibit a mature response to the NK suppressive effects of metaproterenol. In vivo NK function was assessed using the syngeneic mammary adenocarcinoma cell line, MADB106. Following intravenous injection, MADB106 cells seed and colonize only in the lungs, processes shown to be sensitive to NK cells. In-house bred rats aged 33-40 days (prepubescent), 8.5-9, 13-14.5, and 18-19 weeks, plus 5-11 month old NIA-bred Fischer rats (age-matched by assay) were injected subcutaneously with either saline, or 0.8 or 5.0 mg/kg metaproterenol 1 hour before radiolabeled MADB106 cell injection into the tail vein ($4x10^5$ cells/kg). Lungs were removed 22 hours later to assess their radioactive content. Among the vehicle-injected animals, there was a statistically significant age by sex interaction; whereas the females exhibited "mature" levels of tumor retention at 8.5-9 weeks, the males were later, at 13-14.5 weeks. Metaproterenol administration resulted in a statistically significant interaction among factors: age, sex and metaproterenol dose. The sex by metaproterenol interaction previously observed in mature rats, greater male responsiveness to the β-agonist compared to the female, emerged at 13-14.5 weeks. These findings show that mature lung retention levels of the NK-sensitive MADB106 tumor occur by 13-14.5 weeks, as does the mature sex differences in the tumor-promoting impact of metaproterenol on MADB106 lung retention.
Supported by NIH grant NR07742.

DEVELOPMENTAL EFFECTS OF EARLY IMMUNE STRESS ON SOCIAL BEHAVIOR

Douglas Granger
Department of Biobehavioral Health, The Pennsylvania State University, USA

Abstract
Among the enduring issues in developmental science are the origins of individual adaptations to changing environments, and how these influences integrate with maturational processes to produce individual differences in social behavior. It has been argued that integration occurs in response to environmental forces that are predictable and omnipresent but non-obvious. The assumption guiding this program of research is that immune stimuli (e.g., viruses, bacteria) represent a class of non-obvious (not detectable by the five classic senses) features of the physical environment with the potential to affect social development. The origins of individual differences in social development are examined in relation to early stress (neonatal exposure to endotoxin) and social milieu (maternal behavior). The findings of three developmental studies using mice selectively bred for differences in social behavior will be presented that raise novel questions regarding the openness of behavioral systems to effects of non-obvious but omnipresent features of the environment, such as antigenic load, how these effects are integrated to affect social development and psychopathology, and the nature of intrinsic factors that contribute to individual differences in sensitivity to early stressors.

Symposium 2

MODULATION OF BRAIN AND PERIPHERAL HOST DEFENSE BY OPIOIDS, CANNABINOIDS, AND NICOTINE

Organizers: Burt Sharp[1] & Richard Weber[2]

Speakers: Norbert Kaminski[3], Tom Rogers[4], Phil Peterson[5], & Mohan L. Sopori [6]
[1]University of Tennessee, [2]University of Illinois College of Medicine, [3]Michigan State University, [4]Temple University, [5]Hennepin County Medical Center and University of Minnesota, [6]Lovelace Respiratory Research Institute and University of New Mexico, USA

More than two decades of international research have established the immunomodulatory effects of both endogenous and exogenous opioids and cannabinoids, and of nicotine. Commonly acting through 7-transmembrane G protein-coupled receptors expressed by cells involved in brain and peripheral host defense, both opioids and cannabinoids affect intracellular signal transduction pathways that regulate the expression of cytokines, chemokines, cellular proliferation and viral production. In contrast, nicotine affects the peripheral immune system indirectly by interacting with central nicotinic cholinergic receptors. In this symposium, sponsored collaboratively by the Society on NeuroImmune Pharmacology (SNIP) and the PsychoNeuroImmunology Research Society, leading scientists will present basic insights pertaining to the mechanisms and therapeutic implications of immunomodulation by opioids, cannabinoids and nicotine in the brain and periphery. The first presentation will focus on T-cells, highlighting the cannabinoid receptor-dependent and independent effects of these compounds on intracellular signaling. Then, the effects of opioid receptors on chemokine receptor expression and function, which involves regulatory cytokines, will be dissected. In the third presentation, opioid modulation of glial function, through mu, kappa and delta opioid receptors expressed by microglia and astrocytes, will be considered from the perspective of infection, inflammation, and neurodegeneration in the central nervous system. Lastly, mechanisms underlying the anergy-inducing effects of chronic nicotine on peripheral lymphocyte function

will be discussed in light of recent data demonstrating the role of CNS nicotinic cholinergic receptors and the generation of interleukin-1.

