

PROGRAM
29th Annual Meeting
Neve Ilan Resort Hotel
March 12-13, 2009

תכנית

הכינוס השנתי ה- 29
מלון נוה אילן
13-12 מרץ, 2009

עריכת התוכנית: פרופ' אבי סולומון, פרופ' רון עופרי ופרופ' מרדכי רוזנר
עיצוב והבאה לדפוס: יעקב אלבו, הדסה עין-כרם.



העמותה לחקר בריאות העין
ומניעת עיוורון בישראל (ע"ר)



העמותה לחקר בריאות העין
ומניעת עיוורון בישראל (ע"ר)

לראות העמותה לחקר בריאות העין ומניעת עיוורון בישראל

מטרות "לראות"

- * מציאת מזור למחלות עיניים הגורמות לעיוורון באמצעות הגברת המאמץ המחקרי בישראל.
- * העלאת מודעות המוסדות הממשלתיים לחשיבות המכרעת בקידום מחקרים בתחום בריאות העין, כולל מחקרים במחלות "יתומות".
- * העלאת המודעות הציבורית לחשיבות הטיפול המונע.

המועצה המדעית של "לראות"

מרכזת פרויקטים מחקריים הקיימים בישראל בתחום רפואת העיניים, בוחנת ומתקצבת אותם במסגרת המשאבים העומדים לרשותה על פי סדר עדיפויות מוגדר. המועצה פועלת לגיוס מיטב החוקרים מתחומים רלוונטיים וכן להקמת רשת מחקרית בינלאומית. בין חברי המועצה המדעית גורמים בכירים מתחומי הבריאות, האקדמיה והתעשייה. זהו שילוב ייחודי של מומחים הכולל: רופאים ומדענים בתחומי המחקר ורפואת העיניים, ראשי מחלקות למחקר ופיתוח בחברות פרמצבטיות מובילות, בכירים בסקטור העסקי בעלי ראיה כלכלית ומדעית.

כיצד "לראות" פועלת?

העמותה מממשת את מטרותיה באמצעות תרומות מגופים ממשלתיים, מחברות עסקיות ותאגידים, מתורמים פרטיים ומקרנות בישראל ובח"ל.

בין יוזמות עמותת "לראות"

תכנית פעילה למימון מחקרים, סמינר הרצאות בשידור חי, אתר מידע פעיל כולל פורומים של רופאים בכירים ובחירת משרד הבריאות בעמותה כגוף מייעץ בתחום תרופות וטכנולוגיות חדשות.

חברי ההנהלה: אוהד לזב י"ר, פרופ' לבנשטיין ענת, ע"ד נינר אריה, פרופ' ברזילי ארי, ד"ר אביר אריה, אריה יוסי, פרופ' ויינברג דב, פרופ' מרין שאול, פרופ' מרינוב אורי, פרופ' קנת רון, ד"ר שטרק יפית, שביב פרדי, שפיר יאיר, פרופ' אלי חזום, מאיר שטרן, מרק עמוס, ד"ר רונית לוינר, פרופ' חנא גוזרזי, י"ר ועדה מדעית: פרופ' ארי ברזילי

טל. 03-7447710, פקס. 15337447710, רח' אליהו חכים 8, תל אביב 69120, www.eyes.org.il

לזכרו של פרופ' רמי רחמימוב ז"ל

המדען הראשי של משרד הבריאות

דברי אוהד להב – יו"ר עמותת לראות:

חלפו מספר חודשים מאז הלך מאיתנו פרופ' רמי רחמימוב, אך רוחו עדיין שורה עלינו, ועל המפעל שאנו עמלים עליו יום יום.

פרופ' רחמימוב, רמי בשם כולנו, היה איש מדע דגול, חתן פרס ישראל, מומחה בחקר המוח, ומדען בעל חזון ואמונה שלא היסס להלחם באומץ על המחקר המדעי, גם אם כנגד כל מקבלי ההחלטות.

מעל כל אלה היה פרופ' רחמימוב אדם יקר, פתוח וקשוב לסביבתו האנושית, שליווה אותנו, בלב חם ושכל טוב, מיום הקמת העמותה ועד יומו האחרון.

רמי היקר, כולנו חסרים מאוד את חכמתך, את נועם הליכותיך, ואת הדרך שהתווית לנו, ובה אנו צועדים עד היום.

היה שלום חבר יקר,

מערכת הבריאות בישראל, חברי העמותה ואנוכי חסרים אותך עד מאוד.

פרופ' רמי רחמימוב ז"ל סייע בהקמת עמותת לראות ותמך בפעילותה עד יום מותו.

מדברי פרופ' רמי רחמימוב ז"ל:

"המחקר הרפואי הנו בעל חשיבות גדולה מאוד מבחינה חברתית, רפואית וכלכלית.

במדינת ישראל יש כוח אדם מעולה שיכול לקדם רבות את המחקר הרפואי, אך לדאבון לב כולנו, המשאבים הכספיים העומדים לרשות המחקר הרפואי מצומצמים ביותר. לכן אני רואה בברכה רבה את פעילותה של עמותת לראות - לחקר בריאות העין ומניעת עיוורון בישראל, אשר לקחה על עצמה לקדם את המחקר בנושא העיוורון במדינת ישראל ע"י גיוס משאבים, ארגון פעילויות רקע חשובות, ויצירת קשר בין החוקרים במוסדות השונים במדינה.

המשאבים שכבר גויסו למטרה זו תורמים רבות לחקר נושא שיש לו חשיבות עצומה למדינה, הן מבחינת הפתרון הרפואי והחברתי, והן מבחינת הקלת הנטל הכלכלי.

אין ספק שפעילות עמותת לראות - לחקר בריאות העין ומניעת עיוורון בישראל עשויה להביא מזר למאות אלפי חולים במדינת ישראל ומעבר לים, ולקדם את הנושא מבחינה מדעית, רפואית וכלכלית".

יושבי-ראש של האגודה הישראלית לחקר העין והראיה

CHAIRMEN OF THE ISRAEL SOCIETY FOR VISION AND EYE RESEARCH

Prof. Elaine Berman	1979-1982	פרופ' איליין ברמן ז"ל
Prof. Michael Belkin	1983-1985	פרופ' מיכאל בלקין
Prof. Saul Merin	1986-1989	פרופ' שאול מרין
Prof. Shabtay Dikstein	1990-1993	פרופ' שבתאי דיקשטיין
Prof. Fabian Abraham	1994-1996	פרופ' פביאן אברהם ז"ל
Prof. Ido Perlman	1997-1999	פרופ' אידו פרלמן
Prof. Jacob Pe'er	2000-2003	פרופ' יעקב פאר
Prof. Ahuva Dovrat	2004-2006	פרופ' אהובה דברת
Prof. Mordechai Rosner	2007- 2009	פרופ' מרדכי רוזנר

חברי ועד האגודה הישראלית לחקר העין והראיה

**BOARD MEMBERS OF THE ISRAEL SOCIETY FOR
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Prof. Mordechai Rosner	Chairman	יו"ר	פרופ' מרדכי רוזנר
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Prof. Eytan Blumenthal			פרופ' איתן בלומנטל
Prof. Ron Ofri			פרופ' רון עופרי
Dr. Dror Sharon			ד"ר דרור שרון

מרצים זוכים המקבלים פרס על עבודות שהוצגו בכינוס השנתי ה-28, מרץ 2008

RECIPIENTS OF AWARDS FOR THE BEST POSTERS AND TALKS PRESENTED AT THE 28TH MEETING, MARCH 2008

1. אנני רביבו סבח – הטכניון, חיפה
ANNIE REBIBO SABBAAH - TECHNION, HAIFA
Evaluating suppression of nonsense mutations by aminoglycoside antibiotics as an intervention for vision loss in type I usher syndrome
2. רוסלנה אלפר – האוניברסיטה העברית בירושלים
RUSLANA ALPER - HEBREW UNIVERSITY, JERUSALEM
RPE derived from human embryonic stem cells provides functional and structural rescue in the rcs rat model of retinal degeneration
3. לימור אשכנזי – האוניברסיטה העברית בירושלים
LIMOR ASHKENAZY - HEBREW UNIVERSITY, JERUSALEM
Infantile exotropia and long-term neurological outcome
4. ניר ארדינסט – האוניברסיטה העברית בירושלים
NIR ERDINEST - HEBREW UNIVERSITY JERUSALEM
Utilizing functional magnetic resonance imaging to evaluate the foveal function in response to a flash stimulus

הפרסים בחסות:

”לראות” - העמותה למחקר בריאות העין ומניעת עיוורון בישראל



העמותה לחקר בריאות העין
ומניעת עיוורון בישראל (ע"ר)

תודה לחברות שתרמו לכינוס:

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Kivema

**ISRAEL SOCIETY FOR VISION AND EYE RESEARCH
29th ANNUAL MEETING
NEVE ILAN RESORT
PROGRAM AT A GLANCE**

Thursday, March 12, 2009

Registration and Coffee	Exhibition Hall	08:00 – 08:30
Opening Remarks	“SHARON” Hall	08:30 – 08:35
I Poster Session 1	“SHARON” Hall	08:35 – 09:30
II Cornea	“SHARON” Hall	09:30 – 10:30
Poster viewing and Coffee	Exhibition Halls	10:30 – 11:00
III Poster Session 2	“SHARON” Hall	11:00 – 12:00
IV 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 – 15:15
V Retina I	“SHARON” Hall	15:15 – 16:30
Poster viewing and Coffee	Exhibition Halls	16:30 – 17:30
VI Retina II	“SHARON” Hall	17:30 – 18:30
Dinner with Guest (optional)		19:30

Friday, March 13, 2009

Poster viewing and Coffee	Exhibition Hall	08:00 – 08:30
VII Cataract	“SHARON” Hall	08:30 – 09:20
VIII Glaucoma	“SHARON” Hall	09:20 – 10:30
Guest Lecture 2	“SHARON” Hall	10:30 – 11:00
Poster viewing and Coffee	Exhibition Halls	11:00 – 11:30
IX Oncology	“SHARON” Hall	11:30 – 12:20
X Free Papers	“SHARON” Hall	12:20 – 13:20
Concluding Remarks	“SHARON” Hall	13:20 – 13:30

PROGRAM

Thursday, March 12, 2009

Registration 08:00 – 08:30

Opening Remarks 08:30 – 08:35

Prof. Mordechai Rosner

Session I – Poster presentations 1 08:35 – 09:30

Moderators: Prof. Ron Ofri and Dr. Dror Sharon

1. **LONG -TERM FOLLOW-UP OF FUCHS' HETEROCHROMIC IRIDOCYCLITIS PATIENTS**
(1) * RASKIN EYAL (1) KAISERMAN IGOR (2) AMER RADGONDE
(1) BARZILAI MEDICAL CENTER, ASHKELON (2) HADASSAH MEDICAL CENTER, EIN KEREM, JERUSALEM
2. **PENTACAM VS PACHPEN IN MEASURING CORNEAL THICKNESS**
(1) * BAIDOUSI AMJAD (1) TESSLER TZVI (1) AMTIRAT AHED (1) LEVY JAIME (1) LIFSHITZ TOVA
(1) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY MEDICAL CENTER, BEER-SHEVA
3. **THE EFFECT OF MITOMYCIN C ON CORNEAL ENDOTHELIUM IN PTERYGIUM SURGERY**
(1) * BAHAR IRIT (1) KAISERMAN IGOR (1) LEVINGER ELIYA (1) SANSANAYUDH WIWAN (1) SLOMOVIC ALLAN
(1) OPHTHALMOLOGY DEPARTMENT, TORONTO WESTERN HOSPITAL, TORONTO, CANADA
4. **PROGNOSTIC FACTORS IN ACANTHAMOEBA KERATITIS**
(1) * KAISERMAN IGOR (2) BAHAR IRIT (3) MCALLUM PENNY (3) SLOMOVIC ALLAN (3) ROOTMAN DAVID
(1) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON (2) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETAH-TIQVA (3) DEPARTMENT OF OPHTHALMOLOGY, TORONTO WESTERN HOSPITAL, UNIVERSITY OF TORONTO, TORONTO, ONTARIO, CANADA
5. **SAFETY OF CORNEAL COLLAGEN CROSS-LINKING WITH ULTRAVIOLET-A AND RIBOFLAVIN IN PROGRESSIVE**

KERATOCONUS

(1) * GOLDICH YAKOV (2) MARKOVICH ARIE (3) HIRSH AMI (1) AVNI ISAAC (1) ZADOK DAVID

(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER (2) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER (3) ENAIM REFRACTIVE SURGERY CENTERS

6. **COMPLIANCE TO EYE CARE IN GLAUCOMA PATIENTS WITH COMORBID DEPRESSION**

(1) WEISS GUY (1) BURGANSKI-ELIASH ZVIA (1) * BARTOV ELISHA

(1) DEPARTMENT OF OPHTHALMOLOGY, EDITH WOLFSON MEDICAL CENTER

7. **NON-COMPLIANCE WITH OCULAR HYPERTENSIVE TREATMENT IN PATIENTS WITH PRIMARY OPEN ANGLE GLAUCOMA AMONG ARAB POPULATION IN ISRAEL- A CROSS SECTIONAL DESCRIPTIVE STUDY**

