Israel Society for Vision and Eye Research
האגודה הישראלית לחקר העין והראיה

ABSTRACTS

26TH Annual Meeting
Neve Ilan Resort Hotel
March 9-10, 2006

תקצירים

הכינוס השנתי ה-26
מלון נוה אילן
9-10 מרץ, 2006

עריכת התוכנית: פרופ' מרדכי רוזנר, פרופ' אברהם שפירר וד"ר אריה סולומון, מכון העיניים ע"ש גולדשלגר, הפקולטה לרפואה ע"ש סאקלר, אוניברסיטת ת"א, מרכז רפואי שיבא, תל-השומר.

עיצוב והבאה לדפוס: יעקב אלבז, הדסה עין-כרם.

יושבי-ראש של האגודה הישראלית לחקר העין והראיה

פרופ' איליין ברמן ז"ל	1982 - 1979
פרופ' מיכאל בלקין	1985 - 1983
פרופ' שאול מרין	1989 - 1986
פרופ' שבתאי דיקשטיין	1993 -1990
פרופ' פביאן אברהם	1996 - 1994
פרופ' אידו פרלמן	1999 - 1997
פרופ' יעקב פאר	2003 - 2000
פרופ' אהובה דברת	2004

חברי ועד האגודה הישראלית לחקר העין והראיה

פרופ׳ אהובה דברת – יו״ר

פרופ׳ יעקב פאר – מזכיר/גזבר

פרופ׳ אורנה גייר

פרופ׳ מרדכי רוזנר

ד״ר איתן בלומנטל

ד״ר אבי סלומון

ד״ר אריה סלומון

ד״ר רון עופרי

פרופ׳ אברהם שפירר

מרצים זוכים המקבלים פרס השנה על עבודות שהוצגו בכינוס הקודם (הכינוס השנתי ה-25, מרץ 2005):

1. חן יופה-הפקולטה לרפואה תל-אביב, עבור הפוסטר:
"חקר תפקידו של פקס-6 (Pax6) בהתפתחות האפיתל הפיגמנטי של הרשתית (RPE)".

2. ד"ר גריפנר גבריאל – בית הספר לרפואה של האוניברסיטה העברית ירושלים וביה"ח הדסה עין-כרם, עבור ההרצאה:
"פרוליפרציה ונדידה של תאי אפיתל באזור המעבר בין הלחמית לעור, בעפעף של חולדות בריאות"

3. איזנברג דנה – המחלקה לביולוגיה אוניברסיטת חיפה, אורנים, טבעון עבור ההרצאה:
"השפעת משתני הטרף על זיהוי ולכידה בזיקית המובהקת (Chamaeleo chameleon)".

תודה לחברות שתרמו לכינוס:

Pfizer Pharmaceuticals Israel

Ferring - Biotechnology General (Israel) Ltd.

Lemico Ltd.

לוקסמבורג תרופות בע"מ

Medibell Medical Vision Technologies Ltd.

Merck Sharp @ Dohme

Novartis Ophthalmics

Tradis Gat Ltd.

Medivision

Megapharm

Medi-Fisher

ISRAEL SOCIETY FOR VISION AND EYE RESEARCH

XXVI ANNUAL MEETING

NEVE-ILAN RESORT

PROGRAM AT A GLANCE

Thursday, March 9, 2006

Registration and Coffee		**08:00 – 08:30**
Opening Remarks	**("SHARON" Hall)**	**08:30 – 08:35**
Poster Session	**("SHARON" Hall)**	**08:35 – 10:30**
Coffee break	**(Exhibition Hall)**	**10:30 – 11:00**
Retina 1	**("SHARON" Hall)**	**11:00 – 11:30**
Retina 2	**("SHARON" Hall)**	**11:30 – 12:00**
Genetics	**("SHARON" Hall)**	**12:00 – 13:00**
Lunch break	**(Dining Room)**	**13:00 – 14:00**
Guest Lecture 1	**("SHARON" Hall)**	**14:00 – 14:30**
Business Meeting	**("SHARON" Hall)**	**14:30 – 15:00**
Retina 3	**("SHARON" Hall)**	**15:00 – 15:40**
Pediatric & Strabismus	**("SHARON" Hall)**	**15:40 – 16:30**
Poster viewing, Wine & Cheese	**(Exhibition Hall)**	**16:30 – 17:30**
Glaucoma	**("SHARON" Hall)**	**17:30 – 18:30**
Dinner (optional)	**Caravan Restaurant, Abu-Gosh**	**19:30**

Friday, March 10, 2006

Coffee break	**(Exhibition Hall)**	**08:00 – 09:00**
Cataract	**("SHARON" Hall)**	**09:00 – 09:30**
Visual Function	**("SHARON" Hall)**	**09:30 – 10:30**
Guest Lecture 2	**("SHARON" Hall)**	**10:30 – 11:00**
Coffee break	**(Exhibition Hall)**	**11:00 – 11:30**
Oncology	**("SHARON" Hall)**	**11:30 – 12:00**
Cornea	**("SHARON" Hall)**	**12:00 – 13:00**
Refractive Surgery	**("SHARON" Hall)**	**13:00 – 13:50**
Concluding Remarks	**("SHARON" Hall)**	**13:50 – 14:00**

PROGRAMME

Thursday, March 9, 2005

Registration 08:00 – 08:30

Opening Remarks ("SHARON" Hall) 08:30 – 08:35

Prof. Ahuva Dovrat

Session I – Poster presentations 08:35 – 10:30

Moderators: Dr. Avi Solomon and Dr. Eytan Blumenthal

1. **EVALUATING SUPPRESSION OF NONSENSE MUTATIONS BY AMINOGLYCOSIDE ANTIBIOTICS AS AN INTERVENTION FOR VISION LOSS IN TYPE I USHER SYNDROME**
   (1) * MRS. REBIBO ANNIE (1) MRS. RIZEL LEAH (2) DR. AHMED ZUBAIR M. (2) DR. FRIEDMAN THOMAS B. (3) PROF. BAASOV TIMOR (1) DR. BEN-YOSEF TAMAR
   (1) GENETICS DEPARTMENT, THE RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) LABORATORY OF MOLECULAR GENETICS, NATIONAL INSTITUTE ON DEAFNESS AND OTHER COMMUNICATION DISORDERS, NATIONAL INSTITUTES OF HEALTH, ROCKVILLE, MD, USA (3) CHEMISTRY DEPARTMENT, TECHNION, HAIFA

2. **IDENTIFYING THE GENETIC BASIS FOR SYNDROMIC AND NON-SYNDROMIC RETINITIS PIGMENTOSA IN ARAB AND JEWISH FAMILIES FROM NORTHERN ISRAEL**
   (1) * MRS. AUSLENDER NOA (1) MRS. BENAYOUN LIAT (2) DR. ABBASI ANAN (1) MRS. RIZEL LEAH (2) DR. GARZOZI HANNA (3) DR. ALLON-SHALEV STAVIT (1) PROF. PERLMAN IDO (1) DR. BEN-YOSEF TAMAR
   (1) RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) BNAI ZION MEDICAL CENTER, HAIFA (3) HA'EMEK MEDICAL CENTER, AFULA

3. **MUTATIONS ANALYSIS OF DNA EXTRACTED FROM AGE-RELATED CATARACT LENS**
   (1) * DR. CHELES DORINA (2) MRS. AVRAHAM BAT-CHEN R (3) DR. COHEN YORAM (4) DR. EHRLICH RITA (4) DR. PIRIATINSKY BORIS (4) PROF. WEINBERGER DOV (2) DR. GOLDENBERG-COHEN NITZA

(1) TEL-AVIV SOURASKY MEDICAL CENTER (2) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, TEL-AVIV UNIVERSITY AND SACKLER SCHOOL OF MEDICINE, TEL-AVIV (3) SHEBA CANCER RESEARCH CENTER (4) RABIN MEDICAL CENTER, BEILINSON AND GOLDA CAMPUS, PETACH TIKVA

4. **A MISSENSE MUTATION (D144E) IN THE VSX1 GENE IS ASSOCIATED WITH KERATOCONUS IN AN ASHKENAZI JEWISH FAMILY**
(1) * DR. PRAS ERAN (1) DR. ZADOK DAVID (2) DR. GARZOZI HANA (1) DR. AVNI ISSAC
(1) THE DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN (2) THE DEPARTMENT OF OPHTHALMOLOGY, BNEI ZION HOSPITAL, HAIFA.

5. **THE GENETICS OF X-LINKED RETINAL DEGENERATIONS IN THE ISRAELI POPULATION**
(1) * MRS. MIZRAHI-MEISSONNIER LILIANA (1) MR. HUDERA SHMUEL (1) MRS. NEIS RUHAMA (1) DR. BANIN EYAL (1) DR. SHARON DROR
(1) DEPT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIV MED CTR, JERUSALEM

6. **GENETIC ANALYSIS OF LEBER CONGENITAL AMAUROSIS IN THE ISRAELI POPULATION**
(1) * MRS. BIDA LINA (1) MRS. MIZRAHI-MEISSONNIER LILIANA (1) MRS. BEIT-YAACOV ANAT (2) DR. ROSENMANN ADA (1) PROF. MERIN SHAUL (1) DR. BANIN EYAL (1) DR. SHARON DROR
(1) OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) MICHAELSON INSTITUTE FOR VISION REHABILITATION, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM

7. **GENETIC APPROACH TO STRUCTURE-FUNCTION ANALYSIS OF THE DROSOPHILA TRP CHANNEL**
(1) * KOHN ELKANA (1) PROF. MINKE BARUCH
(1) DEPT. OF PHYSIOLOGY, THE HEBREW UNIVERSITY-HADASSAH MEDICAL SCHOOL, JERUSALEM

8. **EARLY DIAGNOSIS OF AGE RELATED MACULAR DEGENERATION BY IDENTIFICATION OF TRITANOPIA**
(1) * MRS. BEN ELI HADAS (1) PROF. HOCHSTEIN SHAUL (2) DR. CHOWERS ITAY
(1) NEUROBIOLOGY DEPARTMENT, INSTITUTE OF LIFE SCIENCES, HEBREW UNIVERSITY, JERUSALEM (2) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH – HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM

9. **CHANGES IN THE REFRACTIVE STATE DURING PREY CAPTURE UNDER LOW LIGHT IN THE NOCTURNAL**

CARDINALFISH APOGON ANNULARIS
(1) MR. HOLZMAN ROI (1) DR. SHASHAR NADAV (2) PROF. HOWLAND HOWARD, C. (3) * PROF. KATZIR GADI
(1) THE INTERUNIVERSITY INSTITUTE FOR MARINE SCIENCES AND DEPARTMENT OF EVOLUTION, SYSTEMATICS AND ECOLOGY, THE HEBREW UNIVERSITY OF JERUSALEM, EILAT (2) NEUROBIOLOGY AND BEHAVIOR, CORNELL UNIVERSITY, ITHACA, NY, USA. (3) BIOLOGY, ORANIM, UNIVERSITY OF HAIFA, TIVON

10. **BINOCULAR PROCESSING MANIPULATING MONOCULAR PERCEPTION**
(1) * MR. YEHEZKEL OREN (1) PROF. BELKIN MICHAEL (2) PROF. SAGI DOV (1) MR. POLAT URI
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, , TEL-AVIV UNIVERSITY , TEL HASHOMER (2) DEPARTMENT OF NEUROBIOLOGY, BRAIN RESEARCH, THE WEIZMANN INSTITUTE OF SCIENCE.

11. **EVALUATION OF THE RETINAL NERVE FIBER LAYER IN CHILDREN WITH ENLARGED CUPPING OF THE OPTIC NERVE**
(1) * DR. NESHER RONIT (2) DR. KRUPSKY SARA (1) DR. GEFFEN NOA (1) DR. EPSTEIN ESTHER (1) DR. KRYSTAL ORNIT (1) DR. RAZ YEHUDIT
(1) DEPARTMENT OF OPHTHALMOLOGY, MEIR MEDICAL CENTER, KFAR SABA (2) DEPARTMENT OF OPHTHALMOLOGY, SHEBA MEDICAL CENTER, TEL HASHOMER

12. **REDUCED FOVEAL CONTRAST SENSITIVITY IN GLAUCOMA**
(1) * MRS. LAHAV KAREN (1) DR. LEVKOVITCH-VERBIN HANI (1) PROF. BELKIN MICHAEL (1) DR. GLOVINSKY YOSSI (1) DR. POLAT URI
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, TEL HASHOMER

13. **PTERYGIUM SURGERY: FIBRIN GLUE VERSUS VICRYL SUTURES FOR CONJUNCTIVAL CLOSURE - LONG TERM RESULTS**
(1) * DR. BAHAR IRIT (1) PROF. WEINBERGER DOV (1) DR. GATON DAN (1) DR. AVISAR RAHAMIM
(1) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER, PETACH TIKVA

14. **COMPARISON OF DIFFERENT TECHNIQUES OF ANTERIOR CHAMBER DEPTH AND KERATOMETRIC MEASUREMENTS**
(1) * DR. ELBAZ URI (1) DR. BARKANA YANIV (1) DR. GERBER YAIR (1) DR. AVNI ISAAC (1) DR. DAVID ZADOK
(1) DEPARTMENT OF OPHTHALMOLOGY ASSAF HAROFEH MEDICAL CENTER, ZRIFIN

15. **THE PATHOLOGICAL MECHANISM OF DELAYED INJURIES**

FOLLOWING SULFUR MUSTARD EXPOSURE: 1. CORRELATION BETWEEN CLINICAL STATUS AND CORNEAL INNERVATION
(1) * DR. KADAR T (1) DR. DACHIR S (1) MRS. COHEN M (1) MRS. GUTMAN H (1) MRS. COHEN L (1) MR. FISHBINE E (1) MRS. SAHAR R (1) DR. BRANDEIS R (1) DR. TURETZ J (1) DR. AMIR A
(1) DEPARTMENT OF PHARMACOLOGY, ISRAEL INSTITUTE FOR BIOLOGICAL RESEARCH

16. **ULTRAVIOLET IRRADIATION ON RABBIT CONJUNCTIVA AND CORNEA - HISTOLOGICAL AND CLINICAL EFFECTS**
(1) * DR. LICHTER HENIA (1) DR. GATON DANI (1) DR. AVISAR INBAL (2) DR. SLODOVINIC DAN (2) DR. SOLOMON ARIEH
(1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, BEILINSON CAMPUS, PETAH TIQVA; (2) DEPARTMENT OF OPHTHALMOLOGY, GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL HASHOMER

17. **TREATMENT OF RECURRENT CORNEAL SALZMANN'S NODULAR DEGENERATION: KERATECTOMY AND MMC**
(1) * DR. ORUCOV FAIK (1) DR. SOLOMON ABRAHAM (1) DR. LANDAU DAVID (1) DR. FRUCHT-PERY JOSEPH
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL

18. **INTRAOPERATIVE MITOMYCIN AND CORNEAL ENDOTHELIUM AFTER PHOTOREFRACTIVE KERATECTOMY**
(1) * DR. ZADOK DAVID (2) DR. MORALES ALBERTO (2) DR. CHAYET ARTURO
(1) 2. DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN (2) CODET-ARIS VISION INSTITUTE, TIJUANA, MEXICO

19. **REFRACTIVE SURGERY AMONG ISRAEL DEFENSE FORCES INDUCTEES**
(1,2) * DR. HOROWITZ JOSEPHA (1,2) DR. MEZER EADY (1) DR. BUCKMAN GILA (3) MRS. SCHOCHAT TZIPPORA (3) DR. SASSON ADI (1) DR. ASHKENAZY YITZHAK (2) PROF. GEYER ORNA
(1) MEDICAL SERVICES CENTER, MEDICAL CORPS, IDF
(2) OPHTHALMOLOGY DEPARTMENT CARMEL MEDICAL CENTER
(3) SURGEAON GENERAL′S HQ, MEDICAL CORPS, IDF

20. **APOPTOSIS MEDIATES PHACOEMULSIFICATION INDUCED INJURY IN CORNEAL ENDOTHELIAL CELLS AS DETERMINED BY A NEW IN VITRO MODEL**
(1) * DR. GEFFEN NOA (2) DR. TOPAZ MORIS (3) DR. KREDY-FARHAN LILLY (1) PROF. ASSIA EHUD I (3) PROF. SAVION NAPHTALI
(1) MEIR MEDICAL CENTER, KFAR SABA, (2) THE PLASTIC SURGERY UNIT, HILLEL–YAFFE HOSPITAL, HADERA, (3) GOLDSCHLEGER EYE RES.INST., SHEBA MED. CTR., TEL

HASHOMER

21. **A POTENTIAL RAT MODEL FOR PROLIFERATIVE DIABETIC RETINOPATHY**
(1) * MRS. BAR YEHUDA TEHILLA (2) PROF. MILLER BENJAMIN (1) PROF. PERLMAN IDO
(1) DEPARTMENT OF PHYSIOLOGY AND BIOPHYSICS, THE RUTH AND BRUCE RAPPAPORT FACULTY OF MEDICINE, TECHNION-ISRAEL INSTITUTE OF TECHNOLOGY (2) THE ALBERTO MOSCONA DEPARTMENT OF OPHTHALMOLOGY, RAMBAM MEDICAL CENTER, HAIFA

22. **A NOVEL MODEL FOR CELL INDUCED OCULAR NEOVASCULARIZATION**
(1) * DR. BACHAR A (2) PROF. MILLER B (3) DR. FLUGELMAN M (4) PROF. PERLMAN I
(1) ALBERT MOSCONA DEPARTMENT OF OPHTHALMOLOGY ALBERT MOSCONA DEPARTMENT OF OPHTHALMOLOGY DEPARTMENT OF CARDIOLOGY , CARMEL HOSPITAL EYE RESEARCH LAB, DEPARTMENT OF PHYSIOLOGY, FACULTY OF MEDICINE, TECHNION

23. **CHARACTERIZATION OF CENTRAL RETINAL ARTERY OCCLUSION IN A MOUSE MODEL**
(1) * DR. BAHAR IRIT (1) DR. KRAMER MICHAL (1) DR. HASANGRILU MURAT (1) DR. ELDAR IDO (2) MRS. AVRAHAM BAT CHEN R. (1) PROF. WEINBERGER DOV (2) DR. GOLDENBERG-COHEN NITZA
(1) RABIN MEDICAL CENTER (2) FMRC, TEL-AVIV UNIVERSITY

24. **THE C1/C2 ALLELES OF TRANSFERRIN CONFER SIMILAR RISK FOR NEOVASCULAR AGE RELATED MACULAR DEGENERATION**
(1) * DR. ABU ASLEH SALEH (1) MRS. LEDERMAN MICHAL (1) DR. AVERBUKH EDWARD (1) DR. HEMO ITZHAK (1) DR. BANIN EYAL (1) DR. SHARON DROR (1) DR. CHOWERS ITAY
(1) OPHTHALMOLOGY DEPARTMENT, HADASSAH MEDICAL CENTER, JERUSALEM

25. **PROLIFERATIVE DIABETIC RETINOPATHY - THE EPIRETINAL MEMBRANES**
(1) * PROF. MILLER BENJAMIN (2) PROF. GREGOR ZDENEK
(1) ALBERTO MOSCONA DEPARTMENT OF OPHTHALMOLOGY; RUTH AND BRUCE RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) MOORFIELDS EYE HOSPITAL, LONDON, UK

26. **NEUROPROTECTIVE EFFECT OF BRIMONIDINE IN A MOUSE MODEL OF ISCHEMIC OPTIC NEUROPATHY**
(1) * DR. HASANREISOGLU MURAT (2) MRS. AVRAHAM BAT-CHEN R. (2) MRS. DADON SHIMRIT (3) DR. COHEN YORAM (1)

PROF. WEINBERGER DOV (2) DR. GOLDENBERG-COHEN NITZA
(1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETACH TIKVA (2) THE KRIEGER EYE RESEARCH LABORATORY, FELSENSTEIN MEDICAL RESEARCH CENTER, PETACH TIKVA (3) SHEBA CANCER RESEARCH CENTER, RAMAT GAN

27. **MECHANISM OF RETINAL GANGLION CELL DEATH IN SECONDARY DEGENERATION OF THE OPTIC NERVE**
(1) * DR. LEVKOVITCH-VERBIN HANI (1) MRS. VANDER SHELLY (1) DR. DARDIK RIMA (1) MRS. NISGAV YAEL (1) PROF. MELAMED SHLOMO
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL HASHOMER

28. **NEUROPROTECTIVE EFFECT OF PN-277 ON LASER-INDUCED RETINAL DAMAGE**
(1) * DR. SHULMAN SHIRI (2) DR. BELOKOPYTOV , MARK (2) DR. DUBINSKY GALINA (2) PROF. ROSNER MORDECHAI (2) PROF. BELKIN MICHAEL
(1) OPHTHALMOLOGY DEPARTMENT,SAPIR MEDICAL CENTRE, KFAR SABA, (2) GOLDSCHLEGER EYE RESEARCH INSTITUTE, TEL-AVIV UNIVERSITY, SHEBA MEDICAL CENTER, TEL HASHOMER

29. **ANALYSIS OF CHOROIDAL NEOVASCULARIZION CASES MISSED BY THE PREFERENTIAL HYPERACUITY PERIMETER AND BY RETINA SPECIALISTS EXAMINATION**
(1) PROF. LOEWENSTEIN ANAT (1) DR. GOLDSTER MICHAELA (2) DR. ALSTER YAIR (2) DR. RAFAELI OMER (3) * PROF. BRESSLER NEIL
(1) OPHTHALMOLOGY, TEL-AVIV MEDICAL CENTER (2) NOTALVISION (3) WILMER INSTITUTE, JOHNS HOPKINS HOSPITAL

30. **NEUROPROTECTIVE TREATMENT WITH NAP REDUCES LASER-INDUCED RETINAL DAMAGE IN RATS**
(1) * BELOKOPYTOV MARK (2) DR. SHULMAN SHIRI (1) DR. DUBINSKY GALINA (3) PROF. GOZES ILLANA (1) PROF. BELKIN MICHAEL (1) PROF. ROSNER MORDECHAI
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, SHEBA MEDICAL CENTER, TEL-AVIV UNIVERSITY, TEL-HASHOMER (2) OPHTHALMOLOGY, SAPIR MEDICAL CENTER, MEIR HOSPITAL, KFAR-SAVA (3) DEPARTMENT OF HUMAN MOLECULAR GENETICS AND BIOCHEMISTRY, SACKLER FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY, TEL-AVIV

31. **INCORPORATION OF NEURONAL PROGENITORS DERIVED FROM HUMAN EMBRYONIC STEM CELLS IN A MOUSE MODEL OF RETINAL DEGENERATION**

(1) * MRS. ALPER-PINUS RUSLANA (1) DR. OBOLENSKY ALEXEY (2) MRS. IDELSON MARIA (1) DR. HEMO ITZHAK (2) PROF. REUBINOFF BENJAMIN (1) DR. BANIN EYAL
(1) CENTER FOR RETINAL AND MACULAR DEGENERATIONS, DEAPRTMENT OF OPHTHALMOLOGY (2) GOLDYNE SAVAD INSTITUTE OF GENE THERAPY AND THE DEPARTMENT OF GYNECOLOGY

32. **NOTCH1 SUPPRESSES CONE-PHOTORECPTOR FATE SPECIFICATION IN THE DEVELOPING MOUSE RETINA**
(1) MRS. YARON ORLY (1) * MR. FARHY CHEN (1) DR. RUTH ASHERY-PADAN
(1) HUMAN GENETICS AND BIOCHEMISTRY

33. **THE EFFECT OF TRIAMCINOLONE ACETONIDE ON THE LEVELS OF VEGF IN RPE CELLS IN VITRO**
(1) * DR. EHRLICH RITA (2) DR. LIVNAT TAMI (2) MRS. BARLIYA TILDA (1) DR. RON YONINA (2) DR. LAVIE GAD (1) PROF. WEINBERGER DOV
(1) OPHTHALMOLOGY DEPARTMENT- RABIN MEDICAL CENTER (2) SHEBA MEDICAL CENTER, TEL-HASHOMER

34. **AXIAL LENGTH MEASUREMENT IN EYES WITH DIABETIC MACULAR EDEMA: A-SCAN ULTRASOUND VERSUS IOLMASTER**
(1) * DR. ATTAS-FOX LIAT (1) DR. ZADOK DAVID (1) DR. AVNI ITZHAK (1) DR. ETING EVA (1) DR. BENHAMOU NATAN (1) DR. PRAS ERAN (1) DR. SEGAL ORI (1) DR. BARKANA YANIV
(1) ASSAF HAROFEH MEDICAL CENTER, DEPARTMENT OF OPHTHALMOLOGY

35. **CYCLOSPORIN A PARTIALLY PREVENTS RETINAL ISCHEMIC INJURY**
(1) * DR. LEVINGER ELIYA (2) DR. FLIESHMAN ANAT (2) PROF. ORON YORAM (1) PROF. GEYER ORNA
(1) DEPARTMENT OF OPHTHALMOLOGY, CARMEL MEDICAL CENTER, HAIFA (2) DEPARTMENT OF PHYSIOLOGY AND PHARMACOLOGY, SACKLER FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY, TEL-AVIV

36. **CLINICAL MEASUREMENTS OF BLOOD FLOW VELOCITY USING THE RETINAL FUNCTION IMAGER (RFI)**
(1) * DR. BEN-CNAAN RAN (2) DR. NELSON DARIN A (2) PROF. ROSNER MORDECHAI (3) PROF. BELKIN MICHAEL (4) PROF. GRINVALD AMIRAM
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL HASHOMER (2) OPTICAL IMAGING, LTD., REHOVOT (3) EYE RESEARCH INSTITUTE, TEL-AVIV UNIVERSITY, TEL HASHOMER (4) WEIZMANN INSTITUTE OF SCIENCE, DEPARTMENT OF NEUROBIOLOGY, REHOVOT

37. **BROTH CULTURES YIELD VERSUS TRADITIONAL APPROACH IN THE WORKUP OF ENDOPHTHALMITIS. A PROSPECTIVE COMPERATIVE STUDY**

* DR. KRATZ ASSAF (1) DR. LEVY JAIME (1) DR. WEINSTEIN ORLY (1) DR. KLEMPERER ITAMAR (1) PROF. LIFSHITZ TOVA
(1) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY MEDICAL CENTER, FACULTY OF HEALTH SCIENCES, BEN-GURION UNIVERSITY OF THE NEGEV, BEER-SHEVA

38. **STAPHYLOLYSIN IN THERAPY OF S. AUREUS EXPERIMENTAL ENDOPHTHALMITIS**

(1) * DR. BAREQUET IRINA (2) DR. MANN ORAN (1) MRS. SAFRIN MARY (1) PROF. KESSLER EFRAT (1) PROF. ROSNER MORDECHAI
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, SHEBA MEDICAL CENTER, TEL-AVIV UNIVERSITY FACULTY OF MEDICINE, TEL HASHOMER (2) DEPT. OF OPHTHALMOLOGY, TEL-AVIV SOURASKY MEDICAL CENTER, TEL-AVIV

39. **INFLAMMATORY CELLS OF THE MURAL LACRIMAL CANAL IN PATIENTS WITH IDIOPATHIC PUNCTUM STENOSIS**

(1) * DR. PORGES YAIR (2) DR. GROISMAN GABRIEL
(1) DIVISION OF OPHTHALMOLOGY , SANZ MEDICAL CENTER, LANIADO HOSPITAL, NETANYA (2) DEPARTMENT OF PATHOLOGY , HILLEL YAFFE MEDICAL CENTER, HADERA

40. **OCULAR INJURIES RELATED TO INDEPENDENCE DAY CELEBRATIONS**

(1) * DR. KRATZ ASSAF (1) DR. PETROV ALENA (1) DR. POLYAKOV PAVEL (1) DR. LEVY JAIME (1) PROF. LIFSHITZ TOVA
(1) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY MEDICAL CENTER, FACULTY OF HEALTH SCIENCES, BEN-GURION UNIVERSITY OF THE NEGEV, BEER-SHEVA

41. **CELLULAR AND MOLECULAR MECHANISMS UNDERLYING OPTIC NERVE DEGENERATION AND THE CREATION OF A NON-PERMISSIVE ENVIRONMENT FOR REGENERATION FOLLOWING AXOTOMY**

(1) * MRS. NITZAN ANAT (2) DR. KERMER PAWEL (3) DR. SHIRVAN ANAT (2) PROF. B&AUML;HR MATHIAS (4) PROF. BARZILAI ARI (1) DR. SOLOMON ARIEH
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, CHAIM SHEBA MEDICAL CENTER (2) DEPARTMENT OF NEUROLOGY, UNIVERSITY HOSPITAL G&OUML;TTINGEN (3) NEURO SURVIVAL TECHNOLOGY (NST) LTD (4) DEPARTMENT OF NEUROBIOCHEMISTRY, GEORGE S. WISE FACULTY OF LIFE SCIENCES, TEL-AVIV UNIVERSITY

42. **SEVERE COUGH AND RETINAL HEMORRHAGE IN INFANTS AND YOUNG CHILDREN**
(1) * DR. MORAD YAIR (2) DR. GOLDMAN MICHAEL (1) DR. ELBAZ URIEL (2) DR. DAGAN ZAHI (1) DR. AVNI ISAAC
(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER (2) DEPARTMENT OF PEDIATRICS, ASSAF HAROFEH MEDICAL CENTER