CANNABINOID REGULATION OF T-CELL NFAT AND IL-2

Norbert E Kaminski , Tong-Rong Jan, Barbara L Faubert Kaplan, & Gautham K Rao
Michigan State University, Pharmacology & Toxicology Dept, USA

Abstract
The calcium-mediated activation of the nuclear factor of activated T cells (NFAT) is a major signal transduction pathway critical for T cell function. NFAT DNA binding, which is essential for the transcriptional regulation of interleukin-2, as well as other T cell-derived cytokines, is disrupted by immunosuppressive cannabinoids. Investigations aimed at elucidating the molecular mechanism responsible for cannabinoid-mediated inhibition of IL-2 gene expression demonstrated disruption of intracellular calcium $[Ca^{+2}]_i$ regulation after cannabinoid treatment. The plant-derived cannabinoids, cannabinol, and to a greater extent, Δ^9-tetrahydrocanabinol (Δ^9-THC), produced a rapid, sustained and concentration dependent rise in $[Ca^{+2}]_i$. Interestingly, ionomycin pretreatment of splenocytes prior to activation inhibited the expression of IL-2 implicating the involvement of a premature calcium rise in the inhibition of IL-2 by cannabinoids. As in mouse leukocytes, Δ^9-THC treatment of HPB-ALL cells also produced a rapid and marked rise in $[Ca^{+2}]_i$ that was attenuated by the CB2 antagonist, SR144528. Conversely, Δ^9-THC induced only a modest rise in $[Ca^{+2}]_i$ which was not attenuated by SR144528 in Jurkat E6.1 cells. Northern blot analysis revealed CB2 mRNA expression in both HPB-ALL and Jurkat E6.1 cells; however, the later exhibited multiple mRNA transcripts all of aberrant size for CB2. Neither of the cell lines expressed CB1 mRNA. Interestingly, SR144528 did not attenuate cannabinoid-mediated inhibition of IL-2 gene expression in splenic T cells or HPB-ALL cells. Collectively, these studies demonstrate that cannabinoids disrupt $[Ca^{+2}]_i$ through a CB2-dependent mechanism but additional cannabinoid-receptor-independent mechanisms are involved in the disruption of IL-2. (Supported by NIH Grant DA07908)

OPIOID MODULATION OF CHEMOKINE RECEPTOR EXPRESSION AND FUNCTION

Thomas J. Rogers
Temple University School of Medicine, USA

Abstract
Both the opioids and chemokines are well established immunomodulatory factors, and recent evidence suggests that the opioid and chemokine receptors cross-talk through a process of heterologous desensitization. We sought to examine the capacity of a number of endogenous and exogenous opioids to modulate the function of several chemokine receptors, including CCR5 and CXCR4, the two major HIV coreceptors. We found that the functional activities of the several, but not all, chemokine receptors was inhibited following opioid pretreatment. Conversely, studies also showed that treatment with several chemokines resulted in the inhibition of opioid receptor function. The nature of the signaling pathways induced by these two families of G protein-coupled receptors results in several critical changes in cellular function. These results have significance for our understanding of the nature of both the regulation of leukocyte trafficking, as well as the regulation of chemokine receptor function in inflammatory disease states.