(1) * MASOUD MUHANNAD (1) IBRAHIM ALI (1) SIMAN-TOV SHLOMI

(1) PIKKEL JOSEPH

(1) DEPARTMENT OF OPHTHALMOLOGY, SIEFF GOVERNMENTAL HOSPITAL, SAFED

8. **MEASUREMENT OF INTRAOCULAR PRESSURE IN KERATOCONIC EYES USING THE OCULAR RESPONSE ANALYZER AND GOLDMANN APPLANATION TONOMETRY**

(1) * GOLDICH YAKOV (1) BARKANA YANIV (1) AVNI ISAAC (1) ZADOK DAVID

(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN

9. **HIGH GLUCOSE (DIABETIC) DAMAGE TO INTACT BOVINE LENSES IN CULTURE**

(1) * BORMUSOV ELVIRA (1) ELIAZ-VOLKOVICH ANAT (2) DOVRAT Yael (1) DOVRAT AHUVA

(1) RAPPAPORT FACULTY OF MEDICINE, TECHNION – ISRAEL INSTITUTE OF TECHNOLOGY, HAIFA (2) KORET SCHOOL OF VETERINARY MEDICINE, THE HEBREW UNIVERSITY OF JERUSALEM

10. **INTRAVENOUS SEDATION VERSUS PLACEBO IN CLEAR CORNEAL PHACOEMULSIFICATION UNDER TOPICAL ANESTHESIA**

(1) * ZALISH MIRIAM (1) MAHLER ORI (2) DUKHAN ALEXANDER (1) YOVEL OREN (1) YEHOSHUA ZOHAR (1) MARCOVICH ARIE

(1) EYE DEPARTMENT, KAPLAN MEDICAL CENTER, REHOVOT, AFFILIATED TO THE HEBREW UNIVERSITY AND HADASSAH MEDICAL SCHOOL JERUSALEM (2) ANESTHESIOLOGY DEPARTMENT, KAPLAN MEDICAL CENTER, REHOVOT, AFFILIATED TO THE HEBREW UNIVERSITY AND HADASSAH MEDICAL SCHOOL JERUSALEM

11. **REDUCING APOPTOSIS IN THE ISCHEMIC RETINA USING HYPERBARIC OXYGEN TREATMENT IN TWO DIFFERENT MOUSE MODELS**
 (1) * GAYDAR VERA (2) EZRACHI DAVID (2) HOFSTETTER SHIR (2) AVRAHAM-LUBIN BAT CHEN R. (2) DRATVIMAN-STOROBINSKY OLGA (2) GOLDENBERG-COHEN NITZA
 (1) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER (2) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, TEL AVIV UNIVERSITY

12. **BAX ABLATION PROTECTS AGAINST RETINAL ISCHEMIA/REPERFUSION INJURY IN TRANSGENIC MICE**
 (1) * DRATVIMAN-STOROBINSKY OLGA (1) DADON-BAR EL SHIMRIT (1) AVRAHAM-LUBIN BAT CHEN R. (2) HOCHHAUSER EDITH (1) GOLDENBERG-COHEN NITZA
 (1) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, RABIN CAMPUS, TEL AVIV UNIVERSITY (2) 3LABORATORY OF CARDIAC RESEARCH, FMRC, RABIN CAMPUS, TEL AVIV UNIVERSITY

13. **TRANSPLANTATION OF RPE-LIKE CELLS DERIVED FROM HUMAN EMBRYONIC STEM CELLS IN DYSTROPHIC RCS RAT EYES: LONG TERM RESULTS AND COMPARISON WITH CELLULAR CONTROLS**
 (1) * ALPER RUSLANA (1) OBOLENSKY ALEXEY (2) IDELSON MARIA (1) HEMO ITSHAK (1) YEOL RUTH (1) PIONTEK ELENA (1) BARZEL ISRAEL (1) SHULMAN MARINA (2) REUBINOFF BENJAMIN (1) BANIN EYAL
 (1) CENTER FOR RETINAL AND MACULAR DEGENERATIONS, DEPARTMENT OF OPHTHALMOLOGY, AND (2) CENTER FOR HUMAN EMBRYONIC STEM CELLS, GENE THERAPY INSTITUTE, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER

14. **NEOVASCULARISATION IN AN APOLIPOPROTEIN E4 TRANSGENIC MOUSE MODEL OF OXYGEN-INDUCED RETINOPATHY**
 (1) * MAHARSHAK IDIT (2) SOLOMON ARIEH (2) ROSNER MORDECHAI (3) WEINBERGER DOV (1) MICHAELSON DANIEL
 (1) DEPT OF NEUROBIOLOGY, TEL AVIV UNIVERSITY (2) DEPT OF OPHTHALMOLOGY, TEL HASHOMER MEDICAL CENTER (3) DEPT OF OPHTHALMOLOGY, THE RABIN MEDICAL CENTER

15. **AAV MEDIATED LEDGF EXPRESSION RESCUES PHOTORECEPTORS IN THE RCS MODEL OF RETINAL DEGENERATION: PROBING THE MECHANISM**
 (1) * RAZ-PRAG DORIT (2) ZENG YONG (2) SIEVING PAUL A. (2) BUSH RON A.
 (1) DEPT. OF NEUROBIOLOGY, GEORGE S. WISE FACULTY OF LIFE SCIENCES, TEL-AVIV UNIVERSITY (2) SECTION FOR TRANSLATIONAL RESEARCH IN RETINAL AND MACULAR DEGENERATION, NIDCD, NIH

16. **CONE DISTRIBUTION AND PATTERN OF CONE LOSS IN THE RD10 MOUSE MODEL OF RETINAL DEGENERATION**
 (1) * GREENWALD YOEL (2) OBOLENSKY ALEXEY (2) BANIN EYAL
 (1) OPHTHALMOLOGY, KAPLAN AND HADASSAH-HEBREW UNIVERSITY MEDICAL CENTERS (2) OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER
17. **FURTHER CHARACTERIZATION OF RETINAL ALTERATIONS IN THE CHEMOKINE RECEPTOR TYPE 2 (CCR2) DEFICIENT MICE**
 (1) * HOROWITZ SMADAR (2) FELDMESSER ESTER (1) OBOLENSKY ALEX (3) SENNLAUB FLORIAN (1) BANIN EYAL (4) ZACK DONALD (1) CHOWERS ITAY
 (1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH - HEBREW UNIVERSITY MEDICAL CENTER (2) WEIZMANN INSTITUTE OF SCIENCE (3) INSTITUT NATIONAL DE LA SANTÉ; ET DE LA RECHERCHE MÉDICALE U872 (4) WILMER EYE INSTITUTE, JOHNS HOPKINS UNIVERSITY SCHOOL OF MEDICINE
18. **CORRELATION BETWEEN PRESSURE MEASUREMENTS IN FLUID FILLED AND GAS INJECTED EYES**
 (1) * HAMMEL NA'AMA (1) LIVNAT TAMI (1) WEINBERGER DOV
 (1) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER; SACKLER SCHOOL OF MEDICINE, TEL-AVIV UNIVERSITY
19. **ANTI-VEGF INTRAVITREAL INJECTIONS IN PATIENTS UNDER CONCURRENT ORAL ANTICOAGULATION OR ANTIPLATELET THERAPY**
 (1) * SEGAL ORI (1) FERENCZ JOSEPH
 (1) MEIR MEDICAL CENTER
20. **ASSOCIATION BETWEEN VITAMIN D AND AGE-RELATED MACULAR DEGENERATION IN MACCABI HEALTHCARE MEMBERS**
 (1) * GOLAN SHANI (2) SHALEV VARDA (2) TREISTER GIORA (1) LOEWENSTEIN ANAT
 (1) TEL-AVIV MEDICAL CENTER (2) MACCABI HEALTHCARE SERVICES
21. **ASTIGMATISM IN CHILDREN - LONG TERM FOLLOW UP**
 (1) * SOCEA SERGIU (1) BREGER YOAV (2) SHAULI YAACOV (3) MEUSHAR AVIVA (1) MEZER EEDY (1) MILLER BENJAMIN (1) MEYER EWY
 (1) ALBERTO MOSCONA DEPARTMENT OF OPHTHALMOLOGY RAMBAM HEALTH CARE CAMPUS (2) MACCABI HEALTH CARE SERVICES (3) CLALIT HEALTH CARE SERVICES
22. **PERIOcular STEROID INJECTION FOR THE MANAGEMENT OF UVEITIS IN CHILDREN**
 (1) * HABOT-WILNER ZOHAR (1) SALLAM AHMED (2) ROUFAS

ATHENA (1) KABASELE PAUL (3) GRIGG JOHN R. (2) MCCLUSKEY PETER (1) LIGHTMAN SUE
(1) DEPARTMENT OF CLINICAL OPHTHALMOLOGY, MOORFIELDS EYE HOSPITAL, LONDON, UNITED KINGDOM (2) DEPARTMENT OF OPHTHALMOLOGY, LIVERPOOL HOSPITAL, SYDNEY, AUSTRALIA (3) SAVE SIGHT INSTITUTE, SYDNEY EYE HOSPITAL, SYDNEY, AUSTRALIA

Session II – Cornea

09:30 – 10:30

Moderators: Prof. Abraham Solomon and Dr. Tamar Kadar

- 09:30-09:40 **THE EFFECT OF TOPICAL TREATMENT WITH THE MATRIX METALLOPROTEINASE INHIBITOR, DOXYCYCLINE, ON OCULAR CHEMICAL INJURY IN RABBITS**
(1) * HORWITZ VERED (1) DACHIR SHLOMIT (1) COHEN LIAT (1) SHALEM YOAV (1) GUTMAN HILA (1) COHEN MAAYAN (1) FISHBINE ELIEZER (1) BRANDEIS RACHEL (1) TURETZ JOSEPH (1) KADAR TAMAR (1) AMIR ADINA
(1) DEPARTMENT OF PHARMACOLOGY, ISRAEL INSTITUTE FOR BIOLOGICAL RESEARCH, NESS ZIONA
- 09:40-09:50 **THE AMINO ACID DERIVATIVE DL-TRIFLUOROLEUCINE ACTS AS A CHEMOREPELLENT TO PSEUDOMONAS AERUGINOSA.**
(1) * KRUGER JOSHUA (2) BLOCK COLIN (1) MAFTSIR GENIA (1) HABIL IYAD (1) SOLOMON ABRAHAM
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER (2) DEPARTMENT OF CLINICAL MICROBIOLOGY AND INFECTIOUS DISEASES, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER
- 09:50-10:00 **ANTI-INFLAMMATORY AND ANTI-VEGF THERAPY FOR NEOVASCULARIZATION ASSOCIATED WITH LIMBAL STEM CELL DEFICIENCY IN RABBITS**
(1) * KADAR TAMAR (1) DACHIR SHLOMIT (1) COHEN LIAT (1) TURETZ JOSEPH (1) FISHBINE ELIEZER (1) SAHAR RITA (1) GUTMAN HILA (1) COHEN MAAYAN (1) SHALEM YOAV (1) HORWITZ VERED (1) AMIR ADINA
(1) DEPARTMENT OF PHARMACOLOGY, ISRAEL INSTITUTE FOR BIOLOGICAL RESEARCH, NESS-ZIONA
- 10:00-10:10 **TOPICAL TREATMENT WITH 1% CYCLOSPORINE FOR CHRONIC ADENOVIRAL KERATITIS**
(1) * LEVINGER ELIYA (2) SLOMOVIC ALANA (2) SANSANAYUDH WIWAN (2) SLOMOVIC ALLAN
(1) DEPARTMENT OF OPHTHALMOLOGY, SOURASKY MEDICAL CENTER, TEL AVIV (2) DEPARTMENT OF OPHTHALMOLOGY, TORONTO WESTERN HOSPITAL,

DONALD K JOHNSON EYE CENTER, UNIVERSITY HEALTH NETWORK, UNIVERSITY OF TORONTO, ONTARIO, CANADA

- 10:10-10:20 **BUSIN GUIDE VERSUS FORCEPS FOR THE INSERTION OF THE DONOR LENTICULE IN DESCEMET STRIPPING AUTOMATED ENDOTHELIAL KERATOPLASTY**
(1) * BAHAR IRIT (1) KAISERMAN IGOR (1) SANSANAYUDH WIWAN (1) LEVINGER ELIYA (1) ROOTMAN DAVID
(1) DEPARTMENT OF OPHTHALMOLOGY, TORONTO WESTERN HOSPITAL
- 10:20-10:30 **PROSPECTIVE EVALUATION OF QUALITY OF CARE AND PATIENT SATISFACTION IN LASIK SURGERY**
(1) * NASSER ORWA (2) BREZIS MAYER (3) FRUCH-PERY JOSEPH (3) SOLOMON AVI
(1) HEBREW UNIVERSITY OF JERUSALEM, FACULTY OF MEDICINE, HADASSAH UNIVERSITY HOSPITAL. JERUSALEM
(2) HADASSAH CENTER FOR CLINICAL QUALITY AND SAFETY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM
(3) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH UNIVERSITY HOSPITAL, JERUSALEM