43. **FLUID CIRCULATION THROUGH VASCULOGENIC MIMICRY PATTERNS IN POSTERIOR POLE UVEAL MELANOMA PATIENTS DETECTED BY INDOCYANININE GREEN LASER SCANNING CONFOCAL ANGIOGRAPHY**
(1) * DR. FRENKEL SHAHAR (2) PROF. FOLBERG ROBERT (2) DR. LEVY JAIME (1) MR. BARSEL ISRAEL (1) PROF. PE'ER JACOB
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) DEPARTMENT OF PATHOLOGY, UNIVERSITY OF ILLINOIS AT CHICAGO, CHICAGO, ILLINOIS, USA

44. **INHIBITOR OF APOPTOSIS PROTEINS (IAP) EXPRESSION IN PRIMARY AND METASTATIC UVEAL MELANOMA**
(1) * MRS. LEDERMAN MICHAL (1) DR. MEIR TAL (1) PROF. PE'ER JACOB (1) DR. CHOWERS ITAY
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM

45. **THE CONTRIBUTION OF SYSTEMIC HEALTH PARAMETERS TO INCREASED INTRAOCULAR PRESSURE**
(1) * DR. BEIRAN ITZIK (2) DR. TEKES-MANOVA DORIT (2) MRS. SHOCHAT TZIPI (2) DR. ASHKENAZI ITZHAK (1) PROF. MILLER BENJAMIN (1,2) DR. MEZER EEDY
(1) ALBERTO MOSCONA DEPARTMENT OF OPHTHALMOLOGY, RAMBAM HEALTH CARE CAMPUS (2) MEDICAL SERVICES CENTER, ISRAEL MEDICAL CORPS, ISREAL DEFENSE FORCES

Coffee break (Exhibition Hall) 10:30 – 11:00

Session II - Retina 1 ("SHARON" Hall) 11:00-11:30

Moderators: Prof. Ayala Polack and Prof. Josef Moisseiev

11:00-11:10 **SERUM CYTOKINES LEVELS IN A MOUSE MODEL OF CENTRAL RETINAL ARTERY OCCLUSION**

(1) * DR. KRAMER MICHAL (1) DR. BAHAR IRIT (2) DR. MONSELISE YEHUDIT (1) DR. HASANGRILU MURAT (1) DR. ELDAR IDO (3) MRS. AVRAHAM BAT CHEN R. (1) PROF. WEINBERGER DOV (3) DR. GOLDENBERG-COHEN NITZA
(1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY, TEL-AVIV (2) LABORATORY OF CLINICAL IMMUNOLOGY, RABIN MEDICAL CENTER (3) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, AND SCHNEIDER'S CHILDREN MEDICAL CENTER, SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY, TEL-AVIV

11:10-11:20 **THE VALUE OF THE FIRST PDT RESULTS IN PREDICTION OF VISUAL OUTCOME**

(1) * DR. REGENBOGEN MICHAEL (1) DR. GOLDSTEIN MICHAELLA (1) DR. HEILWEIL GAD (2) PROF. TREISTER GIORA (1) PROF. LOEWENSTEIN ANAT
(1) DEPARTEMENT OF OPHTHALMOLOGY TEL-AVIV MEDICAL CENTER ICHILOV (2) MACCABI EYE INSTITUTE, TEL-AVIV

11:20-11:30 **INHIBITORY EFFECTS OF HYPERICIN ON RPE CELL MIGRATION AND INVASION**

(1) * MRS. BARLIYA TILDA (2) DR. LIVNAT TAMI (1) DR. LAVIE GAD (3) PROF. WEINBERGER DOV
(1) BLOOD CENTER, SHEBA MEDICAL CENTER, TEL-HASHOMER (2) THROMBOSIS & HEMOSTASIS INSTITUTE, SHEBA MEDICAL CENTER, TEL-HASHOMER (3) DEPARTMENT OF OPHTHALMOLOGY, BEILINSON MEDICAL CENTER, PETAH-TIQVA

Session III - Retina 2 11:30-12:00

Moderators: Prof. Elisha Bartov and Prof. Benjamin Miller

11:30-11:40 **INTRAVITREAL BEVACIZUMAB (AVASTIN) FOR CHOROIDAL NEOVASCULARIZATION**

(1) * DR. LANDA GENNADY (1) PROF. POLLACK AYALA (1) DR. BUKELMAN AMIR (1) DR. KATZ HAIA (1) DR. SHOHAM NIR (1) DR. GREENWALD YOEL (1) DR. HAUSER DAVID
(1) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER, REHOVOT

11:40-11:50 **INTRAVITREAL BEVACIZUMAB (AVASTIN) THERAPY FOR NEOVASCULAR AGE-RELATED MACULAR DEGENERATION: 6 MONTHS RESULTS OF AN UNCONTROLLED, OPEN LABEL CLINICAL STUDY**

(1) * DR. HEILWEIL GAD (1) DR. ADIEL BRAK (2) DR. MOROZ IRIS (1) PROF. LOEWNSTEIN ANAT
(1) DEPARTMENT OF OPHTHALMOLOGY, TEL-AVIV MEDICAL CENTER (2) GOLDSCHLEGER EYE INSTITUTE TEL-HASHOMER

11:50-12:00 **RETINAL ELECTROPHYSIOLOGIC, MORPHOLOGIC AND PENETRATION STUDIES FOLLOWING INTRAVITREAL INJECTION OF BEVACIZUMAB (AVASTIN ®).**

(1) * MR. SHAHAR JONATHAN (2) PROF. AVERY ROBERT L. (3) DR. HEILWEIL GAD (3) DR. BARAK ADIEL (4) DR. ZEMEL ESTHER (5) DR. LEWIS GEOFFREY P. (5) DR. JOHNSON PATRICK T. (5) PROF. FISHER STEVEN K (4) PROF. PERLMAN IDO (3) PROF. LOEWENSTEIN ANAT
(1) SACKLER FACULTY OF MEDICINE, TEL- AVIV UNIVERSITY, TEL-AVIV (2) CALIFORNIA RETINA CONSULTANTS, SANTA BARBARA (3) DEPARTMENT OF OPHTHALMOLOGY, TEL-AVIV MEDICAL CENTER, TEL-AVIV (4) DEPARTMENT OF PHYSIOLOGY AND BIOPHYSICS, RUTH & BRUCE RAPPAPORT FACULTY OF MEDICINE, TECHNION-ISRAEL INSTITUTE OF TECHNOLOGY AND THE RAPPAPORT INSTITUTE, HAIFA (5) NEUROSCIENCE RESEARCH INSTITUTE, UNIVERSITY OF CALIFORNIA, SANTA BARBARA

Session IV - Genetics 12:00-13:00

Moderators: Dr. Ron Ofri and Dr. Ruth Ashery-Padan

12:00-12:10 **HERMANSKY PUDLAK SYNDROME TYPE 6 CAUSED BY A NOVEL MUTATION IN AN EXTENDED, HIGHLY CONSANGUINEOUS, ISRAELI BEDOUIN FAMILY**

(1) * MRS. SCHREYER-SHAFIR NIRA (2) DR. ROSENMANN ADA (3) DR. ANIKSTER YAIR (1) MRS. MAFTZIR GENIA (1) MRS. BEJARANO-ACHACHE IDIT (1) MRS. NUSINKER ZIVA (4) DR. GRADSTEIN LIBE (5) DR. HUIZING MARJAN (1) DR. BLUMENFELD ANAT
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM (2) MICHAELSON INSTITUTE FOR REHABILITATION OF VISION, HADASSAH MEDICAL ORGANIZATION, JERUSALEM
(3) METABOLIC DISEASE UNIT, SAFRA CHILDREN HOSPITAL, SHEBA MEDICAL CENTER, TEL HASHOMER
(4) DEPARTMENT OF OPHTHALMOLOGY, SOROKA MEDICAL CENTER, BEER-SHEVA
(5) NATIONAL HUMAN GENOME RESEARCH INSTITUTE, NATIONAL INSTITUTES OF HEALTH, BETHESDA, MD

12:10-12:20 **GENE EXPRESSION PATTERNS IN PRIMARY AND METASTATIC UVEAL MELANOMA**

(1) * DR. MEIR TAL (1) MRS. DROR RINAT (1) PROF. PE'ER JACOB (1) DR. CHOWERS ITAY
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM

12:20-12:30 **A TEMPORAL MICROARRAY-BASED GENE EXPRESSION ANALYSIS OF THE RAT RETINA**

(1) * DR. BEN-SHLOMO GIL (1) DR. OFRI RON (2) MRS. BANDAH DIKLA (2) MR. SHARON DROR
(1) KORET SCHOOL OF VETERINARY MEDICINE, HEBREW UNIVERSITY OF JERUSALEM, REHOVOT (2) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM

12:30-12:40 **SPATIAL AND TEMPORAL DIFFERENTIAL GENE EXPRESSION ANALYSIS OF PAX6 ALTERNATIVELY-SPLICED TRANSCRIPTS IN THE PIGEON RETINA**

(1) * DR. SHARON DROR (1) MRS. BANDAH DIKLA (1) MR. SWISSA TOMER (2) DR. BEN-SHLOMO GIL (1) DR. BANIN EYAL (2) DR. OFRI RON
(1) HADASSAH-HEBREW UNIV MED CTR, JERUSALEM (2) SCHOOL OF VETERINARY MEDICINE, HEBREW UNIVERSITY OF JERUSALEM, REHOVOT

12:40-12:50 **THE MANY ROLES OF PAX6 IN DEVELOPMENT OF THE OCULAR LENS**
(1) * MR. SHAHAM OHAD (1) DR. ASHERY-PADAN RUTH
(1) DEPARTMENT OF HUMAN GENETICS AND BIOCHEMISTRY, FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY

12:50-13:00 **ALTERED EXPRESSION OF IRON METABOLISM ASSOCIATED GENES IN RETINAL DEGENERATION**
(1) * MRS. DELEON EFRAT (1) MRS. LEDERMAN MICHAL (2) DR. BERENSTEIN EDDY (2) PROF. CHEVION MORDECHAI (1) DR. CHOWERS ITAY
(1) OPHTHALMOLOGY (2) BIOCHEMISTRY

Lunch break 13:00 – 14:00

Session V - Guest Lecture ("SHARON" Hall) 14:00 – 14:30

Prof. Leonard A. Levin MD, PhD

"Axonal Signaling of Retinal Ganglion Cell Death"

Session VI - Business Meeting 14:30 – 15:00

Session VII - Retina 3 15:00 – 15:40

Moderators: Prof. Dov Weinberger and Prof. Anat Levenstein

15:00-15:10 **REPAIR OF OPTIC NERVE ISCHEMIC INJURY IN THE MOUSE BY BONE MARROW DERIVED STEM CELLS**
(1) * MRS. AVRAHAM BAT-CHEN R. (2) PROF. WEINBERGER DOV (1) DR. GOLDENBERG-COHEN NITZA
(1) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, TEL-AVIV UNIVERSITY (2) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER, SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY

15:10-15:20 **BONE MARROW DERIVED STEM CELLS INCORPORATION INTO LASER INJURED RETINA**
(1) * DR. RON YONINA (2) MRS. AVRAHAM BAT-CHEN (1) PROF. WEINBERGER DOV (2) DR. GOLDENBERG COHEN NITZA
(1) OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETACH TIKVA (2) KRIEGER EYE RESEARCH LABORATORY, FMRC, SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY, PETACH TIKVA

15:20-15:30 **ADULT BONE MARROW-DERIVED PROGENITOR CELLS PROMOTE VASCULAR REPAIR IN OXYGEN-INDUCED RETINOPATHY**
(1) * DR. BANIN EYAL (2) DR. RITTER MATTHEW (2) DR. AGUILAR EDITH (2) DR. DORRELL MICHAEL (2) MRS. MORENO STACEY (2) PROF. FRIEDLANDER MARTIN
(1) CENTER FOR RETINAL AND MACULAR DEGENERATIONS, DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER (2) DEPARTMENT OF CELL BIOLOGY, THE SCRIPPS RESEARCH INSTITUTE, LA JOLLA, CALIFORNIA, USA

15:30-15:40 **HUMAN EMBRYONIC STEM CELLS DIFFERENTIATE INTO RETINAL PIGMENT EPITHELIUM CELLS**
(1) MRS. IDELSON MARIA (2) * DR. OBOLENSKY ALEXEY (2) MRS. ALPER RUSLANA (1) PROF. REUBINOFF BENJAMIN (2) DR. BANIN EYAL
(1) GENE THERAPY INSTITUTE (2) CENTER FOR RETINAL AND MACULAR DEGENERATIONS, DEPARTMENT OF OPHTHALMOLOGY, HADASSAH HEBREW UNIVERSITY MEDICAL CENTER

Session VIII - Ped. Ophthalmol. & Strab. 15:40 – 16:30

Moderators: Prof. Abraham Spierer and Dr. Moshe Snir

15:40-15:50 **THE USE OF A SINGLE MADDOX ROD FOR THE MEASUREMENT OF CYCLODEVIATION**
(1) DR. ALMOG YEHOSHUA (1) * DR. TON YOKRAT
(1) OPHTHALMOLOGY MEIR HOSPITAL

15:50-16:00 **THE EFFECT OF THALIDOMIDE ON OXYGEN INDUCED RETINOPATHY IN A MOUSE MODEL**
(1) * DR. KATZ GABRIEL (2) DR. RABINOWITZ RONEN (1) PROF. ROSNER MORDECHAI (2) DR. PRI-CHEN SARA (1) PROF. SPIERER ABRAHAM
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL-HASHOMER (2) SACKLER FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY

16:00-16:10 **DIODE LASER TREATMENT OF RETINOPATHY OF PREMATURITY**
(1) * DR. AXER-SIEGEL RUTH (1) DR. MAHARSHAK IDIT (1) DR. EHRLICH RITA (1) DR. FRILLING RONIT (1) PROF. WEINBERGER DOV (1) DR. SHALEV BENJAMIN (2) PROF. SIROTA LEA (1) DR. SNIR MOSHE
(1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL

CENTER, BEILINSON CAMPUS, PETAH TIQVA (2)NEONATAL INTENSIVE CARE UNIT, SCHNEIDER CHILDREN'S MEDICAL CENTER OF ISRAEL, PETAH TIQVA

16:10-16:20 **INCIDENCE AND SEVERITY OF ROP IN TWO CONSECUTIVE ISRAELI POPULATION GROUPS - A SINGLE CENTER RETROSPECTIVE STUDY.**
(1) * DR. SHOHAM NIR (2) PROF. SHINWELL ERIC (1) PROF. POLLACK AYALA (1) DR. LEIBA HANA
(1) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER, REHOVOT (2) NEONATAL INTENSIVE CARE UNIT, KAPLAN MEDICAL CENTER, REHOVOT

16:20-16:30 **SUTURES MATERIAL IN PEDIATRIC CATARACT SURGERY: A COMPARISON OF VYCRIL VERSUS MERSILEN**
(1) * DR. BAR-SELA SHAI M. (1) PROF. SPIERER ABRAHAM
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL-HASHOMER, AND SACKLER FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY, TEL-AVIV

Session IX - Poster viewing, Wine & Cheese

(Exhibition Hall + Posters Hall) 16:30 – 17:30

Session X - Glaucoma 17:30 – 18:30

Moderators: Prof. Shlomo Melamed and Prof. Orna Geyer

17:30-17:40 **DO TOPICAL BETA-BLOCKERS INCREASE THE RISK FOR DEPRESSION IN GLAUCOMA PATIENTS?**
(1) * DR. KAISERMAN IGOR (2) MRS. KAISERMAN NADIA (3) DR. ELHAYANY ASHER (3) DR. VINKER SHLOMO
(1) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON
(2) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH MEDICAL CENTER, JERUSALEM
(3) DEPARTMENT OF FAMILY MEDICINE, CLALIT HEALTH SERVICES, CENTRAL DISTRICT, REHOVOT

17:40-17:50 **DOES CHANGING EYE TEST ORDER WITH 24-2 SITA STANDARD RESULT IN A MEANINGFUL CHANGE IN TEST RESULTS?**
(1) * DR. BARKANA YANIV (2) DR. MORA RICARDO (2) DR. TELLO CELSO (3) DR. LIEBMANN JEFFREY (2) DR. RITCH ROBERT
(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFE MEDICAL CENETR, ZERIFIN (2) DEPARTMENT OF OPHTHALMOLOGY, THE NEW YORK EYE & EAR

INFIRMARY, NEW YORK, NY (3) DEPARTMENT OF OPHTHALMOLOGY, MANHATTAN EYE EAR & THROAT HOSPITAL, NEW YORK, NY

17:50-18:00 **RETINAL NERVE FIBER LAYER SPLIT BUNDLES ARE TRUE ANATOMICAL VARIANTS**
(1) * DR. BLUMENTHAL EYTAN (1) DR. KALINER EHUD (1) DR. COHEN MATAN (1) DR. MIRON HAGAI (1) DR. KOGAN MICHAEL
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM

18:00-18:10 **SALAGEN DOES NOT AFFECT INTRAOCULAR PRESSURE AND PUPIL DIAMETER IN GLAUCOMA PATIENTS.**
(1) * DR. WOLF ALVIT (1) DR. AREV-FISHELZON TAGIL (1) DR. WOLFSON YULIA (1) DR. LEVINGER ELIA (1) PROF. GEYER ORNA
(1) DEPARTMENT OF OPHTHALMOLOGY, CARMEL MEDICAL CENTER, HAIFA

18:10-18:20 **MEASUREMENTS OF OPTIC DISC SIZE WITH HRT II, OCT3 AND FUNDUSCOPY ARE NOT INTERCHANGEABLE**
(1) * DR. BARKANA YANIV (2) DR. HARIZMAN NOGA (3) DR. LIEBMANN JEFFREY (2) DR. RITCH ROBERT
(1) OPHTHALMOLOGY, ASSAF HAROFE MEDICAL CENTER, ZRIFIN (2) OPHTHALMOLOGY, NEW YORK EYE AND EAR INFIRMARY, NEW-YORK, NY (3) OPHTHALMOLOGY, MANHATTAN EYE, EAR AND THROAT HOSPITAL

18:20-18:30 **DOSE THE AQUEOUS HUMOR HAVE A ROLE IN THE MAPK INTRACELLULAR SIGNALING IN GLAUCOMA?**
(1) MRS. SHMUELEVICH ALLA (1) * DR. BEIT-YANNAI ELIE
(1) Clinical Pharmacology Department, The faculty of Health Sciences, Ben-Gurion University of the Negev

Dinner – 19:30, Caravan Restaurant, Abu-Gosh (optional)

Friday, March 10, 2006

Coffee break (Exhibition Hall) **0**8:00 – 09:00

Session XI - Lens and Cataract 09:00 – 09:30

Moderators: Prof. Ehud Assia and Dr. Yaakov Rosenman

09:00-09:10 **IS THE ANTIOXIDANT N-ACETYL-L-CYSTEINE EFFICIENT IN REDUCING OR PREVENTING DIABETIC DAMAGE TO THE EYE LENS?**
(1) * DOVRAT YAEL (2) DR. BORMUSOV ELVIRA (2) PROF. DOVRAT AHUVA
(1) KORET SCHOOL OF VETERINARY MEDICINE, THE HEBREW UNIVERSITY OF JERUSALEM (2) RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA

09:10-09:20 **THE EFFECT OF TOPICAL SODIUM DICLOFENAC TREATMENT ON MACULAR THICKNESS POST PHACOEMULSIFICATION IN DIABETIC AND NON DIABETIC PATIENTS**
(1) * DR. SCHWARTZ SHULAMIT (1) DR. BENHAMOU NATHANAEL (1) DR. CHETRIT NOA (1) DR. AVNI ISAAC (1) DR. ZADOK DAVID
(1) OPHTHALMOLOGY DEPARTEMENT, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN

09:20-09:30 **REPOSITIONING AND SCLERAL FIXATION OF THE SUBLUXATED LENS CAPSULE USING AN INTRAOCULAR ANCHORING DEVICE**
(1) * DR. TON YOKRAT (2) DR. MICHAELI ADI (1) PROF. ASSIA EHUD
(1) MEIR MEDICAL CENTER, KFAR SABA (2) TEL-AVIV MEDICAL CENTER

Session XII - Visual Function 09:30 – 10:30

Moderators: Dr. Uri Polat and Prof. Ido Perlman

09:30-09:40 **DEPENDENCY BETWEEN LIGHT INTENSITY AND CHICK'S REFRACTIVE DEVELOPMENT UNDER LIGHT DARK CYCLE**
(1) * DR. COHEN YUVAL (2) PROF. BELKIN MICHAEL (1) DR. AVNI ISAAC (2) DR. POLAT URI
(1) ASSAF HAROFEH MEDICAL CENTER, SACKLER FACULTY OF MEDICINE, TEL-AVIV UNIVERSITY, ZERIFIN (2)

GOLDSCHLEGER EYE RESEARCH INSTITUTE, TEL-AVIV UNIVERSITY, TEL HASHOMER

09:40-09:50 **A COMPARATIVE STUDY OF HARDY-RAND-RITTLER 4TH EDITION AND ISHIHARA COLOR PLATES FOR DETECTION OF COLOR-VISION DEFECTS IN OPTIC NEUROPATHY**

(1) * DR. HABOT-WILNER ZOHAR (1) DR. HUNA-BARON RUTH (1) DR. GLOVINSKY YOSEPH

(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL-HASHOMER

09:50-10:00 **PERCEPTUAL DEFICITS IN A SIMULATED-SCOTOMA MODEL OF HUMAN EYE INJURY**

(1) * DR. BRANDEIS R (1) DR. PERI D (1) DR. TURETZ J (1) MRS. EGOZ I

(1) DEPT. OF PHARMACOLOGY, ISRAEL INSTITUTE FOR BIOLOGICAL RESEARCH

10:00-10:10 **THE EFFECT OF HYPERBARIC TREATMENTS ON THE RATE OF REFRACTIVE CHANGES AMONG DIABETIC PATIENTS**

(1) DR. BEN ZION ITAY (2) * DR. GARZOZI HANNA (3) DR. BEIRAN IZAK (4) DR. MELAMED YEHUDA

(1) HILLEL YAFFE MEDICAL CENTER, HADERA (2) BNEI-ZION MEDICAL CENTER, HAIFA (3) RAMBAM MEDICAL CENTER, HAIFA (4) ELISH-RAMBAM

10:10-10:20 **THE EFFECT OF HYPERBARIC OXYGEN TREATMENT ON VISUAL ACUITY AFTER ACUTE CRAO**

(1) * DR. SCHWARTZ SHULAMIT (1) DR. ZADOK DAVID (2) DR. VISHNEVSKIA- DAI VIKTORIA (3) DR. NAHUM GAL (1) DR. AVNI ISAAC

(1) DEPARTEMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTRE, ZERIFIN (2) DEPARTEMENT OF OPHTHALMOLOGY, SHEBA MEDICAL CENTER, TEL HASHOMER (3) HYPERBARIC INSTITUTE, ASSAF HAROFEH MEDICAL CENTRE, ZERIFIN

10:20-10:30 **EXPRESSION OF ACHE RESADTHROUGH ISOFORM CORRELATES WITH RETINAL PHOTIC STRESS IN A RAT MODEL**

(1,2) * DR. KEHAT RINAT (2) DR. ZEMEL ESTHER (3) PROF. SOREQ HERMONA (1) PROF. PERLMAN IDO

(1) BNEI ZION MEDICAL CENTER (2) TECHNION-ISRAEL INSTITUTE OF TECHNOLOHY (3) HEBREW UNIVERSITY

Session XIII - Guest Lecture ("SHARON" Hall) 10:30 – 11:00

Prof. Leonard A. Levin MD, PhD

"Differentiation of a Retinal Ganglion Cell Line for Studies of Neuroprotection"

Coffee break (Exhibition Hall) 11:00 – 11:30

Session XIV - Oncology 11:30 – 12:00

Moderators: Prof. Jacob Pe'er and Prof. Mordechai Rosner

11:30-11:40 **SURVIVAL OF UVEAL MELANOMA PATIENTS AFTER SURGERY FOR LIVER METASTASES**

(1) * DR. FRENKEL SHAHAR (2) DR. NIR ITZHAK (3) DR. LOTEM MICHAL (4) PROF. FOLBERG ROBERT (1) PROF. PE'ER JACOB

(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) DEPARTMENT OF SURGERY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (3) DEPARTMENT OF ONCOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (4) DEPARTMENT OF PATHOLOGY, UNIVERSITY OF ILLINOIS AT CHICAGO, CHICAGO, ILLINOIS, USA

11:40-11:50 **POTENTIAL BLOOD MARKERS FOR DETECTION OF METASTATIC UVEAL MELANOMA – A COMPARATIVE ANALYSIS**

(1) * PROF. BARAK VIVIAN (2) DR. FRENKEL SHAHAR (3) PROF. FOLBERG ROBERT (4) DR. MAJUMDAR DIBYEN (1) DR. KALICKMAN INA (2) PROF. PE'ER JACOB

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11:50-12:00 **OPTICAL COHERENCE TOMOGRAPHIC CHARACTERISTICS OF COMBINED HAMARTOMA OF THE RETINA AND RETINAL PIGMENT EPITHELIUM (RPE) IN 11 PATIENTS**

(1) * DR. VISHNEVSKIA-DAI VICKTORIA (2) DR. SHIELDS CAROL. L (2) DR. MASHAYEKHI ARMAN (2) DR. MATERIN

MIGUEL. A (2) DR. SHIELDS JERRY. A
(1) GOLDSHLEGER EYE INSTITUTE TEL-AVIV UNIVERSITY SHEBA MEDICAL CENTER, TEL-HASHOMER (2) OCULAR ONCOLOGY SERVICE, WILLS EYE HOSPITAL, PHILADELPHIA USA

Session XV - Cornea 12:00 – 13:00

Moderators: Dr. Isaac Avni and Prof. Joseph Frucht-Pery

12:00-12:10 **INCREASED EXPRESSION OF INFLAMMATORY CYTOKINES AND CATHEPSINS IN THE CORNEAL EPITHELIUM IN KERATOCONUS**
(1) * DR. IVANIR YAIR (1) DR. ORUKOV FAIK (1) PROF. PE`ER JACOB (1) PROF. FRUCHT-PERY JOSEPH (1) DR. SOLOMON ABRAHAM
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM

12:10-12:20 **DRUG MODDIFICATION OF ANGIOGENESIS IN A RAT CORNEA MODEL**
(1) * DR. SCHWARTZ SHULAMIT (2) DR. BARAK ADIEL (1) DR. AVNI ISAAC
(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTRE, TEL-AVIV UNIVERSITY, ZRIFIN (2) DEPARTMENT OF OPHTHALMOLOGY, TEL-AVIV SORASKY MEDICAL CENTRE, TEL-AVIV UNIVERSITY, TEL-AVIV

12:20-12:30 **THE PATHOLOGICAL MECHANISM OF DELAYED INJURIES FOLLOWING SULFUR MUSTARD EXPOSURE: 2. A CORRELATION OF CLINICAL STATUS WITH GROWTH CAPACITY OF EPITHELIAL CELLS AND INFLAMMATORY MARKERS OF THE CORNEA**
(1) * DR. AMIR A (1) DR. DACHIR S (1) MRS. COHEN L (1) MRS. COHEN M (1) MRS. GUTMAN H (2) MR. SHALEM Y (1) DR. BRANDEIS R (1) DR. KADAR T
(1) DEPATMENT OF PHARMACOLOGY ISRAEL INSTITUTE FOR BIOLOGICAL RESEARCH (2) LSRI

12:30-12:40 **METALLO-COMPLEXES AUGMENT TREATMENT OF PSEUDOMONAS KERATITIS**
(1) * MRS. LOZINSKI ALINA (2) DR. BERENSHTEIN EDUARD (2) PROF. CHEVION MORDECHAI (1) DR. BANIN EYAL
(1)) CENTER FOR RETINAL AND MACULAR DEGENERATIONS, DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) DEPARTMENT OF CELLULAR

BIOCHEMISTRY AND HUMAN GENETICS, HADASSAH-HEBREW UNIVERSITY MEDICAL SCHOOL, JERUSALEM

12:40-12:50 **THE EFFECT OF 4TH GENERATION FLUOROQUINOLONES ON THE HEALING SPEED OF CORNEAL EROSIONS IN AN ANIMAL MODEL**
(1) * DR. BAREQUET IRINA (1) DR. HABOT-WILNER ZOHAR (1) DR. LAVINSKY FABIO (1) MRS. ZIV HANA (1) PROF. BELKIN MICHAEL (1) PROF. ROSNER MORDECHAI
(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, SHEBA MEDICAL CENTER, TEL-AVIV UNIVERSITY FACULTY OF MEDICINE, TEL HASHOMER

12:50-13:00 **EFFECT OF ADJUNCTIVE STEROID TREATMENT ON THE OUTCOME OF BACTERIAL KERATITIS**
(1) * DR. ORUCOV FAIK (1) DR. SOLOMON ABRAHAM (1) DR. LANDAU DAVID (1) DR. STRASSMAN EYAL (1) PROF. FRUCHT-PERY JOSEPH
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM

Session XVI - Refractive Surgery 13:00 – 13:50

Moderators: Dr. Hanna Garzozi and Prof. Tova Lifshitz

13:00-13:10 **BINOCULAR FUNCTION FOLLOWING MONOVISION CORRECTION WITH LASER IN SITU KERATOMILEUSIS (LASIK) SURGERY IN PRESBYOPIC PATIENTS.**
(1) * DR. SHOLOHOV GALINA (1) DR. LEVARTOVSKY SHMUEL (2) DR. LEVINGER ELIYA (3) DR. LEVINGER SHMUEL (4) DR. LEIBA HANA
(1) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON (2) DEPARTMENT OF OPHTHALMOLOGY, CARMEL MEDICAL CENTER, HAIFA (3) ENAIM INSTITUTE, JERUSALEM (4) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER, REHOVOT

13:10-13:20 **EARLY RECOVERY AFTER SIMULTANEOUS BILATERAL PHOTOREFRACTIVE KERATECTOMY FOR MYOPIA**
(1) * DR. COHEN RAMI (2) DR. ZADOK DAVID (2) DR. SCHIMMEL DEBORAH (2,3) DR. VARSSANO DAVID
(1) SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY (2) MAROM BAZEL MEDICAL CENTER, TEL-AVIV (3) DEPARTMENT OF OPHTHALMOLOGY, TEL-AVIV MEDICAL CENTER, TEL-AVIV

13:20-13:30 **INFORMATION SOURCES AND THEIR USE BY PATIENTS UNDERGOING KERATOREFRACTIVE SURGERY**
(1) * DR. ABU-ELHEGA AIMAN (2) DR. SHEHADEH MASHA'OUR RANEEN (2) DR. GARZOZI HANNA
(1) HAEMEQ MEDICAL CENTER, AFULA (2) BNAI ZION MEDICAL CENTER, HAIFA

13:30-13:40 **ROLE OF EPITHELIAL HYPERPLASIA IN REGRESSION FOLLOWING LASER ASSISTED SUBEPITHELIAL KERATOMILEUSIS (LASEK)**
(1) * MR. MANSOUR AHMAD (1) DR. KEHAT RINAT (1) DR. SHEHADEH-MASHA'OUR RANEEN (1) DR. GARZOZI HANNA, J.
(1) DEPT. OF OPHTHALMOLOGY, BNEI ZION MEDICAL CENTER, HAIFA

13:40-13:50 **THE EFFECT OF INTRALASIK ON CORNEAL SENSITIVITY AND TEAR FUNCTION**
(1,2) * DR. BAREQUET IRINA (2) DR. HIRSH AMI (2) DR. LEVINGER SAMUEL
(1) GOLDSCHLEGER EYE INSTITUTE, TEL-AVIV UNIVERSITY SACKLER FACULTY OF MEDICINE, SHEBA MEDICAL CENTER TEL-HASHOMER (2) ENAIM REFRACTIVE SURGERY CENTERS

Concluding Remarks 13:50 – 14:00

Prof. Ahuva Dovrat

ABSTRACTS

POSTERS

EVALUATING SUPPRESSION OF NONSENSE MUTATIONS BY AMINOGLYCOSIDE ANTIBIOTICS AS AN INTERVENTION FOR VISION LOSS IN TYPE I USHER SYNDROME

(1) * MRS. REBIBO ANNIE (1) MRS. RIZEL LEAH (2) DR. AHMED ZUBAIR M. (2) DR. FRIEDMAN THOMAS B. (3) PROF. BAASOV TIMOR (1) DR. BEN-YOSEF TAMAR

(1) GENETICS DEPARTMENT, THE RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) LABORATORY OF MOLECULAR GENETICS, NATIONAL INSTITUTE ON DEAFNESS AND OTHER COMMUNICATION DISORDERS, NATIONAL INSTITUTES OF HEALTH, ROCKVILLE, MD, USA (3) CHEMISTRY DEPARTMENT, TECHNION, HAIFA

Introduction: Type 1 Usher syndrome (USH1) is a recessively-inherited condition, characterized by profound prelingual deafness, vestibular areflexia, and prepubertal onset of retinitis pigmentosa (RP). USH1 can be caused by mutations of at least seven different genes, including PCDH15, encoding protocadherin 15. Interestingly, while nonsense mutations of PCDH15 cause USH1, missense mutations of the same gene are associated with nonsyndromic deafness. Based on this finding we hypothesize that normal retinal function, compared to normal hearing, requires lower level of protocadherin 15 activity. A possible strategy for partially restoring protocadherin 15 activity in USH1 patients harboring nonsense mutations is suppression of nonsense codons by aminoglycoside antibiotics.
Patients / Methods: We use a transcription/translation assay of a reporter plasmid harboring various PCDH15 nonsense mutations, in order to test their suppression in vitro. To test suppression ex vivo, we transfect cells with PCDH15 partial cDNA harboring different nonsense mutations and treat them with aminoglycosides. We are also involved in development of new aminoglycoside derivatives with higher efficiency and lower toxicity.
Results: We demonstrated up to 91% suppression of different PCDH15 nonsense mutations by aminoglycosides in vitro. We are now evaluating suppression of these nonsense mutations ex vivo, using transfected cells in culture. To date we tested over twenty new aminoglycosides and found one paromomycin derivative, named NB30, which demonstrates high suppressive efficiency with lower toxicity.
Conclusions: The research described here will have important implications for development of targeted interventions that are effective for patients with USH1 and other genetic conditions caused by various nonsense mutations.

IDENTIFYING THE GENETIC BASIS FOR SYNDROMIC AND NON-SYNDROMIC RETINITIS PIGMENTOSA IN ARAB AND JEWISH FAMILIES FROM NORTHERN ISRAEL

(1) * MRS. AUSLENDER NOA (1) MRS. BENAYOUN LIAT (2) DR. ABBASI ANAN (1) MRS. RIZEL LEAH (2) DR. GARZOZI HANNA (3) DR. ALLON-SHALEV STAVIT (1) PROF. PERLMAN IDO (1) DR. BEN-YOSEF TAMAR

(1) RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) BNAI ZION MEDICAL CENTER, HAIFA (3) HA'EMEK MEDICAL CENTER, AFULA

Introduction: Retinitis Pigmentosa (RP) is the most common form of hereditary retinal degeneration, characterized by night-blindness and gradual loss of the visual field. The molecular causes of RP are strikingly heterogeneous. There are autosomal dominant, autosomal recessive and X-linked forms. To date over 40 loci and genes have been implicated in non-syndromic RP. However, in more than 50% of RP patients, the underlying genes are yet to be found. RP can also appear as part of various syndromes, one of them is Usher Syndrome (USH).USH can be caused by mutations in at least 11 different genes.

Patients / Methods: Linkage analysis is performed with polymorphic markers linked to each of the mapped loci for the relevant disease, in order to prove or to rule out whether the phenotype in each family is linked to one of these loci. If linkage is identified, we search for the causative mutation. In the case of exclusion of linkage to all known genes and loci for a certain disease, and given a family big enough to support a significant LOD score, a genome wide screening approach will be taken to map the causative gene.

Results: To date, we ascertained over 30 Arab and Jewish families from diverse ethnic origins, with either RP or USH. In three of the USH families we found known mutations (The R245X mutation of PCDH15 and the N48K mutation of USH3A). In two of the USH families we found evidence for linkage to the USH2A locus. Mutation analysis in these families is underway.

Conclusions: The research described here will have important implications for carrier screening, genetic counseling and better understanding of the pathophisiology of RP in various populations.

MUTATIONS ANALYSIS OF DNA EXTRACTED FROM AGE-RELATED CATARACT LENS

(1) * DR. CHELES DORINA (2) MRS. AVRAHAM BAT-CHEN R (3) DR. COHEN YORAM (4) DR. EHRLICH RITA (4) DR. PIRIATINSKY BORIS (4) PROF. WEINBERGER DOV (2) DR. GOLDENBERG-COHEN NITZA

(1) TEL-AVIV SOURASKY MEDICAL CENTER (2) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, TEL-AVIV UNIVERSITY AND SACKLER SCHOOL OF MEDICINE, TEL-AVIV (3) SHEBA CANCER RESEARCH CENTER (4) RABIN MEDICAL CENTER, BEILINSON AND GOLDA CAMPUS, PETACH TIKVA

Introduction: Recently, Nishimoto et. al. found that mice deficient in the DLAD gene are incapable of degrading nuclear DNA during lens cell differentiation, and develop cataract. The loss of organelles is believed to ensure the transparency of the lens, but the molecular mechanism behind this process is not known. DLAD, a Dnase II- like acid DNase, is responsible for the degradation of nuclear DNA and DLAD mRNA was reported as highly expresses in human and mouse lens starting in late stage of embryogenesis. When DNA is left undigested in the lens, it causes cataracts of the nucleus lentis, blocking the light path. The aim of this study is to search for possible DLAD mutations in age related human cataract.
Patients / Methods: We tested 31 surgically excised capsules from nuclear age related cataract lenses for DLAD gene mutations. All the patients in this study underwent slit lamp examinations and their cataracts were classified according to Lens Opacities Classification Scale grading system (LOCS grading system). DNA Isolation: DNA was extracted using standard SDS/proteinase K digestion followed by phenol-chloroform extraction and ethanol precipitation. Detection of DLAD mutations: DLAD 6 exons using PCR amplification followed by direct sequencing of the PCR products. PCR primer sequences were designed using Primer 3 program.
Results: Overall, no new DLAD mutations were identified. The snips identified are reported in the literature. Frequencies of the snips of exons 1, 2 and 6 were similar to those reported in the Chinese and Japanese, but not in Afro-American populations.
Conclusions: We found no new mutations in any of the DLAD exons. The genotype frequencies of the snips located were similar to the Chinese and Japanese, but not Afro-American populations. The frequency of the snip in exon 5 has not been reported before. In contrast to the findings in the mouse model, no mutation was associated with human cataractogenesis. Further studies are needed to evaluate the role of DLAD in different types of cataract, and in the congenital and pediatric group.

A MISSENSE MUTATION (D144E) IN THE VSX1 GENE IS ASSOCIATED WITH KERATOCONUS IN AN ASHKENAZI JEWISH FAMILY

(1) * DR. PRAS ERAN (1) DR. ZADOK DAVID (2) DR. GARZOZI HANA (1) DR. AVNI ISSAC

(1) THE DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN (2) THE DEPARTMENT OF OPHTHALMOLOGY, BNEI ZION HOSPITAL, HAIFA.

Introduction: Purpose: To identify the genetic defect associated with keratoconus (KC) in an Ashkenazi Jewish family, and to asses the phenotypic expression of mutation carriers.
Patients / Methods: A three generation Ashkenazi Jewish family with KC was ascertained. The diagnosis of KC was made on the basis of clinical examination, and corneal topography. Candidate loci analysis was performed using micro-satellite polymorphic markers in close proximity to 7 previously associated KC loci and genes. Mutation analysis of the VSX1 gene was performed by direct sequencing of PCR-amplified exons, and a BseR1 restriction assay. In selected cases, where the genotype was consistent with KC additional effort to detect subtle corneal changes was made by using computerized Orbscan measurements.
Results: Co-segregation between KC and a polymorphic marker close to the VSX1 gene was observed. Sequencing the coding region of the VSX1 gene revealed a C to G substitution at position 8 of exon 2, resulting in a change of aspartic acid at position 144 to glutamic acid (D144E). Mutation carriers manifest a wide spectrum of corneal shapes ranging from apparently normal dome to severe KC.
Conclusions: This constitutes the first description of a molecular defect associated with KC in Israel. Genotype/phenotype correlation reveals variable corneal expressions within mutation carriers.

THE GENETICS OF X-LINKED RETINAL DEGENERATIONS IN THE ISRAELI POPULATION

(1) * MRS. MIZRAHI-MEISSONNIER LILIANA (1) MR. HUDERA SHMUEL (1) MRS. NEIS RUHAMA (1) DR. BANIN EYAL (1) DR. SHARON DROR

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Introduction: At least seven loci on the X-chromosome have been associated with X-linked cone-rod degeneration (XLCRD) or retinitis pigmentosa (XLRP). Only two of these genes, RPGR and RP2, have thus far been identified. RPGR is a major retinal disease gene responsible for about 75% of XLRP cases and for an unknown percentage of XLCRD cases in a mutation-specific manner. Our aim is to study the genetic causes of X-linked retinal degenerations in the Israeli population.
Patients / Methods: Clinical evaluation included detailed family history, full ophthalmologic exam, assessment of refractive error, color vision testing, and full-field electroretinography. A haplotype analysis was performed by studying DNA markers located within and around the RPGR gene and the COD2 locus. Mutation analysis was performed by either the SSCP technique or direct sequencing.
Results: We have recruited 27 families with XLRP and 9 with XLCRD. Among XLRP patients, SSCP analysis of RPGR exons 1-14 revealed one missense mutation in a family with dominant XLRP inheritance. Sequencing analysis of ORF15, the terminal RPGR exon, in XLRP families revealed two null mutations, one of which is novel. Each of these three mutations was found in a single family. A sequencing analysis of the RP2 gene did not yet reveal any mutations. Only one mutation in the 3' region of ORF15 was identified in patients with XLCRD. Linkage analysis of the remaining XLCRD families excluded RPGR in 3 of families and gave indication for the involvement of the COD2 locus in these families. Aiming to identify the COD2 gene, we tabulated all known genes in this region and collected information regarding the expression pattern and protein function. Out of 33 evaluated genes, we consider two as candidates for the disease. A mutation analysis of these genes is in progress.
Conclusions: Our results indicate that, similar to other inherited diseases, the mutation spectrum of X-linked retinal degenerations in the Israeli population is unique and differs from other populations. This is based on the low number of mutations identified in RPGR, the lack of RP2 mutations, and the involvement of the COD2 locus which was reported so far in only one family with XLCRD.

GENETIC ANALYSIS OF LEBER CONGENITAL AMAUROSIS IN THE ISRAELI POPULATION

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(1) OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM, ISRAEL (2) MICHAELSON INSTITUTE FOR VISION REHABILITATION, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM, ISRAEL.

Introduction: Leber Congenital Amaurosis (LCA) is the most severe hereditary nonsyndromic retinal disease. LCA is a heterogeneous condition inherited mainly in autosomal recessive mode and caused by at least 10 genes, seven of which have been identified to date. One of the major LCA genes is crumbs homolog 1 (CRB1) responsible for 10-13% of LCA cases in the European population.
Patients / Methods: Patients with LCA were identified within the DNA bank of the Center for Retinal and Macular Degenerations at Hadassah Medical center. Clinical evaluation included detailed family history, a full ophthalmologic exam, assessment of refractive error, and full-field electroretinography. Genomic DNA was evaluated for mutations using the LCA microarray. Mutations were verified by sequencing analysis. Three genetic markers within the CRB1 gene were chosen for segregation analysis. The genotype was determined by a restriction enzyme analysis.
Results: Out of 384 families thus far recruited to our DNA bank, 25 manifested LCA (8 Jewish, 15 Arab, and 2 Bedouin families) and 4 had early-onset retinal degeneration. Patients from 15 of these families were evaluated for all known LCA mutations. The analysis revealed one CRB1 mutation and many nonpathogenic sequence changes in LCA genes. The CRB1 mutation (V578E) was identified in a large Muslim family and cosegregated with the disease. A mutation analysis of CRB1 in two other families revealed two mutations, one of which is novel. To study the role of CRB1 in the remaining families, we developed an assay for homozygosity testing in 18 consanguineous families. The analysis revealed seven index patients who were homozygote for all three CRB1 markers. A cosegregation analysis of the corresponding families excluded CRB1 as the cause of disease in two of these families.
Conclusions: Our results show that LCA is more prevalent in the Arab population (~12% of Arab families with inherited retinal disease) compared to the Jewish population (only ~3%). In addition, our analysis suggests that LCA mutations in the Israeli population are unique to a specific family. This complicates genetic diagnosis and counseling in LCA families, and suggests that novel, presently unknown LCA genes, are the cause of disease in many Israeli LCA patients.

GENETIC APPROACH TO STRUCTURE-FUNCTION ANALYSIS OF THE DROSOPHILA TRP CHANNEL

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Introduction: The Drosophila TRP is the prototypical member of the TRP channel protein family. TRP is a Ca2+ permeable channel that is expressed in many tissues and cell types. The light-induced current in Drosophila phototransduction is mediated by the TRP and TRP-like channels, whose gating and detailed channel properties are still unclear.

Patients / Methods:

Results: In order to explore TRP channel properties, point mutations were induced by chemical mutagenesis. White-eyed homozygous flies were selected from red-eyed heterozygous lines and were screened for defects in phototransduction using electroretinogram (ERG). Six mutant lines with the trp phenotype were isolated. These mutant strains failed to complement with the trp343 null mutant, suggesting that the mutation is in the trp gene. The full genomic sequence of the gene of these lines was amplified by PCR, cloned into expression vector and sequenced. The level of the TRP protein was assayed by Western blot analysis. Full length TRP protein was detected in 2 of the 6 lines and the amount of the protein was compared to that of wild-type flies, while in the other mutants no protein was detected.

Conclusions: The classical method of forward genetics has proved to be useful to obtain new information about the channel properties. Here we report on a new point mutation in the trp gene, with the trp phenotype, however, the rate of decline of the light response to baseline during light and the speed of the recovery from inactivation, are significantly reduced relative to the null mutant. In addition a considerable amount of full-length protein is still found. Further work needs to be done to fully characterize this mutant, which offers great potential for understanding the gating and mechanisms of the TRP channel. We thank Charles Zuker for supplying the flies and Vered Ben-Naim for help in the molecular genetic studies.

EARLY DIAGNOSIS OF AGE RELATED MACULAR DEGENERATION BY IDENTIFICATION OF TRITANOPIA

(1) * MRS. BEN ELI HADAS (1) PROF. HOCHSTEIN SHAUL (2) DR. CHOWERS ITAY

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Introduction: Despite the fact that early diagnosis of age related macular degeneration (AMD) is mandatory to obtain maximal benefit from current treatments, the vast majority of AMD patients first present to the ophthalmologist at advanced stages of the disease. We aim to develop a self-administered set of psychophysical card tests for early diagnosis of non-neovascular AMD based on the identification of tritanopia defects. Such defects are common in AMD patients.
Patients / Methods: A series of test cards were developed to detect tritanopic defects and difficulties in detecting decreased color saturation. In order to test color vision specifically, all tests used iso-luminant stimuli. Tests were then presented to patients with known tritanopia, patients with non-neovascular AMD, and age and visual acuity matched healthy controls.
Results: 12 AMD patients, 5 patients with tritanopia, and 21 healthy controls were shown modified colored Amsler grids, Pelli-Robson gratings and triangle charts that we have developed. AMD and tritanopia patients more commonly failed to recognize blue-yellow gratings at relatively high contrast levels compared to the same saturation and frequency in a red-green chart as well as compared with healthy individuals.
Conclusions: These preliminary data suggest that detection of tritanopia may be valuable for early diagnosis of AMD. The novel tests that we developed may serve as a screening tool to identify individuals at increased risk for AMD.

CHANGES IN THE REFRACTIVE STATE DURING PREY CAPTURE UNDER LOW LIGHT IN THE NOCTURNAL CARDINALFISH APOGON ANNULARIS

(1) MR. HOLZMAN ROI (1) DR. SHASHAR NADAV (2) PROF. HOWLAND HOWARD, C. (3) * PROF. KATZIR GADI

(1) THE INTERUNIVERSITY INSTITUTE FOR MARINE SCIENCES AND DEPARTMENT OF EVOLUTION, SYSTEMATICS AND ECOLOGY, THE HEBREW UNIVERSITY OF JERUSALEM, EILAT (2) NEUROBIOLOGY AND BEHAVIOR, CORNELL UNIVERSITY, ITHACA, NY, USA. (3) BIOLOGY, ORANIM, UNIVERSITY OF HAIFA, TIVON

Introduction: Many nocturnal and crepuscular fish use vision to feed and function under low light levels. However, little is known about their ability to accommodate or their visual acuity under these light levels
Patients / Methods: We used Infrared Photoretinoscopy to track the refractive state of the eye during prey capture under low light in Apogon annularis, a nocturnal reef fish. Anatomical measurements of the eyes allowed calculations of visual acuity.
Results: Changes in the refractive state were observed in ~ 75% of the prey capturing strikes, preceding the strikes by 30 ms. These changes were rare between strikes or when prey was absent. Anatomical measurements indicated that the number of photo-detection units in a retinal image greatly exceeded the minimal number needed to detect prey.
Conclusions: We conclude that nocturnal vision in A. annularis is sufficiently sensitive to allow accommodation during prey capture.

BINOCULAR PROCESSING MANIPULATING MONOCULAR PERCEPTION

(1) * MR. YEHEZKEL OREN (1) PROF. BELKIN MICHAEL (2) PROF. SAGI DOV (1) MR. POLAT URI

(1) GOLDSCHLEGER EYE RESEARCH INSTITUTE, , TEL-AVIV UNIVERSITY , TEL HASHOMER (2) DEPARTMENT OF NEUROBIOLOGY, BRAIN RESEARCH, THE WEIZMANN INSTITUTE OF SCIENCE.

Introduction: Bias in perceived grouping was examined in conditions where the two eyes receive different spatial external distortion.
Patients / Methods: Stimuli, 10x10 dots matrix with Horizontal and Vertical spacing defining the direction of grouping (V/H), were presented, in random order either to both eyes (binocular), or only to one (monocular) while the other exposed to background luminance. Cylindrical lens were used to introduce a constant distortion along one direction. Bias and reaction times were measured under three dichoptic conditions: (1) binocular and monocular without distortion (2) orthogonal distortions between the eyes presented simultaneously or in different trials (3) as in (2) but with monocular distortion to one eye. Task was to distinguish between vertical and horizontal groupings without feedback.
Results: (1) a) without induced distortion (N=14) subjects show no bias, with sharp transitions between V/H groupings, b) at the transition, reaction time increased, with binocular being slower than monocular by 160 ms (2) orthogonal distortions between the eyes showed (N=10) no binocular bias but large monocular bias (3) monocular distortions (N=11) showed a bias in the treated eyes while untreated eyes showed orthogonal bias. Binocular reaction time was slower than the monocular reaction time by 150 ms. The bias with the dichoptic presentation was found to be the average of the two opposite monocular groupings.
Conclusions: The finding that the reaction time of the binocular level is slower (~150 ms), and that monocular distortion bias of the untreated eye, suggest that the binocular level manipulating the perception of the monocular level.

EVALUATION OF THE RETINAL NERVE FIBER LAYER IN CHILDREN WITH ENLARGED CUPPING OF THE OPTIC NERVE

(1) * DR. NESHER RONIT (2) DR. KRUPSKY SARA (1) DR. GEFFEN NOA (1) DR. EPSTEIN ESTHER (1) DR. KRYSTAL ORNIT (1) DR. RAZ YEHUDIT

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Introduction: The purpose of this study was to evaluate the thickness of retinal nerve fiber layer (RNFL) in children with enlarged cupping of the optic nerve, attempting to identify those with RNFL alterations. In such cases narrowing of RNFL may serve as an indicator of pre-perimetric glaucoma.
Patients / Methods: Nineteen children who were followed periodically by their ophthalmologist due to large cup/disc ratio and intraocular pressure below 21 mm Hg underwent complete ocular examination. Visual field testing (fastpac, 24-2) was conducted when possible. The control group consisted of 12 children with cup/disc ratio of less than 0.5 who underwent similar testing. The nerve fiber layer thickness was evaluated by scanning laser polarimetry (GDx VCC; LDT). The parameters of the study group were compared to those of controls, in addition to automated built-in evaluation in reference to the closest age group (above 18), provided by the GDx software.
Results: Children 5 - 18 years old were included in the study group. Ten children (20 eyes) could perform visual field, 17 fields of which were reliable. All visual fields were normal. Average thickness parameters were lower in children with enlarged cupping while the vertical diameter was higher. Nerve fiber indicator was normal but higher in the study group. Vert. dia. Horiz. dia. TSNIT mean Sup. mean Inf. mean TSNIT stdv. Inter-eye symm. NFI Study 2018 1823 50.7 62.9 58.4 21.6 0.87 24.9 Control 1603 1822 56.1 68.5 64.8 23.2 0.85 15.1
Conclusions: Children with larger vertical discs have lower RNFL thickness, probably due to wider spread of the nerve fibers around the disc. RNFL thickness may be helpful in the evaluation of children with enlarged cupping.

REDUCED FOVEAL CONTRAST SENSITIVITY IN GLAUCOMA

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Introduction: Contrast sensitivity (CS) was reported to be impaired in glaucomatous neuropathy. Here we used a simple computerized rapid test for foveal CS to demonstrate difference between glaucomatous and non-glaucomatous patients under mesopic and photopic conditions.
Patients / Methods: Glaucoma patients (eyes=33) and age matched controls (eyes= 24) with visual acuity of 20/30 or better underwent a comprehensive ocular examination, including perimetry. We used two computerized procedures of psychophysical testing: transient presentation of the target in a two temporal alternative forced-choice procedure; and a static method in four forced- choice procedures. The targets were Gabor patches with spatial frequencies of 1.5-9 cpd. The mesopic testing was conducted in a completely darkened room, with the monitor covered with neutral density filter, allowing luminance of only 0.03 cd/m2.
Results: Significantly lower foveal CS was found in glaucomatous patients under both photopic and mesopic conditions in all spatial frequencies ($p<0.05$). The difference between photopic and mesopic CS values were lower in the glaucoma group than the controls, corresponding to difficulty in night vision in glaucomatous patients. Both the transient and static methods yielded similar results and were significantly correlated ($p<0.05$).
Conclusions: The results indicate that disruption of central visual function may occur in glaucoma, despite good visual acuity. The similarity between the results of the two testing methods implies that the static method, being the shorter and easier out of the two, may be used in future research. Further research is necessary to establish CS testing role in the screening and monitoring of glaucoma.

PTERYGIUM SURGERY: FIBRIN GLUE VERSUS VICRYL SUTURES FOR CONJUNCTIVAL CLOSURE - LONG TERM RESULTS

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Introduction: Pterygia are wing-shaped conjunctival encroachments onto the cornea. They generally occur on the nasal side, and are more frequent in areas exposed to ultraviolet (UV) radiation. Surgical removal is the treatment of choice. Our aim was to compare the long-term results of conjunctival closure in pterygium surgery using fibrin adhesive versus Vicryl sutures.
Patients / Methods: A comparative prospective randomized clinical trial was performed on 81 patients (81 eyes) with primary nasal pterygium. Surgery in all patients consisted of the bare sclera technique combined with intraoperative mitomycin C. Patients were randomized to undergo conjunctival closure with a fibrin tissue adhesive (Quixil) (n=42) or 8-0 Vicryl absorbable interrupted sutures (n=39). Clinical assessment was performed on days 1, 3, 10 21 and 3,6,12 months after surgery . All patients completed a questionnaire at each follow up visit grading pain, discomfort and satisfaction with the procedure. The groups were compared for operative time, ocular signs and symptoms, overall satisfaction , cosmetic outcome and recurrence rate.
Results: Average operative time was 16 minutes in the fibrin glue and 28 minutes in the Vicryl suture group ($p<0.05$). Significantly less pain, photophobia, foreign body sensation, irritation, epiphora, itching, local hyperemia, conjunctival chemosis and dry eye sensation were noted in the subjects treated with glue than in controls ($p<0.05$). There were no complications during the follow-up period in the glue-treated patients. One of the patients in the suture group had a medically treatable corneal dellen a week after operation. At the end of follow up recurrent pterygium developed in 5 (11.9%) eyes of the fibrin glue treated eyes and in 3 (7.7%) of the vicryl sutured conjunctiva.
Conclusions: The use of fibrin glue in pterygium surgery significantly reduces operative time and patient symptoms of pain. Long term follow-up results shows that patients treated with fibrin glue developed a higher recurrence of the disease.

COMPARISON OF DIFFERENT TECHNIQUES OF ANTERIOR CHAMBER DEPTH AND KERATOMETRIC MEASUREMENTS

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Introduction: Purpose : To compare measurement results of anterior chamber depth and corneal curvature with Pentacam (Oculus, Wetzlar, Germany) to the IOL Master (Carl Zeiss Jena GmbH) Echoscan – US - 1800 (Nidek Technologies, Tokyo, Japan), and Automated keratometer ARK 700A (Nidek Technologies, Fremont, CA) measurements.

Patients / Methods: In 22 eyes (11 patients), with no previous eye surgery or pathology we prospectively obtained ACD measurements and corneal curvature readings using the Pentacam - Scheimpflug photography system ,and compared it to the ACD measurements of the IOL Master and Echoscan – US – 1800 (A – scan), and to the corneal curvature readings of the IOL Master and Automated keratometer.