OPIOID MODULATION OF ACTIVATED GLIAL CELLS: KEEPING THE 'FRIENDLY' FIRE FRIENDLY

Phillip K Peterson[1,2], Shuxian Hu[1, 2], Genya Gekker[1, 2], Wen Sheng[1, 2] James Lokensgard[1, 2]
[1]Minneapolis Medical Research Foundation, [2]University of Minnesota, USA

Abstract
Although glial cells have been long recognized for their supportive functions within the central nervous system (CNS), it has only been in the past decade that their involvement in brain damage has been considered. Because microglia and astroglia express opioid receptors, our research team has postulated that opioids could be exploited for treatment of certain CNS infections and neuroinflammatory disorders. In studies of purified primary microglial cell and astrocyte cultures, we have shown that these glial cells produce abundant amounts of cytokines and chemokines. When cocultured with neurons, these and other mediators produced by activated glial cells induce apoptotic neuronal cell death. Microglial cells are the only brain cell type that supports HIV-1 replication, and activated microglia play a pivotal role in development of AIDS dementia. We have shown that kappa-opioid receptor ligands suppress HIV-1 replication and inhibit production of the neurotoxin, quinolinic acid, by HIV-1-infected microglia. Recent findings in our laboratory implicate chemokines and cytokines produced by herpes simplex virus-1-stimulated microglia in the neuropathogenesis of herpes encephalitis. Reports on the effects of morphine in animal models of herpes encephalitis suggest that opiates could have therapeutic potential in this serious brain infection. In addition to CNS viral infections, activated glia have been implicated in a number of neurodegenerative and neuroinflammatory disorders. Thus, activated microglia and astrocytes may represent neuropharmacologic targets for a variety of CNS diseases.

ROLE OF IL-1β IN NICOTINE-INDUCED IMMUNOSUPPRESSION

Mohan L. Sopori
Lovelace Respiratory Research Institute, Albuquerque, NM, USA

Increasing evidence suggests a bidirectional communication between the central nervous system and the immune system through shared signal molecules such as cytokines and neurotransmitters. Chronic ICV administration of relatively very small concentrations of nicotine (NT) into the lateral ventricle of normal or adrenalectomized LEW rats causes a significant reduction in the antibody-forming cell response to SRBC and impairs the antigen receptor-mediated signaling in T cells. These effects are blocked by the nicotinic acetylcholine receptor (nAChR) antagonist mecamylamine or by ganglionic blockers chlorisondamine and hexamethonium. These data suggest that some of the effects of NT on the immune system are mediated through nAChRs in the brain and are dependent on the autonomic nervous system but independent of the activation of the hypothalamus-pituitary-adrenal axis. Interestingly, chronically NT-treated animals exhibit enhanced expression of IL-β mRNA in the brain, and this increase is kinetically correlated to the induction of immunosuppression. In addition, unlike the control wild-type mice, IL-β receptor KO mice do not exhibit NT-induced immunosuppression. Furthermore, ICV administration of IL-1 induces Fyn kinase activity in the spleen that is abrogated by pretreatment with chlorisondamine. These data suggest that IL-1β may have a critical role in mediating the effects of NT on the neuroimmune communication.

Symposium 3

DEPRESSION AND IMMUNITY: NEW LIGHT ON A DARK SUBJECT

Organizer: Steven Schleifer[1]

Speakers: Michael Irwin[2], Michael Maes[3], Zul Merali[4], & Andrew Miller[5]

[1] UMDNJ-New Jersey Medical School, [2] ULCA Neuropsychiatric Institute, [3]University Hospital of Maastricht, The Netherlands, , [4]University of Ottawa, Canada
[5]Emory University, Georgia, USA

Abstract

Depressive disorders are among the most extensively studied clinical psychiatric conditions in the history of psychoneuroimmunology. While there is some consensus about certain immune correlates of depression, the literature is challenged by considerable variability in the findings. This variation may be due to: 1) a lack of homogeneity in the diagnostic entity, 2) mediation by complex symptom dimensions, such as sleep, quality of mood disturbance, and clinical course, 3) modulation by age, gender and other comorbid psychiatric conditions, and 4) medication effects. The clinical context of depressive disorders (e.g., dysthymia vs. major depression), differences between disorders (e.g., anxiety vs. depression) and distinction from "subclinical" depressive states (e.g., normal grief) will be reviewed, highlighting immune-related observations. Mediating variables will be discussed, especially sleep disturbance, which is implicated in immune dysregulation and typically found in depression. Studies with clinical populations have demonstrated immune changes associated with sleep loss and with abnormal sleep architecture, with bidirectional associations. Bidirectional links between depression and immunity have also been demonstrated in persons exposed to cytokines. Interferon-alpha causes depression, fatigue, cognitive dysfunction, and pain; interleukin-6 and TNF-alpha have been implicated in mediating these interferon-induced symptoms. The implications for the treatment of immune-based depression will be considered. Finally, the panel will wrestle with the question of whether depressive disorders should be described in a global manner as associated with activation vs. suppression of the immune system.