Poster viewing and coffee (Exhibition Halls) 10:30 – 11:00

Session III – Poster presentations 2 11:00 – 12:00

Moderators: Prof. Avraham Spierer and Prof. Eitan Blumenthal

23. **EFFECT OF DISINSERTION OF RECTUS EYE MUSCLES ON AQUEOUS HUMOR COMPOSITION IN RABBITS**
(1) * BEN SIMON GUY (1) SPIERER ABRAHAM
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, TEL HASHOMER
24. **GENE EXPRESSION PATTERNS OF PERIPHERAL BLOOD CELLS SUBPOPULATIONS IN ACUTE OPTIC NEURITIS: THE ROLE OF CD19+ B CELLS**
(1) * FELDMAN ANNA (2) GUREVICH MICHAEL (3) HUNA-BARON RUTH (4) ACHIRON ANAT
(1) MULTIPLE SCLEROSIS CENTER, SHEBA MEDICAL CENTER (2) GOLDSHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER (3) SACKLER MEDICAL SCHOOL, TEL AVIV UNIVERSITY (4) SACKLER MEDICAL SCHOOL, TEL AVIV UNIVERSITY
25. **INCREASED SCOTOPIC AND PHOTOPIC A-WAVE AMPLITUDE ELECTRORETINOGRAM RESPONSES AFTER INTRAVITREAL BEVACIZUMAB INJECTION IN PATIENTS WITH NEOVASCULAR**

AGE-RELATED MACULAR DEGENERATION

(1) * ROTENSTRIECH YGAL (1) SKAAT ALON (1) MOROZ IRIS (1) DAI VIKI (1) SOLOMON ARIE

(1) GOLDSCHLAGER EYE INSTITUTE, SHEBA MEDICAL CENTER

26. **EVALUATING SUPPRESSION OF NONSENSE MUTATIONS BY AMINOGLYCOSIDE ANTIBIOTICS AS AN INTERVENTION FOR VISION LOSS IN TYPE I USHER SYNDROME**
(1) * REBIBO SABBAH ANNIE (2) NUDELMAN IGOR (2) BAASOV TIMOR (1) BEN-YOSEF TAMAR
(1) DEPARTMENT OF GENETICS, THE RAPPAPORT FAMILY INSTITUTE FOR RESEARCH IN THE MEDICAL SCIENCES, FACULTY OF MEDICINE, TECHNION-ISRAEL INSTITUTE OF TECHNOLOGY, HAIFA (2) DEPARTMENT OF CHEMISTRY, INSTITUTE OF CATALYSIS SCIENCE AND TECHNOLOGY, TECHNION, HAIFA
27. **POLYMORPHISM IN THE WRN GENE IN PATIENTS WITH SENILE CATARACT**
(1) * EHRENBERG MIRIAM (2) DRATVIMAN-STOROBINSKY OLGA (2) AVRAHAM-LUBIN BAT-CHEN R. (2) GOLDENBERG-COHEN NITZA (1) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER (2) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, RABIN CAMPUS, TEL AVIV UNIVERSITY
28. **MOLECULAR CHARACTERIZATION OF CERKL: A GENE UNDERLYING AUTOSOMAL RECESSIVE SEVERE RETINAL DEGENERATION WITH EARLY MACULAR INVOLVEMENT.**
(1) * VEKSLIN SHARON (1) NEVET JUDITH (1) AUSLENDER NOA (1) BEN YOSEF TAMAR
(1) DEPARTMENT OF GENETICS AND THE RAPPAPORT FAMILY INSTITUTE FOR RESEARCH IN THE MEDICAL SCIENCES, FACULTY OF MEDICINE, TECHNION, HAIFA
29. **LAMIN A IS NOT ASSOCIATED WITH AGE-RELATED NUCLEAR CATARACT**
(1) * SADIKOV TAMILLA (2) COHEN YORAM (1) AVRAHAM-LUBIN BAT CHEN R. (2) SIMON AMOS (1) GOLDENBERG-COHEN NITZA (1) THE KRIEGER EYE RESEARCH LABORATORY, FMRC, RABIN CAMPUS, TEL AVIV UNIVERSITY (2) SHEBA CANCER RESEARCH CENTER, SHEBA MEDICAL CENTER, TEL HASHOMER
30. **FINE MAPPING FOR THE POSTERIOR POLAR CATARACT GENE IN MOROCCAN JEWS**
(1) * PRAS ERAN (2) ABU ALMOGIT (1) AVNI ISAAC (2) PRAS ELON (2) NETZER IRIS (2) WOLF-REZNIK HAIKE
(1) THE DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN (2) THE DANEK GARTNER INSTITUTE OF HUMAN GENETICS, SHEBA MEDICAL CENTER, TEL HASHOMER.
31. **C3 POLYMORPHISM IS ASSOCIATED WITH INCREASED RISK FOR**

AGE RELATED MACULAR DEGENERATION IN THE ISRAELI POPULATION

(1) * LEDERMAN MICHAL (1) CHOWERS ITAY
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH - HEBREW UNIVERSITY MEDICAL CENTER

32. **GENETIC ANALYSIS OF FAMILIAL KERATOCONUS IN THE JEWISH POPULATION OF THE NEGEV**
(1) PETROV ALENA (2) GRADSTEIN LIBE (2) LIFSHITZ TOVA (2) BIRK OHAD (2) OFIR RIVKA
(1) DEPARTMENT OF OPHTHALMOLOGY SOROKA UNIVERSITY MEDICAL CENTER, BEER-SHEVA (2) GENETICS INSTITUTE SOROKA UNIVERSITY MEDICAL CENTER, BEER-SHEVA
33. **THE ROLE OF ABCA4 IN HEREDITARY RETINAL DISEASES IN THE ISRAELI POPULATION**
(1) * BIDA LINA (1) BEIT-YAAKOV ANAT (1) HUDARA SHMUEL (1) HASHOUL JRARD (1) BANIN EYAL (1) SHARON DROR
(1) HADASSAH HEBREW UNIVERSITY MEDICAL CENTER
34. **AN ANCIENT FOUNDER SPLICE-SITE RPE65 MUTATION IN NORTH-AFRICAN JEWISH PATIENTS WITH LEBER CONGENITAL AMAUROSIS**
(1) * BANDAH DIKLA (2) STROM TIM (1) BANIN EYAL (1) SHARON DROR
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) INSTITUTE OF HUMAN GENETICS, NEUHERBERG, GERMANY
35. **PREVALENCE OF CHRONIC DISEASES AMONG PATIENTS UNDERGOING CATARACT SURGERY**
(1) * KAISERMAN IGOR (2) NEMET ARIE (3) VINKER SHLOMO (4) LEVARTOVSKY SHMUEL
(1) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON, FACULTY OF HEALTH SCIENCES, BEN-GURION UNIVERSITY, BEER-SHEVA (2) DEPARTMENT OF OPHTHALMOLOGY, SHAARE ZEDEQ MEDICAL CENTER, JERUSALEM (3) DEPARTMENT OF FAMILY MEDICINE, CLALIT HEALTH SERVICES, CENTRAL DISTRICT, REHOVOT (4) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON, FACULTY OF HEALTH SCIENCES, BEN-GURION UNIVERSITY, BEER-SHEVA
36. **PROGNOSTIC FACTORS IN POSTERIOR OPEN GLOBE INJURIES (ZONE-III INJURIES).**
(1) * KNAIZER BORIS (1) LEVY JAIME (2) ROSEN SHIRLEY (1) BELFAIR NADAV (1) KLEMPERER ITAMAR (1) LIFSHITZ TOVA
(1) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY MEDICAL CENTER, BEN-GURION UNIVERSITY OF THE NEGEV, BEER-SHEVA (2) DEPARTMENT OF EPIDEMIOLOGY, FACULTY OF HEALTH SCIENCES, BEN-GURION UNIVERSITY OF THE NEGEV, BEER-SHEVA

37. **SEMA-3A DISRUPTS THE REGENERATION PROCESS OF GOLDFISH AND RAT OPTIC NERVES FOLLOWING CONTROLLED INJURY**
 (1) ROSENZWEIG SHIRA (1) * RAZ-PRAG DORIT (1) NITZAN ANAT (1) PAZ MA'AYAN (2) JESERICH GUNNAR (3) NEUFELD GERA (1) BARZILAI ARI (4) SOLOMON ARIEH S.
 (1) DEPARTMENT OF NEUROBIOLOGY, GEORGE S. WISE FACULTY OF LIFE SCIENCES, TEL-AVIV UNIVERSITY (2) DEPARTMENT OF NEUROBIOLOGY, UNIVERSITY OF OSNABR&UUML;CK, 49069 OSNABR&UUML;CK, GERMANY (3) CANCER AND VASCULAR BIOLOGY RESEARCH CENTER, RAPPAPORT RESEARCH INSTITUTE IN THE MEDICAL SCIENCES, THE BRUCE RAPPAPORT FACULTY OF MEDICINE, TECHNION (4) GOLDSCHLEGER EYE RESEARCH INSTITUTE, HAIM SHEBA MEDICAL CENTER, TEL HASHOMER
38. **HOLOGRAPHIC DYNAMIC CONTROL OF NEURONAL POPULATIONS IN THE RETINA**
 (1) * FARAH NAIROUZ (1) REUTSKY INNA (1) GOLAN LIOR (1) SHOHAM SHY
 (1) FACULTY OF BIOMEDICAL ENGINEERING, THE TECHNION – I.I.T., HAIFA
39. **INTRAVITREAL INJECTION OF CILIARY NEUROTROPHIC FACTOR TO ENHANCE IN SITU DIFFERENTIATION OF STEM-CELL-FACTOR-MOBILIZED BONE-MARROW-DERIVED STEM CELLS**
 (1) * AVRAHAM-LUBIN REVITAL BAT CHEN (2) WEINBERGER DOV (1) GOLDENBERG-COHEN NITZA
 (1) THE KRIEGER EYE RESEARCH LABORATORY, FELSENSTEIN MEDICAL RESEARCH CENTER, PETAH TIQVA; SACKLER SCHOOL OF MEDICINE, TEL AVIV UNIVERSITY, TEL AVIV; (2) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER PETAH TIQVA;
40. **EXPRESSION AND LOCALIZATION OF CLAUDINS, TIGHT JUNCTION MEMBRANE PROTEINS, IN THE MOUSE EYE**
 (1) * ELKOUBY NAOR LIRON (1) BEN-YOSEF TAMAR
 (1) DEPARTMENT OF GENETICS, THE RAPPAPORT FAMILY INSTITUTE FOR RESEARCH IN THE MEDICAL SCIENCES, FACULTY OF MEDICINE, TECHNION-ISRAEL INSTITUTE OF TECHNOLOGY, HAIFA
41. **THE EFFECT OF TOPICAL STEROIDS ON BLOOD GLUCOSE PROFILE IN DIABETIC PATIENTS**
 (1) * BAHAR IRIT (2) VINKER SHLOMO (3) KAISERMAN IGOR
 (1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETAH-TIQVA (2) DEPARTMENT OF FAMILY MEDICINE, CLALIT HEALTH SERVICES, CENTRAL DISTRICT, REHOVOT (3) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON, FACULTY OF HEALTH SCIENCES, BEN-GURION

UNIVERSITY, BEER-SHEVA

42. **MODULATION OF VEGF EXPRESSION BY RPE CELLS DEPENDS ON THE MODE OF α V INTEGRIN INHIBITION**
(1) * LIVNAT TAMI (1) DARDIK RIMA (1) WEINBERGER DOV
(1) DEPARTMENT OF OPHTHALMOLOGY, BEILINSON MEDICAL CENTER AND TEL-AVIV UNIVERSITY SCHOOL OF MEDICINE, TEL AVIV
43. **RETINAL NERVE FIBER LAYER THICKNESS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS).**
(1) * AREV-FISHELZON TAGIL (1) SAGIV ODED (1) MATHALONE NURIT (1) WOLFSON JULIA (1) IRMANS ANA (2) PELED RON (1) GEYER ORNA
(1) CARMEL MEDICAL CENTER, HAIFA (2) SLEEP LABORATORY, THE TECHNION, RAPPAPORT FACULTY OF MEDICINE, HAIFA