Results: Eleven patients (22 eyes) were enrolled in the study. The mean patient age was 56.3 ± 14 years (range 23-71 years). The mean keratometric measurements of the 22 eyes with the IOL Master ,Automated keratometer and the Pentacam were : 43.9 ± 1.3 D (range 41.3-46.6 D), 43.5±1.3 D (range 41-46.3 D), and 43.5 ± 1.4 D (range 40.4 – 46.7 D), respectively. The mean ACD measurements of the 22 eyes with the IOL Master , Echoscan – US - 1800 ,and the Pentacam (including central corneal thickness values) were : 3.0 ± 0.4 mm (range 2.4-3.5 mm), 3.0 ± 0.4 mm (range 2.4-3.6 mm), and 3.1 ± 0.4 mm (range 2.4-3.6 mm), respectively. Statistical analysis using the bland altmann plot and calculated values of the 95% Limit of Agreement (LOA) demonstrated high concordance between the above instruments measuring ACD and corneal curvature.

Conclusions: When measuring ACD one should be aware of the different point of reference between the different devices (e.g. the Pentacam measures ACD from the posterior surface of the cornea while A-Scan and IOL Master measures ACD from the anterior corneal pole). Concerning this fact, there is high concordance between the ACD measurement of the Pentacam, the A-scan and the IOL Master. Moreover high concordance exists between the corneal curvature readings of the pentacam, the automated keratometer and the IOL Master.

THE PATHOLOGICAL MECHANISM OF DELAYED INJURIES FOLLOWING SULFUR MUSTARD EXPOSURE: 1. CORRELATION BETWEEN CLINICAL STATUS AND CORNEAL INNERVATION

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Introduction: To further study the pathological mechanism of sulfur mustard (SM)-induced delayed keratopathy by comparing corneal innervation in impaired and non-impaired eyes, one month following exposure and correlate it with clinical parameters.
Patients / Methods: Animal Care and Use Committee approval at IIBR was obtained. Rabbit eyes were exposed to SM vapor and a clinical follow-up was carried out for one month, using slit-lamp biomicroscopy and pachymetry. At the end of the monitoring period, animals were euthanized, eyes enucleated and processed for histochemical evaluation. Eyes were divided into two groups of clinically impaired and non-impaired according to the appearance of neovascularization (NV) and/or bullae. The structure and density of nerves was studied in whole mount corneas, stained for cholinesterase activity. Quantitative analysis of neural density in central and peripheral corneal regions was carried out, by measurement of total nerve fiber length per defined area, using the Image-Pro software. Pearson correlation was performed between the clinical scores and neural density.
Results: Delayed injuries were observed in 50% of the SM-exposed corneas, representing two sub-populations of impaired and non-impaired. In general, SM exposed corneas were characterized by a large heterogeneity in neural density among different corneas and among different regions within the same cornea, in contrast to the homogeneous pattern of innervation observed in naïve corneas. Clinically impaired corneas displayed poor innervation and the total fiber length was significantly smaller compared to naïve ($p<0.025$) and non-impaired ($p<0.009$) corneas, in both peripheral and central regions. The non-impaired corneas displayed even denser nerve network than naïve corneas, due to the extensive regenerative process. Significant correlations were found between neural density and corneal clinical score for both periphery ($r=-0.75$, $p<0.01$) and central ($r=-0.55$, $p<0.05$) corneal regions.
Conclusions: The reduced corneal innervation and the abnormal healing of corneal sensory nerves following SM exposure play a key role in the pathogenesis of long-term injuries that characterize 50% of the exposed eyes in our rabbit model. The prolonged damage of corneal nerves may stimulate chronic inflammation and may induce deficit of neurotrophic factors that are essential to maintain the integrity of normal corneal epithelium, thus indicate potential therapy.

ULTRAVIOLET IRRADIATION ON RABBIT CONJUNCTIVA AND CORNEA - HISTOLOGICAL AND CLINICAL EFFECTS

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Introduction: Exposure to ultraviolet radiation from the sun or from man-made devices is associated with an increased risk of cataract, macular degeneration, photokeratitis, pterygium, and climatic droplet keratopathy. The purpose of this study was to examine the histological and clinical effects of controlled ultraviolet (UV) irradiation according to duration and amount of exposure on the conjunctiva and cornea of rabbit eyes.
Patients / Methods: The eyes of ten rabbits were irradiated with a UV lamp for 0.5-4 h daily and examined by slit-lamp microscopy. Two weeks later, the eyes were enucleated and examined histologically and immunohistochemically.
Results: Conjunctival redness, corneal erosion, edema and opacity increase with longer UV exposure as well as over time (days). However, conjunctival discharge decreased over time. Histologically, the corneal epithelium has lost its typical multilayer appearance, mainly of the basal cell layer. In addition there was significant lymphocytic infiltration of the subepithelial conjunctiva (diffuse and focal) as well as focal tissue necrosis in rabbits exposed for more than 2 hours.
Conclusions: Daily UV irradiation for 2 h or longer increases damage to rabbit eyes. This probably represents a threshold of tolerable radiation to the conjunctival and cornea. This study shows that UV irradiation leads to lymphocytic infiltration in the conjunctival subepithelium, which may represent an immune reaction to damaged epithelial cells.

TREATMENT OF RECURRENT CORNEAL SALZMANN'S NODULAR DEGENERATION: KERATECTOMY AND MMC

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Introduction: To report on the outcome of the Salzmann's nodular degeneration excision.
Patients / Methods: Seven patients aged 65 to 78, who had recurrent Salzmann's nodular degeneration episodes after previous excision, underwent excision of the corneal lesions. The lesions were dissected / peeled from the corneal surface. Mitomycin C, 0.02%, soaked on a sponge, was placed for 1-2 minutes on the exposed stromal surface. Patients were closely followed until the corneal epithelialization was complete, thereafter 1 month, 3 months and one year.
Results: During the follow-up period, the corneas remained clear. In two eyes, there was mild stromal haze, but no recurrence of the Salzmann nodular degeneration lesions. The visual acuity improved from finger counting to 20/100 - 20/40.
Conclusions: Excision of Salzmann's nodular degeneration lesions and adjunctive use of MMC prevented the recurrence of Salzmann's nodular degeneration nodules on the corneal surface.

INTRAOPERATIVE MITOMYCIN AND CORNEAL ENDOTHELIUM AFTER PHOTOREFRACTIVE KERATECTOMY

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Introduction: To determine whether there is an increased risk to the corneal endothelium when using mitomycin C (MMC) following laser surface ablation.
Patients / Methods: In this prospective double-masked randomized study, we analyzed the corneal endothelium preoperatively and postoperatively in 18 eyes of 9 patients who underwent either MMC or balanced salt solution (BSS) supplemented surface refractive surgery. Following laser ablation, 1 eye was randomly assigned to intraoperative topical MMC 0.02% treatment for 30 seconds, and the fellow eye, the control eye, was treated in a standard fashion using topical BSS. Pre-operative pachymetry and endothelial cell count were performed and compared with postoperative measurements after 1 month, and 3 months.
Results: There was no significant difference in the preoperative endothelial cell count between the 2 groups: MMC group 2835 ± 395, control group 2779 ± 492, $p = 0.62$. In the control group, at 1 month and 3 months the difference in the endothelial cell count was not statistically significant ($p = 0.27$, $p = 0.11$, respectively). However, in the MMC group the endothelial cell loss was statistically significant: at 1 month 14.7 ± 5.1 %, and at 3 months 18.2 ± 9.0 % ($p = 0.0006$, $p = 0.003$, respectively).
Conclusions: The use of intraoperative topical MMC 0.02% for 30 seconds following corneal laser surface ablation may affect the endothelial cell count.

REFRACTIVE SURGERY AMONG ISRAEL DEFENSE FORCES INDUCTEES

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Introduction: Little is known about refractive surgery among soldiers and if it affects combat fitness. A surgical procedure that can eliminate dependency on corrective eyewear may be advantageous for combat military personnel. Yet, there are several side effects associated with refractive surgery that may have a detrimental effect on the visual performance of combat soldiers. The current study examines the incidence of refractive surgery among candidates for military service in the IDF between the years 1998-2005 as well as the percentage of soldiers who were accepted and persevered in combat units.

Patients / Methods: The study subjects were ametropes found among all military service candidates examined by the IDF during the years 1998-2005. The incidence of recruits who underwent refractive surgery was evaluated by calculating the percentage of the recruits who underwent surgery out of all ametropic recruits. Their ability to perform combat duties was determined by comparing the percentage of operated and eyeglass wearing recruits that remained in their combat units until completing military service.

Results: The percentage of the recruits who underwent refractive surgery out of all ametropic recruits rose steadily between the years 1998-2005 and was 0.15% in 1998 and 0.83% in 2005. The percentage of operated recruits assigned to combat duty rose during the study period, from 27% in 1998 to 69% in 2005. The drop-out rate from combat units was about the same in the eyeglass wearing and operated-on groups (30% and 20% respectively).

Conclusions: There is a significant rise in the number of military service candidates who undergo corneal refractive surgery before being recruited into the IDF. The similar high percentage of operated and eyeglass wearing soldiers who serve uninterruptedly in combat units indicates that this procedure causes no restriction in performing their duties.

APOPTOSIS MEDIATES PHACOEMULSIFICATION INDUCED INJURY IN CORNEAL ENDOTHELIAL CELLS AS DETERMINED BY A NEW IN VITRO MODEL

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Introduction: It is well known that phacoemulsification has harmful effect on corneal endothelium. The damage is attributed to mechanical and thermal injury. It was recently suggested that the damage is mediated by chemical injury caused by free radicals that are generated in the process and induce apoptosis. The purpose of this study was to examine the chemical injury caused by phacoemulsification to tissue cultures of bovine corneal endothelial cells.
Patients / Methods: In order to exclude the mechanical effects of phacoemulsification, the cells were grown on a special Biofolie gas-permeable membrane. A frame holding the membrane with a confluent monolayer of cells was placed in a chamber with irrigation solution into which the phacoemulsification tip was introduced. The amount of apoptosis following the treatment was evaluated using the anti-Caspase 3 antibodies. The degree of apoptosis following phacoemulsification and Hydrogen-Peroxide treatment were compared and the protective effect of Ascorbic acid was evaluated.
Results: Exposure to Phacoemulsification induced apoptosis of corneal endothelial cells. The amount of detected apoptotic cells increased with the incubation time following the treatment reaching maximal amounts after 48 h at which 333±29 per 1mm² were stained by the antibody compared to 23±5. The average number of apoptotic cells induced by exposure for 48h to H2O2 at concentrations of 50 and 100 μM was 345±6 and 376±1, respectively. Comparison between sham and exposure to phacoemulsification, when fixation is made after 48 h, reveals that the number of apoptotic cells increases significantly from 45±12 to 333±28 (P=1.4E-09). The addition of ascorbic acid (10^{-3} M) reduced the apoptotic cells count to 219±15 (P=5.1E-06). The reduction is even greater when the concentration is higher (10^{-2} M) - 129±29 (P=1.9E-08).
Conclusions: We established a new model of membranes covered with cultured bovine corneal endothelial cells. This model enables us to avoid the mechanical damage caused by turbulent currents heating the cells on hard plates and mimics the conditions in vivo. We have proved that exposure to phacoemulsification energy induces a cellular cascade leading to apoptosis and addition of ascorbic acid to the irrigation solution reduces significantly the amount of apoptotic cells probably due to its free radicals scavenging properties.

A POTENTIAL RAT MODEL FOR PROLIFERATIVE DIABETIC RETINOPATHY

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Introduction: Studying retinal neovascularization due to diabetes is hindered by the lack of a proper animal model. Attempts to use vascular endothelial growth factor (VEGF) to induce angiogenesis in animal models did not lead to consistent results. The aim of this preliminary study was to establish an animal model for retinal angiogenesis, using Streptozotocin-induced diabetic rats injected intravitreally with an adeno-virus carrying the gene for VEGF (Ad-VEGF).
Patients / Methods: The right eyes of 16 SPD rats, 8 of which were streptozotocin-induced diabetic and 8 served as control, were injected with either Ad-VEGF or saline. Visual function was evaluated using electroretinogram and fundus morphology was photographed using a fundus camera. 3-4 months after injection animals were sacrificed for further analysis; FITC-dextran angiography was carried by transcardial perfusion and the vascular bed was imaged using fluorescence microscopy. Retinal histology was demonstrated on 16um cryosections following Hematoxilin & Eosin staining.
Results: Retinae of healthy rats injected with Ad-VEGF showed slight abnormalities in blood vessels morphology and a decreased ERG response, compared to controls. Retinae of diabetic rats had increased blood vessel permeability and decreased ERG response. The retinas of diabetic rats injected with Ad-VEGF exhibited substantial abnormalities in retinal structure and clear neovascular pathological changes.
Conclusions: Inducing over-expression of VEGF by gene transfer in diabetic rats has the potential for creating an animal model of proliferative diabetic retinopathy.

A NOVEL MODEL FOR CELL INDUCED OCULAR NEOVASCULARIZATION

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Introduction: Ocular neovascularization is a cause for blindness in the Western World. Severe visual loss in Diabetic retinopathy is caused by ischemia induced proliferation of new vessels. VEGF is thought to be a primary mediator of intraocular angiogenesis as well as vascular permeability. Currently there is no model that simulates proliferative retinopathy to its full extent. Our aim was to create a model that could be used for investigating possible therapeutic options for retinal neovascularization. Induction of proliferative retinopathy was produced by injecting autologous cells carrying the VEGF165 gene in the rabbit eye.

Patients / Methods: Smooth muscle cells were harvested from 6 rabbits from jugular vein segments. They were isolated and expended. Pseudo-typed retroviral-based vector was used to transfer the VEGF165 gene to the cells. The autologous smooth muscle cells carrying the VEGF165 transgene were injected into the left vitreous cavity of the rabbits. The right eye was used as a control. Three rabbits were injected with 1 million cells and three were injected with 2.5 million cells. One cell produces about 1 picogram of VEGF. By doing so, we planned to achieve intraocular neovascularization using the cells as the base in a pro-angiogenic environment with the continuous VEGF expression. The rabbits were followed up for 5 to 8 weeks by fundus examination and by weekly fluorescein angiography and color photography. ERG was performed at baseline, immediately after injection and at necropexy. Intravitreal VEGF levels were measured at the end of the experiment. The eyes were then studied histologically.

Results: Over expression of VEGF using autologous smooth muscle cells in this rabbit model produced retinal neovascularization. The extent and duration of this effect was directly related to the amount of the injected cells.

Conclusions: It is feasible to produce intra-ocular neovascularization by injecting intravitreally autologous smooth muscle cells carrying the VEGF transgene. Further investigation is needed to establish this model. After mastering this very first step we plan to further manipulate this basic model. We do believe that once this model will be established, it can be used for efficacy and long-term safety of therapeutic modalities for ocular neovascular diseases.

CHARACTERIZATION OF CENTRAL RETINAL ARTERY OCCLUSION IN A MOUSE MODEL

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Introduction: Central retinal artery occlusion (CRAO) is an ophthalmologic emergency that causes a major irreversible visual loss. Our purpose was to characterize the molecular changes in the retina following induction of CRAO in a mouse model.
Patients / Methods: CRAO was induced by laser activation of intravenous injected rose Bengal at the origin of the central artery emerging from the optic nerve head. The right eye was treated while the left served as control. A clinical fundus exam was performed 1, 3, 6, 12 and 24 hours following CRAO induction and also on 7 and 21 days. Fluorosceine angiography (FA) was performed 3,6 and 24 hours after CRAO induction. The animals were sacrificed and the eyes were prepared for histology, immunohistochemistry staining and apoptosis assay by TUNEL. RNA was extracted from the retina on different time intervals to analayze levels of expression of the following genes: HIF 1α (hypoxia induced factor), VEGF (vascular endothelial growth factor), and EPO1 (erythropoietin 1), using real time-polymerase chain reaction (RT-PCR).
Results: 6 hours following CRAO induction the retina was edematous with interrupted blood perfusion. FA revealed reduced arterial perfusion in the first 3 hours from the induction. Reperfusion was demonstrated after 24 hours. Histological sections showed nuclear loss in the inner retinal layers (RGC and INL) and apoptosis was maximal 24 hours post induction. Gene expression analysis revealed elevated expression of HIF 1α and VEGF levels within the first 3 hours that decreased to baseline 24 hours from the induction. EPO1 levels were elevated at 12 and 24 hours.
Conclusions: This is a valid model of CRAO, that enables to measure changes in the levels of expression of ischemia-related genes. This model might serve in the future to examine the efficacy and efficiency of various therapeutic strategies.

THE C1/C2 ALLELES OF TRANSFERRIN CONFER SIMILAR RISK FOR NEOVASCULAR AGE RELATED MACULAR DEGENERATION

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Introduction: Background: Recent studies suggested that iron and the proteins which are involved in its metabolism are potentially associated with the pathogenesis of age related macular degeneration as well as with other neurodegenerative disorders. It was further suggested that the C2 allele of transferrin confers increased risk for Alzheimer disease. The purpose of this study was to assess whether different alleles of transferrin are also associated with an increased risk for neovascular AMD.
Patients / Methods: Methods: DNA was extracted from blood samples of patients with neovascular AMD and unaffected, age matched, controls. Genotyping for the transferrin alleles was performed by restriction enzyme analysis as well as by automatic sequencing. The distribution of the different alleles of transferrin in the AMD group was compared with the control group using Fisher Exact test.
Results: Results: Of the 104 neovascular AMD patients, 54 (52%) were homozygote for the transferrin C1 allele, 7 (7%) were homozygote for transferrin C2 allele, and 43 (41%) were heterozygote. Of the 79 controls, 45 (57%) were homozygote for the C1 allele, 7 (9%) were homozygote for transferrin C2 allele, and 27 (34%) were heterozygote. Neither of the alleles was significantly associated with the disease.
Conclusions: Conclusion: Unlike the risk for Alzheimer disease, our data suggests that the transferrin C1 and C2 alleles confer similar risk for neovascular AMD. However, the existence of interactions of transferrin alleles with other susceptibility alleles for AMD such as those of apolipoprotein E cannot be excluded and deserves further evaluation.

PROLIFERATIVE DIABETIC RETINOPATHY - THE EPIRETINAL MEMBRANES

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Introduction: To better understand the pathobiology of proliferative diabetic retinopathy (PDR), we studied the morphology of epiretinal membranes (ERMs) that were removed from eyes of patients with advanced diabetic eye disease during closed intraocular microsurgery.
Patients / Methods: Twenty membranes were excised from the retinal surface in patients that needed pars plana vitrectomy for complications of PDR such as non-clearing vitreous hemorrhage, tractional retinal detachment with macular involvement and combined tractional and rhegmatogenous retinal detachment. The ERMs were removed by intra-ocular forceps, fixated, stained by Alcian blue, embedded in paraffin wax, sectioned, stained histochemically and immunohistochemically and then studied by light microscopy.
Results: The ERMs from this group of diabetic patients were typically composed of major vascular element, many fibroblasts, minor glial component and collagen as the principal extra cellular matrix. The Alcian blue stained the mucopolysaccharides and thus enabled orientation of the membranes. Hypertrophy of endothelial cells in the neovascular complex of the ERMs was observed, as well as silicone oil droplets in the re-proliferationg fibrocellular membranes. The intensity of VEGF localization in the membranes depended on the age of the ERM; it faded when the proliferating tissue aged, as the vascular element became scarce and the collagenous component became prominent.
Conclusions: Based on our observations of the morphology of diabetic ERMs we propose a hypothesis for the role of glial cells and retinal new vessels in initating the epiretinal fibrovascular proliferation in diabetic eyes. Reproliferation of ERMs after vitrectomy and silicone oil injection as well as the role of growth factors such as VEGF and PEDF in proliferation and re-proliferation, will be discussed.

NEUROPROTECTIVE EFFECT OF BRIMONIDINE IN A MOUSE MODEL OF ISCHEMIC OPTIC NEUROPATHY

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Introduction: To elucidate the potential protective effect of brimonidine following induction of ischemic optic neuropathy (AION) in a mouse model.
Patients / Methods: AION was induced in C57Bl/6 mice by localized illumination of a photosensitive rose Bengal agent at 12, 24, or 48 hours following intraperitoneal (IP) injection of a single dose of brimonidine or 3 days of treatment with topical brimonidine 2% eyedrops. The animals were euthanized on day 1, 3, or 21 after injury for tissue staining and RT-PCR analysis of ischemia-, angiogenesis-, and oxidative-stress-induced gene expression. Findings were compared with untreated AION eyes and with the contralateral healthy eyes of the treated mice.
Results: In the untreated AION-induced eyes, at day 1 following rAION induction, levels of endothelial nitric oxide synthase (eNOS), vascular endothelial growth factor (VEGF), glutathione peroxidase-1 (Gpx), copper-zinc superoxide dismutase-1 (SOD1), hypoxia inducible factor 1, alpha subunit (HIF-1 alpha) and pigment epithelium derived factor (PEDF) were reduced to 45%, 80%, 80%, 80%, 89% and 90% of levels in healthy control eyes. Heme-oxygenase-1 (HO-1) levels did not change. On day 3, eNOS, PEDF, and Gpx returned to normal levels, but VEGF, HO-1, and SOD1 increased 1.2-, 1.4-, and 1.5-fold, respectively. On day 21 following AION induction, the levels of expression of all genes returned to normal, except for HO-1 and, to a lesser extent, PEDF, which were high. By contrast, in eyes treated with bromonidine, there was no change in PEDF, and HO-1 increased, but to a lesser extent. This effect was true for both modes of treatment, but more significant for the IP route. Topical treatment was also associated with reduced levels of VEGF, endothelial-specific receptor tyrosine kinase (Tie2), HIF-1-alpha, Gpx, and SOD1. Interestingly, IP treatment had an inverse effect on VEGF and Tie2 at 21 days.
Conclusions: Brimonidine treatment causes changes in the level of expression of various ischemia-, oxidative-stress-, and angiogenesis-induced genes following AION induction. The neuroprotective effect of these changes will be discussed and the preferred route of administration evaluated. These results are in agreement with previously published data, and should be further applied to patients diagnosed with AION in the acute phase.

MECHANISM OF RETINAL GANGLION CELL DEATH IN SECONDARY DEGENERATION OF THE OPTIC NERVE

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Introduction: We had previously applied a partial optic nerve transection model, which can morphologically separate between primary and secondary optic nerve degeneration, to demonstrate secondary degeneration in the optic nerve of monkeys and rats. We now used that model to investigate the mechanism of retinal ganglion cell (RGC) death in secondary degeneration.
Patients / Methods: Secondary degeneration of the optic nerve was induced in 117 rats. The apoptosis rate was evaluated by Hoechst staining at several time points, and the involvement of various signal transduction pathways by immunohistochemistry, western blotting and RT-PCR. The neuroprotective effect of minocycline on secondary degeneration was also measured.
Results: Significant apoptosis was detected in areas of secondary degeneration from 11-90 days (3.1±1.6% – 18.0±5.7%) after partial transection of the optic nerve ($p<0.01$ for days 11, 18, 38 and 90). We detected a significant early increase in immunolabeling for p-ERK at 3 days, a 2 fold increase in p-SAPK/JNK at 11 days and significant activation of the transcription factor p-c-jun from 1 38 days($p<0.05$ for each time point). These results were confirmed by western blotting. Levels of caspase-3 were mildly elevated (1.5 fold) between 1-7 days and then declined to baseline. p-AKT activation was detected by western blotting at 1 week ($p=0.06$). RT-PCR detected significant upregulation of pro-apoptotic genes from the p-53 pathway (Ei24 and , and of cdk2 in both secondary and primary degeneration (p)αGadd45<0.03). These genes were overexpressed from days 7-36 by 2-6 folds. The pro-survival gene IAP-1 (a caspase inhibitor) was simultaneously and significantly up-regulated (3 fold at 7 and 14 days, $p<0.01$), but the Bcl-2 gene was significantly down-regulated from 7-36 days during secondary and primary degeneration ($p<0.01$). Minocycline was significantly neuroprotective in primary degeneration (78% RGC survival with minocycline compared to 65% with saline) and less in secondary degeneration (p=NS) .
Conclusions: Secondary degeneration leads to significant apoptosis of RGC long after injury to the optic nerve. This process involves activation of the MAP kinase and p-53 signal transduction pathways, as in glaucoma and complete optic nerve transection. The pro-survival factors p-AKT and IAP-1 are simultaneously up-regulated, while the Bcl-2 gene is down-regulated.

NEUROPROTECTIVE EFFECT OF PN-277 ON LASER-INDUCED RETINAL DAMAGE

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Introduction: The retinal lesion induced by laser photocoagulation increases many times over by secondary degeneration processes whereby tissues adjacent to the primary lesion are damaged . This process involves release of noxious compounds, mainly glutamate, from the primarily injured cells that spread to the neighboring cells and harm them. We attempted to prevent these processes by immune neuroprotection by vaccination that activates T cells to neutralize damaging compounds and induce secretion of growth promoting factors. We used PN 277, a novel compound whose efficacy in immune neuroprotection was previously demonstrated in models of retina, optic nerve, brain, and spinal cord lesions. The purpose of our study was to test the neuroprotective ability of PN277 to reduce the spread of laser-induced retinal lesions
Patients / Methods: Standard argon laser lesions were created in 36 DA pigmented rats divided into two groups: a PN277 treated group (by subcutaneous injection seven days before lasering) and a control group. Histological and morphological evaluations of the lesions 3, 20, and 60 days after the injury were preformed.
Results: Significant reduction in photoreceptor loss after 60 days was seen in the retinas of the pre-immunized animals as demonstrated by lesion size reduction ($P=0.007$), cell density in the whole lesion ($P=0.013$) and in the center of the lesion ($P<0.001$).
Conclusions: The results show that pre-immunization with PN277 is neuroprotective in unmyelinated (gray matter) neural tissue such as the retina. This approach may be of clinical significance in ameliorating laser-induced retinal injuries in humans.

ANALYSIS OF CHOROIDAL NEOVASCULARIZION CASES MISSED BY THE PREFERENTIAL HYPERACUITY PERIMETER AND BY RETINA SPECIALISTS EXAMINATION

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Introduction: The PHP multicenter clinical trial has demonstrate the good specificity (88%) and sensitivity (82%) in detecting recent onset CNV and differentiating them from intermediate AMD. The purpose of this study was to determine the clinical characteristics of CNV cases that did not demonstrate a significant visual field defect on the PHP (false negative cases) and to determine the clinical significance of these findings.

Patients / Methods: A comparison of parameters indicating age, size of lesion, number of days with symptoms, visual acuity, lesion location, type of lesion amount of leakage, existence of blood, existence of previous laser treatment and existence of blocked fluorescence was made between the eyes with true positive PHP as compared to eyes with false negative PHP. A comparison of parameters indicating age, visual acuity and total drusen size was made between the eyes with true negative PHP as compared to eyes with false positive PHP was made.

Results: Comparing the eyes with true positive PHP results and the eyes with false negative results, CNV was more likely to be missed by the PHP if there was no classic component in the CNV lesion or if there was a relatively good visual acuity. ($p=0.05$). Comparing the eyes with true positive diagnosis by the retina specialists and the eyes with false negative results, CNV was more likely to be missed by the PHP if they were not subfoveal, did not contain blood, and if there was a relatively good visual acuity.

Conclusions: False negative PHP result is more likely to occur where there is no classic component to the lesions, and when there is a relatively good visual acuity. Since in any event treatment of occult lesions in not warranted until there is decrease progression and a visual acuity of 6/15 or less, this fact does not hamper the use of the PHP in monitoring AMD patients.

NEUROPROTECTIVE TREATMENT WITH NAP REDUCES LASER-INDUCED RETINAL DAMAGE IN RATS

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Introduction: The retinal damage induced by laser photocoagulation is multiplied by secondary degeneration processes whereby tissues adjacent to the primary lesion are damaged. NAP is an 8 amino acid peptide derived from activity-dependent neuroprotective protein (ADNP). It exhibits direct neuroprotection in cell culture, animal models (review - J Mol Neurosci. 2004;24:67) and against retinal ganglia cell degeneration associated with nutrient deprivation (IOVS. 2005;46:933). However, no in vivo retinal neuroprotection studies were reported to-date. The purpose of this study was to test the neuroprotective ability of NAP to reduce the spread of laser-induced retinal damage.

Patients / Methods: Standard argon laser lesions (514 & 544 nm, 200 mm, 0.1 W, 0.05 second) were created in 72 DA pigmented rats divided into four groups: two NAP treated groups (intravenously or intravitreally immediately after the photocoagulation) and two control groups treated similarly by saline. The lesions were evaluated histologically and morphometrically 3, 20 and 60 days after the injury.

Results: Preliminary results showed that when administered 24 hours after the lesion, NAP had no effect. In contrast, the immediate intravitreal injection of NAP had significant ameliorative effect ($P < 0.01$) 3 days after the laser injury. Furthermore, the immediate intravenous administration of NAP reduced the cell loss in the whole lesion at all tested time points, though only sixty days after photocoagulation this reduction was statistically significant ($P < 0.01$). When measured in the central lesion zone, cell loss was reduced at all the time points. The systemic administration of NAP had also significant ameliorative effect ($P < 0.01$) on the diameter of the lesion 20 and 60 days after the injury.