CYTOKINE–INDUCED DEPRESSION: IMPLICATIONS FOR THE ROLE OF THE IMMUNE SYSTEM IN MOOD DISORDERS

Andrew H. Miller
Department of Psychiatry, Emory University School of Medicine, USA

Abstract

Treatment with interferon- (IFN) alpha for infectious diseases and cancer causes profound behavioral toxicity including depressed mood, anhedonia, fatigue, anorexia, pain, and cognitive dysfunction. In a double-blind, placebo-controlled study, we demonstrated that the antidepressant, paroxetine, given two weeks before initiation of IFN-alpha treatment and continued for the first 12 weeks of IFN-alpha therapy, reduced the incidence of major depression from almost 50% in the placebo group to approximately 10% in the paroxetine group. A dimensional analysis of symptom evolution and response to paroxetine in these patients revealed that while mood and cognitive changes appeared relatively late during IFN-alpha treatment and were paroxetine responsive, neurovegetative symptoms appeared early and were relatively resistant to paroxetine treatment. These data suggest at least two distinct behavioral syndromes during immune activation with IFN-alpha: a mood disorder (with cognitive dysfunction) and a neurovegetative syndrome. More recent explorations of neural correlates of IFN-alpha-induced behavioral/cognitive changes using positron emission tomography and functional magnetic resonance imaging reveal marked IFN-alpha-induced reductions in activity of the pre-frontal cortex consistent with findings seen in depression. Interestingly, these changes are accompanied by marked increases in activity of striatal regions, indicating that altered fronto-striatal circuitry may drive IFN-alpha-induced mood disturbances. Taken together, the data demonstrate that cytokine administration leads to behavioral changes that overlap with, but in certain instances are distinct from, major depression. Moreover, the neural correlates of cytokine administration may provide important clues to nervous system pathways involved in cytokine-induced symptom expression and possibly mood disorders in general.

BEHAVIORAL AND NEUROCHEMICAL EFFECTS OF CYTOKINES IN ANIMAL STUDIES: IMPLICATIONS FOR HUMAN DEPRESSION

Zul Merali[1,2] & Hymie Anisman[1,3]
[1]Institute of Mental Health Research, [2]Psychology, University of Ottawa,
[3]Neuroscience, Carleton University. Ottawa, Ontario, Canada

Abstract
Although human studies have implicated cytokines in promotion or exacerbation of depressive illness, it is uncertain whether they provoke processes similar to those underlying clinical depression. This review assesses the behavioral and neurochemical effects of interleukin (IL)-2, IL-1b and necrosis factor-a (TNF-a) in animal studies, and contrasts these to those of stressors. Unlike stressors, systemic IL-2 does not appreciably influence hypothalamic-pituitary-adrenal (HPA) neuroendocrine functioning, but elicited behavioral effects reminiscent of those provoked by traditional stressors, including anhedonia (diminished pleasure gained from otherwise rewarding stimuli). Chronically administered IL-2 markedly influenced central monoamine activity, elicited protracted anhedonic effects, and disrupted cognitive processes, including spatial working memory. Unlike IL-2, systemic IL-1b increased HPA activity and altered central norepinephrine and serotonin functioning at hypothalamic and extrahypothalamic sites. In addition, IL-1b also induced an anhedonic effect. In humans, mitogen elicited production of IL-1b was increased in depressed patients, particularly in chronic depression (dysthymia), independent of the presence of typical vs atypical symptoms. However, this effect was not entirely eliminated with antidepressant treatment. While the data provisionally support a role for these cytokines in depression, it needs to be cautioned that their effects have been evaluated in a limited number of situations, and too few animal studies involved chronic cytokine treatment. Moreover, given the biphasic dose-response effects of IL-2, coupled with the potential neurotoxic effects of cytokines, it is still premature to conclude that the behavioral effects are, in fact, reflective of a depressive effect comparable to that elicited by conventional stressors.