Session IV - Genetics

12:00-13:00

Moderators: Prof. Eyal Banin and Dr. Tamar Ben-Yosef

- 12:10-12:20 **CONE DYSFUNCTION AND CONGENITAL DAY BLINDNESS IN AWASSI SHEEP IS CAUSED BY A MUTATION IN THE CNGA3 GENE**
(1) * OFRI RON (1) SHAMIR MERAV (2) OBOLENSKY ALEXEY
(2) BANIN EYAL (3) BRENNER ORI (4) REICHER SHAY (4) SEROUSSI EYAL (4) GOOTWINE ELISHA
(1) KORET SCHOOL OF VETERINARY MEDICINE, HEBREW UNIVERSITY OF JERUSALEM, REHOVOT (2) CRMD, DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER (3) WEIZMANN INSTITUTE OF SCIENCE (4) AGRICULTURAL RESEARCH ORGANIZATION, THE VOLCANI CENTER, MINISTRY OF AGRICULTURE
- 12:20-12:30 **GENETIC ANALYSIS OF BEST DISEASE IN THE ISRAELI AND PALESTINIAN POPULATIONS**
(1) MIZRAHI-MEISSONNIER LILIANA (1) BITNER HANNA (1) MERIN SAUL (1) BANIN EYAL (1) * SHARON DROR
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM
- 12:30-12:40 **THE SPECTRUM OF RETINAL DISEASE CAUSED BY NR2E3 MUTATIONS IN ISRAELI AND PALESTINIAN PATIENTS**
(1) * BANIN EYAL (1) BANDAH DIKLA (2) MANZAR ASHAB (1) MERIN SAUL (1) SHARON DROR
(1) OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER (2) OPHTHALMOLOGY, SHAARE-ZEDEK MEDICAL CENTER, JERUSALEM

12:40-12:50 **INTRAFAMILIAL GENETIC HETEROGENEITY IN ISRAELI CONSANGUINEOUS FAMILIES WITH RETINAL DEGENERATION**

(1) BENAYOUN LIAT (2) SPIEGEL RONEN (1) AUSLANDER NOA (1) NEVET JUDITH (1) ABBASI ANNAN H. (1) RIZEL LEAH (2) HUJEIRAT YASIR (3) SALAMA IHSAN (4) GARZOZI HANNA J. (2) ALLON-SHALEV STAVIT (1) * BEN-YOSEF TAMAR

(1) GENETICS DEPARTMENT, RAPPAPORT FACULTY OF MEDICINE, TECHNION, HAIFA (2) GENETICS INSTITUTE, HA'EMEK MEDICAL CENTER, AFULA (3) CLALIT, HEALTH SERVICES, NORTH DISTRICT (4) OPHTHALMOLOGY DEPARTMENT, BNAI ZION MEDICAL CENTER, HAIFA

12:50-13:00 **RELATIVE IMPORTANCE OF MAJOR RISK SINGLE NUCLEOTIDE POLYMORPHISMS (SNPS) FOR THE DEVELOPMENT OF AGE RELATED MACULAR DEGENERATION (AMD) IN ISRAEL**

(1) * CHOWERS ITAY (2) WEINSTEIN ORLY (3) POLLACK AYALA (4) AXER-SIEGEL RUTH (1) LEDERMAN MICHAL (1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY MEDICAL CENTER, BEER SHEVA (3) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER, REHOVOT (4) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETAH TIQVA

13:00-13:10 **GENOTYPE AND ETHNICITY CORRELATIONS IN CONGENITAL GLAUCOMA IN ISRAEL**

(1) * WOLF ALVIT (2) SHOCHT CHEN (2) BERCOVICH DANI (1) LEVINGER ELIA (1) GEYER ORNA (1) OPHTHALMOLOGY DEPARTMENT, CARMEL MEDICAL CENTER, HAIFA (2) THE HUMAN MOLECULAR GENETICS, MIGAL – GALILEE. BIO-TECHNOLOGY CENTER; TEL HAI ACADEMIC COLLE, KIRYAT-SHMONA

Lunch break

13:00 – 14:00

Guest Lecture 1

14:00 – 14:30

Prof. José-Alain Sahel

Institut de la Vision, UMRS-968, INSERM, Paris, France

TITLE: Cone viability factors: molecular and functional studies

Business Meeting

14:30 – 15:00

In memory of Prof. Rami Rahamimov

15:00 – 15:15

Prof. Benjamin Sredni - Chief Scientist, Ministry of Health

Session V – Retina I

15:15 – 16:30

Moderators: Prof. Dov Weinberger and Dr. Adiel Barak

15:15-15:25 **EFFECT OF INTRAVITREAL BEVACIZUMAB (AVASTIN) ON THE GROWING RABBIT EYE**

(1) * SIEGEL RUTH (1) HERSCOVICI ZVI (1) HASANREISOGLU MURAT (1) KREMER ISRAEL (2) BENJAMINI YOAV (3) SNIR MOSHE

(1) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER (2) DEPARTMENT OF STATISTICS, SACKLER FACULTY OF EXACT SCIENCES, TEL AVIV UNIVERSITY (3) PEDIATRIC OPHTHALMOLOGY UNIT, SCHNEIDER CHILDREN'S MEDICAL CENTER OF ISRAEL

15:25-15:35 **EVALUATION OF THE METABOLIC EFFECTS OF INTRAVITREAL KENALOG IN RABBITS**

(1) * BARAK YOREH (2) ZEMEL ESTHER (1) LANG YARON (1) DEPARTMENT OF OPHTHALMOLOGY, HA'EMEK MEDICAL CENTER, AFULA (2) THE RUTH AND BRUCE RAPPAPORT FACULTY OF MEDICINE TECHNION, AND THE RAPPAPORT INSTITUTE, HAIFA

15:35-15:45 **INFLUENCE OF NON-TOXIC DOSES OF BEVACIZUMAB AND RANIBIZUMAB**

(1) BARAK ADIEL (2) * BARZILAI AYA (2) GEORGE JACOB (1) LOWENSEIN ANAT

(1) OPHTHALMOLOGY, TEL AVIV MEDICAL CENTER (2) VASCULAR BIOLOGY LAB, TEL AVIV MEDICAL CENTER

15:45-15:55 **RISK ASSESSMENT AFTER MORE THAN 2500 AVASTIN INJECTIONS**

(1) * SEGAL ORI (1) RUBOVITZ ALEX (1) GILADI GILA (1)
FERENCZ JOSEPH
(1) MEIR MEDICAL CENTER

15:55-16:05 **THE EXPRESSION AND POSSIBLE ROLE OF
ACETYLCHLINESTERASE (ACHE) FOLLOWING PHOTIC-
STRESS IN THE MOUSE RETINA**
(1) * HERTZ RIVI (1) HEINRICH RONIT (1) ZEMEL ESTHER (1)
MANN IRIT (1) PERLMAN IDO
(1) THE RUTH AND BRUCE RAPPAPORT FACULTY OF
MEDICINE TECHNION, AND THE RAPPAPORT INSTITUTE,
HAIFA

16:05-16:15 **PROANGIOGENIC POTENTIAL OF MICROVASCULAR
ENDOTHELIAL CELLS (EC) IS ENHANCED BY
COCULTURE WITH RETINAL PIGMENT EPITHELIAL
(RPE) CELLS**
(1) LIVNAT TAMI (1) DARDIK RIMA (1) * WEINBERGER DOV
(1) DEPARTMENT OF OPHTHALMOLOGY, BEILINSON
MEDICAL CENTER AND TEL-AVIV UNIVERSITY SCHOOL OF
MEDICINE, TEL AVIV

16:15-16:25 **TRAUMATIC BRAIN INJURY INDUCED
NEUROPROTECTION OF RETINAL GANGLION CELLS TO
OPTIC NERVE CRUSH**
(1) * BEN SIMON GUY (2) HOVDA DAVID (2) HARRIS NEIL (2)
GOMEZ-PINILLA FERNANDO (3) GOLDBERG ROBERT
(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL
CENTER, TEL HASHOMER (2) DIVISION OF NEUROSURGERY,
DEPARTMENT OF SURGERY, UNIVERSITY OF CALIFORNIA
AT LOS ANGELES SCHOOL OF MEDICINE, LOS ANGELES,
CALIFORNIA, USA (3) 1. JULES STEIN EYE INSTITUTE AND
DEPARTMENT OF OPHTHALMOLOGY, DAVID GEFFEN
SCHOOL OF MEDICINE AT UCLA, LOS ANGELES, ALIFORNIA,
USA

Poster viewing and coffee (Exhibition Halls) 16:30 – 17:30

Session VI - Retina II

17:30 – 18:30

Moderators: Prof. Anat Lowenstein and Prof. Ruth Siegel

- 17:30-17:40 **VISION IMPROVEMENT IN TREATMENT TRIAL WITH 9-CIS BETA-CAROTENE FOR RETINITIS PIGMENTOSA PATIENTS**
(1) * ROTENSTREICH YGAL (2) SHAISH AVIV (2) HARATS DROR (3) FERMAN-ATTAR GILI (1) BELKIN MICHAEL (1) GOLDSHLAGER EYE INSTITUTE, SHEBA MEDICAL CENTER (2) INSTITUTE OF LIPID AND ATHEROSCLEROSIS RESEARCH, SHEBA MEDICAL CENTER (3) OPHTHALMOLOGY DEPARTMENT, EDITH WOLFSON MEDICAL CENTER
- 17:40-17:50 **TREATMENT OF CONGENITAL STATIONARY NIGHT BLINDNESS WITH AN ALGA CONTAINING HIGH DOSE OF 9-CIS BETA CAROTENE**
(1) * ROTENSTREICH YGAL (2) SHAISH AVIV (2) HARATS DROR (3) PRAS ERAN (1) BELKIN MICHAEL (1) GOLDSCHLAGER EYE INSTITUTE, SHEBA MEDICAL CENTER (2) INSTITUTE OF LIPID AND ATHEROSCLEROSIS RESEARCH, SHEBA MEDICAL CENTER (3) OPHTHALMOLOGY DEPARTMENT, ASSAF HAROFE MEDICAL CENTER
- 17:50-18:00 **COMPARISON BETWEEN THE FORESEE HOME PERIMETER AND THE AMSLER GRID, IN PATIENTS WITH AGE RELATED MACULAR DEGENERATION**
(1) * LOWENSTEIN ANAT (2) FERENCZ J.R (3) YESHURUN ITAMAR (4) LANG YARON (5) LIFSHITZ TOVA (6) KARP JACOB (7) POLLACK AYALA (8) SEIGAL RUTH (1) TEL - AVIV MEDICAL CENTER, TEL - AVIV (2) SAPIR MEDICAL CENTER, KFAR - SABA (3) WOLFSON MEDICAL CENTER, HOLON (4) HAEMEK MEDICAL CENTER, AFULA (5) SOROKA MEDICAL CENTER, BEER - SHEVA (6) MACHON MOR MEDICAL CENTER, RAMAT - GAN (7) KAPLAN MEDICAL CENTER, REHUVOT (8) RABIN MEDICAL CENTER, PETACH - TIKVA
- 18:00-18:10 **CYSTOID FOVEAL OEDEMA IN SYMPTOMATIC INNER LAMELLAR MACULAR HOLES**
(1) * OPHIR AVINOAM (1) FATUM SAMIA (1) OPHTHALMOLOGY, HILLEL-YAFFE MEDICAL CENTER
- 18:10-18:20 **THE RATES OF THROMBOEMBOLIC EVENTS IN MACCABI HEALTHCARE ADULT MACULAR DEGENERATION MEMBERS.**
(1) * GOLAN SHANI (2) SHALEV VARDA (2) TREISTER GIORA (1) GOLDSTEIN MICHAELA (1) LOEWENSTEIN ANAT

(1) TEL AVIV MEDICAL CENTER (2) MACCABI

18:20-18:30 **THE ROLE OF MICROPARTICLES (MPS) IN DIABETIC RETINOPATHY**

(1) * TSIMERMAN GALA (2) BACHAR ANAT (2) MILLER BENJAMIN (1) BRENNER BENJAMIN (1) AHARON ANAT (1) THROMBOSIS AND HEMOSTASIS UNIT, RAMBAM HEALTH CARE CAMPUS, HAIFA (2) DEPARTMENT OF OPHTHALMOLOGY, RAMBAM HEALTH CARE CAMPUS, HAIFA