Conclusions: The results show that systemic treatment with NAP is neuroprotective in vivo on unmyelinated (gray matter) neural tissue such as the retina. The proposed mechanism of NAP's neuroprotection involves microtubules, key cytoskeletal elements in the living cell, implying a broad protective efficacy (J Biol Chem. 2004;279:28531). The results described above suggest that NAP development may be of clinical significance in the treatment of retinal lesions in humans. The intravenous formulation of NAP, termed AL-208, is under clinical development by Allon Therapeutics that supported this research.

INCORPORATION OF NEURONAL PROGENITORS DERIVED FROM HUMAN EMBRYONIC STEM CELLS IN A MOUSE MODEL OF RETINAL DEGENERATION

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Introduction: The purpose of the present study was to examine the potential of human embryonic stem cell (hESC) -derived neural progenitors (NPs) to survive, integrate and differentiate into retinal cells following in-vivo intraocular transplantation into the eyes of rd10 mice, which serve as a model for progressive retinal degeneration
Patients / Methods: Seventy-three rd-10 mice which manifest a rapid retinal degeneration due to a mutation in the PDE gene and thirty-one C57/Bl6 mice were included. Spheres enriched for neural progenitors (NPs) were derived from hESC (HES-1 cell line) by culturing in-vitro. Between 100,000-120,000 NPs, or saline, were injected into the vitreal and/or sub-retinal space of 104 adult and newborn mice eyes. Fellow eyes in each animal served as controls. Survival of the transplanted cells, their migration, proliferation and differentiation were assessed by histomorphology and immunohistochemistry up to 2 months post-injection.
Results: Following in-vivo intraocular transplantation, viable grafts were found in approximately 30% of rd10 eyes and 20% of control C57/Bl6 eyes. Transplanted cells migrated from the main grafts and integrated in the host retina. A small percentage of engrafted cells were Ki67-positive, indicating that they were still proliferating. In intra- and sub-retinal grafts, engrafted human cells expressing the retinal marker recoverin and the photoreceptor-specific markers NRL, red-green opsin and blue opsin were observed.
Conclusions: Human ES cell-derived NPs have the developmental potential to differentiate into retinal cells in rodent eyes manifesting retinal degeneration. hESC-based cell therapy of retinal disease may perhaps be possible in the future.

NOTCH1 SUPPRESSES CONE-PHOTORECPTOR FATE SPECIFICATION IN THE DEVELOPING MOUSE RETINA

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Introduction: The vertebrate retina is an excellent model system for the study of cell type specification in the CNS. During retinal development, Notch1 mediates local cell-cell signaling and is implicated in the onset of differentiation, cell fate decisions, survival and proliferation of retinal progenitor cells (RPCs). However, direct retinal phenotype analysis of Notch1 loss of function has been precluded due to lethality of Notch1 null mice at E9.5.
Patients / Methods: In order to investigate the role of Notch1 in retinogenesis we employ a conditional knockout approach (the Cre/loxP system) to delete Notch1 from the distal retina. Two transgenic lines were employed in this study for obtaining somatic inactivation of Notch1 exclusively in the RPCs: Notch1f/f mice and the a-Cre transgenic line. The third line employed for cell-lineage tracing of the mutant cells was the Z/AP reporter line, in which a human alkaline phosphatase (hAP) reporter gene is detected in cells that expressed the Cre.
Results: Histological and molecular analysis of Notch1f/f;a-Cre mice revealed that the retina is smaller and exhibits abnormal organization, namely rosette like structures, mostly composed of mutant cells. A significant reduction of Notch1- RPCs pool was found utilizing the Z/AP mouse line, allowing the identification of mutated cells. Decreased number of proliferating RPCs together with the expression of a subset of proneuronal genes, i.e., Math5, Ngn2 and NeuroD indicate cell cycle exit and premature differentiation. Most importantly, the majority of the Notch1- retinal cells adopt cone-photoreceptor fate evident from the expression of cone specific markers such as cone-opsin and cone-arrestin. This cone fate expansion appears to be on the expense of other early- and late-born retinal cell types.
Conclusions: Together, these findings reveal an unexpected role of Notch signaling in directly controlling neuronal cell-type composition, and suggest a model by which, during normal retinogenesis, Notch1 functions to suppress cone-photoreceptor fate, allowing for the specification of the diversity of retinal cell types.

THE EFFECT OF TRIAMCINOLONE ACETONIDE ON THE LEVELS OF VEGF IN RPE CELLS IN VITRO

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Introduction: In the last few years the use of Triamcinolone acetonide (TA) became wide in various retinal diseases including age related macular degeneration. Our purpose was to investigate the anti-angiogenic effect of TA.
Patients / Methods: We used humen retinal pigment epithelium cell culture (ARPE). We induced chemical hypoxia with the use of CoCl2. TA was added in the concentrations 0.1, 0.01 and 0.001mg/ml to cells cultures hypoxia induced for 24 and 48 hours. We investigated mRNA of VEGF, PEDF bFGF with the use of RT-PCR. We also examined in western blot the protein levels of VEGF and HIF-1α. Zymography was performed in order to examine the activity of MMP9 and MMP2. We also performed MTT cell proliferation assay to investigate cell death after we added TA with and without hypoxia
Results: We found high levels of HIF-1α in ARPE cells with hypoxia, TA did not change the levels of HIF-1α. No significant change was found in levels of VEGF , mRNA and protein levels. We found decreased levels of VEGF 189 with hypoxia and TA in concentrations of 0.01 and 0.001mg/ml. No change was found in mRNA levels of PEDF and bFGF. We found similar activity of MMP9 and MMP2 with and without hypoxia and with the various concentrations of TA. In the MTT assay we found decrease in cell viability with the add of TA 0.1mg/ml after 6 days (P=0.018 Chi-square). Cell viability decreased with hypoxia but no change was found with TA 0.1-0.001mg/ml
Conclusions: We managed to show increase in HIF-1α after induction of hypoxia. We found that TA did not change the levels of HIF-1α. We did not find a change in the levels of VEGF, PEDF and bFGF with and without hypoxia and TA. We found some decrease in the levels of VEGF 189 with TA 0.01-0.001mg/ml. Further investigation is needed in order to find the anti-angiogenic mechanism of TA in retinal ischemic diseases

AXIAL LENGTH MEASUREMENT IN EYES WITH DIABETIC MACULAR EDEMA: A-SCAN ULTRASOUND VERSUS IOLMASTER

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Introduction: The IOLMaster measures axial length (AL) from the corneal vertex to the RPE. It was calibrated to equal immersion A-scan ultrasound (US) that measures AL to the vitreoretinal interface - in healthy eyes. We compared AL measurements using US and IOLMaster in eyes with diabetic clinically significant macular edema (ME).
Patients / Methods: We prospectively measured AL using US and IOLMaster in 16 eyes with ME and 16 healthy eyes. Foveal thickness was measured with optical coherence tomography (OCT). The difference between both methods and its correlation to foveal thickness was evaluated.
Results: Mean ±SD foveal thickness in healthy and diabetic eyes was 202.8±30.9 µm (range 156-240 µm) and 476.9±124.6µm (range 331-758 µm), respectively. Mean AL by US and IOLMaster in healthy eyes was 23.0±0.85 mm (range 22.0-24.8 mm) and 23.1±0.86 mm (range 22.1-24.9 mm), respectively ($p<0.001$); in diabetic eyes it was 22.7±0.71 mm (range 21.5-23.6 mm) and 23.0±0.66 mm (range 22.0-23.9 mm), respectively ($p<0.001$). Mean±SD difference in AL between the two methods was 64 ± 49 µm (range –30 to 130 µm) in healthy eyes and 285 ± 144 µm (range 64 to 540 µm) in diabetic eyes. Correlation between AL difference and foveal thickness was poor in both healthy and diabetic eyes.
Conclusions: AL measurements using US and IOLMaster in patients with diabetic ME differ significantly. This is likely due to refractively-significant ME which is measured differently by the two methods. We suggest that in these patients the degree of ME be taken into account when calculating IOL power prior to cataract extraction. If concomitant or future treatment is expected to reduce ME then IOLMaster will provide a more clinically accurate AL measurement.

CYCLOSPORIN A PARTIALLY PREVENTS RETINAL ISCHEMIC INJURY

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Introduction: Retinal ischemia results in a marked damage, including thinning and disarrangement of retinal layers and loss of ganglion cells (GCs). Ocular pathologies that produce ischemia, such as acute and chronic glaucoma or retinal artery occlusion, can cause irreversible impairment of vision and blindness. The elucidation of the mechanism(s) of retinal ischemic injury may help developing novel therapeutic modalities for these diseases. Ischemic damage to retinal tissues results in an inflammatory response, which may either contribute to the damage or to the recovery of partially injured cells. Inflammatory response resembles the immune response and the two responses share many molecular characteristics. Here we report that temporary inhibition of the immune response decreases the ischemic damage to rat retina
Patients / Methods: Sparague-Dawley male rats (250-350 g) were used throughout. Retinal ischemia was produced by 90min increase of intraocular pressure, via a 27G needle inserted into the anterior chamber and connected to a sterile saline infusion maintained at 160cm above the eye level. Ischemia was verified by direct ophthalmoscopy. The animals were divided into three groups: Untreated controls, rats injected IP with 20, and 50mg/kg of CA 4h before ischemia. 7 days post-ischemia the animals were sacrificed, the eyes dissected out, fixed, sectioned and stained with hematoxyllin-eosin. The thickness of individual retinal layers and the number of ganglion cells were determined at four locations in each retina. These parameters were normalized by dividing the value for each parameter determined in ischemic retina by a corresponding value in fellow, non-ischemic eye in the same animal.
Results: As previously reported, ischemia caused major decrease in retinal layers thickness and in the number of ganglion cells. Most affected parameters were the inner nuclear layer (INL), the inner plexiform layer (IPL) and the number of GCs. Pre-treatment with CA decreased ischemia-induced ganglion cell loss (from 29+3% of non-ichemic controls, to 47+9 and 59+10% for 20 and 50mg/kg CA, $p<0.03$ and 0.01, respectively). Similar effect of 50mg/kg CA was seen for INL (from 62+3 to 73+5%, $p<0.05$) and IPL (from 30+3 to 59+9%, $p<0.01$).
Conclusions: Transient suppression of the immune response with CA partially protects rat eyes from ischemic changes in retinal morphology.

CLINICAL MEASUREMENTS OF BLOOD FLOW VELOCITY USING THE RETINAL FUNCTION IMAGER (RFI)

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Introduction: The Retinal Function Imager (RFI) is an experimental non-invasive, easy to use, direct qualitative and quantitative imaging method that extends the utility of fundus imaging by providing information about retinal blood flow velocity, blood oximetry, metabolic state and hidden vasculature. The aim of this work was to test the RFI in clinical use for the first time. In this work we concentrated on characterizing blood flow velocity in retinal arteries and veins and evaluating blood flow velocity measures in normal subjects.
Patients / Methods: 14 healthy volunteers (mean age 30.7 years) were used as subjects. All had a complete eye examination which was normal. For each subject one eye was tested. Each subject had at least two sessions of imaging using the RFI at different time points.
Results: The mean segment velocity was 5.4 ± 3.5 mm/sec for secondary and tertiary arteries and 4.1± 2.7 mm/sec for secondary and tertiary veins. The mean "global velocity" was 5.0 mm/sec with high variability (SD 17%). After adjusting for the heartbeat cycle phase, global velocity variability decreased from 17% to 10%.
Conclusions: The RFI provides a non-invasive, simple and reliable technique for retinal blood flow velocity measurement, and has the potential to become an important tool for diagnosis and follow-up in ophthalmology.

BROTH CULTURES YIELD VERSUS TRADITIONAL APPROACH IN THE WORKUP OF ENDOPHTHALMITIS. A PROSPECTIVE COMPERATIVE STUDY

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Introduction: The Bactec Peds Plus F is a broth medium usually used for culturing body fluids in pediatric departments. It has a capability to grow both bacteria and fungi requiring only minute amounts of specimen volume. In a previous study, presented in the 24th annual meeting of ISVER, we showed that this medium yielded high growth rates, compared to the traditional method of culturing, when used in cases with infectious kreatitis. The purpose of this study was to elucidate whether the Bactec broth can be also used for vitreous cultures in cases with clinically suspected endophtalmitis, and to compare yields between this method and the traditional method.
Patients / Methods: All consecutive cases with clinically suspected endophtalmitis in our institution from July 2003 were included in the study. All cases were cultured both in the Bactec Peds Plus F broth and the traditional method.
Results: 13 cases were included in this study. The overall growth rate for the Bactec broth was 28.4% higher than the rate of the traditional method (69.2% and 53.9% respectively). In two cases, one of acute-onset post operative endophthalmitis due to streptococcus mitis and one of delayed-onset post operative endophthalmitis due to candida albicans, there was a positive growth only in the Bactec broth.
Conclusions: Our results show that Bactec Peds Plus F broth can be used successfully also in the work-up of clinically suspected endophthalmitis. The method has, apparently, several advantages over the traditional method: time-savings, as only one medium needs to be inoculated, transportation to the laboratory is simpler as there is no need for immediate incubation, and there is no need to keep and maintain a supply of fresh agar media. This method is especially suitable for office settings and remote clinics, but also can be used in hospital setting, as an adjunct, to increase the growth yield.

STAPHYLOLYSIN IN THERAPY OF S. AUREUS EXPERIMENTAL ENDOPHTHALMITIS

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Introduction: To study the effect of staphylolysin, a staphylolytic protease secreted by Pseudomonas aeruginosa, in the management of Staphylococcal endophthalmitis.
Patients / Methods: Endophthalmitis was induced in rats by intravitreal injection of 50 freshly grown methicillin-resistant S. aureus cells. Six hours post-infection, rats were randomly assigned to treatment and control groups. Each received an intravitreal injection of 10 ul of staphylolysin (0.5 mg/ml; treatment group) or BSA (0.5 mg/ml; control). Forty-eight hours after the induction of endophthalmitis, vitreous samples were collected and processed for bacterial counting.
Results: A statistically significant reduction in bacterial counts was observed in the staphylolysin-treated eyes as compared with controls ($p<0.05$).
Conclusions: Staphylolysin appears to be effective in reducing the bacterial counts of S. aureus in this experimental endophthalmitis model. Further studies should be performed, to assess the potential of staphylolysin as a new therapeutic tool in the management of staphylococcal endophthalmitis that can overcome the problem of resistance to antibiotics.

INFLAMMATORY CELLS OF THE MURAL LACRIMAL CANAL IN PATIENTS WITH IDIOPATHIC PUNCTUM STENOSIS

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Introduction: Acquired punctum stenosis is a very common condition in which the opening of the lacrimal canal undergoes occlusion gradually over time. Various mechanisms have been reported in the past including dry eyes, post–infection, irradiation, pharmacological agents, cicatricial inflammations, burns, tumors, eye-lid malposition and idiopathy. The aim of the study was to verify and to evaluate the presence of inflammatory process in the wall of the lacrimal canal in patients presenting with constant epiphora due to lacrimal punctum stenosis. .
Patients / Methods: We conducted a prospective clinico-pathological study in which 66patients (49 females, 27 males, mean age 65±13Y) with constant epiphora due to idiopathic lacrimal punctum stenosis underwent punctoplasthy. The specimens were taken for histo-pathological evaluation during surgery with the main objective to identify the presence of inflammatory sub epithelial cells in the mural lacrimal canal.
Results: Inflammatory cells were found in 62(94%) of the specimens. Chronic inflammatory process consisted mainly from lymphocytes and plasma cells were found in most 44(67%) specimens. Co- existing acute and chronic (mixed type) inflammatory process involving neutrophiles and lymphocytes was found in 18(27%) specimens and only 4(6%) specimens were free of inflammatory cells. Even-though most of the specimens (40, 60.6%), showed mild or no (4, 6%) inflammation in almost 33.5% (22) specimens the inflammation level was graded between moderate (17 specimens, 25.8%) to severe (5 specimens, 7.6%). Although anatomical success (the remaining of an open punctum), was achieved in 62(94%) patients, functional success (no epiphora), was recorded in only 49(74.25%) patients. The main causes for the functional failure group were naso-lacrimal obstruction (13, 19.7%), canalicular stenosis (2, 3%) and restenosis (2, 3%). Among those, 11(64.7%) showed chronic and mixed type (acute and chronic) inflammation in 5(29.4%) patients.
Conclusions: High incidence rate (94%) of inflammatory cells was identified in specimens of mural lacrimal canal taken during punctoplasthy. Evaluation of the inflammation level among those showed mostly chronic process however acute inflammatory component was detected in more than one fourth (27%) of all patients. The high incidence inflammation involving the mural lacrimal canal can signifies for possible contribution of those to the forming mechanism of acquired lacrimal obstruction.

OCULAR INJURIES RELATED TO INDEPENDENCE DAY CELEBRATIONS

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Introduction: Each year, following Israel's Independence Day celebrations, patients are referred to our medical center as a result of ocular injuries from artificial snow spray, fireworks, plastic hammers, stick-lights (plastic tubes filled with fluorescent liquid), and more. The purpose of this study is to describe and characterize the ocular injuries that are directly related to the use of these devices.
Patients / Methods: The files of all patients who were referred to the Department of Ophthalmology in Soroka University Medical Center, between 1998 and 2005, during a three days period around Israel's Independence Day were screened. If a patient sustained an injury from a device used during the celebration, then data relating to the age, sex, injured eye, diagnosis, the device that caused the injury and treatment were collected.
Results: 437 patients were included in this study; fifty-two patients had suffered an ocular injury directly due to a celebration device. The patients' mean age was 17.56 years; 61.54% of the patients were male. The three most common ocular injuries were corneal erosions, conjuctival erosions, and superficial punctate keratitis (38.46%, 28.85%, and 23.08%, respectively). Other injuries included corneal and conjuctival foreign bodies, traumatic iritis, elevated intraocular pressure, and subconjuctival hemorrhage. The devices responsible for most of the injuries were artificial snow spray and fireworks. Ocular injuries from stick-light liquid, plastic hammers, and balloon explosions were seen as well. Almost all patients (96.15%) required medical treatment. Patients injured by celebration devices accounted for 27.88% of all patients seen during the Independance Day itself in our Ophthalmology Department; in several years, the rate approached 40%.
Conclusions: Strict enforcement of rules and legislation regarding the use of celebration devices must be kept. The public's awareness of the harm that these devices can cause should be strengthened.

CELLULAR AND MOLECULAR MECHANISMS UNDERLYING OPTIC NERVE DEGENERATION AND THE CREATION OF A NON-PERMISSIVE ENVIRONMENT FOR REGENERATION FOLLOWING AXOTOMY

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Introduction: Optic nerve axons fail to regenerate after injury or as a result of chronic disease due to non-permissive environmental cues, and disrupted activation of the genetic program that activates the growth process. In an attempt to understand the mechanisms that underlie the generation of a non-permissive environment, we analyzed the molecular and cellular alterations that occur in response to acute optic nerve injury.
Patients / Methods: A rat model of complete transection of the optic nerve that spares the vascular supply and the neural scaffold was used. The response of the optic nerve and retinas to axotomy was studied by immunological and biochemical investigations.
Results: Optic nerve axotomy led to massive cell invasion at the site of injury that spread along both sides of the nerve. The invading cells were microglia, oligodendrocytes, and to a lesser extent astrocytes. A marked induction of type 3 semaphorin was evident in response to axotomy, especially in the area of the scar, and persisted for the 28 days of the experiment. The molecular events associated with axotomy were studied by measuring the levels of the pro-apoptotic protein Bax, p38 and ERK1/2. The levels of Bax were elevated 3 days post-axotomy and then declined. Optic nerve axotomy led to the activation of p38 and ERK1/2. In addition, acute nerve injury led to morphological alterations in oligodendrocytes, astrocytes and the extracellular matrix, disrupting the delicate internal organization of the optic nerve.
Conclusions: We suggest that cell invasion, semaphorin induction and disruption of the internal organization of the optic nerve contribute to the generation of the non-permissive environment that prevents axonal regeneration and leads to neuronal loss.

SEVERE COUGH AND RETINAL HEMORRHAGE IN INFANTS AND YOUNG CHILDREN

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Introduction: Retinal hemorrhage is a common finding in child abuse, mainly in the "shaken baby syndrome", and it is considered as almost pathognomonic for this syndrome. In adults there have been several reports on retinal hemorrhage following Valsalva maneuvers, termed "Valsalva retinopathy or maculopathy". This rapid rise in intra-abdominal and intra-thoracic pressure can be seen with coughing, vomiting and weight lifting. To our knowledge, there have been no published studies investigating retinal hemorrhage secondary to cough in children. Therefore, the goal of the present study was to investigate whether severe persistent coughing may cause retinal hemorrhage in young children.

Patients / Methods: All children aged 3 months to 2 years admitted to the department of Pediatrics, Assaf Harofeh Medical Center, Israel, with severe coughing were eligible for study. Severe coughing was defined as: 1) Cough lasting ≥ 3 days. 2) The reason for referral to the emergency department was coughing. 3) The child needed hospitalization. All children have had an ocular examination. External ocular examination was performed using a slit-lamp bio microscopy. Fundus examination was done using an indirect ophthalmoscope and a 20-diopter lens. We applied Hanley's "rule of 3" in calculating our sample size, meaning that if we examined 100 patients and found no retinal hemorrhage, than the 95% confidence interval (CI) would be 0 to 3%.

Results: One hundred consecutive patients who met the inclusion criteria were enrolled in the study. Of the 100, 63 were male. Patient's age ranged from 3 to 24 months, with a mean age of 8.85 months (±6.08 months) and a median age of 7 months. The duration of coughing before the ocular examination ranged from 3 to 31 days, with a mean of 8.11 days (± 6.34 days), and a median of 6 days. No retinal hemorrhages were detected in any of the 100 patients (0 of 100, 95% CI: 0%- 3%).

Conclusions: If one finds retinal hemorrhage in infants and young children with cough, child abuse must be excluded

FLUID CIRCULATION THROUGH VASCULOGENIC MIMICRY PATTERNS IN POSTERIOR POLE UVEAL MELANOMA PATIENTS DETECTED BY INDOCYANININE GREEN LASER SCANNING CONFOCAL ANGIOGRAPHY

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Introduction: Vasculogenic mimicry patterns, formed by highly invasive tumor cells, connect to endothelial cell lined blood vessels and have been shown to contain fluid in vitro and in vivo. This study was designed to determine if fluid leaks into vasculogenic mimicry patterns without circulation or if fluid circulates in these patterns.
Patients / Methods: Indocyanine green laser scanning confocal angiography (Heidelberg HRA, Heidelberg Engineering) was performed on five patients with posterior choroidal melanoma. Blood was drawn from the contralateral arm of the ICG injection site before and 1 minute after the injection of ICG. The time to first filling of retinal vessels and vasculogenic mimicry patterns, and the time at which no fluorescence could be detected by the HRA instrument were recorded. After fluorescence was no longer detected in either of these two circulations, the patient's pooled blood was imaged by the Heidelberg HRA.
Results: Looping vasculogenic mimicry patterns were detected focally in all 5 patients. Fluorescence in retinal vessels was noted 24.9 ± 6 seconds after injection, and was undetetable by the Heidelberg HRA within 10:04.5 ± 1:38 minutes post injection. Looping vasculogenic mimicry patterns were first detected 18.7 ± 5 seconds after injection and were detectable until 3:54 ± 0:55 minutes post injection. Blood drawn before ICG injection did not autofluoresce and the ICG-containing blood pooled in the tube continued to fluoresce for at least 1 month post injection.
Conclusions: Vasculogenic mimicry patterns are not part of the endothelial cell lined vascular system and fluid must enter these patterns through leakage. However, the rapid infusion of ICG into these patterns after injection and the disappearance of fluorescence detectable by the Heidelberg HRA suggest that fluid circulates in these patterns and does not accumulate as a stagnant pool.

INHIBITOR OF APOPTOSIS PROTEINS (IAP) EXPRESSION IN PRIMARY AND METASTATIC UVEAL MELANOMA

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Introduction: Inhibitor of Apoptosis Proteins (IAP) have been implicated in the pathogenesis of variety of tumors including skin melanoma. We aim to characterize the expression of IAP genes in primary uveal melanoma tumors, liver metastasis from uveal melanoma, and uveal melanoma cell lines.

Patients / Methods: Samples were collected from six primary uveal melanomas, seven liver metastasis from uveal melanoma, four uveal melanoma cell lines, and normal retina, retinal pigmented epithelium (RPE), and choroid tissues. The expression of eight genes (XIAP, apollon, ML-IAP, Survivin, Naip, Birc2, Birc3, and Birc8) from the IAP family was assessed through RT-PCR. Microcirculation patterns were determined by immunostaining for laminin and by PAS staining on cryosections. Ki-67 immunostaining was preformed to exhibit cells undergoing proliferation, and cell type was assessed by conventional histology.

Results: Generally, IAP genes were expressed in melanoma and normal tissue samples (RPE, retina) excluding a minority of samples. An outstanding variability of XIAP and apollon gene expression was distinguished. These genes were expressed in a third and two thirds of the tumor samples, respectively. Neither of these genes was expressed in the normal tissues. By contrast, Survivin and ML-IAP were expressed in each of the tumor samples. Interestingly, none of the IAP family genes was expressed in normal choroid tissue.

Conclusions: Members of the IAP family show altered expression in primary and metastatic uveal melanoma. These proteins can inhibit tumor cell apoptosis and, thus, may potentially modulate uveal melanoma growth rate.

THE CONTRIBUTION OF SYSTEMIC HEALTH PARAMETERS TO INCREASED INTRAOCULAR PRESSURE

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Introduction: The aim of the study was to evaluate the association between elevated intraocular pressure (IOP) and several systemic health parameters in Israel.
Patients / Methods: We retrospectively reviewed the charts of 1,563 IDF soldiers, age 35 years old or older, who underwent routine periodical medical examination between 1991 and 2004. None of the subjects had undergone medical treatment for glaucoma or ocular hypertension. High IOP (>21mmHg) was correlated with age, sex, arterial blood pressure, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, body mass index (BMI), and smoking habits. Pearson correlation coefficients and multivariate linear regression were used.
Results: Statistically significant positive correlation was found between high BMI and high IOP ($r=0.11677$, $p<0.0001$). No correlation was detected between a high IOP and the other parameters.
Conclusions: BMI was positively correlated with IOP levels. Former reports showed variable results regarding BMI correlation to glaucoma incidence and to be positively correlated with retinal thickness. The finding that a high BMI has a protective effect against glaucoma progression advocates taking BMI into consideration in the assessment of glaucoma suspects.

PAPERS

RETINA 1

SERUM CYTOKINES LEVELS IN A MOUSE MODEL OF CENTRAL RETINAL ARTERY OCCLUSION

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Introduction: Central Retinal Artery Occlusion (CRAO) is a devastating disease leading to blindness. Recently, elevated inflammatory markers suggested the role of inflammation in ischemic processes of various organs, including the eye. The aim of this study is to measure cytokines levels in the serum, following induction of central retinal artery occlusion in a mouse model.
Patients / Methods: CRAO was induced in C57BL6 male mice, using laser photoactivation of intravenously injected rose-bengal, over the central retinal artery at the optic nerve head. In each mouse the right eye was treated and the left eye was kept for control. Clinical examination and fluorescein angiography (FA) were performed. Blood sampling, and histopathologic evaluation were performed at 1, 3, 6, 12 hours and 1, 7, and 21 days after CRAO induction. Levels of (MIP)-2, interleukin (IL) -6, and TNF-α were measured in serum samples using ELISA.
Results: CRAO was fully evident clinically 6 hours following induction. FA revealed compromised arterial perfusion at 6 hours, with reperfusion at 24 hours. Normal perfusion was demonstrated in the control eyes. Nuclear loss in the inner retinal layers (RGC and INL) with maximal loss after 21 days was evident on histology. Serum MIP-2 levels peaked at 1 hour, decreasing to control levels at 12 hours. IL-6 levels were elevated between 3 and 12 hours decreasing at 24 hours. TNF- α showed an early peak at 1 hour and a late rise at 1 and 7 days.
Conclusions: Pro-inflammatory cytokines are elevated in the serum after induction of CRAO in mice. The pattern is similar to previously described in CRAO in humans and in ischemic optic neuropathy, both clinical and experimental. These findings may suggest an inflammatory role in the pathogenesis of the ischemic damage to the retina.

THE VALUE OF THE FIRST PDT RESULTS IN PREDICTION OF VISUAL OUTCOME

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Introduction: With the rise of many different treatment options for neovascular age related macular degeneration (AMD) including combined Photodynamic Therapy (PDT) and triamcinolone, and various anti-angiogenic drugs, we wanted to evaluate the prognostic value of the first PDT result and to asses the role of further PDT treatments if the first PDT fails to show stabilization or improvement.
Patients / Methods: 80 charts of patients who underwent their first PDT from 3/2002 – 10/2004 due to all types of subfoveal Choroidal Neovascularization (CNV) secondary to AMD were reviewed. Patients who suffered from any other ocular disease, as well as patients treated with prior laser, or who had combined therapy of PDT and triamcinolone were excluded. Visual acuity (VA), lesion size and lesion type, at baseline, 3 months and 12 months, were recorded. Statistical analysis was performed using Pearson Correlation Coefficients to find any correlation between success of the first treatment and stabilization in VA and reduced lesion size at 12 months. First treatment success was determined as improvement or stabilization in VA or in lesion size.
Results: 46 patients were included in the analysis. There were 18 females and 27 males. No statistically significant correlation was found between first PDT success (when defined as change in lesion size) and stabilization or improvement in VA after 12 months (P=0.0562), or between stabilization or improvement of VA after the first PDT and reduction in lesion size after 12 months (p=0.1377). Variant analysis between subgroups of CNV was unable to detect any statistical significant correlation.
Conclusions: No correlation was found between the results of the first PDT and the outcome determined by visual acuity or lesion size at 12 months. Therefore the decision upon continuing PDT should not be influenced by the results of the first PDT, and treatment indications should not differ from the ones suggested by the prior multi-center studies.