SOLUBLE CELL ADHESION MOLECULES, INFLAMMATORY CYTOKINES, AND DEPRESSION IN ACUTE CORONARY SYNDROME PATIENTS

Michael Irwin, Francois Lespérance, & Nancy Frasure-Smith.

Cousins Center for Psychoneuroimmunology, UCLA, Los Angeles, CA
University of Montreal, Montreal Heart Institute and McGill University, Canada

Indicators of chronic low-grade inflammation and endothelial activation, such as interleukin-6 (IL-6) and soluble intracellular adhesion molecule (sICAM) are linked to both the incidence and prognosis of patients with coronary artery disease. As elevations of markers of inflammatory activation have also been documented in patients with depression, we evaluated the relationships between depression, endothelial activation, inflammation, and cardiovascular disease risk by measuring plasma levels of sICAM and IL-6 in 510 acute coronary syndrome (ACS) patients (mean age=59.8; 18.6% women). Presence and histories of affective, anxiety, and substance use disorders was assessed by SCID interview with same day blood samples obtained in the morning after an overnight fast. 37 persons met criteria for current major depression and 84 patients reported a lifetime history of major depression. Patients with current depression had significantly $p<0.05$) higher plasma levels of sICAM compared to non-depressed patients (209.2 ± 55.4 vs. 188.3 ± 56.2 ng/ml; $p<.05$). Moreover, there was a significant ($p<0.05$) interaction between current and prior history of major depression for sICAM in which patients with a current, first episode of depression (221.11 ± 57.8 ng/ml) showed higher levels of sICAM than those patients with a history of recurrent major depression (186.2 ± 46.1 ng/ml), even after controlling for potential confounders (smoking, age, sex, and past cardiac history). Plasma levels of IL-6 were not associated with current or history of depression. In summary, a molecular marker of endothelial activation, sICAM, is elevated in depressed coronary

syndrome patients. These data have implications for understanding the pathophysiologic mechanisms of increased risk for adverse events in cardiovascular disease patients with depression and depressive symptoms.

Symposium 4

NEW INSIGHTS FROM MOLECULAR BIOLOGY

Organizer: Keith W. Kelley[1]

Speakers: Serge Rivest[1], Suzanne Broussard[1], Zsuzsanna Fabry[3], & Richard Weindruch[3]

[1]University of Illinois at Urbana-Champaign, [2] Laval University, Canada,
[3] University of Wisconsin-Madison, USA

Abstract

Cytokine biology now encompasses nearly all aspects of physiology, including immunology, endocrinology, neuroscience, psychology, reproduction and cardiology. Bacteria and fungi possess specific elements that generate pathogen-associated molecular patterns (PAMPs) recognized by cells of the immune system. Toll-like receptors (TLRs) in both *Drosophila* and mammals recognize these PAMPs. New evidence indicates that TLR4 responds to products from gram-negative bacteria, such as LPS. TLR2 likely responds to PAMPs from gram-positive bacteria and yeast. The TLRs share significant homology in their intracellular domains with the type I IL-1 receptor (IL-1R). Expression of IL-1R/TLR superfamily members in both circumventricular organs and the parenchyma of the brain are critical to understanding neural-immune relationships. Serge Rivest from the Laboratory of Molecular Endocrinology, Laval University in Quebec, Canada, will present new findings on this topic. Suzanne Broussard from the University of Illinois at Urbana-Champaign will provide a new view of the mechanisms by which two pro-inflammatory cytokines, TNFα and IL-1β, regulate skeletal muscle cells. She will focus on the cross-talk between growth factor and pro-inflammatory cytokine receptors. Zsuzsanna Fabry, Department of Pathology at Wisconsin, will discuss new discoveries on chemical signaling at the blood-brain-barrier and T cell recruitment into the central nervous system. Finally, Richard Weindruch from the UW Department of Medicine will demonstrate novel applications of gene-expression profiling for assessing aging in both mice and primates using cDNA micro-chip arrays. This technology is revealing how aging and diet regulate CNS steady-state transcripts involved in inflammation, oxidative stress, mitochondrial electron transport and oxidative phosphorylation.