Dinner with the guest (Optional) 19:30

Friday, March 13, 2009

Poster viewing and Coffee (Exhibition Halls) 08:00 – 08:30

Session VII – Cataract 08:30 – 09:20

Moderators: Prof. Ehud Asia and Dr. Guy Kleinmann

- 08:30-08:40 **THE POTENTIAL OF DESFERRIOXAMINE AND ZINC-DESFERRIOXAMINE FOR REDUCTION OF LENS OXIDATIVE DAMAGE.**
(1) * DOVRAT AHUVA (1) BORMUSOV ELVIRA (1) SCHAAL SHLOMIT (1) BEIRAN ITZCHAK (2) CHEVION MORDECHAI (1) RAPPAPORT FACULTY OF MEDICINE, TECHNION INSTITUTE OF TECHNOLOGY, HAIFA. (2) THE HEBREW UNIVERSITY, HADASSAH MEDICAL SCHOOL, JERUSALEM
- 08:40-08:50 **THE PROTECTIVE EFFECT OF DIFFERENT OPHTHALMIC VISCOELASTIC DEVICES ON CORNEAL ENDOTHELIAL CELLS DURING PHACOEMULSIFICATION IN A RABBIT MODEL – PART I**
(1) * TAL KFIR (2) BEN ELIAHU SHMUEL (2) EZOV NATI (3) MILSHTEIN ASHER (3) KLEINMANN GUY
(1) HADASSAH MEDICAL SCHOOL (2) HARLAN BIOTECH ISRAEL (3) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER
- 08:50-09:00 **THE PROTECTIVE EFFECT OF DIFFERENT OPHTHALMIC VISCOELASTIC DEVICES ON CORNEAL ENDOTHELIAL CELLS DURING PHACOEMULSIFICATION IN A RABBIT MODEL – PART II**
(1) * BEN ELIAHU SHMUEL (2) TAL KFIR (1) EZOV NATI (2) MILSHTEIN ASHER (2) KLEINMANN GUY
(1) HARLAN BIOTECH ISRAEL (2) KAPLAN MEDICAL CENTER, OPHTHALMOLOGY DEPARTMENT
- 09:00-09:10 **CEFUROXIME- IS IT SAFE ENOUGH TO BE USED ON A LARGE SCALE BASIS IN CATARACT SURGERY?**
(1) * SHAHAR JONATHAN (2) ZEMEL ESTHER (1) HEILWEIL GAD (2) PERLMAN IDO (1) LOEWENSTEIN ANAT
(1) OPHTHALMOLOGY DEPARTMENT, TEL AVIV MEDICAL CENTER, SACKLER FACULTY OF MEDICINE, TEL AVIV UNIVERSITY (2) THE RUTH AND BRUCE RAPPAPORT FACULTY OF MEDICINE TECHNION, AND THE RAPPAPORT INSTITUTE, HAIFA

09:10-09:20 **CORNEAL ENDOTHELIAL MORPHOLOGIC FEATURES IN TOXIC ANTERIOR SYNDROME IN AN ISRAELY POPULATION**
(1) * AVISAR RAHAMIM (2) WEINBERGER DOV
(1) RMC, CAMPUS GOLDA, HASHARON HOSPITAL PETAH TIKVA (2) RMC, CAMPUS BELINSON, PETH TIKVA

Session VIII - Glaucoma

09:20-10:20

Moderators: Prof. Orna Geyer and Dr. Hani Levkovitch-Verbin

- 09:20-09:30 **INTERACTIONS BETWEEN TRABECULAR MESHWORK CELLS AND LENS EPITHELIAL CELLS – A POSSIBLE MECHANISM IN INFANTILE APHAKIC GLAUCOMA**
(1) MICHAEL INBAL (2) SHMOISH MICHAEL (3) WALTON DAVID .S. (1) * LEVENBERG SHULAMIT
(1) TECHNION- ISRAEL INSTITUTE OF TECHNOLOGY, FACULTY OF BIO-MEDICAL ENGINEERING, HAIFA (2) TECHNION- ISRAEL INSTITUTE OF TECHNOLOGY, BIOINFORMATICS KNOWLEDGE UNIT, THE LORRY I. LOKEY INTERDISCIPLINARY CENTER FOR LIFE SCIENCES AND ENGINEERING, HAIFA (3) GLAUCOMA SERVICE, MASSACHUSETTS EYE AND EAR INFIRMARY, AND HARVARD MEDICAL SCHOOL, BOSTON, MASSACHUSETTS.
- 09:30-09:40 **THE EFFECT OF AGING ON RGCS SUSCEPTIBILITY TO ELEVATED INTRAOCULAR PRESSURE**
(1) * VANDER SHELLY (1) MELAMED SHLOMO (1) LEVKOVITCH-VERBIN HANI
(1) THE SAM ROTHBERG MOLECULAR BIOLOGY LAB, GOLDSCHLEGER EYE INSTITUTE, TEL-HASHOMER
- 09:40-09:50 **SYSTEMIC TREATMENT WITH RASAGILINE, A SELECTIVE MONOAMINE OXIDASE INHIBITOR, IS NEUROPROTECTIVE IN GLAUCOMATOUS RAT EYES**
(1) * VANDER SHELLY (1) MELAMED SHLOMO (1) LEVKOVITCH-VERBIN HANI
(1) THE SAM ROTHBERG MOLECULAR BIOLOGY LAB, GOLDSCHLEGER EYE INSTITUTE, TEL-HASHOMER
- 09:50-10:00 **TOPICAL GLAUCOMA THERAPY AS A RISK FACTOR FOR NASOLACRIMAL DUCT OBSTRUCTION**
(1) * SEIDER NIR (1) MILLER BENJAMIN (1) BEIRAN ITZCHAK
(1) THE ALBERTO MOSCONA DEPARTMENT OF OPHTHALMOLOGY, RAMBAM MEDICAL CENTER, HAIFA
- 10:00-10:10 **CONJUNCTIVAL WOUND HEALING PROBLEMS RELATED TO AHMED GLAUCOMA VALVE SURGERY**
(1) * GEFFEN NOA (1) TROPE E GRAHAM (1) SMITH MICHAEL
(1) ANRAKU AYAKO (1) ALASBALI TARIQ (1) BUYS M YVONNE

(1) UNIVERSITY OF TORONTO, DEPARTMENT OF
OPHTHALMOLOGY AND VISION SCIENCES, TORONTO
WESTERN HOSPITAL, TORONTO, ONTARIO, CANADA

10:10-10:20 **AUTOLOGOUS PATCH GRAFT IN AQUEOUS GLAUCOMA
SURGERY**

(1) * GOLAN OREN (1) AREV-FISHELZON TAGIL (1) HOD YAIR
(1) SEGEV EITAN (1) GEYER ORNA
(1) CARMEL MEDICAL CENTER, HAIFA

10:20-10:30 **COMPUTER-GUIDED PATTERNED LASER
TRABECULOPLASTY**

(1) * PALANKER DANIEL, (2) TURATI MAURICIO, (2) MORALES
ADOLFO, (2) QUIROZ-MERCADO HUGO, (3) SCHUELE GEORG,
(3) MARCELLINO GEORGE
(1) DEPARTMENT OF OPHTHALMOLOGY, STANFORD
UNIVERSITY, STANFORD, CA (2) ASSOCIATION TO PREVENT
BLINDNESS IN MEXICO (APEC) (3) OPTIMEDICA
CORPORATION, SANTA CLARA, CA.

Guest Lecture 2

10:30 – 11:00

Prof. José-Alain Sahel

Institut de la Vision, UMRS-968, INSERM, Paris, France

**TITLE: Seeing the invisible: High resolution imaging of subcellular
structures in the human retina**

Poster viewing and coffee (Exhibition Halls) 11:00 – 11:30

Session IX – Oncology

11:30 – 12:20

Moderators: Prof. Jacob Pe'er and Dr. Igor Kaiserman

- 11:30-11:40 **TREATMENT OF UVEAL MELANOMA BY IRREVERSIBLE ELECTROPORATION - FINITE ELEMENT MODEL STUDY**
(1) * MANDEL YOSSI (2) PE'ER JACOB (2) FRENKEL SHAHAR
(1) RUBINSKY BORIS
(1) SCHOOL OF COMPUTER SCIENCE & ENGINEERING,
HEBREW UNIVERSITY, JERUSALEM (2) OPHTHALMOLOGY,
HADASSAH HEBREW UNIVERSITY MEDICAL CENTER,
JERUSALEM
- 11:40-11:50 **THE IMPACT OF TREATING PRIMARY POSTERIOR UVEAL MELANOMA ON SERUM BIOMARKER LEVELS**
(1) * BARAK VIVIAN (2) FRENKEL SHAHAR (2) HENDLER
KAREN (1) KALICKMAN INA (2) PE'ER JACOB
(1) ONCOLOGY, HADASSAH-HEBREW UNIVERSITY MEDICAL
CENTER, JERUSALEM (2) OPHTHALMOLOGY, HADASSAH-
HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM
- 11:50-12:00 **RUTHENIUM-106 PLAQUE BRACHYTHERAPY FOR THICK POSTERIOR UVEAL MELANOMAS**
(1) * KAISERMAN NADIA (2) KAISERMAN IGOR (1) HENDLER
KAREN (1) FRENKEL SHAHAR (1) PE'ER JACOB
(1) DEPARTMENT OF OPHTHALMOLOGY, HADASSAH-
HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2)
DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL
CENTER, ASHKELON, ISRAEL
- 12:00-12:10 **THE EFFICACY OF COLOR DOPPLER ULTRASOUND FOR DIAGNOSIS AND FOLLOW-UP OF ORBITAL HEMANGIOMA IN CHILDREN**
(1) * SPIERER ORIEL (2) KESSLER ADA (1) STOLOVITCH
CHAIM (1) LEIBOVITCH IGAL (1) SHALEV BENJAMIN (1)
DOTAN GAD (1) NEUDORFER MEIRA
(1) DEPARTMENT OF OPHTHALMOLOGY, TEL-AVIV
SOURASKY MEDICAL CENTER (2) ULTRASOUND UNIT, TEL-
AVIV SOURASKY MEDICAL CENTER
- 12:10-12:20 **OUR EXPERIENCE WITH ANTERIOR APPROACH AND CRYOEXTRACTION FOR SURGERY OF ORBITAL CAVERNOUS HEMANGIOMA**
(1) * PRIEL AYELET (1) ROSEN NACHUM (1) BEN SIMON GUY
J (1) ROSNER MORDECHAI
(1) THE GOLDSCHLEGER EYE INSTITUTE, SACKLER SCHOOL
OF MEDICINE, TEL-AVIV UNIVERSITY, SHEBA MEDICAL
CENTER, TEL-HASHOMER

Session X – Free Papers

12:20 – 13:20

Moderators: Prof. Gadi Katzir and Dr. Snir Moshe

- 12:20-12:30 **CESSATION OF THE OCULOCEPHALIC REFLEX IN NORMAL HEALTHY BABIES**
(1) * SNIR MOSHE (2) HASANREISOGLU MURAT (1) GOLDENBERG-COHEN NITZA (1) FRILING RONIT (2) WEINBERGER DOV (2) AXER-SIEGEL RUTH
(1) PEDIATRIC OPHTHALMOLOGY UNIT, OPHTHALMOLOGY DEPARTMENT, SCHNEIDER CHILDREN'S MEDICAL CENTER (2) OPHTHALMOLOGY DEPARTMENT, RABIN MEDICAL CENTER
- 12:30-12:40 **MACULAR VOLUME AND RNFL THICKNESS ARE CORRELATED WITH CLINICAL OCULAR EXAMINATION AND DISEASE STATUS IN PATIENTS WITH MULTIPLE SCLEROSIS**
(1) * WOLFSON YULIA (1) HOROWITZ JOSEPHA (2) MILLER ARIEL
(1) OPHTHALMOLOGY, CARMEL MEDICAL CENTER (2) NEUROLOGY, CARMEL MEDICAL CENTER
- 12:40-12:50 **"HIDE AND SEE(K)"- A CHAMELEON'S VISUALLY GUIDED RESPONSE TO THREAT.**
(1) * LUSTIG AVICHAJ (2) KATZIR GADI
(1) DEPT. OF NEUROBIOLOGY & ETHOLOGY, HAIFA UNIVERSITY (2) DEPT. OF NEUROBIOLOGY & ETHOLOGY, HAIFA UNIVERSITY, DEPT. OF SCIENCE EDUCATION, ORANIM, UNIV. OF HAIFA
- 12:50-13:00 **UTILIZING FUNCTIONAL MAGNETIC RESONANCE IMAGING OF THE VISUAL CORTEX TO EVALUATE MACULAR FUNCTION IN RESPONSE TO A FLASH STIMULUS**
(1) * ERDINEST NIR (1) SOLOMON ABRAHAM (1) BANIN EYAL (2) BICK ATIRA (3) GOMORI JOHN MOSHE (4) RAZ NOA (4) LEVIN NETTA
(1) DEPARTMENTS OF OPHTHALMOLOGY, HADASSAH HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (2) DEPARTMENTS OF NEUROBIOLOGY, LIFE SCIENCE INSTITUTE, HEBREW UNIVERSITY JERUSALEM (3) DEPARTMENTS OF RADIOLOGY, HADASSAH HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM (4) DEPARTMENTS OF NEUROLOGY, HADASSAH HEBREW UNIVERSITY MEDICAL CENTER, JERUSALEM
- 13:00-13:10 **IN VITRO SYNERGY OF MOXIFLOXACIN AND CEFUROXIME AGAINST OCULAR CLINICAL ISOLATES**
(1) * BAREQUET IRINA (1) HABOT-WILNER ZOHAR (1) MOISSEIEV JOSEPH (2) KELLER NATHAN

(1) GOLDSCHLEGER EYE INSTITUTE, SHEBA MEDICAL CENTER, SACKLER FACULTY OF MEDICINE, TEL AVIV UNIVERSITY, TEL HASHOMER (2) DEPARTMENT OF CLINICAL MICROBIOLOGY, SHEBA MEDICAL CENTER, SACKLER FACULTY OF MEDICINE, TEL AVIV UNIVERSITY, TEL HASHOMER

13:10-13:20

INTRAVITREAL INJECTIONS OF NEUROTROPHIC FACTORS SECRETING MESENCHYMAL STEM CELLS ARE NEUROPROTECTIVE IN POST OPTIC NERVE TRANSECTED RAT EYES

(1) * LEVKOVITCH-VERBIN HANI (2) SADAN OFER (1) VANDER SHELLY (2) BARHUM YAEL (2) MELAMED ELDAD (2) OFFEN DANIEL (1) MELAMED SHLOMO (1) THE SAM ROTHBERG MOLECULAR BIOLOGY LAB, GOLDSCHLEGER EYE INSTITUTE, TEL-HASHOMER (2) LABORATORY OF NEUROSCIENCES, FELSENSTEIN MEDICAL RESEARCH CENTER, TEL-AVIV UNIVERSITY

Concluding Remarks

13:20 – 13:30

Prof. Mordechai Rosner

ABSTRACTS

Session I – Poster presentations 1

LONG -TERM FOLLOW-UP OF FUCHS' HETEROCHROMIC IRIDOCYCLITIS PATIENTS

(1) * RASKIN EYAL (1) KAISERMAN IGOR (2) AMER RADGONDE
(1) BARZILAI MEDICAL CENTER, ASHKELON (2) HADASSAH
MEDICAL CENTER, EIN KEREM, JERUSALEM

Introduction: Fuchs heterochromic iridocyclitis (FHI) or Fuchs' uveitis syndrome is a chronic anterior segment inflammatory syndrome of unknown etiology, it accounts for 2 to 3% of all uveitis cases and is frequently overlooked. The purpose of this work is to analyze the clinical manifestations and the ocular associations of 19 patients with Fuchs' Heterochromic Iridocyclitis (FHI) who were followed for a mean period of 46.7 ± 62 months.