INHIBITORY EFFECTS OF HYPERICIN ON RPE CELL MIGRATION AND INVASION

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Introduction: RPE cells provide structural and metabolic support for the visual function of neuronal retina and are usually in a stationary state. However, RPE can be activated for proliferation and migration in response to signaling by chemotactic agents and growth factors which are released to the blood in response to trauma or in pathological conditions. Migration of RPE has beneficial role by aggregating along injured capillaries. Negative outcomes of RPE migration and proliferation occur following excessive activity resulting in formation of epiretinal membranes. Understanding RPE cell behavior in response to stimulation by these factors and devising methods to control this migration are thus important. Our group focused on hypericin as a potential tool for preventing epiretinal membrane formation. Hypericin exhibits antimetastatic activities resulting from potent anti-angiogenic activities in-vivo. Hypericin induces Hsp90 chaperone ubiquitination destabilizing its client proteins such as HIF-1α and MMP2. HIF-1 activates many genes including mediators of cell migration. We examined the effects of hypericin on migration and invasion of RPE cells. We report here that hypericin may act as an inhibitor of ocular pathologies such as diabetic retinopathy, PVR and the wet form of AMD.
Patients / Methods: Migration and invasion of RPE cells were induced by stimulation with either VEGF or SDF-1α. The effects of hypericin on activated-RPE cells invasion were monitored as pseudopodia formation by the cells in 3D collagen gels and on cell migration by the wound healing assay.
Results: SDF-1α induced more extensive protrusion of pseudopodia from RPE cells in 3D collagen gel assays than VEGF. In both stimulations the number of pseudopodia formed was higher than in untreated cells. The migration assays confirmed the invasion assay findings. Activated RPE cells migrated through the scratch line in larger numbers and crossed longer distances compared to controls. Hypericin was found effective in decreasing cell migration and induced morphological transition of these cells to tubular forms.
Conclusions: Our results suggest that hypericin may be effective in prevention of activated RPE cell migration and invasion. This reagent has the potential of being utilized in prevention of ocular pathologies such as PVR and diabetic retinopathy.

INTRAVITREAL BEVACIZUMAB (AVASTIN) FOR CHOROIDAL NEOVASCULARIZATION

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Introduction: To evaluate the outcome of intravitreal bevacizumab (Avastin) for eyes with choroidal neovascularization (CNV) either not eligible or unresponsive to other treatments such as photodynamic therapy (PDT). This report will focus on the result of intravitreal Avastin for serous pigment epithelial detachment (SPED) with occult CNV secondary to age-related macular degeneration (AMD).
Patients / Methods: This IRB approved, open-label, uncontolled clinical study enrolled patients with CNV either not eligible or unresponsive to other treatments such as PDT. Patients underwent EDTRS visual acuity assessment, tonometry, ophthalmic examination, optical coherence tomography (OCT) and fluorescein angiography. Inform consent was signed by each patient. Intravitreal bevacizumab of 1.25 micrograms was injected under sterile conditions. Need for retreatment was based on visual acuity measurement, evidence of subretinal fluid on clinical examination and OCT, and /or leakage on fluorescein angiography.
Results: Included were 17 eyes of 17 patients. Seven eyes had SPED, 5 eyes had CNV due to AMD and received previous PDT, 3 eyes had large hemorrhagic lesion due to AMD and 2 eyes had angioid streaks. Out of 7 eyes with SPED, 3 eyes showed improvement of visual acuity and one ot those showed improvement of 4 lines. Four eyes maintained vision through follow up. All demonstrated absorption of subretinal fluid by fundoscopy, decreased leakage by angiography and reduction of retinal thickness by OCT. Ten eyes demonstrated mild conjuctival injection on the first postoperative day which resolved spontaneously during the subsequent days. None of the eyes showed increased intraocular pressure. Beneficial effect was also observed in other groups of eyes ..
Conclusions: Intravitreal bevacizumab showed improvement and stabilization of vision in this small group of eyes with SPED. As there is effective proven treatment for eyes with SPED and occult CNV, further evaluation of this treatment is under investigation.

INTRAVITREAL BEVACIZUMAB (AVASTIN) THERAPY FOR NEOVASCULAR AGE-RELATED MACULAR DEGENERATION: 6 MONTHS RESULTS OF AN UNCONTROLLED, OPEN LABEL CLINICAL STUDY

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Introduction: Aim: to evaluate the safety and efficacy of intravitreal bevacizumab (avastin, Genentech inc.) for the treatment of subfoveal choroidal neovascularization (CNV) in patients with AMD.
Patients / Methods: Methods: 12 AMD patients with subfoveal CNV that did not respond to PDT treatment were enrolled in the study. Patients were treated at baseline with intravitreal bevacizumab and than followed up for 6 months at regular intervals. Ophthalmologic evaluation included protocol VA measurements, ocular examinations along with optical coherence tomography (OCT) imaging and fluorescein angiography (FA). Retreatment of intravitreal bevacizumab was performed (up to 3 injections) if there was evidence of recurrent leakage from the CNV.
Results: Results: no serious ocular or systemic adverse events were identified through the 6 months follow up. Visual acuity revealed stabilization or improvement in 80% patients. FA revealed marked reduction in leakage in all patients, and OCT imaging revealed stable or decreased retinal thickness in all patients.
Conclusions: Conclusion: Bevacizumab therapy given intravitrealy for neovascular AMD was well tolerated and effective through a 6 months follow up period. These results albeit promising need to be validated in multicentered controlled trials

RETINAL ELECTROPHYSIOLOGIC, MORPHOLOGIC AND PENETRATION STUDIES FOLLOWING INTRAVITREAL INJECTION OF BEVACIZUMAB (AVASTIN ®).

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Introduction: Intravitreal bevacizumab (Avastin; Genentech Inc., San Francisco, CA) is a new promising treatment for age-related macular degeneration, used widely off-label. Our aim was to evaluate retinal penetration and toxicity of bevacizumab.
Patients / Methods: 10 albino rabbits were injected intravitreally with 0.1 ml (2.5 mg) of avastin to one eye and 0.1 ml saline into the fellow eye. To assess toxicity, electroretinogram (ERG) was recorded 3-hours, 3-days, 1-, 2-, 4-weeks after injection. At the termination of the ERG follow-up (4-weeks), the visual evoked potential (VEP) was recorded. Then, the rabbits were sacrificed and the retinas prepared for histological examination at the light microscopy level. To assess retinal penetration of the humanized bevacizumab antibody, donkey anti-human IgG conjugated to the fluorochrome Cy3 was added to 100 μm thick vibratome sections. Muller cell and microglia reactivity was assessed by labeling retinal sections with anti-vimentin and the isolectin B4, respectively. Images were collected using a laser scanning confocal microscope.
Results: The ERG responses of the control and experimental eyes were similar in amplitude and pattern throughout the follow-up period. The flash VEP responses of the experimental eyes were of normal pattern and amplitude and did not differ from those recorded by stimulation of the control eye alone. No morphological changes were observed in any of the examined eyes. Full thickness retinal penetration to the RPE (but not within the RPE) was present at 24 hours and 7 days and was essentially absent at 4 weeks. No reactivity was observed in either Muller cells or microglia; the intermediate filament expression in Muller cells and the distribution of microglia were similar to uninjected control, as well as saline injected eyes.
Conclusions: Bevacizumab was found to be non-toxic to the rabbit retina. Full thickness retinal penetration may explain observed clinical effects of intravitreal bevacizumab. Although extrapolation to humans should be done with caution, our study supports the safe use of intravitreal bevacizumab injection.

HERMANSKY PUDLAK SYNDROME TYPE 6 CAUSED BY A NOVEL MUTATION IN AN EXTENDED, HIGHLY CONSANGUINEOUS, ISRAELI BEDOUIN FAMILY

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Introduction: Hermansky-Pudlak syndrome (HPS) is a rare autosomal recessive disorder characterized by oculocutaneous albinism (OCA) and platelet storage pool deficiency, resulting from abnormal formation of intracellular organelles of the lysosomal lineage. The diagnosis is based on absence of dense bodies in platelet whole mount electron microscopy. Seven autosomal human genes and fifteen murine models for HPS, causing various phenotypes of HPS, have been described.
Patients / Methods: An extended, highly consanguineous Israeli Bedouin family with at least 20 individuals exhibiting a unique phenotype of OCA was studied. Haplotype analysis and homozygosity mapping of candidate genes and loci were employed. Linked candidate genes were tested for mutations by sequence analysis. The novel HPS6 mutation 1066insG was further confirmed by restriction enzyme digest. Gene expression was evaluated using semi-quantitative RT-PCR. Trafficking of lysosomal lineage organelles (protein activity) was determined by confocal microscopy of LAMP1 (lysosomal associated membrane protein 1) in fibroblasts.
Results: Electron microscopy of platelets from patients revealed absence of dense bodies, confirming the diagnosis of HPS. The 7 human HPS genes and additional 6 genes corresponding to murine HPS models were evaluated. Initially, linkage to HPS1 was observed, but no mutations were identified. Linkage to a 4.48Mb region distal to HPS1 was observed. Several candidate HPS genes are mapped to this region. Sequence analysis identified a novel insertion mutation, 1066insG, in HPS6. The mutation co-segregated with the phenotype, confirming the 1066insG mutation as the causative mutation. The level of expression of the intronless HPS6 gene was not affected by the mutation. However, confocal microscopy revealed abnormal distribution of LAMP1 in fibroblasts from the patients, indicating abnormal trafficking of lysosomal lineage organelles.
Conclusions: So far, only a single patient with HPS6 has been identified. Our finding of a novel mutation in the extended family is the second mutation in HPS6. This finding enables pre-marital and prenatal genetic testing for diagnosis and counseling in this Bedouin population. Two major genetic isolates of HPS1 and HPS3 patients were diagnosed in Puerto Rico. The extended Israeli Bedouin family is the largest isolate of non-Puerto-Rican HPS patients, and comprises about one third of the undiagnosed non-Puerto-Rican HPS patients worldwide.

GENE EXPRESSION PATTERNS IN PRIMARY AND METASTATIC UVEAL MELANOMA

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Introduction: Systemic metastases develop in about 50% of uveal melanoma patients. Such metastases are associated with death within several months following its diagnosis. We aim to characterize the molecular pathways which lead to the development and growth of metastasis in uveal melanoma patients.
Patients / Methods: Seven primary uveal melanomas and seven liver metastases from uveal melanoma were included in the study. Microarray analysis was performed using a spotted oligonucleotide microarray containing 36,000 features and by applying a reference sample study design. Genes with metastasis associated expression pattern were identified using the Significance Analysis for Microarray (SAM) algorithm. Primary tumors and metastasis were characterized in terms of cell type (by conventional histology), microcirculation patterns (on PAS stained sections), proliferative activity of tumor cells (using Ki-67 immunostaining), and apoptosis rate (on TUNEL stained sections).
Results: Microarray analysis identified 193 features that showed metastasis associated expression pattern at the False Discovery Rate (FDR) level of 0%. Nine of these features showed increased mRNA levels in metastasis compared with primary tumors while 184 showed decreased mRNA levels in metastasis. Consistent with findings from other cancer types, genes which show increased expression in metastasis are involved in protein synthesis. Genes which show decreased expression in metastasis belongs to variety of functional classes. SAM assessment and hierarchical clustering revealed that gene expression patterns differ between primary and metastatic lesion significantly more than across tumors classified according to prognostic factors such as microcirculation patterns and cell type.
Conclusions: Multiple genes show altered expression patterns in liver metastasis from uveal melanoma compared with primary uveal melanoma. Further studies will demonstrate the potential role of these genes in the development and growth of such liver metastasis.

A TEMPORAL MICROARRAY-BASED GENE EXPRESSION ANALYSIS OF THE RAT RETINA

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Introduction: Our aim: performing a comprehensive gene expression analysis of the rat retina at 5, 11, and 20 weeks of age, with the aim of identifying differentially expressed genes, and particularly those expressed in retinal ganglion cells (RGCs).
Patients / Methods: Retinal samples of albino Lewis rats were dissected at 5, 11, and 20 weeks of age. Total RNA was extracted using the RNeasy kit (Qiagen). Retinal samples were labeled with either cy3 or cy5 dyes and hybridized to Agilent rat microarrays. A dye-swap analysis was performed for each microarray hybridization. The data was statistically analyzed with the aim of comparing expression levels for the 3 different age groups. a detailed analysis of a subset of genes was done by collecting gene expression data from previous EST and SAGE analyses of human, mouse, and rat tissues.
Results: The microarray data obtained from the rat retina contain many known retinal genes and are in agreement with previous studies. However, among the genes showing the highest expression levels in our analysis, a relatively high number (70 out of 118) of non-annotated genes were observed. 841 of the genes showed differential expression levels between at least two of the three time points, and clustered into 7 different groups. The most common cluster contained 484 genes and showed no change in expression level between age 5 and 11 weeks while the expression level at the age of 20 weeks was higher comparing to 5 and 11 weeks. A bioinformatics analysis of the 841 differentially expressed genes revealed that at least 21 of them are expressed in RGCs. Among these genes were Stathmin, known to be involved in axongenesis, with relatively high expression levels in the young retina, and TWIK-2, a potassium channel, with a low expression level in the young retina.
Conclusions: Our analysis revealed many novel and known retinal genes that show differential expression pattern in the rat retina; some of these are already known to be expressed in RGCs. We have recently shown that the pattern electroretinogram in rats, which is correlate with RGC function, changes with age . We are therefore performing quantitative real-time RT-PCR analysis on a subset of genes that are expressed in ganglion cells and show differential microarray expression that might correlate with RGC function.

SPATIAL AND TEMPORAL DIFFERENTIAL GENE EXPRESSION ANALYSIS OF PAX6 ALTERNATIVELY-SPLICED TRANSCRIPTS IN THE PIGEON RETINA

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Introduction: The molecular events leading to the development of different retinal regions, and particularly the macula, are poorly understood. We have chosen the pigeon retina, which includes both a central macula and a large nasal macular-like region (the red area), as a model for studying spatial and temporal retinal gene expression. Our purpose is to perform a detailed analysis of PAX6 alternatively-spliced isoforms in different regions of the pigeon retina along development.
Patients / Methods: Different retinal regions were dissected with the pecten as an indicator of topography. Total RNA was extracted and used to produce cDNA by reverse transcriptase. Degenerative primers were designed to amplify PAX6 in pigeon. The TA-cloning procedure was applied followed by sequencing of insert DNA. Quantitative real time RT-PCR analysis was performed on ABI-PRISM 7000 with cDNA samples from different retinal and brain regions.
Results: We have determined the sequence of the canonical pigeon PAX6 open reading frame, which is highly similar to human and chicken PAX6. A screen for alternatively-spliced isoforms has revealed so far 3 alternatively-spliced sites producing 14 different transcripts, and potentially 14 functional PAX6 proteins. This includes the PAX(5a) isoform with an addition of an exon with 42 nucleotides, a skipping of 201 nucleotides in exon 6, and skipping of 18 nucleotides in exon 11. We have designed primers specific for each PAX6 isoform and verified that all 14 isoforms exist in the pigeon retina and brain. Quantitative real-time RT-PCR analysis revealed that all isoforms have a higher expression level in the retina versus brain, and that 4 isoforms show significant differential expression in either adult versus young retina or macular red area versus peripheral retina.
Conclusions: Our analysis revealed two important findings regarding the involvement of PAX6 in retinal development: at least 14 different PAX6 isoforms exist in the pigeon retina, and at least 4 of which show differential expression levels in different retinal regions. The precise biological significance of the splice variants is currently unknown, but in view of the important role of PAX6 in ocular and brain development, we predict that some of these isoforms are involved in retinal, and possibly macular, development.

THE MANY ROLES OF PAX6 IN DEVELOPMENT OF THE OCULAR LENS

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Introduction: Pax6 is a fundamental transcription factor in the development of the eye. With complete loss of Pax6, eyes fail to develop, while when only one copy of the gene is disrupted, severe ocular abnormalities are observed. In the mouse these abnormalities include microphthalmia, aniridia and cataracts. Lens development in vertebrates can be divided into several steps, the first of which is an induction of part of the head surface ectoderm by the optic cup, to form a lens placode. This placode later invaginates and detaches to form a lens vesicle. In animals that lack Pax6 expression, the lens placode does not form and lens development is aborted. In later stages of development, cells from lens vesicle form an epithelium of dividing cells, which can proliferate or differentiate into the lens fibers that make up the bulk of the lens. In this study, we focused on the role of Pax6 in the context of the developing lens in later stages.
Patients / Methods: Using the cre/loxP conditional knock-out approach we deleted Pax6 specifically in the lens, subsequent to the lens vesicle stage. Mice carrying a cre-recombinase transgene under a modified alphaA crystallin promoter were crossed with mice carrying floxed alleles of Pax6. Eyes were examined morphologically and histologically, as well as for molecular markers.
Results: Mice carrying a mutation in both alleles had severely reduced, and morphologically abnormal lenses. Epithelial cells were aggregated in the transitional zone and some were apoptotic. Surprisingly, heterozygous animals for a lens-specific mutation showed no obvious phenotype. This is in contrast to heterozygotes with a Pax6 conditional mutation that predates lens induction and full heterozygote knockouts, which develop smaller lenses and a residual lens stalk.
Conclusions: We show distinct roles of Pax6 in different stages of lens development. While Pax6 is essential for lens induction, it is also crucial for proper differentiation and morphology in later development. From the lack of the characteristic heterozygote phenotype we can conclude that the dosage requirement for Pax6 in the lens is critical for the induction or invagination stages but not for growth and differentiation of the lens in later stages.

ALTERED EXPRESSION OF IRON METABOLISM ASSOCIATED GENES IN RETINAL DEGENERATION

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Introduction: Iron associated injury has been implicated in several neurodegenerative disorders among them age related macular degeneration (AMD). We aim to characterize the expression of iron metabolism associated genes during retinal degeneration in order to gain insight into the potential involvement of iron in the degenerative process.
Patients / Methods: Retinal expression pattern of transferrin and ceruloplasmin was studied at 2, 3 and 6 weeks of age in rd10 mice, which often serve as an animal model for this devastating pathology. The findings were compared to those in wild type controls. mRNA levels were measured using quantitative real time RT-PCR (QPCR) while protein expression was assessed using immunohistochemistry (IHC).
Results: Retinal transferrin mRNA levels were similar in rd10 and controls at 2 weeks of age (0.9 ± 0.43 in arbitrary units vs. 0.79 ± 0.43, respectively. $p=0.54$). Up regulation of transferrin mRNA was detected in rd10 retinas compared with controls once the degenerative process had initiated at 3 weeks of age (3.1 ± 1.5 vs. 1 ± 0.44, respectively, $p=0.00045$). At six weeks of age there was further up regulation of transferrin mRNA expression in rd10 retinas (6.9 ± 1.1 vs. 1.27 ± 0.39, respectively. $p=1.38E-10$). Ceruloplasmin mRNA levels also showed up regulation in rd10 retinas starting at the age of 3 weeks. Immunohistochemistry showed diffuse transferrin expression throughout the retina layers with marked up regulation of the protein in the inner retina at 6 weeks of age.
Conclusions: Retinal mRNA expression and protein levels of transferrin and ceruloplasmin significantly increase in rd10 mice during the degenerative process compared with controls. These data combined with previous reports suggest that alteration in iron metabolism characterize retinal degeneration from variety of primary insults.

REPAIR OF OPTIC NERVE ISCHEMIC INJURY IN THE MOUSE BY BONE MARROW DERIVED STEM CELLS

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Introduction: Assess the incorporation and differentiation potential of adult bone marrow-derived stem cells (BMSC) as a potential therapeutic approach to replenish dead RGC following ischemic optic neuropathy.
Patients / Methods: Mononucleate and low-density bone marrow cells (mBMC) from GFP donors were injected intravenous and intravitreal at various times after induction of ischemic optic neuropathy injury (AION). AION was induced by laser photosensitization of intravenously-injected rose Bengal over the optic disk in syngeneic C57/B6 mice. The contralateral eyes served as negative controls for AION induction. The recipients were euthanized at variable times. The eyes were harvested and used for flat mount retina and immunohistochemistry preparations.
Results: When intravitreous injection, significant numbers of cells were detected in the eyes with and without retinal injury. Following intravenous infusion of the cells, mBMC-GFP cells were seen only in eyes that have undergone rAION. The pattern of incorporation was, as expected, diffuse with selective accumulation in the central retina and along blood vessels. The incorporation occurred within several days and was still detectable one month following the induction.
Conclusions: This study shows that mBMC home to the ischemia-injured retina from the peripheral blood, and incorporate selectively at the sites of injury. The cells were viable after sustained periods of time and expressed various markers of non-hematopoietic differentiation. The differentiation patterns and functionality of the cells are currently under investigation.

BONE MARROW DERIVED STEM CELLS INCORPORATION INTO LASER INJURED RETINA

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Introduction: Various retinal diseases lead to visual debilitation and blindness. Laser treatment is used in many retinal diseases. Repair of the injury and restoration of retinal activity following laser treatment are not yet available and directly relevant to the preservation of vision. The purpose of this study is to assess the incorporation of adult bone marrow-derived stem cells (BMSC) as a potential therapeutic approach to ameliorate retinal laser injury.

Patients / Methods: Mononucleate cells derived from the bone marrow of GFP mice (BMSC) were injected intravenously and into the vitreous body of syngeneic B6 mice after laser injury to the retina. Laser burns were performed using argon laser. The animals were euthanized at variable times for preparation of flat mount retinas and cryosections, with the contralateral healthy eye serving as a negative control for the laser-induced injury.

Results: Following laser injury, GFP positive BMSC were detected in all layer of the retina that underwent laser injury. The cells accumulated preferentially in the scar margins. The GFP-BMSC were not detected in the control eyes. Intravenously infused cells reached the injured retina more efficient than intravitreously-infused cells, indicating superior homing to the lesion area of cells circulating in the peripheral blood. The GFP-BMSC were detected as early as 4 days after injection, and were stably incorporated in the retina after 3 months. Some of the cells showed changes in morphology with an apparent transition from large cells to smaller elongated cells, reminiscent of photoreceptors. Differentiation patterns of these cells are currently under investigation

Conclusions: Mononucleate BMSC show specific homing to sites of injury after laser-induced damage, whereas there is no homing to healthy retina. Blood circulating cells reach the lesion more efficiently than cells in the vitreous body. These cells incorporate in all layers of the retina and are retained for long periods of time.

ADULT BONE MARROW-DERIVED PROGENITOR CELLS PROMOTE VASCULAR REPAIR IN OXYGEN-INDUCED RETINOPATHY

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Introduction: Conventional approaches to treating neovascular diseases of the eye involve the use of laser ablation and angiostatic compounds to inhibit new blood vessel growth. Recent reports suggest that progenitor cells derived from the bone marrow participate and can augment vascular repair in diseased tissue. Our purpose was to evaluate the ability of bone marrow-derived cells to enhance healing of oxygen-induced retinopathy (OIR) in a mouse model.

Patients / Methods: Lineage-negative (Lin-) cells were isolated from bone marrow of adult actGFP C57BL/6J mice and injected intravitreally into one eye of C57BL/6J mice at postnatal day two (P2)-P7. In fellow control eyes, either injection of vehicle alone or no injection was performed. To induce OIR, mice were transiently exposed to hyperoxic conditions (75% oxygen) between P7-P12, then returned to room air. At P17, Retinas were isolated, blood vessels were stained with isolectin Griffonia Simplicifolia, confocal fluorescent images of retinal whole-mounts were acquired, and the area of vascular obliteration and extent of pre-retinal neovascularization were quantified. Immunohistochemistry was used to identify cell types associated with the healing process.

Results: Injection of Lin- cells dramatically enhanced vascular recovery following exposure to hyperoxia. At P17, 57% of the Lin- injected eyes exhibited fully developed superficial and deep vascular networks with few abnormal neovascular tufts. Only 4% of PBS-injected contralateral eyes demonstrated such full recovery. At P17, the average area of obliteration was reduced by over 75% in the Lin- injected eyes. Long term (up to 6 months) follow-up revealed no tumor formation and histological preservation of the neural retina in Lin- injected eyes.In an effort to better understand the nature of the functional cells in these experiments, we used single markers and FACS to isolate specific, highly pure subpopulations. We demonstrate that purified CD44hi cells retain the function of the mixed population of Lin- cells in this model, thus identifying an active and definable subpopulation.

Conclusions: Intravitreal injection of bone marrow-derived progenitor cells markedly accelerates vascular healing of OIR, with reduced abnormal neovascularization and rapid revascularization of the retina. The findings support the concept of cell therapy using specific subpopulations of BM–derived cells in ischemic retinopathies.

HUMAN EMBRYONIC STEM CELLS DIFFERENTIATE INTO RETINAL PIGMENT EPITHELIUM CELLS

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Introduction: Dysfunction, injury, and loss of retinal pigment epithelium (RPE) cells is a prominent feature of Age Related Macular Degeneration (AMD) and also occurs in subtypes of Retinitis Pigmentosa (RP). Our objective was to evaluate the ability of human embryonic stem cells (hESC) to give rise to RPE cells in-vitro and to examine their survival in-vivo following intraocular transplantation into rodent eyes.
Patients / Methods: Spontaneous differentiation of hESC was induced by culturing embryonic bodies (EBs) in suspension for at least 4 weeks. Clusters of pigmented cells within the EBs were mechanically dissected, plated and further cultured on laminin-coated slides. Phenotype of the cells was characterized by Immunostaining and PCR for RPE-associated markers. For in-vivo transplantation, a cell suspension enriched with pigmented cells was derived from the cultures and delivered into the sub-retinal or intravitreal space of RCS rats which manifest an RPE-specific mutation and in albino Sprague-Dawley rats. Survival of engrafted cells and expression of RPE-specific markers were examined by immunohistology 3.5 weeks after transplantation.
Results: Prolonged differentiation of hESC within EBs gave rise to clusters of pigmented cells. Nearly 70% of EBs contained such pigmented clusters. Expression of the RPE-specific transcription factor MiTF-A was increased in pigmented EBs from the fifth week of incubation. Following plating on laminin, pigmented cells assumed a characteristic polygonal RPE-like shape and expressed the RPE-associated markers CRALBP, RPE65, MiTF-A, Bestrophin, and ZO-1. Following intraocular transplantation, hESC-derived pigmented cells survived and were present in intraviteal, intraretinal, and subretinal grafts. A large fraction of transplanted cells were pigmented, and expressed mature RPE markers such as RPE65 and ZO-1.
Conclusions: hESC cells can give rise to pigmented cells in-vitro which demonstrate an RPE-like morphology and express RPE-specific markers. Following intraocular transplantation, the cells survive for at least 3.5 weeks and maintain their RPE-like qualities. This may be a first step towards the future use of hESC as an unlimited source for renewing or replacing degenerating RPE cells.

THE USE OF A SINGLE MADDOX ROD FOR THE MEASUREMENT OF CYCLODEVIATION

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Introduction: Cyclodeviation is generally measured with the double Maddox rod test. The line seen through one of eyes serves as a horizontal reference. The angle of cyclodeviation is measured by instructing the patient to rotate the Maddox rod placed in front of the other eye until the two lines are perceived parallel. In the examination room there are usually a lot of horizontal reference lines namely the intersection between the wall and the floor or windows and doors frames. Assuming that cyclodeviation is present, the patient could use the above mentioned horizontal reference clues and the cyclodeviation could be equally measured using only a single Maddox rod. We compared the cyclodeviation as measured with the conventional double Maddox rod test to the angle of cyclodeviation obtained with a single Maddox rod.
Patients / Methods: 27 patients with superior oblique palsy were included. For each patient 4 measurements of excyclodeviation were taken: A-with two Maddox rod while the sound eye serves as a horizontal reference. B-with two Maddox rod while the fellow eye serves as a horizontal reference. C-one Maddox rod in front of the sound eye. D-one Maddox rod in front of the fellow eye.
Results: The mean angle of excyclodeviation for the 27 patients for the four different types of measurements A, B, C and D were: 7.30±2.3, 7.20±2.6, 6.60±2.4 and 6.90±2.5 respectively. The differences between all combinations of the four measurements were very small and with no statistical significance. Only 3 of patients had a difference of more than 2 seconds of arc in the angle of excyclodeviation between the double and the single Maddox rod test.
Conclusions: Cyclodeviation can be determined with a single Maddox rod placed in front of one eye. This test yields practically equal results to the conventional double Maddox rod test.