MECHANISMS INVOLVED IN THE CEREBRAL INNATE IMMUNITY

Serge Rivest[1]
[1]Laval University, Canada

Abstract

There is an elegant, innate immune response that takes place in a well organized and coordinated manner in the CNS during systemic infection, which is associated with a constitutive expression of the LPS receptor

CD14 and toll-like receptor 4 (TLR4) in the circumventricular organs (CVOs) and other structures devoid of blood-brain barrier (BBB). Circulating LPS also causes a rapid increase in NF-kB, CD14, TLR2, cytokines, chemokines and members of the complement system in these leaky structures, whereas a delayed response can be found in parenchymal cells located in the anatomical boundaries of the CVOs and thereafter in microglia across the brain parenchyma. The basal expression of CD14 and TLR4 in the CVOs is likely to be a determinant mechanism in the proinflammatory signal transduction events that originate from these structures during innate immune response. The cerebral innate immunity is likely to be an essential player in the etiology of inflammatory CNS disorders resulting from infection as well as those assumed to have an immune etiology, such as multiple sclerosis. On the other hand, molecules of the innate immunity have been found to trigger the production of neurotrophic factors and promote neurorepair and remyelination in response to brain injuries, trauma and toxin-induced demyelination. This lecture will present both sites of the story and highlight the mechanisms involved in the "good" and "bad" role of the inflammatory response in the brain that either protect neurons or is the direct cause of the neurodegenerative disorders.

A NOVEL MECHANISM BY WHICH TNF RECEPTORS PROMOTE MUSCLE WASTING.

Suzanne R. Broussard [1], Keith W. Kelley[1]
[1]Laboratory of Immunophysiology, University of Illinois

Abstract
Muscle wasting occurs in a wide variety of diseases resulting in decreased quality of life and a reduction in patient survival. In both AIDS and cancer patients, proinflammatory cytokines are tightly linked to the loss of lean muscle that is not reversible by nutritional intervention. It is well documented that TNFα decreases lean muscle mass at least in part by increasing protein degradation. Here we present an alternative mechanism by which proinflammatory cytokines cause muscle wasting. Regeneration is the normal process responsible for maintenance of healthy muscle. Central to this process is the fusion of muscle progenitor cells, known as myoblasts, into myotubes. Based upon our recent cell culture experiments, it is likely that proinflammatory cytokines decrease the ability of IGF-I to promote muscle regeneration. TNFα significantly impairs the ability of IGF-I to induce protein synthesis in myoblasts. Indeed, TNFα inhibits IGF-I from increasing expression of myogenin, a transcription factor required for myoblast fusion. TNFα also reduces the ability of IGF-I to activate at least two of its downstream docking molecules, IRS-1 and IRS-2. The proinflammatory cytokine IL-1β acts like TNFα to inhibit IGF-I-induced tyrosine phosphorylation of IRS-1 and IRS-2. We provide evidence that ceramide, a downstream product that is induced by both TNFα and IL-1β, may be a common intermediate by which they inhibit IGF-I receptor signaling in myoblasts. Collectively, these data support the idea that TNFα and IL-1β promote muscle wasting by impairing activation signals induced by the tyrosine kinase IGF-I receptor. (Supported by NIMH-51569)

RECRUITMENT AND FUNCTION OF T CELLS IN THE BRAIN

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Abstract
The central nervous system (CNS) is regarded as an immune-privileged site due to the existence of several protective anatomical and physiological barriers. Recently it has become evident that there are bi-

directional interactions between the brain and the immune system that are augmented under pathological conditions. Changes in the balance between pro- and anti-inflammatory factors in the CNS determine the localization, intensity and course of immune responses in the brain. Our goal has been to define the mechanisms involved in entry, accumulation and function of antigen specific CD4 and CD8 T cells in the CNS. These mechanisms are crucial for initiating adaptive immune responses in the nervous tissue. Using T cell receptor transgenic animals, their respective antigens and tetramer technology we demonstrate that CNS introduction of antigens initiate very strong immune responses in the CNS, and that CD4 and CD8+ T cells persist in the brain parenchyma much longer than was previously appreciated. We can interfere with the recruitment of T cells into the CNS at the blood-brain barrier by blocking adhesion molecules. The function of these cells can also be modified by changes in the pro- and anti-inflammatory environment in the nervous tissue. An understanding of T cell trafficking and function in the nervous tissue can provide novel therapies for prevention and treatment of damage in the CNS resulting from immune responses.