Patients / Methods: We retrospectively reviewed the medical charts of all patients with FHI who sought treatment in Hadassah Medical Center between 1989 and 2008. Nineteen patients (22 eyes) were identified. Demographic data such as gender and age of presentation, clinical characteristics and ocular associations were collected.

Results: Data of 19 patients were reviewed; eleven were males (57.9%), and mean age at presentation was 26.1 ± 8.9 years (range 9 -46 years). One eye was affected in 16 patients (84.2%). The median interval between the first visit to seek medical care and the diagnosis of FHI was 1.3 years (range 0 -37.7 years). Two patients (10.5%) had coexisting Usher syndrome. The most common presenting symptom was visual deterioration in 40.9% of eyes. The logMAR visual acuity at diagnosis was 0.67 ± 0.79 (20/93) and the worst acuity during follow-up was 0.85 ± 0.84 (20/141). At last visit, mean visual acuity was 0.29 ± 0.45 (20/39). During the course of follow-up, 81.8% of eyes developed cataract and 18.2% developed glaucoma.

Conclusions: FHI in Israeli patients is characterized by an early age of presentation. Patients sought medical help mainly due to visual deterioration. Coexistence of usher syndrome and FHI was seen in 2 patients. Previous studies suggested abnormal immunity to rubella virus (RV) in retinitis pigmentosa patients where elevated serum ELISA IgG antibodies to RV were found in these patients. Recent publications have provided evidence for the possible involvement of RV in the pathogenesis of FHI. The association of Usher syndrome with FHI in two of our cohort implicates a possible role for RV in the coexistence of the two conditions.

PENTACAM VS PACHPEN IN MEASURING CORNEAL THICKNESS

(1) * BAIDOUSI AMJAD (1) TESSLER TZVI (1) AMTIRAT AHED (1)
LEVY JAIME (1) LIFSHITZ TOVA
(1) DEPARTMENT OF OPHTHALMOLOGY, SOROKA UNIVERSITY
MEDICAL CENTER, BEER-SHEVA

Introduction: The measurement of central corneal thickness (CCT) has become increasingly important in ophthalmic practice . Ultrasonic pachymetry is the most common clinical technique for measuring CCT. In recent years, several optical technologies have been introduced for measuring corneal thickness. In this study we compare the results of CCT Measured by two devices, the OCULUS Pentacam (a non-contact optical system) ,and the Accutome Pachpen (an ultrasonic pachymeter) in glaucomatous and nonglaucomatous participants, and correlate differences in results with keratometry and refraction.

Patients / Methods: Twenty five Consecutive patients (Male 11, Female 14) were examined at the outpatient eye clinic of Soroka University Medical Center from August to November 2008. Central corneal thickness measurements obtained with the Pentacam system were compared with those of the PachPen. Refraction and keratometry were also taken. A paired t-test was used to compare between two measurement systems. For each patient, the difference between the two measurements was calculated and then a t-test for independent variables was used to evaluate the association between the level of difference for gender, age group, glaucoma status,keratometry and refraction.

Results: Mean age was 60.30 ± 19 . There were 14 glaucoma patients and 11 nonglaucomatous participants. Mean values of CCT for each instrument were $555.29 \pm 42.4 \mu\text{m}$ and $554.65 \pm 38\mu\text{m}$ using the ultrasonic PachPen and the Pentacam, respectively. There was no correlation between gender, keratometry, refraction or glaucoma status and the differences in CCT measurements between the two instruments.

Conclusions: The CCT values obtained by Pentacam and Pachpen correlated well and showed few outliers. The new Pentacam system provides a reliable, easy to-use, noncontact method for measuring CCT.

THE EFFECT OF MITOMYCIN C ON CORNEAL ENDOTHELIUM IN PTERYGIUM SURGERY

(1) * BAHAR IRIT (1) KAISERMAN IGOR (1) LEVINGER ELIYA (1)
SANSANAYUDH WIWAN (1) SLOMOVIC ALLAN
(1) OPHTHALMOLOGY DEPARTMENT, TORONTO WESTERN
HOSPITAL, TORONTO, CANADA

Introduction: Mitomycin C (MMC) was shown to be a useful adjunct to pterygium surgery, especially in preventing recurrence. It is used worldwide both for primary and recurrent pterygium excisions. Unfortunately, this substance poses a risk of devastating complications, such as scleral necrosis, and secondary infections. The aim of this study was to evaluate the changes in endothelial cell counts in patients after pterygium surgery with MMC 0.02% and to compare them with patients undergoing pterygium excision without MMC.

Patients / Methods: A prospective nonrandomized study was performed. 43 consecutive patients were included. Sixteen patients underwent pterygium surgery with conjunctival autograft and MMC for recurrent pterygium and 27 patients underwent Pterygium excision without MMC for primary pterygium removal (control group) . Endothelial images were acquired at the center of the cornea with a specular microscope before surgery and at one week, one month, and three months following surgery.

Results: Mean preoperative endothelial cell counts were 2330 ± 318 cells/mm² in the Pterygium excision without MMC group and 2486 ± 327 cells/mm² in the pterygium excision with MMC group ($p=0.13$). One month after surgery, the pterygium with MMC group showed a significant endothelial cell loss of 6% which was not present in the control group ($p=0.03$). Three months after surgery, endothelial cell loss was reduced to 4%. ($p=0.08$ compared with the control) In the pterygium excision with MMC group, endothelial cell volume was increased (at 1 and 3 months) and the percentage of hexagonal cells was reduced (at 1 month).

Conclusions: The use of topical MMC during recurrent pterygium surgery was found to have a deleterious effect on corneal endothelium 1 month following surgery. Judicious use of this drug is therefore recommended.

PROGNOSTIC FACTORS IN ACANTHAMOEBA KERATITIS

(1) * KAISERMAN IGOR (2) BAHAR IRIT (3) MCALLUM PENNY (3) SLOMOVIC ALLAN (3) ROOTMAN DAVID

(1) DEPARTMENT OF OPHTHALMOLOGY, BARZILAI MEDICAL CENTER, ASHKELON (2) DEPARTMENT OF OPHTHALMOLOGY, RABIN MEDICAL CENTER, PETAH-TIQVA (3) DEPARTMENT OF OPHTHALMOLOGY, TORONTO WESTERN HOSPITAL, UNIVERSITY OF TORONTO, TORONTO, ONTARIO, CANADA

Introduction: Purpose: To assess the factors influencing visual prognosis after acanthamoeba keratitis (AK) over an 8 year period, in a tertiary care setting in Canada

Patients / Methods: A retrospective case note review was undertaken of all AK cases treated in the cornea clinic at the Toronto Western Hospital between January 1999 and December 2006. A diagnosis of AK was made on the basis of positive culture results with a corresponding clinical presentation. Cases of AK that were seen for a single consultation, and not followed at our hospital, were excluded from the study. We calculated the effect of the various risk factors on final visual acuity, as well as on the length and type of treatment.

Results: Forty two eyes of 41 patients with AK were identified. The mean follow up was 19.7 ± 21.0 months. 64% of cases had more than one identified risk factor for AK, the most common risk factor being contact lens wear (92.9% of eyes). At presentation, median best spectacle corrected visual acuity (BSCVA) was 20/200 (20/30 – hand movements). 95% had pain, 59.5% had an epithelial defect, 59.5% had a ring infiltrate, 38.1% had keratoneuritis, 28.6% had a dense stromal infiltrate, 11.9% had pseudodendrites and 7.1% had a hypopyon. Initial treatment was with 2 or more anti-amoebal agents in 85.7% of eyes. Median BSCVA after treatment was 20/40 (20/20 – counting fingers). Patients with infection acquired by swimming or related to contact lenses had significantly better final visual acuity ($p=0.03$ and $p=0.007$ respectively). Neuritis and pseudodendrites were also associated with better final visual acuity ($p=0.04$ and $p=0.05$ respectively). Having had an epithelial defect on presentation was associated with worse final BSCVA ($P=0.0006$). Multivariate regression analysis found initial visual acuity ($p=0.002$), infections related to swimming ($p=0.01$) and the presence of an epithelial defect ($p=0.03$) to significantly influence the final visual acuity.

Conclusions: We demonstrated an increasing number of AK cases at our institution. We identified several prognostic parameters that can help clinicians evaluate the expected visual damage of the AK infection and thus tailor treatment accordingly.

SAFETY OF CORNEAL COLLAGEN CROSS-LINKING WITH ULTRAVIOLET-A AND RIBOFLAVIN IN PROGRESSIVE KERATOCONUS

(1) * GOLDICH YAKOV (2) MARKOVICH ARIE (3) HIRSH AMI (1) AVNI ISAAC (1) ZADOK DAVID

(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER (2) DEPARTMENT OF OPHTHALMOLOGY, KAPLAN MEDICAL CENTER (3) ENAIM REFRACTIVE SURGERY CENTERS

Introduction: To assess the possible damage to ocular tissue during treatment of keratoconus with ultraviolet A (UVA)-Riboflavin corneal collagen cross-linking (CXL).

Patients / Methods: Fourteen eyes of 14 patients aged 28.2 ± 5.9 (mean \pm SD) years with progressive keratoconus were treated with UVA-Riboflavin CXL. Corneal endothelium was assessed with the endothelial specular microscope. Macula was assessed with biomicroscopy examination and with optical coherence tomography using macular thickness protocol. Patients were examined preoperatively, at week 1, month 1, 3, 6, 9 and 12 after treatment.

Results: Comparative preoperative and postoperative results showed stable endothelial cell density (2730 cells/mm², 2793 cells/mm² and 2640 cells/mm² - preoperatively, at month 6 and 12, respectively) and stable central foveal thickness (203 μ m, 202 μ m and 205 μ m - preoperatively, at month 6 and 12, respectively). No morphologic abnormalities were noted.

Conclusions: UVA-Riboflavin CXL appears to be a safe procedure that do not cause damage to corneal endothelium and macula.

COMPLIANCE TO EYE CARE IN GLAUCOMA PATIENTS WITH CO-MORBID DEPRESSION

(1) WEISS GUY (1) BURGANSKI-ELIASH ZVIA (1) * BARTOV ELISHA
(1) DEPARTMENT OF OPHTHALMOLOGY, EDITH WOLFSON
MEDICAL CENTER

Introduction: Chronic diseases are associated with depression in many cases. Studies regarding chronic diseases demonstrated the relation between co-morbid depression and lack of compliance to medical treatment. In this study we investigated the association between depressive symptoms in glaucoma patients and their level of compliance to anti-glaucoma medications.

Patients / Methods: Eighty-three patients participated in this cross-section study, of which 76 were included. Diagnosis of glaucoma, age of 18 years and above and use of eye drops were the inclusion criteria. The subjects underwent ophthalmic evaluation and completed the Morisky 8-item Medication Adherence questionnaire and Center for Epidemiologic Studies Depression Scale (CES-D), evaluating their compliance and depression, respectively. Differences between groups were assessed with Student's t-test and Mann-Whitney test, and correlation analysis with Chi-Square tests and Spearman's rho.