THE EFFECT OF THALIDOMIDE ON OXYGEN INDUCED RETINOPATHY IN A MOUSE MODEL

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Introduction: Oxygen therapy is a well-recognized risk factor for retinopathy of prematurity (ROP). We tested if treatment with thalidomide could reduce the severity of oxygen-induced retinopathy in a mouse model of ROP.
Patients / Methods: ROP was induced in mice by exposure to 75% oxygen for 5 days from postnatal day 7 to 12. 43 baby mice (wild type) were divided into three groups of room air reared, oxygen exposed and oxygen exposed with thalidomide treatment. Fluorescein angiography of retinal vasculature was performed before scarifying the mice and retinal whole mounts of the enucleated eyes were evaluated in a masked fashion for features of retinopathy using the Modified Retinopathy Scoring System (MRSS). The parameters scored included blood vessel growth, tufts formation, neovascularization, vessel constriction and tortuosity. Count of blood vessel tufts was performed on H&E-stained retinal sections.
Results: The retinopathy score by MRSS of the thalidomide treated mice was similar to that of untreated mice that were exposed to oxygen (9.3±1.91 vs 10.15±1.63). The neovascularization count was also similar between the two groups (10.43±5.6 vs 9.6±4.83). In the room air reared control group the retinopathy score was close to 0 and the neovascularization count was also very low (2.92±2.14).
Conclusions: Our model did not show a significant ameliorating effect of thalidomide on the oxygen induced retinopathy. The causes might be an ineffective level of the active drug in the retina, or involvement of factors not influenced by thalidomide in the process.

DIODE LASER TREATMENT OF RETINOPATHY OF PREMATURITY

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Introduction: Purpose: To report the anatomical and refractive outcomes of infrared diode laser photocoagulation (DLPC) treatment of threshold ROP.
Patients / Methods: Retrospective review of consecutive prematures treated from 10/1997 till 12/2004.
Results: 100 neonates (194 eyes); mean birth weight: 833.9 (SD 250.3) gr; mean gestational age: 26 (SD 1.9) weeks; mean follow up 26 months; 63% had zone 1 or posterior zone 2 ROP. A mean of 1750 (SD 970) laser applications were performed in each eye. Additional 2 DLPC rows posterior to the ridge were delivered in 31% of the eyes. Second DLPC treatment was delivered 18% of the eyes. Mean pre and post treatment IOP were 15 (SD 4) and 33.6 (SD 11) mm Hg respectively. 184 eyes (94.8%) had favorable anatomical results. 5 stage 4A eyes (2.6%) were successfully operated; 5 eyes (2.6%) developed stage 5 ROP. Refractive data was available in 117 eyes: 29% had myopia of >-2.5; 18% had high myopia. Strabismus was found in 24 of 70 babies (33%). Correlations were found among gestational age, and the corrected age at treatment, the zone of ROP, the number of laser applications, and the spherical equivalent.
Conclusions: DLPC is a safe and effective treatment for ROP. Neonates with smaller GA require earlier and more aggressive laser treatment, and may develop higher refractive error.

INCIDENCE AND SEVERITY OF ROP IN TWO CONSECUTIVE ISRAELI POPULATION GROUPS - A SINGLE CENTER RETROSPECTIVE STUDY.

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Introduction: Introduction: Recent studies have been controversial regarding changes in the incidence of ROP (retinopathy of prematurity). Our aim was to retrospectively analyze the incidence of ROP in two consecutive populations, in five year periods, in a well defined geographical area in Israel.
Patients / Methods: Methods: Infants born between January 1st 1995 until December 31st 1999 (group A) and January 1st 2000 until December 31st 2004 (group B) with a birth weight (Bwt) of 1500 grams or less were studied retrospectively. The incidence of ROP, with its various stages (mild 1,2 and severe 3,4), was compared between the two population groups. Multiple logistic regression analysis was performed to account for the influence of potential confounding variables.
Results: Results: There were 259 premature babies in group A and 284 babies in group B. Mean gestational age (GA) in group A was 29.2 weeks and 29.4 weeks in group B. Mean Bwt was 1088 grams and 1136 grams in groups A and B respectively.The incidence of ROP in group A was 43.4%, and 22.3% in group B ($p=0.005$). The incidence of mild ROP was 33.83% in group A and 15.44% in group B ($p=0.001$). The severity of ROP decreased significantly. The incidence of severe ROP was 8.5% in group A and 5.7% in group B ($p=0.001$). Analyzing the risk factors for the development of ROP the following dependent variables were found: GA, Bwt, number of days on oxygen (O2) and number of days on ventilatory support (IMV-intermittent mandatory ventilation). A significant overall interaction effect was found between incidence of ROP and the antenatal steroid ($p<0.011$). A significant interaction effect was found in the IMV variable ($p=0.0001$).
Conclusions: Conclusions: The incidence of ROP, significantly declined over the years. There was also a decrease in the severity of the disease. Bwt, GA, O2 and IMV were found to be risk factors for ROP, according to logistic regression analysis. Premature infant whose mothers didn't receive antenatal steroids were ventilated for longer durations and had ROP more often.

SUTURES MATERIAL IN PEDIATRIC CATARACT SURGERY: A COMPARISON OF VYCRIL VERSUS MERSILEN

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Introduction: The purpose of this study is to evaluate the refractive astigmatism in children who underwent cataract extraction and foldable intraocular lens implantation, using Vycril versus Mersilen sutures.
Patients / Methods: Forty five children (60 eyes) had cataract surgeries with intraocular lens implantation using clear corneal incision between 1999 and 2005 in our department. Mean age was 4.4 ± 3.4 years. Group 1 consisted of 35 patients (45 eyes), who had congenital cataract surgery between 1999 and 2004, using Mersilen sutures. Group 2 consisted of twelve patients (15 eyes), who had congenital cataract surgery between 2004 and 2005, using Vycril sutures. The refraction was tested and recorded at 1 week, 3 months and 5 months post-operatively. The paired t-test was used to compare between the refraction results in the different groups.
Results: Mean astigmatism 1 week postoperatively was 2.4 ± 2.2 Diopter (D) and 1.4 ± 1.1 D in groups 1 and 2, respectively. Thereafter, the astigmatism reduced to 0.9 ± 0.8D and 1.0 ± 0.6 D in groups 1 and 2, respectively, 5 months after surgery. A significant difference was found between the two groups regarding the astigmatism values at 1 week after surgery: P=0.03. However, a significant difference was not found between the two groups concerning the astigmatism values at 5 months after surgery.
Conclusions: Children, who underwent extraction of congenital cataract and foldable intraocular lens implantation using Mersilen sutures, had a significant higher astigmatism 1 week after the surgery, than those who had the same surgery, using Vycril sutures. This difference disappeared at 5 months after the operation. Therefore, we recommend the use of Vycril sutures for pediatric cataract surgeries.

DO TOPICAL BETA-BLOCKERS INCREASE THE RISK FOR DEPRESSION IN GLAUCOMA PATIENTS?

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Introduction: Depression is a common disorder, with lifetime incidence estimated at about 21.3% in women and 12.7% in men. Association of systemic and ophthalmic beta-blockers with depression is widely accepted in clinical medicine, and their use is avoided for that reason. Indeed, depression and mood changes are listed among other side effects of ophthalmic beta-blockers by their manufacturers. The purpose of this study was to investigate the effect of topical beta-blockers on the prevalence of depression among glaucoma patients.
Patients / Methods: We followed the electronic medical records of all the members in the Central District of Clalit Health Services older than 20 years (317,469 members) and documented all anti-glaucoma prescriptions (n=274,023) and all anti-depressants prescriptions (n=16,948), filled by glaucoma patients in the district between January 1st, 2001 and December 31st, 2003. We included only those patients that filled at least 6 consecutive anti-glaucoma prescriptions at least once every 2 months (n=6,597; 5,846 (88.6%) were treated with topical beta-blockers). Depressed patients were defined as patients that filled at least four prescriptions for antidepressants during the study period (n= 810, 12.3% of all glaucoma patients).
Results: No significant demographic differences were noted between glaucomatous patients treated and not treated with topical beta-blockers. 12.21% (12.69% after age-adjustment) of those treated with beta-blockers, and 12.70% of those not treated with beta-blockers, were also receiving drug therapy for depression (p=0.7, chi square test). When stratified by age, treatment with topical beta-blockers did not influence the prevalence of depression at any age group. Logistic regression analysis revealed a significant effect of age, place of birth and gender on the prevalence of depression but not of consumption of topical beta-blockers.
Conclusions: Consumption of topical beta-blockers by glaucoma patients does not increase the risk of depression.

DOES CHANGING EYE TEST ORDER WITH 24-2 SITA STANDARD RESULT IN A MEANINGFUL CHANGE IN TEST RESULTS?

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Introduction: The 24-2 SITA standard (SS) algorithm is potentially less affected by patient fatigue due to reduced test duration. We evaluated whether the order of the eyes tested with 24-2 SS affects field sensitivity or reliability in routine clinical practice.
Patients / Methods: Consecutive glaucoma patients or suspects with 2 prior sets of 24-2 SS VFs taken OD first ("test 1" and "test 2") were enrolled. The next VF test was performed OS first ("test 3"). All were experienced with automated perimetry and had visual acuity > 20/60. Duration between exams was less than 20 months. For each eye, MD was compared among the 3 successive exams using Repeated Measures ANOVA. A field was considered reliable if all indices were less than 20%.
Results: 49 persons (30 F, 19 M) were enrolled (mean age 70.4±11.8 years). Mean MD was OD: -5.98±5.30 dB and OS: -5.64±4.71 dB. There was no statistically significant change in MD between the 3 field tests of each eye. Testing the left eye first had no effect on exam reliability. Overall, unreliable fields were obtained in 30.6% of right eyes and 33.3% of left eyes. Fixation loss was responsible for unreliability in almost all cases (87.0% of right eyes and 95.9% of left eyes).
Conclusions: In this cohort of experienced visual field takers, changing the order of eyes tested with the SS 24-2 algorithm did not have a significant effect on mean deviation or test reliability. Inter-eye fatigue may not be clinically significant with this algorithm. Fixation loss remains a problem even with the short 24-2 SS algorithm and was not affected by eye order in this study.

RETINAL NERVE FIBER LAYER SPLIT BUNDLES ARE TRUE ANATOMICAL VARIANTS

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Introduction: The presence of retinal nerve fiber layer (RNFL) split bundles was recently described in normal eyes scanned using scanning laser polarimetry (GDx-VCC). In this study we set out to ascertain whether RNFL split bundles are indeed normal anatomical variants, or perhaps imaging artifacts.
Patients / Methods: 14 normal eyes obtained post-mortem were processed histologically using the "Umbrella technique". Peripapillary RNFL thickness measurements were plotted from ring sections of 3.0, 3.5, 4.0 and 4.5mm diameter. The presence of superior and/or inferior split RNFL patterns was sought.
Results: 4/14 eyes demonstrated a distinct histological pattern of RNFL split bundles, of which all four were found superiorly and none inferiorly.
Conclusions: This study provides histological evidence validating that RNFL split bundles are indeed a true anatomical finding, rather than an imaging artifact, and that it appears to be a relatively common variant in normal eyes.

SALAGEN DOES NOT AFFECT INTRAOCULAR PRESSURE AND PUPIL DIAMETER IN GLAUCOMA PATIENTS.

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Introduction: Topical pilocarpine has been used as an ophthalmologc agent for decades. Miosis and ocular hypotension are the well-known ocular effects of this preparation. Oral pilocarpine (Salagen) has recently been approved for the treatment of dry mouth (xerostomia), however the influence of this drug formulation on the pupil diameter and intraocular pressure has not been reported so far. We investigated the effect of Salagen on the pupil diameter and intraocular pressure in 32 glaucomatose eyes of 22 patients.
Patients / Methods: Patients received a single dose of Salagen (5mg) after the appropriate washout period. Intraocular pressure and pupil diameter using a NeurOptics digital infrared pupilometer were measured before and 4 hours after dosing.
Results: The mean (+/- SD) intraocular pressure and pupil diameter were 32.3+/-8.1 mmHg and 4.51+/-1.22mm before treatment with salagen. Four hours after Salagen, the mean intraocular pressure reduction was 3.78+/-4.8 mmHg (10%, $P<0.0001$) and the mean reduction of pupil diameter was 0.05+/-0.28mm.
Conclusions: Salagen did not lead to miosis in glaucoma patients. Although the statistical analysis shows some small significant decrease in intraocular pressure, it is not beneficial in the management of glaucoma patients.

MEASUREMENTS OF OPTIC DISC SIZE WITH HRT II, OCT3 AND FUNDUSCOPY ARE NOT INTERCHANGEABLE

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Introduction: Disc size is a major determinant of other disc parameters such as cup size and cup-disc-ratio. The purpose of this study was to assess the interchangeability of optic disc size measurements using slit-lamp funduscopy, OCT3 and HRT-II in clinical practice.

Patients / Methods: Measurements of vertical disc diameter (VDD) following pupillary dilation were obtained with the three methods. A single investigator, masked to the imaging results, measured VDD at the slit-lamp using a 78D lens to project a narrow beam within the scleral ring and reading its size on the scale of the slit-lamp. Another masked investigator measured VDD using the interactive measurements option of the HRT. VDD was directly read from OCT printouts. True agreement between methods in measuring VDD was assessed using Bland-Altman graphs and 95% limits of agreement (LoA). Disc area was obtained from OCT and HRT printouts. Discs were classified as small, average or large; for this purpose we defined average size according to diameter or area as 1) mean ± SD or 2) middle tertile. Agreement in classification of discs as small, average or large was assessed using kappa statistics.

Results: 48 patients (24 females, 24 males) were enrolled (mean age 53.4 ± 14.3). VDD (mean±SD) was 1.58±0.15, 1.70±0.22, and 1.90±0.24 mm with funduscopy, HRT and OCT, respectively. Very large LoA were observed: -0.29 to +0.70 mm for OCT and HRT, -0.07 to 0.71 mm for OCT and funduscopy, and -0.29 to 0.53 mm for HRT and funduscopy. There was poor agreement (kappa<0.4) in classification of disc size as small, average or large whether disc diameter or area was compared and using either definition of disc size.

Conclusions: Despite good reported reliability of individual methods, we observed a large range of differences in estimating disc size with HRT, OCT and funduscopy. This precludes interchangeable use of these measurements in clinical practice, and does not allow simple conversion formulas to be proposed. In addition, there is poor agreement between these methods in classifying disc size as small, average, or large. At present, estimation of both absolute and relative disc size can only be defined separately for each measurement modality.

DOSE THE AQUEOUS HUMOR HAVE A ROLE IN THE MAPK INTRACELLULAR SIGNALING IN GLAUCOMA?

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Introduction: Introduction: Glaucoma is a group of optic nerve neuropathies affecting millions of people worldwide. Primary open-angle glaucoma (POAG) is age-related and the most abundant form of the disease. The major risk factor for POAG is an elevated intraocular pressure (IOP). All forms of glaucoma involve retinal ganglion cell death with progressive and irreversible visual field loss. The possible cause of ganglion cell death is oxidative stress induced apoptosis. The induction of mitogen –activated protein kinases (MAPK) signaling cascades by oxidative stress determents the cell fate. Purpose: To investigate our hypothesis, whether elevated ocular pressure causes oxidative stress followed by induction of MAPK cascade within the aqueous humor.
Patients / Methods: Methods: Slow and persistent elevation of intraocular pressure was achieved by once weekly injection of 2% hyaluronic acid into anterior chamber of the left rat eye, while the right eye was kept untreated .The aqueous humor from injected and control eyes was analyzed for the presence of activated and total MAPK (ERK1/2 and JNK p38) by Western blot analysis at different time points.
Results: Results: Mean intraocular pressure was significantly elevated, (injected eyes 19.7mmHg±3.85 versus control 8.6 mmHg ±1.88) during one month of weekly injections. There were no pressure elevations in the parent eyes or in saline injected eyes. A 50% elevation in total Erk1 and a 6 fold increase in Erk2 were found in the aqueous humor driven from the elevated IOP eyes. Only the active phosphorylated form of Erk1 was found with the same intensity in control and elevated IOP. Two bands corresponding to pan-JNK were found in both groups: at 46KDa & 54KDa. Phosphorylated-JNK, the activated JNK form, was found only at 54KDA. No differences in JNK protein expression were found between groups.
Conclusions: Conclusions: In this study we were able to present the presence of several member of the MAPK signaling pathway, in the aqueous humor. Our data shows some differences in the expression of theses intracellular cell signaling proteins due to elevated IOP. We hypothesize that the aqueous humor is serving as a signaling medium, and might have role in the changes found in the trabecular meshwork.

IS THE ANTIOXIDANT N-ACETYL-L-CYSTEINE EFFICIENT IN REDUCING OR PREVENTING DIABETIC DAMAGE TO THE EYE LENS?

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Introduction: Purpose: To investigate the mechanisms of cataract formation under diabetic conditions, and to examine the direct effects of the antioxidant N-acetyl-L-cysteine on diabetic cataractogenesis.
Patients / Methods: Bovine lenses from animals up to 1 year old were used in this study. The lenses were divided into three groups: (a) control lenses, (b) lenses incubated with high glucose levels (450 mg%), (c) lenses incubated with high glucose levels and N-acetyl-L-cysteine. Lenses were incubated for a period of two weeks in an organ culture system. Lens optical quality was analyzed every 24 hours. At the end of the culture period the lenses were photographed using an inverted microscope. Lens epithelium was analyzed for cell morphology changes, and lens proteins were analyzed by SDS gel electrophoresis.
Results: High levels of glucose in the culture medium caused optical damage to bovine lenses, increased lens volume due to swelling, caused damage to lens epithelial cells, and caused aggregation of lens proteins. N-acetyl-L-cysteine increased lens transparency and prevented lens protein aggregation, but caused damage to lens morphology as seen by inverted microscopy.
Conclusions: High glucose levels cause damage to the eye lens. N-acetyl-L-cysteine partially protects the lens from this damage. This work was supported in part by the Guzik Ophthalmology Research Fund.

THE EFFECT OF TOPICAL SODIUM DICLOFENAC TREATMENT ON MACULAR THICKNESS POST PHACOEMULSIFICATION IN DIABETIC AND NON DIABETIC PATIENTS

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Introduction: Pseudophakic macular oedema remains a troublesome problem being one of the most frequent causes of visual decrease after cataract surgery. Although it occurs more frequently in patients suffering from vascular incompetence (HTN and diabetes) and after complicated operations (posterior capsular rupture and/or vitreous loss), it also occurs after uncomplicated cataract surgeries. The novel technique of optical coherence tomography (OCT) enables us to measure reliably retinal thickness, being a sensitive tool for the detection , quantification and follow up of early macular changes even without oedema. We conducted a prospective randomized clinical trial to investigate the influence of uneventful phacoemulsification surgery on macular thickness of diabetes mellitus (DM) and non-DM patients and the role of NSAIDS eye drops (sodium diclofenac) in modifying macular oedema

Patients / Methods: Fifty five consecutive patients (17 patients without DM [group 1] and 18 patients with DM [group 2]) with senile cataract awaiting cataract surgery were chosen and assigned randomly into two groups: groups 1A and 2A received only steroid eye drops 4 times a day for 4 weeks and groups 1B and 2B received post operative sodium diclofenac and steroids eye drops 4 times a day each for 4 weeks at least. Patients with DM had mild to moderate retinopathy without any pre existing macular oedema. All patients underwent an uneventful cataract surgery. macular thickness measurements by OCT were performed before and 1month after the operation.

Results: Mean preoperative macular thickness measurements in group 1A, 1B, 2A, and 2B were 207.2 ± 21.02 µm, 195.46 ± 12.05 µm, 205 ± 24.08 µm and 200.5 ± 33.7 µm, respectively ($p>0.6$). Mean postoperative increases in retinal thickness were 10.57± 18.97µm, 26.92 ± 79.43µm, 16.5 ± 16.68µm, and 9.5 ± 15.32µm respectively. Postoperative retinal thickness increase was statistically significant for both groups of DM patients (1A $p= 0.057$, 1B $p= -0.245$, 2A $p= 0.0027$ and 2B $p=0.036$).

Conclusions: Using OCT, sub clinical macular thickness changes can be noted after uneventful phacoemulsification surgery in DM and non-DM patients. Our findings suggest that topical sodium diclofenac doesn't have a statistically significant influence on the postoperative increase in retinal thickness in DM and non-DM patients.

REPOSITIONING AND SCLERAL FIXATION OF THE SUBLUXATED LENS CAPSULE USING AN INTRAOCULAR ANCHORING DEVICE

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Introduction: Lenticular subluxation presents a surgical challenge. We describe a new device for central repositioning and scleral fixation of the capsular bag of subluxated lenses in experimental models.
Patients / Methods: The device is a PMMA one-plane implant consisting of 2 handles, which grasp the edges of the capsulorrhexis, and a base for scleral fixation with a single 10-0 or 9-0 polypropylene suture. A temporary safety suture may be used until the device is secured to the scleral wall. The device was implanted in porcine eyes and in living New Zealand White rabbit eyes. An animal model of lens subluxation was achieved by partial tearing of the zonules. Capsular centering, implant stability, and inflammatory reaction were evaluated 2 to 4 weeks post implantation.
Results: The device was implanted in 7 porcine eyes and in 9 rabbit eyes. Lens subluxation model was created in 4 porcine eyes and in 2 rabbit eyes. The device effectively pulled the capsular bag to the center and remained stable up to 4 weeks thereafter. Large zonular dialysis was managed by using 2 devices. Successful Intraocular lens implantation was done repeatedly in the presence of the device. The implant was well tolerated in all rabbit eyes. Histopathological examination of the enucleated eyes revealed no inflammatory reaction or adhesions.
Conclusions: Experimental studies on the capsular anchoring device for subluxated lenses confirmed the safety and efficacy of the new device. A capsular tension ring can be inserted separately to further stabilize the capsular bag.

DEPENDENCY BETWEEN LIGHT INTENSITY AND CHICK'S REFRACTIVE DEVELOPMENT UNDER LIGHT DARK CYCLE

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Introduction: Light intensity modulates the chicks' refractive error induced by continuous light rearing, mainly via fine regulation of corneal curvature (Cohen et al., ARVO 2005). The emmetropization process is a fine tune of the refractive state by altering the refractive components toward near zero refraction. In chicks, the emmetropia is reached after 8 weeks. We provided cyclic illumination conditions and examined the effect of light intensity on the progression of emmetropization.
Patients / Methods: Forty chicks were reared under 12h/12h dark/light cycle at three different light intensities: 10 000 lux (n = 13, high intensity group), 500 lux (n = 14, medium intensity group), and 50 lux (n = 13, low intensity group). Their eyes were followed for 90 days by repeated retinoscopy, keratometry, as well as measurements in vivo by ultrasound and, after enucleation, by caliper.
Results: The time to emmetropization is light intensity dependent and reached after 50 days for the low intensity group and 60 days for the medium intensity group. The high intensity group had stable hyperopia of +1.1 ± 0.05 D from day 60 onwards and did not become emmetropic even on day 90. The low intensity group developed myopia of -2.4 ± 0.26 D on day 90. Corneal curvature differ significantly between the groups and directly correlates with light intensity (R^2=0.185, P<0.000), the higher the intensity the steeper is the cornea. The various light intensities also produced significant other changes in the components of the anterior and posterior segments
Conclusions: Light intensity is an environmental factor that modulates the process of emmetropization, the lower the intensity the faster emmetropia is reached. Thus, low ambient light under light/dark cycle is a risk factor for myopia development. It seems that the dependency between visual experience and the development of refractive errors is determined at least by three parameters, the age of the chick (residual plasticity), it's current refractive error (residual modifiable power) and the environmental inducer (in this case -light intensity).

A COMPARATIVE STUDY OF HARDY-RAND-RITTLER 4TH EDITION AND ISHIHARA COLOR PLATES FOR DETECTION OF COLOR-VISION DEFECTS IN OPTIC NEUROPATHY

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Introduction: Color vision testing is one of the most useful components of the sensory neuro-ophthalmologic examination. The Hardy-Rand-Rittler 4th edition (HRR) was published in 2002, but there is insufficient information about its sensitivity to color vision defects in cases with optic neuropathy. The purpose of our study was to compare the sensitivity of the HRR and Ishihara color plates to color-vision defects in patients with optic neuropathy.
Patients / Methods: This study was a prospective study. The study group included 44 patients (49 eyes) with newly diagnosed optic neuropathy. The control group included 43 patients (65 eyes) with no optic nerve or retinal macular disorders. A complete ophthalmologic and general history was obtained. The patients underwent a comprehensive eye examination and color-vision test examinations in a randomized order. The Ishihara test score was set as the number of identified plates out of 12 and the HRR test score was set as the number of identified plates out of 6.
Results: The mean Ishihara score in the study group was 10.5 ±3 and 11.75±0.4 in the control group, while the mean HRR score was 2.39±1. 7 in the study group and 5.29±0.54 in the control group. With a cut-off score of 11/12 and 10/12 the Ishihara test achieved specificity- sensitivity levels of 91%-69% and 100%-51% respectively. The HRR test achieved a better specificity-sensitivity balance of 92%-86% and 97%-84% for cut-off scores of 4.5/6.0 and 4.0/6.0 respectively.
Conclusions: HRR 4th edition is more sensitive in detecting acquired dyschromatopsia due to optic neuropathy than the Ishihara plates.

PERCEPTUAL DEFICITS IN A SIMULATED-SCOTOMA MODEL OF HUMAN EYE INJURY

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Introduction: Macular scotomas, affecting visual functioning, characterize many eye and neurological diseases like AMD (Acquired Macular Degeneration), diabetes mellitus, multiple sclerosis, macular hole and others. In this work, foveal visual field defects were modeled, and their effects were evaluated on stimulus detection and aiming.
Patients / Methods: The modeled occluding scotomas, of different size, were superimposed on the stimuli presented on the computer display, and were stabilized on the retina using a mono Purkinje Eye-Tracker. The detection task consisted of a triple conjunctive visual search display of: size (in visual angle), contrast and background (simple, low-level features vs. complex, high-level features). Search/aiming accuracy as well as R.T. measures used for performance evaluation.
Results: Artificially generated scotomas slowed stimulus detection time in complex background search situation more than in simple background. Detection speed was dependent on scotoma size and size of stimulus. In contrast, visually guided aiming was more sensitive to scotoma effect in simple background search situation than in complex background. Both stimulus aiming R.T. and accuracy (precision targeting) were impaired, as a function of scotoma size and size of stimulus.
Conclusions: The data can be explained by models distinguishing between saliency-based, parallel and serial search processes, guiding visual attention, which are supported by underlying retinal as well as neural mechanisms.

THE EFFECT OF HYPERBARIC TREATMENTS ON THE RATE OF REFRACTIVE CHANGES AMONG DIABETIC PATIENTS

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Introduction: Hyperbaric oxygenation HBO is a common treatment both for emergency medicine as well as for chronic treatments. The usual protocol course is breathing 100% oxygen in a "depth" of 10 meter under water (2 ATA). The duration of each treatment is 90 minutes and done on a daily basis. One of the most common indications for treatment is non healing wound of diabetic patients ("the diabetic foot"). Its known that prolonged HBO treatments cause a myopic change in refraction. From the literature we know that the myopic shift is usually temporary, reversing back to basic refraction within a few weeks of cessation of the HBO course.
Patients / Methods: 50 diabetic patients treated chronically (atleast 30 treatments) with HBO for NHW were included. Hyperbaric oxygen therapy was given for 90 minutes daily at a pressure of 2ATA. Follow-up examinations were carried out every 10 treatments. Refraction was assessed automatically and by the monocular subjective refraction method. We included separately: SPHER, SE, CYLINDER, AXIS. For each parameter, a regression equation was calculated after plotting the myopic change against time. A single examiner performed all the measurements; the examiner was blind for the previous results.
Results: There was a statistically significant myopic shift in both SPHER, SE(0.57D, 0.64D). The change accrued from the first examination and remained throughout the follow-ups at a steady rate. Both eyes behaved similarly. We did not find a correlation between the myopic shift and: gender, age, basic refraction.
Conclusions: A positive correlation was found between HBO treatments and a myopic shift in the refraction. The correlation was statistically significant for: SPHER, SE. The myopic shift was noted already in the first examination and continued in a similar rate throughout the treatment course. There was no difference in the myopic shift rate and or progress between the eyes (right Vs left). Also gender, age, or basic refraction on admission had no effect on the myopic change. Interestingly a finding of this work was found in comparing Phakic Vs Pseudophakic eyes, we found the rate of shift as well as the amount of shift to be similar between the groups.