GENE EXPRESSION PROFILE OF THE AGING BRAIN

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Abstract

Aging of the brain leads to impairments in cognitive and motor skills and is the major risk factor for several common neurological disorders such as Alzheimer's disease and Parkinson's disease. Recent studies suggest that brain aging is associated with subtle morphological and functional alterations in specific neuronal circuits, as opposed to large scale neuronal loss. In fact, aging of the CNS in diverse mammalian species shares many features such as atrophy of pyramidal neurons, synaptic atrophy, decrease of striatal dopamine receptors, accumulation of fluorescent pigments, cytoskeletal abnormalities and reactive astrocytes and microglia. In order to provide the first global analysis of brain aging at the molecular level, we employed oligonucleotide arrays representing 6347 genes to determine the gene expression profile of the aging neocortex and cerebellum in mice (Lee et al., *Nature Genet*. 25:294-7, 2000). Aging resulted in a gene expression profile indicative of a marked inflammatory response, oxidative stress, and reduced neurotrophic support in both brain regions. At the transcriptional level, brain aging in mice displays striking parallels with human neurodegenerative disorders. Caloric restriction, which retards the aging process in mammals, selectively attenuated the age-associated induction of genes encoding inflammatory and stress responses. Long-term caloric restriction also shifted expression levels for several genes which did not change with aging. These studies are beginning to provide a global view of transcriptional patterns associated with the aging process and its retardation.

Early Bird Workshop

CONVERGING PERSPECTIVES: EAST MEETS WEST

Speakers: Cai Song[1], Hoon Ryu[2]

Discussants: Duck-Hee Kang[3], Richard Kradin[4]

[1]University of British Colombia, [2]Harvard University, [3]University of Alabama, [4]Harvard University

Abstract

A primary focus on the underlying physiological mechanisms of disease in Western science led some to downgrade the influence of mental and emotional processes, but this bias has been changing. There is now more appreciation for the value of a healthy life style in preventive medicine, and the benefits of some alternative therapies has led to a greater acceptance of psychological influences on bodily functions. Thus, it seems timely to consider the insights that may be gained from the knowledge and scientific traditions developed by other cultures. For this special educational session, two Asian scientists will share their unique perspectives with us. Cai Song, known to PNIRS for her research on cytokine biology, will review some of the major tenets of Chinese medicine, which she learned while gaining her medical degree in China. Duck-Hee Kang will lead the discussion at the end of her short lecture. Hoon Ryu will then present findings on how Asian meditation and exercise practices affect physiological responses and may improve health. He has examined the effects of a Korean practice, ChunDoSunBup Qi-training, on growth hormone and neutrophil functioning. Richard Kradin will serve as the discussant for this presentation. While this short session can provide only a few illustrative examples, it will serve to broaden our understanding of this important area of knowledge.

INSIGHTS FROM TRADITIONAL CHINESE MEDICINE

Cai Song[1]

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Abstract

The Nobel Prize winner W. Heisenberg said that the nature we observe is not nature itself. What we discover is revealed by the way that we ask the question. Traditional Chinese Medicine (TCM) and Western Medicine progressed using different ways to ask questions. Western Medicine developed from Reductionism describes the body as a machine separable into parts, and diseases due to a malfunctioning of its physical or chemical components. TCM was influenced by Dao and Buddha's philosophies that led Chinese scientists to emphasize the relationship between the universe, nature, life and health. TCM theories include energy metabolism, Yin and Yang balance, 5-element forming, and organ/energy channel interaction. Four principles (entirety, interconnectivity, order, and dynamic states) in TCM appear similar to principles of the modern systems theory. The relationship between the brain and immune system in diseases is also viewed from this framework. Four materials were related to immune functions: Qi , Xie, Jing, and Yie. Stress and emotion can change their metabolism and distribution in the body. Conversely, different illness may present different emotion disturbance. TCM has an emphasis on homeorhesis. The brain and immune system interact in a system comprised of two-dialectical sides: defense and pathogenic, Yin and Yang, over-acting and deficient, superficial and internal, hot and cold, and phenomenon and root cause. The perspectives and wisdom developed by TCM provides an alternative ways of viewing the relationship between the brain and body, may offer insights for PNI research.