Results: Fifteen patients (19.7%) were classified as "non-compliant" (Morisky cutoff <10). Sixteen patients (21.1%) suffered from depression (CES-D cutoff ≥ 16), similar to the prevalence of depression in the general Israeli population. We did not find a statistically significant higher prevalence of depression comparing the non-compliant and compliant patients (26.7% vs. 19.7%, $p=0.55$). However, we did find a significant correlation between the level of depression and the level of non-compliance ($r=-0.23$, $p=0.043$).

Conclusions: We found a similar rate of depression in glaucoma patients as in the general Israeli population. The presence of depression was not associated with the presence of non-compliance, but the level of depression was associated with the level of non-compliance. Depression might have a role in patients' compliance to glaucoma medication.

NON-COMPLIANCE WITH OCULAR HYPERTENSIVE TREATMENT IN PATIENTS WITH PRIMARY OPEN ANGLE GLAUCOMA AMONG ARAB POPULATION IN ISRAEL- A CROSS SECTIONAL DESCRIPTIVE STUDY

(1) * MASOUD MUHANNAD (1) IBRAHIM ALI (1) SIMAN-TOV SHLOMI
(1) PIKKEL JOSEPH
(1) DEPARTMENT OF OPHTHALMOLOGY, SIEFF GOVERNMENTAL HOSPITAL, SAFED

Purpose: To evaluate the non-compliance treatment rates among primary open angle glaucoma (POAG) Arab patients in Israel, and to verify the possible intervenable factors behind it.

Patients and Methods: A cross sectional study was carried out at the Ophthalmologic Clinics of three Arabic cities in the center of Israel. During their visit at health care clinic, four hundred consecutive Arab glaucoma patients were interviewed by an ophthalmologist and assigned to complete a questionnaire. The questionnaire was developed systematically based on a pilot test. Items included information about age, gender, number of drugs, and multiple reasons for non-compliance with drug therapy.

Results: Among 400 participants (197 men, 203 women), the general rate of non-compliance, for both genders, was 50%. Interestingly, there was a positive correlation between age and number of drugs. On the other hand, compliance rates were not affected by either gender or number of drugs. Instead, the compliance was significantly higher in younger patients (age<50), and in older patient (age> 80), 63%, and 77%, respectively (p<0.05). Moreover, Factors associated with non-adherence included inadequate knowledge (32%), underestimation of the severity of the disease (25.5%), and denial 15.5%.

Conclusions: Non-compliance was found to be common among Arabic population in Israel. More attention to issues of non-adherence in this population (lack of knowledge and underestimation) could improve adherence substantially.

MEASUREMENT OF INTRAOCULAR PRESSURE IN KERATOCONIC EYES USING THE OCULAR RESPONSE ANALYZER AND GOLDMANN APPLANATION TONOMETRY

(1) * GOLDICH YAKOV (1) BARKANA YANIV (1) AVNI ISAAC (1) ZADOK DAVID

(1) DEPARTMENT OF OPHTHALMOLOGY, ASSAF HAROFEH MEDICAL CENTER, ZERIFIN

Introduction: To establish correlations between intraocular pressure measurements obtained with the Goldmann applanation tonometer (GAT) and the ocular response analyzer (ORA) in patients with keratoconus (KC).

Patients / Methods: One hundred and two eyes of 59 patients with KC (39 M, 20 F, mean age 27.8 ± 6.8 years) were included in prospective study. IOP's were measured by GAT and ORA. The ORA provided a Goldmann correlated IOP (IOPg) and a corneal correlated IOP (IOPcc). Assessment of refractive status, visual acuity, axial length, corneal topography and pachymetry, and endothelial status was done. Regression and correlation analyses were performed between three tonometry methods and between tonometry methods and other ocular parameters. Bland-Altman plots were constructed.

Results: Mean GAT, IOPg and IOPcc values were 10.9 ± 2.0 , 9.5 ± 2.8 and 13.31 ± 2.5 mmHg. Pearson analysis showed weak significant correlation between the three methods (GAT versus IOPg: $r = 0.33$, $P < 0.001$, GAT versus IOPcc: $r = 0.40$, $P < 0.001$). The differences between GAT and IOPg (1.4 ± 2.7 mmHg, 95% limits of agreement = -3.9 to 6.8 mmHg) and between GAT and IOPcc (-2.4 ± 2.6 mmHg, 95% limits of agreement = -7.5 to 2.8) were statistically significant and were significantly correlated with corneal curvature. No strong correlation with corneal thickness was found.

Conclusions: Both IOPg and IOPcc have good agreement with GAT in patients with keratoconus. Differences between both sets of measurements seem to be affected mainly by corneal curvature.

HIGH GLUCOSE (DIABETIC) DAMAGE TO INTACT BOVINE LENSES IN CULTURE

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Introduction: Purpose: to follow the steps of the damage from high glucose concentration (diabetes). One of the major causes for cataract (loss of lens transparency) is Diabetes mellitus. The mechanisms of diabetes induced cataract are still not known in spite of many studies conducted. Researchers indicated that one cause for damage by high glucose is elevated activity of the enzyme aldose reductase, which produces alcohol from glucose. Alcohol accumulates in the lens and causes damage to lens cells and proteins. Other scientists indicated higher oxidation and glycation of lens proteins.

Patients / Methods: Methods: Intact bovine lenses were exposed in organ culture conditions to 450 mg % glucose, which simulates acute diabetes. Control lenses were incubated without glucose. Incubation time in culture was 15 days. Lens optical quality was assessed throughout the 15 days of the culture using our unique laser system. At the end of the culture period, lenses were analyzed by inverted microscopy. The lens epithelial layer was used for analysis of the enzymes hexokinase and ATPase activities using histochemical methods. Lens proteins were analyzed by SDS gel electrophoresis.

Results: Results: We characterized the damage-pathways from high glucose concentration by observing (a) changes in lens optical quality, (b) structural damage caused to the lens, (c) changes in the activity of lens epithelial enzymes, (d) following protein modifications. High glucose concentrations caused damage to lens optical quality, Increased Hexokinase and ATPase activities, which indicate increased metabolism and enhanced amounts of reactive oxygen species. Separation of lens proteins by electrophoresis showed a reduction in lens soluble proteins, changes in β crystalline with no change in α crystalline.

Conclusions: Conclusions: High glucose levels cause optical damage to the eye lens. High glucose concentrations (diabetes) increase the metabolism in the lens and might increase oxidation. Changes in lens proteins β crystalline cause damage to lens optical quality. This study was supported in part by a grant from the Esther and Chaim Coppel Trust and by the Guzik Ophthalmology Research Fund

INTRAVENOUS SEDATION VERSUS PLACEBO IN CLEAR CORNEAL PHACOEMULSIFICATION UNDER TOPICAL ANESTHESIA

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Introduction: Cataract surgery (phacoemulsification) is performed mostly under local/topical anesthesia. Sedation during this procedure is a debatable question. In an attempt to answer this question, patients that received intravenous (IV) sedation were compared with patients that received placebo IV, in regard to subjective preoperative, intraoperative and postoperative pain and anxiety, and surgeon satisfaction during phacoemulsification.

Patients / Methods: A double blind prospective randomized clinical trial was performed in 50 eyes of 50 patients that underwent clear corneal phacoemulsification by two surgeons. All the patients were anesthetized with topical application of oxybuprocaine hydrochloride 0.4% drops and 2% lidocaine jelly. Intracameral injection of 0.1 ml of preservative free lidocaine 1% was performed prior to phacoemulsification. The patients were randomized to receive either intravenous sedation of 1 mg midazolam and 25 mcg fentanyl, or placebo. After the surgery, the patients and surgeons completed a questionnaire. The patients had to grade their pain and anxiety scores from 0 to 5: 0=none, 1=mild, 2=mild to moderate, 3= moderate, 4=moderate to severe, 5=severe. The surgeons graded patient cooperation and ease during surgery.

Results: The final results were collected from 21 patients in the sedation group and 25 patients in the placebo group. The patients in the sedation group reported less preoperative and intraoperative anxiety (0.52 versus 1.85) and pain (0.33 versus 1.85) compared to the placebo group. Detailed results and statistical analysis will be presented.

Conclusions: Intravenous sedation significantly reduced patient anxiety and pain during clear corneal phacoemulsification done under topical and intracameral anesthesia.

REDUCING APOPTOSIS IN THE ISCHEMIC RETINA USING HYPERBARIC OXYGEN TREATMENT IN TWO DIFFERENT MOUSE MODELS

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Introduction: To investigate the therapeutic effect of the hyperbaric oxygen chamber (HBO) following induction of ischemic injury in models of central retinal artery occlusion (CRAO) or optic nerve crush.

Patients / Methods: Mice underwent CRAO (n=40) or crush induction (n=40); 20 of each group underwent HBO treatment. CRAO was induced by laser activation after rose-Bengal administration intravenously. Crush injury was induced by compressing the optic nerve posterior to the globe. Mice treated by HBO were exposed to 100% oxygen, in 2 atm for 90 minutes immediately after induction, and daily thereafter for up to 14 days. Histological sections of the retina were examined for apoptosis; mRNA expression levels of Bax, Bcl-2, and Caspase-3 were analyzed using quantitative real-time PCR, in the treated and untreated induced ischemic retinae.

Results: Maximal retinal cell loss of the inner retinal layers on day 21 were 60% and 80% in the CRAO and crush injury respectively. In the HBO treated groups, cell loss was only 30% and 40% respectively. Molecular analysis revealed an increase of bax (X4), caspase 3 (X2) and bcl-2 (X1.5) on day 1 in crush models, but not in the HBO treated mice. On day 3 all levels reverted to baseline (caspase 3, bax) or lower (bcl-2 0.67), similar to the levels measured in the HBO treated mice. CRAO showed similar trends, with 2-fold increase in the bcl-2 level on day 3 in the HBO treated group.

Conclusions: HBO-treatment protects neuronal cells from apoptosis. Histologically and molecularly we demonstrated reduced apoptosis and tissue preservation. These results encourage further clinical trials in patients with ischemic neuronal damage.

BAX ABLATION PROTECTS AGAINST RETINAL ISCHEMIA/REPERFUSION INJURY IN TRANSGENIC MICE

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Introduction: Optic nerve crush results in apoptosis of the retinal ganglion cells followed by secondary degeneration and progressive damage of the optic nerve. Bax is a pro-apoptotic member of the Bcl-2 family of proteins which regulates apoptosis. Suppression/inhibition of the BAX gene/or Bax protein in animal models was found to reduce ischemic injury in the heart and liver via inhibition of necrotic and apoptotic pathways. To measure apoptosis in the optic nerve and retina following optic nerve crush injury in mice transgenic for the BAX^{-/-} gene.

Patients / Methods: Three groups of mice -- homozygous (Bax^{-/-}), heterozygous (Bax^{+/-}), and wild-type (Bax^{+/+}) -- were euthanized at 1, 3, and 7 days following optic nerve crush injury. Retinal and optic nerve sections were analyzed for apoptosis using the TUNEL assay, and protein expressions of caspase-3 and bcl-2 were detected by immunohistochemistry stains and Western blot. mRNA extracted from the retina and optic nerves was analyzed for levels of Bax, bcl-2 and caspase-3 with with reverse transcriptase polymerase chain reaction using gene-specific primers.

Results: At 1 to 3 days after crush injury, apoptosis was maximal in the wild-type mice, with lower values in the heterozygous mice. No apoptotic cells were detected at these time points in the homozygous transgenic mice. On histological analysis, apoptosis was localized to the retinal ganglion cell layer.

Conclusions: Following crush injury in a mice model apoptosis occurs simultaneous in the optic nerve and retina. The total absence of apoptosis in the mice transgenic for BAX indicates that the Bax protein plays a crucial role in the induction phase of apoptosis, and suppressing its expression can reduce the damage.

TRANSPLANTATION OF RPE-LIKE CELLS DERIVED FROM HUMAN EMBRYONIC STEM CELLS IN DYSTROPHIC RCS RAT EYES: LONG TERM RESULTS AND COMPARISON WITH CELLULAR CONTROLS

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Introduction: Cell therapy is a promising strategy for the treatment of retinal degenerations. In this study we examined long term survival and effect of pigmented RPE-like cells derived from Human Embryonic Stem Cells (hESCs) following transplantation into dystrophic RCS rat eyes with RPE dysfunction. To determine whether other cell types can confer similar effects, human tenon fibroblasts and mitotically-inhibited human foreskin fibroblasts were similarly transplanted.

Patients / Methods: Differentiation of hESCs into pigmented RPE-like cells was induced by culturing embryoid bodies (EBs) under specific in-vitro conditions. A cell suspension enriched with such pigmented cells, or human fibroblasts, was injected into the subretinal space of RCS rat eyes at the age of 3 weeks. Fellow eyes served as control. Retinal function was assessed using electroretinography (ERG). Survival and location of the grafts were followed in vivo using a Heidelberg fundus camera. Expression of RPE-specific markers and thickness of the host photoreceptor layer were examined using histological and immunohistochemical techniques.