THE EFFECT OF HYPERBARIC OXYGEN TREATMENT ON VISUAL ACUITY AFTER ACUTE CRAO

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Introduction: Central retinal artery occlusion (CRAO) is a devastating ophthalmic emergency. A massive irreversible damage causes an acute visual loss with little chance of improvement. This led to a constant search for a treatment modality that improves visual outcome better than the natural history of the disease. The purpose of this study was to evaluate the effect of hyperbaric oxygen treatment (HBT) on visual acuity in patients, presenting with acute CRAO.
Patients / Methods: Retrospective case series single referral centre study Forty consecutive patients with acute non arthritic CRAO were treated with HBO in our Hyperbaric Institute. Three atmosphere absolute (ATA), 100% oxygen for 90 minutes were given every eight hours in the beginning and once daily afterwards. Treatment was discontinued if no improvement was observed after three treatments. Visual acuity was recorded before and after treatment.
Results: Mean visual acuity at admission was 3.08±0.78 log MAR (range 0.7to 5) . Mean time interval between the onset of symptoms and the beginning of HBT treatments was 8.95 ± 4.39 hours . Patients received a mean of 4.56 ± 1.78 consecutive treatments . Visual acuity improvement after treatment was recorded in 18 patients (45%). The mean change of visual acuity before and after treatment was 0.5± 0.86 log MAR for the whole group and 1.31 ± 0.52 log MAR for those that improved (p =0.001). A moderate negative correlation (r= -0.44) was found between the change in visual acuity before and after treatment and the time interval between the onset of symptoms and the beginning of HBT treatments (p= 0.0047). Logistic regression models elicit a sub optimal cut-off value of eight hours from the onset of symptoms to the beginning of HBT treatments that would best predict post-treatment improvement. Age and number of treatment had a negligible correlation with visual outcome.
Conclusions: The improvement in visual acuity as demonstrated in our study has little clinical significance in those patients with good vision in their fellow eye. Although our study is the largest one published, further large scale prospective randomized studies are needed in order to establish the exact usefulness of HBT modality in the treatment of acute CRAO

EXPRESSION OF ACHE RESADTHROUGH ISOFORM CORRELATES WITH RETINAL PHOTIC STRESS IN A RAT MODEL

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Introduction: Acetylcholinesterase (AChE) is involved in neurite growth and stress responses. We have previously shown that AChE stress related isoform- AChE-readthrough isoform - expression is increased in the photoreceptors of the rat retina following exposure to damaging light. The purpose of this study was to study the correlation of AChE-readthrough isoform expression with the degree of photic stress.
Patients / Methods: Rats raised in 12/12 hour light/dark cycle prior to and following damaging light exposure. Several groups were compared with varying duration of damaging light exposure: 4, 10, 24, 30 hrs. An additional control group consisted of rats that were not exposed to damaging light. Electroretinograms (ERGs) were recorded at 1 and 14 days after light exposure, and rats were sacrificed and their eyes enucleated. AChE-readthrough mRNA was examined by in-situ hybridization and cytochemical staining assessed AChE activity.
Results: ERG recordings correlated with the duration of light exposure. At durations of 4 and 10 hrs, ERG deteriorated but fully recovered. At exposure of 24 hrs there was a slight recovery, but a permanent deficit persisted. Exposure of 30 hrs resulted in a significant permanent deficit. AChE-readthrough mRNA and activity levels in the photoreceptors were increased in light exposed groups as compared with controls. AChE expression correlated with the degree of functional damage. Thus, at 4 hrs of exposure no significant change was noted. However, at longer durations of exposure we could note an increased expression of AChE. The higher degree of structural and functional damage correlated with a higher degree of AChE expression in the photoreceptors.
Conclusions: AChE-readthrough isoform expression increased in light exposed groups, as compared with controls. However, this increase was higher as photic stress increased. Thus, induction of AChE-readthrough isoform expression correlates with the degree of retinal damage. Together with our previous results showing that antisense treatment attenuate this damage, this data support the causal detrimental role of AChE in photoreceptor damage.

SURVIVAL OF UVEAL MELANOMA PATIENTS AFTER SURGERY FOR LIVER METASTASES

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Introduction: The liver is the main site of metastases in uveal melanoma patients. The purpose of this study was to evaluate the post hepatectomy survival of uveal melanoma patients with liver metastasis as a function of their disease free interval, the number of lesions found during the abdominal surgery and the surgical result (disease free or residual disease).
Patients / Methods: A retrospective analysis of data of our uveal melanoma patients in the years 1995-2005. Patients were grouped by disease free interval of ≤1, 1-5 and >5 years; by the number of lesions found during surgery: 1 metastasis, 2-3 metastases, and >3 metastases; and by the surgical borders at the end of the surgery: R0 and R1/R2. Kaplan Meyer survival analysis and ANOVA were done using JMP 5.0.
Results: Out of 431 patients with uveal melanoma on our database, 40 are known to have metastasis. Surgical data was available for 31. Overall survival was 21.6 ±4.1 months (mean ±SE). Post hepatectomy survival by disease free interval was 14.5 ±4.1, 21.1 ±4.8 and 26.3 ±11.2 months for patients who developed metastasis ≤1, 1-5 and in >5 years, respectively (Log Rank Chi square 0.6630, DF 2, p 0.7179). Post hepatectomy survival by the number of lesions found during surgery was 32.7 ±9.5, 27.4 ±19.3 and 13.4 ±1.7 months for patients with 1, 2-3 and >3 metastases, respectively (Log Rank Chi square 4.8361, DF 2, p 0.0891). Patients with one metastasis have statistically longer survival than those with >3 (R square 0.1189, F 6.0553, p 0.0208). Patients with clean surgical borders survive longer than those with residual disease (33.3 ±8.6 vs. 13.1 ±1.8 months, Log Rank Chi square 5.2167, DF 1 p 0.0224).
Conclusions: It is possible to significantly extend the life expectancy of patients who develop isolated hepatic metastasis by complete resection of the lesion.

POTENTIAL BLOOD MARKERS FOR DETECTION OF METASTATIC UVEAL MELANOMA – A COMPARATIVE ANALYSIS

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Introduction: Serum Osteopontin levels have been shown to increase in metastatic uveal melanoma. This study was designed to compare Osteopontin, with two other potential serum markers for metastatic uveal melanoma: S-100 and MIA.
Patients / Methods: Serum levels of Osteropontin, S-100 and MIA were examined in sera from 15 patients with proven metastatic uveal melanoma and of 37 patients under follow-up for more than 10 years without metastatic disease. Among the 15 patients with metastases, serum samples from 8 patients before and after the diagnosis of metastases were assayed. Serum from 30 healthy age- and sex-matched individuals were also examined as a control group for this part. The three biomarkers were measured by commercially available ELISA kits (Assay Designs, Ann Arbor, MI).
Results: The serum levels of S100 protein were 2 fold higher in patients with metastatic melanoma to the liver than in the control disease-free population (Kruskal Wallis chi-square = 4.0859, df=1, p=0.0432), but there was no significant difference between serum levels in patients before and after metastasis (sign test, p=0.4531). Serum levels of MIA neither discriminated between patients with patient with metastatic melanoma and controls (Kruskal Wallis chi-square = 2.4210, df=1, p=0.1197), nor between patients before and after metastasis (sign test, p=0.7266).
Conclusions: The nearly 2 fold increase in S-100 levels is of borderline significance. In this study MIA was not found to be a useful serum marker for metastatic uveal melanoma. Elevation in serum osteopontin remains the only serum marker tested among three common biomakers for melanoma that appears to identify patients with metastatic uveal melanoma to the liver.

OPTICAL COHERENCE TOMOGRAPHIC CHARACTERISTICS OF COMBINED HAMARTOMA OF THE RETINA AND RETINAL PIGMENT EPITHELIUM (RPE) IN 11 PATIENTS

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Introduction: Combined hamartoma of the retina and retinal pigment epithelium (RPE) is an uncommon fundus tumor with classic clinical features. It was described by Schachat et as a typically elevated pigmented lesion with vascular tortuosity, vitreoretinal interface changes, and lipid exudates. The vitreoretinal interface changes of the disease can lead to formation of wrinkling retinopathy or in more severe forms to vascular dragging and in time ending in progressive loss of visual acuity due to tractional distortion of the fovea. Insufficient understanding of the nature of the vitreoretinal interface changes led to a controversy around the question of the benefits and timing of surgical removal of the tractional component. In order to learn more of the vitreoretinal interface abnormalities and retinal microarchitecture of combined hamartoma of the retina and RPE we examined 11 consecutive patients with the disease using OCT.

Patients / Methods: Eleven consecutive patients who had a combined hamartoma of the retina and RPE imaged with OCT.

Results: In all cases, the tumor was gray-brown unilateral and unifocal. The initial symptom at presentation was blurred vision (n = 6), strabismus (n = 2), or asymptomatic (n = 3). The meridional location of the tumor was macula (n = 8), inferior (n = 1), and nasal (n = 2). The mean basal dimension of the tumor was 7 mm and the mean thickness of the tumor as measured by ultrasonography was 1.8 mm. Choroidal neovascularization was not present. A distinct epiretinal membrane with secondary retinal folds and striae was observed on OCT in 10/12 patients. In all cases the retina was anatomically disorganization with loss of identifiable retinal layers. In some cases the retina was drawn into folds or striae .The mean retinal thickness at the site of the lesion was 766 µm. The adjacent flat retina appeared to be of normal thickness and anatomical configuration

Conclusions: Optical coherence tomography can provide important information regarding the vitreoretinal interface and retinal microstructure of this tumor and could influence surgical decisions.

INCREASED EXPRESSION OF INFLAMMATORY CYTOKINES AND CATHEPSINS IN THE CORNEAL EPITHELIUM IN KERATOCONUS

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Introduction: To evaluate the expression of inflammatory cytokines and cathepsins in the corneal epithelium in keratoconus.
Patients / Methods: Tissue sections were prepared from corneal buttons removed during penetrating keratoplasty from 29 patients who had keratoconus. Tissue sections from 10 age-matched eyes enucleated because of uveal melanoma served as a control. Expression of Interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-alpha, and cathepsins (Cath) B, S, L and D in the corneal epithelium was evaluated by immuno-histochemistry. Digital image analysis was performed to quantify the expression of the various cytokines and cathepsins. A mean intensity stain index (ISI), based on the staining density and the area stained, was calculated from digital images. The mean ISI of sections from patients with keratoconus was compared to that of controls. Clinical parameters were recorded from the patients' charts including the age at diagnosis of keratoconus, years with keratoconus prior to surgery, age at surgery, and maximal central topographic K reading. The correlation between the clinical parameters and the ISI values for each protein was evaluated by factor analysis and Spearman's rank correlation.
Results: A significantly higher mean protein expression in the corneal epithelium of keratoconus corneas was demonstrated for IL-1beta (ISI score in keratoconus +/- 15,214 in normal controls, p=0.00017),±43,086 +/- 32,829 compared to 9,354 IL-6 (31.63 +/- 22.4 in keratoconus vs. 10.76 +/- 21.41 in controls, p=0.024), 1,467,971 in±TNF-alpha (3,402,263 +/- 1,516,245 in keratoconus vs. 1,933,844 10.30 in keratoconus vs. 3.51 +/- 5.87 in±controls, p=0.021) and Cath B (11.08 controls, p=0.011). Higher though not significant expression was also demonstrated for Cath D, Cath L and Cath S, who had 1.3, 2.1, and 1.9 folds higher ISI values compared to controls, respectively. A highly significant correlation was found between the patients age at diagnosis of keratoconus and the ISI values of IL-1beta (r=0.8047, p=0.0072) and TNF-alpha (r=0.8047, p=0.0039).
Conclusions: The corneal epithelium in keratoconus expresses high levels of cytokines and enzymes, which are associated with inflammation and tissue degradation. These agents are associated with patients' age, and may be responsible for the progression in corneal thinning and increased steepness, eventually leading to corneal transplantation.

DRUG MODDIFICATION OF ANGIOGENESIS IN A RAT CORNEA MODEL

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Introduction: The discovery that angiogenesis plays a key role in a large number of human diseases has led to a constant search for angiogenesis modulators. Ophthalmic medications, especially in glaucoma therapy uses a variety of substances that mimic the activity of natural humoral and local neural transmitters .We conducted a pilot study to evaluate the influence of some widely used anti glaucoma agents on angiogenesis in a novel rat cornea model.

Patients / Methods: design-in vivo assay using an animal model. Slow release polymer Elvax pellets containing basic fibroblast growth factor (bFGF) were used to induce angiogenesis in 32 rats using a novel rat corneal micropocket assay. After an observation period of seven days angiogenesis was measured and compared in five different groups of rats. The only difference being the anti glaucoma drug therapy given during the experiment including prostaglandins, beta blockers, alpha 2 agonists and carbonic anhydrase inhibitors.

Results: Biomicroscopic observation disclosed that all pellets induced neovascular reaction in the rat's corneal stroma. A growth index using the maximal linear vessel growth divided by pellet- limbus distance was measured. When comparing the normal, Latanoprost, Dorzolamide , Brimonidine and Timolol malate we found growth index of 1.655+0.1554, 1.977+0.1834, 1.844+0.1888, 2.034+0.3759, 1.650+0.1407 respectively (data represent mean + SD). These results confirm the hypothesis that anti glaucoma drugs delivered topically modifies the normal angiogenic response. Among them prostaglandins shows the most prominent excitatory effect (one way ANOVA test, p= 0.03).

Conclusions: This is the first study that explores the use of ophthalmic topical medication in angiogenesis modulation. We showed the excitatory effect of several widely used anti glaucoma therapy on the angiogenic process in a model of a rat corneal angiogenesis assay. Our results imply the need for a rational use of selected drugs in different ophthalmic pathological processes such as neovascular glaucoma, taking into account their influence on angiogenesis. Further investigation is still needed.

THE PATHOLOGICAL MECHANISM OF DELAYED INJURIES FOLLOWING SULFUR MUSTARD EXPOSURE: 2. A CORRELATION OF CLINICAL STATUS WITH GROWTH CAPACITY OF EPITHELIAL CELLS AND INFLAMMATORY MARKERS OF THE CORNEA

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Introduction: Ocular exposure to sulfur mustard (SM) vapor causes a severe acute lesion in all exposed eyes (rabbits). Nevertheless, the delayed injury appears in ~50% of the eyes. We aimed to study correlation of appearance of delayed lesion to impaired epithelium, and to ongoing inflammation, indicating therapeutic strategies.
Patients / Methods: Animal Care and Use Committee approval at IIBR was obtained. Rabbit eyes were exposed to SM vapor and a clinical follow up was carried out one month. Rabbits were euthanized and corneas processed for biochemical analyses and for primary corneal epithelial cell cultures from center (C) or periphery (P) of the corneas (ex vivo PRCEC). Proliferative capacity of epithelial cells was measured from cell yield of successful explants. Protein was measured by the Bradford method, CGRP by ELISA of acetic acid extracted corneas and PGE by RIA.
Results: At one month post exposure, neovascularization appeared in 50% of corneas, thus two corneal populations were evident: non-impaired and impaired. In ex vivo PRCEC prepared from exposed corneas the yield of cells from C of all exposed corneas was smaller than the yield from control corneas. SM exposed corneas were 20% heavier than controls ($p<0.005$), and impaired corneas were 20% heavier than non- impaired ($p<0.01$). PGE content of corneas was 3 fold higher than controls ($p<0.001$), and impaired corneas had 50% higher PGE than non-impaired corneas ($p<0.05$). CGRP content was 5 fold higher in SM exposed corneas ($p<0.001$) but no significant difference was found between impaired and non-impaired corneas.
Conclusions: Persistent inflammation correlated with the clinical status of individual eyes during the delayed phase of the lesion. Such signs corroborate our suggestion that the pathological healing process and the ongoing inflammation in impaired eyes contribute to the delayed lesion. Our findings imply that supplement of growth factors and anti- inflammatory drugs during the delayed phase of the lesion might be beneficial.

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METALLO-COMPLEXES AUGMENT TREATMENT OF PSEUDOMONAS KERATITIS

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Introduction: Pseudomonas aeruginosa is an opportunistic bacterial pathogen capable of causing severe corneal infections. Formation of biofilms may be one of its principal pathogenic features, contributing to resistance against antibiotic treatment and the host immune response. Recent data suggests that iron may play an important role in the formation and stability of pseudomonas biofilms. Our objective was to evaluate whether depletion of iron using gallium-desferrioxamine (Ga/DFO) complexes could be useful in the treatment of pseudomonas keratitis.

Patients / Methods: Keratitis was induced in the eyes of 53 NZW rabbits by application of contact lenses contaminated by P. aeruginosa. Once a 3-4mm infiltrate formed, topical treatment with eye drops was initiated with eyes randomly assigned to one of five treatment groups: sham, 0.5% gentamicine (GM) alone, 0.5% GM+3 mg/ml Ga/DFO, 0.5% GM+DFO or 0.5% GM+GaCl3. Drops were administered according to an intensive regimen, similar to that used clinically, for 96 hours. Progression of the infiltrate and epithelial defect were quantified in a masked fashion from digital color and fluorescein photographs up to two weeks following infection, and extent of corneal opacification, iris injection and hypopion formation were graded. Eyes were then enucleated and processed for histo-pathologic evaluation. .

Results: In sham-treated keratitis eyes, infection spread rapidly. Within 48 hours total opacification of the cornea occurred, and endophthalmitis developed in most cases. In all GM-treated groups the course of disease was less severe, with the most significant rescue occurring in the Ga/DFO-treated eyes. Infiltrate, erosion and final scar areas in these eyes were reduced by approximately 50% compared to GM alone, a difference which was statistically significant. Addition of either DFO or GaCl3 to GM yielded an effect that did not differ significantly from GM alone, with DFO+GM somewhat better than GM alone and GaCl3+GM slightly worse. Ga/DFO+GM eyes also demonstrated marked reduction in corneal opacification, iris injection and hypopion formation. Histologically, Ga/DFO+GM corneas presented less leukocyte infiltration, neo-vascularization and fibrosis than other experimental groups.

Conclusions: Addition of the Ga/DFO complex to GM significantly improves corneal healing of P.aeruginosa induced keratitis. Modulation of iron availability may be a novel means to augment efficacy of antibiotic treatment.

THE EFFECT OF 4TH GENERATION FLUOROQUINOLONES ON THE HEALING SPEED OF CORNEAL EROSIONS IN AN ANIMAL MODEL

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Introduction: To compare the effect of gatifloxacin and moxifloxacin (two new fourth-generation fluoroquinolones) on the epithelial healing rate of corneal erosion in an animal model.
Patients / Methods: Uniform 6 mm central corneal erosion was created in 15 rabbits. The rabbits were randomized to receive gatifloxacin, moxifloxacin, or saline (control). Drops were administered every 15 minutes for an hour, then hourly for 3 hours, and afterwards, four times daily. Eyes were examined and photographed every 12 hours until the erosion had healed completely. The photographs were used for the measurement of the area of residual epithelial defects and for evaluation of the speed of healing.
Results: The control eyes healed on average after 57 hours, the moxifloxacin-treated eyes healed on average after 62.4 hours, and the gatifloxacin treated eyes healed on average after 67.2 hours. The differences were not statistically significant.
Conclusions: Both gatifloxacin and moxifloxacin produced insignificant delay of the healing of corneal erosions in this rabbit model. Therefore, either of these new fluoroquinolones can be considered for antibiotic prophylaxis in the treatment of corneal erosions. An additional study with larger groups is planned to more firmly establish these findings.

EFFECT OF ADJUNCTIVE STEROID TREATMENT ON THE OUTCOME OF BACTERIAL KERATITIS

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Introduction: To evaluate the outcome of bacterial keratitis treated with or without adjunctive topical steroids in additional to the traditional antibiotics use.
Patients / Methods: We retrospectively reviewed the records of patients who were hospitalized with bacterial keratitis in the department of Ophthalmology at Hadassah Hospital between 1999-2004. We did not include patients with incomplete records. 65 files were reviewed and 41 files with complete data were included in the study. Age, sex , ulcer size and localization, microbiological etiology, epithelization time and the final best corrected visual acuity were evaluated. Patients were divided into two groups. Group 1: 21 patients with bacterial keratitis who received standard therapy of fortified cephazolin 5% and gentamicin 1.4% drops. Group 2: of 20 patients who received the same topical antibiotics and adjunctive drops of dexamethasone phosphate 0.1% were added after identification of the bacteria or improved symptomatology when the cultures were negative.
Results: There were no differences between age, sex, ulcer size and localization and visual acuity at the first examination (p = 0.306 to 0.588). Contact-lens wear was the most common predisposing factor in both groups (33.3% and 20%). Corneal cultures were positive in 57.1% in group 1 and in 45% in group 2 .The most common isolated organisms were P. aeruginosa (33.3% in Group 1 and 44.4% in Group 2) and Staphylococci species (33.3% in Group 1 and 11.1% in Group 2). Treatment with topical steroids began between days 2-12 (mean 6.0±3.1 day) and they were applied for 4 to 180 days (mean 50.4±42.5 day). Most of cases received steroid 4 times daily. Steroid were gradually tapered and discontinued. Complete epithelization was achieved after 3 to 30 days (mean 9.4±7.1 days) in Group 1 and 3 to 38 days (mean 14.5±10.9 days) in Group 2 (p=0.119). Final visual acuities were 0.49±0.38 (between hand motion and 1.0) in Group 1 and 0.34±0.34 (between light perception and 0.90) in Group 2. The number of gained Snellen lines were 2.6±3.1 in Group 1 and 1.7±.2.4 in Group 2 (p=0.370) .
Conclusions: The use of adjunctive steroids did not significantly improve the outcome of microbial keratitis.

BINOCULAR FUNCTION FOLLOWING MONOVISION CORRECTION WITH LASER IN SITU KERATOMILEUSIS (LASIK) SURGERY IN PRESBYOPIC PATIENTS.

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Introduction: Monovision is a method of presbyopic correction whereby the dominant eye is usually corrected for distance vision and the nondominant eye corrected for near. This strategy can be performed with spectacles, contact lenses, or refractive surgery. In our study, presbyopic patients were treated with LASIK. The effect of this treatment on binocular visual function was studied.
Patients / Methods: A prospective study was performed on 20 patients age 40 years and older who underwent monovision LASIK surgery. Fusion and stereopsis were measured before LASIK surgery and at one month and 3 months following surgery.
Results: No difference was found in fusion for distance and near, before surgery and at one month and 3 months following surgery. Stereopsis values before LASIK surgery were 53.5 ± 81.92(SD) and 60.5 ± 78.07 (SD) seconds of arc for distance and near respectively with optical correction. One month following surgery, mean stereopsis values without optical correction, were reduced to 150 ± 124.19 (SD) and 117 ± 177.09 (SD) seconds of arc at distance and near respectively. However, with the appropriate optical correction stereopsis returned to the preoperative values (57 ± 32.84 (SD) and 55 ± 40.30 (SD)). Three months after surgery mean stereopsis values without optical correction were 188.5 ± 141.43 (SD) and 58.4 ± 32.84 (SD) seconds of arc at distance and near respectively; and 57.8 ± 88.48 (SD) at distance and 42 ± 5.10 (SD) at near with the appropriate optical correction.
Conclusions: Monovision LASIK surgery in the pesbyopic patient did not alter fusion. Best stereoacuity results for distance were achieved only with optical correction. Stereoacuity for near could be achieved after one month only with optical correction while after 3 months no optical correction was needed.

EARLY RECOVERY AFTER SIMULTANEOUS BILATERAL PHOTOREFRACTIVE KERATECTOMY FOR MYOPIA

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Introduction: Broad beam excimer laser PRK patients had relatively slow visual recovery, thus prompting most surgeons at that time to perform sequential PRK procedures. Hence people are attracted to a procedure like laser in situ keratomileusis (LASIK) although it has higher risk profile (i.e. corneal ectasia, mycobacterium keratitis, diffuse lamellar keratitis and flap complications). In this trial we tried to assess the early visual and refractive recovery after simultaneous bilateral PRK using a flying small spot laser.

Patients / Methods: A prospective nonrandomized clinical trial included one hundred sixty two consecutive myopic eyes, of 81 patients at Marom Bazel Refractive Surgery Clinic, Tel- Aviv, Israel. Two surgeons (D.V., D.Z.) performed simultaneous bilateral myopic photorefractive keratectomy (M-PRK) using the Allegretto 200 excimer laser (WaveLight Laser Technologie AG, Erlangen, Germany). Emmetropia was the goal for all eyes. Mean preoperative spherical equivalent and astigmatism were -3.37 ± 1.62 D (range, - 0.75 to –8.25 D) and -0.71 ± 0.73 D (range, -0.5 to -3.5 D), respectively. Main outcome measures were uncorrected visual acuity (UCVA) at 1 week and 1 month after surgery. Secondary outcome measures were manifest refraction and best-spectacle corrected visual acuity (BCVA) at 1 week and 1 month after surgery.

Results: One week after surgery the mean decimal UCVA was 0.78 ± 0.19 (range 0.25 to 1.3), UCVA of 20/40 or better and 20/25 or better were achieved by 95.1% and 65.4% of eyes, respectively. One month after surgery the mean decimal UCVA was 0.91 ± 0.19(range 0.20 to 1.3), UCVA of 20/40 or better and 20/25 or better were achieved by 97.5% and 87.0% of eyes, respectively.

Conclusions: Traditional PRK performed utilizing a modern excimer laser provides fast visual rehabilitation for most patients. The gap in speed of visual recovery between surface ablation and LASIK seems to narrow

INFORMATION SOURCES AND THEIR USE BY PATIENTS UNDERGOING KERATOREFRACTIVE SURGERY

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Introduction: Patient's input is critical to clinical management in patients undergoing keratorefractive surgery. However, little is known about the range of sources patients use to learn about their ophthalmic condition, which sources they find most useful, and how this relates to their understanding.
Patients / Methods: Cross-sectional survey of the patients undergoing keratorefractive surgery in one keratorefractive medical center, Haifa, Israel. A statistical analysis was performed
Results: : One hundred and six patients participated. Most patients received information from more than one source, with the refractive surgeon being the most frequently reported (99. 06%). Other important sources were another ophthalmologist (76, 08%), family members and friends (66.34%), internet (54.55%), newspapers (48.51%), optometrist (46.74%) and television (40.00%). The refractive surgeon was ranked as the best source by most of the patients. 84% of participants were satisfied from the refractive surgery. A significant statistical correlation was found between the presence of a perceived good information source and patient's satisfaction.
Conclusions: The findings emphasize the key role of the ophthalmic refractive surgeon in improving patient education directly, as well their responsibilities and opportunities to do so through supporting information through other sources, especially colleagues in ophthalmology and the internet.

ROLE OF EPITHELIAL HYPERPLASIA IN REGRESSION FOLLOWING LASER ASSISTED SUBEPITHELIAL KERATOMILEUSIS (LASEK)

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Introduction: Refractive surgery has emerged as a safe, effective and predictable technique for correcting refractive errors. Several techniques are used, each with different advantages and disadvantages. PRK (Photorefractive Keratectomy) may be hampered by post operative regression, related to epithelial hyperplasia. The goal of this work was to examine whether LASEK (Laser assisted subepithelial keratomileusis), which is also a surface ablation is associated with post-operative regression and epithelial hyperplasia.

Patients / Methods: Consecutive patients undergoing LASEK surgery were evaluated before, and at 1 and 3 month following operation. The demographic details, best corrected visual acuity, keratometry, and refractive error were recorded. Central corenal thickness (CCT) and corneal epithelial thickness (CET) were measured using the Corneo –Gage Plus high frequency (50 MHz) ultrasonic pachymeter. Data were analysed using ANOVA.

Results: 18 patients (36 eyes) were evaluated, 10 females and 8 males at the age of 31.6±10.6 years. Mean refractive error was -4.46±2.09 D , and -0.08± 0.3 D prior to surgery and at one month following surgery respectively ($p<0.001$). There was no significant refractive change between one month and 3 month following surgery. Thus the refractive error difference was-0.02±0.2 D, and keratometry difference was -0.21±1.66 (p=NS) between one and three month following surgery. Pachymetry showed a significant increase in CET at one month following LASEK m increase, pμ(0.47±0.77 <0.001), as compared to CET before surgery. Between one month and 3 month following surgery CET decreased (difference -0.19±0.4, $p<0.01$), but was still significantly higher than the value recorded prior to surgery. CCT did not change between one month and three month following surgery.

Conclusions: This study showed that the refractive correction achieved by LASEK surgery was stable, and was not associated with myopic regression in the first three month. Interestingly, we could note, small yet significant changes in CET, that were not associated with refractive changes. This cohort will be evaluated at 6 and 12 month to assess for late regression.

THE EFFECT OF INTRALASIK ON CORNEAL SENSITIVITY AND TEAR FUNCTION

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Introduction: The purpose of the study was to evaluate the changes in corneal sensitivity and tear function following myopic IntraLASIK.
Patients / Methods: Corneal sensitivity (filament), tear break-up time and Schirmer II tests were performed preoperatively, and at 1 week and 2 months postoperatively.
Results: Seventy-eight eyes from 41 consecutive patients (21 women and 20 men) who underwent IntraLASIK for myopia or myopic astigmatism were evaluated. The mean corneal sensitivity was preoperatively 5.04 ± 1.33, decreased only slightly to 4.84 ± 2.03 at 1 week and increased to 4.92 ±1.21 mm at 2 months postoperatively. Mean tear break-up time was 7.9 ± 2.6 seconds preoperatively, and postoperatively 8.7 ± 3.9 at 1 week and 10.5 ± 4.9 at 2 months. Mean Schirmer score was 13.9 ± 5.9 mm preoperatively, and postoperatively 12.6 ± 5.5 at 1 week, and 13.4 ± 4.9 mm at 2 months.
Conclusions: The reduction in corneal sensitivity and tear function tests were minimal after myopic IntraLASIK. Despite the relatively narrow hinge, the thin flap in IntraLASIK appears to have a beneficial effect on corneal sensitivity and dry eye signs.