ENDOCRINE AND IMMUNE EFFECTS OF CHUNDOSUNBUP QI-TRAINING

Hoon Ryu[1] , Myung Soo Lee[2], Seong Min Jeong[2,3], & Nam Il Mo[3]

[1]Dept.of Neurology, Harvard Medical School, USA, [2]Dept. of Immunology and Qi Medicine, Wonkwang University, IkSan, [3]ChunDoSunBup Institute, KangWonDo, Republic of Korea

Abstract
Qi-training is an oriental, traditional psychosomatic training not only for the development of physical balance through isotonic, isometric slow motions but also for the psychological stabilization through meditation. The uniqueness of Qi-training has been distinguished from physical exercise. Cross-disciplinary approaches using methods from psychology, neuroendocrinology, and immunology have greatly enhanced our understanding of the mechanisms through which Qi-training influences the holistic health state in men. We first hypothesized that part of the beneficial effects of Qi-training on psychological and immunological functions were mediated via neuroendocrine responses. We found that ChunDoSunBup (CDSB) Qi-training acutely affects the secretion of growth factors in man. The plasma levels of growth hormone (GH), insulin like growth factor (IGF)-I and insulin like growth factor binding protein (IGFBP)-3 increased during and after CDSB Qi-training. The increase in IGF-I was associated with the secretion of GH. Next, the possible role of GH secreted by the stimulation of Qi-training was investigated in neutrophils in vivo and in vitro. Our study showed that CDSB Qi-training is sufficient to elevate the capacity of circulating neutrophils to produce microbicidal reactive oxygen intermediates (superoxide anion, O_2)) upon stimulation in aged men. This respiratory function is rapidly activated in the isolated neutrophils during and immediately after Qi-training. The novel effect of GH on neutrophils was further investigated in vitro employing native, purified and recombinant GH. GH primed and increased the function and adhesion of neutrophils through the phosphorylation of Janus kinse (JAK)2, signal transducer and activator of transcription (STAT) 3, focal adhesion kinas (FAK), and paxillin. Taken together, in vitro and in vivo data suggest that the endogenous GH induced by Qi-training may enhance resistance to infection and inflammation through the modulation of innate defense function.

GEORGE F. SOLOMON MEMORIAL SESSION

Gail Ironson[1], Margaret Kemeny[2], & Robert Ader[3]
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Abstract
On October 8, 2001 PNIRS lost an esteemed colleague, George Solomon, M.D. This memorial session will review both his scientific and personal contributions. Dr. Solomon is widely considered to be one of the key founders of psychoneuroimmunology. In 1964 he published a classic study comparing psychological profiles of sisters with a similar biological predisposition for rheumatoid arthritis (RA), but one had symptoms and the other did not. With Alfred Amkraut he showed that neural lesions disrupted immune responses in rats, thus linking the hypothalamus to immunity. They also conducted a seminal study on coping behavior showing that fighting mice were significantly better at rejecting virally induced tumors. More recently he conducted innovative studies demonstrating that immune responses are preserved in elderly people of good health and spirit. Similarly, Dr. Solomon had an abiding interest in factors that sustained the health of HIV-infected individuals. With Gail Ironson and Lydia Temoshok, he showed the importance of the doctor-patient relationship, spirituality, emotional expression, life engagement, and NK cytotoxicity. On a personal level Dr. Solomon was known for his generous mentoring, his compassion and youthful exuberance, and his integrity. He will be sorely missed. More information about Dr. Solomon can be found in his autobiography "From Psyche to Soma and Back: Tales of Biopsychosocial Medicine" (2000). His extensive annotated bibliography of 634 references, which supports 243 postulates can be found on www.pnirs.org.