Results: Up to 4 months of age, dark-adapted mixed cone-rod ERG responses were significantly better preserved in eyes engrafted with RPE-like cells compared with control non-treated fellow eyes and with fibroblast-transplanted eyes. At the highest intensity, mean b-wave amplitude in RPE-transplanted eyes at this age was $74.6 \pm 21.3 \mu\text{V}$ (n=6) versus $23.3 \pm 4.9 \mu\text{V}$ in fellow control eyes. Transplanted RPE-like cells expressing RPE-specific markers could be identified up to the age of 5 months, and were associated with better survival of host photoreceptors in vicinity of the grafts. No tumors developed in any of the RPE-transplanted eyes. In contrast, transplanted human tenon fibroblasts proliferated extensively and were associated with a marked inflammatory response and disruption of retinal structure. Mitotically-inhibited fibroblasts did not confer functional or structural rescue.

Conclusions: RPE-like cells derived from hESCs survive for at least four months after transplantation into RCS rat eyes, and are associated with better preservation of function and structure of the dystrophic host retina. A different cell type, human fibroblasts, does not confer a similar effect. The results support the possible future use of hESC-derived RPE for the treatment of retinal and macular degenerations caused by RPE dysfunction.

NEOVASCULARISATION IN AN APOLIPOPROTEIN E4 TRANSGENIC MOUSE MODEL OF OXYGEN-INDUCED RETINOPATHY

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Introduction: Apolipoprotein E (apoE) is the most abundant brain lipoprotein. There are three major apoE isoforms. ApoE3, the most prevalent isoform, contains Cysteine and Arginine at positions 112 and 158, respectively. ApoE4 and apoE2 contain respectively two Arginines and two Cysteines at these positions. ApoE4 is the most prevalent risk factor for Alzheimer's disease, such that the age of disease onset is inversely related to the gene dosage of this protein. ApoE4 is also a detrimental factor in stroke and vascular diseases and it has been suggested that its pathological effects are mediated by impairments in cellular maintenance and repair. Paradoxically, it has been suggested that in Age- related Macular Degeneration apoE4 is protective. The objective of the present study was to develop a transgenic mouse model for studying the effects of apoE4 on retinal neovascularisation induced by hyperoxygenation followed by normoxia.

Methods: 7-day -old (P7) targeted replacement mice in which the endogenous mouse apoE was replaced by human apoE4 were placed in 75% oxygen for 5 days. On P12 the animals were returned to room air for another 5 days, and subsequently sacrificed at P17-P21. Control mice were targeted replacement apoE4 mice with normally developing retinal vasculature exposed to room air from birth until postnatal day 17. Quantification of the number of new blood vessel was performed with hematoxylin & eosin (H&S) staining of paraffin-embedded eye sections. In addition, fluorescein-conjugated dextran angiography of retinal vasculature was performed, and retinal whole mounts were prepared to score features of retinopathy. The parameters that were scored included extra retinal neovascularization and degree of central vasoconstriction.

Results: Histological assessment of the retinas of the oxygen-treated mice revealed marked increase in the levels of newly formed retinal blood vessels. Fluorescein angiography measurements revealed marked central vasoconstriction and newly formed extra-retinal neovascularisation. These findings show that exposure of the apoE4 mice to elevated levels of oxygen followed by normoxia, induces neovascularisation in the retina.

Conclusion: This model is currently being applied to transgenic mice which express other human apoE alleles such as apoE3 and the extent to which they are affected by the different human apoE genotypes will be explored in the future.

AAV MEDIATED LEDGF EXPRESSION RESCUES PHOTORECEPTORS IN THE RCS MODEL OF RETINAL DEGENERATION: PROBING THE MECHANISM

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Introduction: Lens epithelium derived growth factor (LEDGF) is up-regulated in response to stress and enhances survival of neurons in the retina and optic nerve, as well as a wide range of other cells such as fibroblasts and keratinocytes. We investigated photoreceptor rescue in the RCS rat retinal degeneration model following Ledgf delivery with an adeno-associated virus (AAV) and explored the mechanism of rescue.

Patients / Methods: Thirty-six RCS and nine P23H rats were evaluated following bilateral subretinal injections of AAV-Ledgf in one eye and buffer injected in contralateral eyes as control. Retinal function was evaluated 8 weeks later by the electroretinogram and compared to photoreceptor cell layer count. mRNA and protein levels of LEDGF and known stress related factors were compared in treated and control retinas to explore mechanism of LEDGF rescue. Nine RCS rats were treated with adenovirus-heat shock protein 27 (ad-Hsp27) and examined for rescue.

Results: Significant photoreceptor rescue was evident functionally and morphologically in 65-100% of the RCS rats treated at early ages of up to 7 weeks. Cell rescue was more prominent in the superior retinal hemisphere which has a slower natural degeneration rate in untreated eyes. Although many of the heat shock proteins and other stress related genes showed significant elevation in the AAV-Ledgf treated eyes, all increases were moderate. Transduction of retinal cells with an ad-Hsp27 also resulted in photoreceptor rescue. AAV-Ledgf elicited no photoreceptor functional rescue in transgenic P23H rhodopsin rat retina.

Conclusions: Chronic LEDGF treatment via AAV-Ledgf administration gave successful rescue of photoreceptors in the RCS rat retina. AAV-Ledgf treatment retarded cell death by about two weeks. Induction of heat shock proteins also gave photoreceptor rescue. However we did not find compelling evidence that LEDGF rescue was associated with up-regulation of heat shock proteins.

CONE DISTRIBUTION AND PATTERN OF CONE LOSS IN THE RD10 MOUSE MODEL OF RETINAL DEGENERATION

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Introduction: We characterized the distribution of cone photoreceptors in the retina of rd10 mice during retinal development and subsequent retinal degeneration. The blue and red/green cone distribution in rd10 mice was compared with that of age matched C57BL/6J mice, the appropriate wild type correlate for this animal. The information gleaned from this study could serve as a basis for the exploration of interventions aimed at preventing cone photoreceptor death in this important model of human retinitis pigmentosa.

Patients / Methods: Cone immunofluorescence studies were performed on retinal whole mounts of rd10 and C57BL6 mice between the ages of 2 to 6 weeks. Retinas were incubated with primary anti-red/green opsin (rabbit polyclonal, 1:200; Chemicon International) and anti-blue-sensitive opsin (goat polyclonal, 1:200; Santa Cruz Biotechnology). Specimens were then incubated with appropriate secondary antibodies (CYTM2-conjugated donkey anti-rabbit IgG (1:200) and rhodamin RedTM-X-conjugated donkey anti-goat IgG (1:200); Jackson ImmunoResearch Laboratories). Imaging was performed with an Olympus fluorescent microscope and whole mount images were compiled using Photoshop software. Density and eccentricity data were analyzed using ImagePro software.

Results: Red/green (R/G) and blue (B) cone distribution was mapped for rd10 and C57BL6 mice at 2, 4 and 6 weeks of age (n=6-10 at each data point). At early ages, R/G cones were most dense in the periphery, while B cones were more evenly distributed in both rd10 and C57BL6 mice. In the rd10 mouse progressive loss of both cone photoreceptor types occurred. The degenerative process began centrally and extended peripherally. At 6 weeks of age, survival of R/G cones in rd10 mice was limited to the superior and far-periphery of the retina and survival of B cones to the inferior retina.

Conclusions: Red/Green and Blue cone distribution across the retina is not homogenous in rd10 or in normal C57BL6 mice. The degenerative process in rd10 mice progresses from the center of the retina peripherally, with the majority of cones lost by 6 weeks of age. The accurate mapping of cone density loss across the retina and along time in rd10 mice is crucial for proper analysis of the effect of novel therapies aimed at promoting cone survival in this model.

FURTHER CHARACTERIZATION OF RETINAL ALTERATIONS IN THE CHEMOKINE RECEPTOR TYPE 2 (CCR2) DEFICIENT MICE

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Introduction: The CCR2 deficient mouse was reported to manifest retinal alterations which share similarities with age related macular degeneration (AMD) in humans. We aim to further characterize retinal alterations in this mouse strain and to evaluate its similarities to the human disease.

Patients / Methods: CCR2 deficient mice and age and genetic background matched wild type mice (WT) were studied. Mice were evaluated at the age of 7, 12, and 16-23 months by conventional histology and immunohistochemistry. Bioinformatic analysis of microarray study of gene expression of this mouse strain which we have previously performed conducted.

Results: At 16-23 months of age CCR2 deficient mice showed enrichment of microglial cells in the subretinal space according to immunostaining for CD11b. At the same time point drusen were not identified in sequential histological sectioning of CCR2 deficient mice eyes. Bioinformatic analysis demonstrated similarities between retinal gene expression signatures of CCR2 deficient and expression signature reported from other mice models for retinal degeneration and ageing mice retina.

Conclusions: Aged CCR2 deficient mice show retinal alterations. While drusen are not a common histological finding in these mice, fundoscopic drusen-like lesions are often observed and are associated with altered retinal immune response. Combined with previous studies these data suggest that drusen are not required for development of retinal alterations associated with the perturbed immune response and complement activation present in this mouse strain.

CORRELATION BETWEEN PRESSURE MEASUREMENTS IN FLUID FILLED AND GAS INJECTED EYES

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Introduction: Intraocular gas injection is a routine procedure in vitro-retinal surgery. Expansion of the intraocular gas may cause uncontrolled rise in intraocular pressure (IOP) and may lead to retinal vascular occlusion or damage the optic nerve head. The objective of this study was to correlate IOP measurements obtained using an intraocular pressure transducer, with simultaneous applanation tonometry, in fluid filled and gas-injected enucleated porcine eyes.

Patients / Methods: A 23-gauge needle connected to a pressure transducer and to a bottle of saline was inserted through the sclera into the vitreous cavity. IOP was varied from 10 to 50 mmHg in steps of 5 mmHg by adjusting the height of the saline reservoir. For each pressure setting, several comparative measurements of IOP were taken simultaneously using the transducer and the TonoPen XL. The pressure measurements were applied in fluid filled and 0.5-1cc gas-injected enucleated porcine eyes.

Results: In the fluid filled eyes increasing the height of the saline reservoir led to a linear increase in intraocular pressure as indicated by the two measurements methods: intraocular transducer and the TonoPen XL over the range of 10-50 mmHg. However, comparative repeated measurements taken by the TonoPen XL were always higher than the pressures recorded by the transducer and overestimated by 30%. In contrast, no difference between intraocular measurements by the transducer and TonoPen XL measurements were detected in 0.5-1.0cc gas injected eyes.

Conclusions: The variability of pressure measurements by applanation tonometry depends on fluid and gas content of the eye. In fluid filled porcine eyes, applanation tonometry overestimates the intraocular transducer measurements by 30%. IOP measurements with applanation tonometry of gas injected eyes were correlative, linear and similar to intraocular transducer measurements

ANTI-VEGF INTRAVITREAL INJECTIONS IN PATIENTS UNDER CONCURRENT ORAL ANTICOAGULATION OR ANTIPLATELET THERAPY

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Introduction: The aim of this prospective study was to evaluate the safety of intravitreal injections in patients under concurrent oral anticoagulation or antiplatelet therapy.

Patients / Methods: 351 patients who underwent consecutively 472 intravitreal injections of Avastin or Lucentis were included in the study. Post-injection intra- or extraocular hemorrhages were documented.

Results: Small subconjunctival hemorrhages were noted in 16 % of the eyes. No patient experienced a subtotal subconjunctival or vitreous hemorrhage. There was no difference between patients who received oral anticoagulation, those with antiplatelet therapy and control.

Conclusions: This study suggests that intravitreal injections can be performed without cessation of oral anticoagulation or antiplatelet therapy.

ASSOCIATION BETWEEN VITAMIN D AND AGE-RELATED MACULAR DEGENERATION IN MACCABI HEALTHCARE MEMBERS

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Introduction: Several potential risk factors for the development and progression of AMD have been identified, such as age, cigarette smoking, hypertension and family history . Recently, inflammation has received attention as a potential risk factor for this disease. A number of studies suggest an anti-inflammatory role for vitamin D in vitro and in vivo. Furthermore, there is recent evidence of vitamin D being a potent inhibitor of angiogenesis by its effects on endothelial cells and by interrupting angiogenesis related signaling pathways. The primary purpose of this research was to examine the relationship between serum vitamin D level and prevalent AMD.

Patients / Methods: Retrospective cohort study carried out in Maccabi Healthcare Services. participants were patients aged 60 years or older, whose vitamin D levels were taken for routine examination since 2000 and were evaluated for the existence of AMD. Mean level of 25-OH vitamin D in ng/ml were calculated in the 2 groups and we evaluated the proportion of the tests in which the level were low (<16ng/mL) and in which it was high (>74ng/mL).

Results: Since 2000 a total of 9189 MHS members whose levels of vitamin D were identified (12.5% out of MHS with AMD and 9.5% out of non AMD Maccabi healthcare members) The total study cohort was comprised of 1045 AMD patients and 8124 non-AMD controls. The mean level of 25-OH vitamin D in the AMD group was 24.1 ± 9.41 and in the controls 24.13 ± 9.50 . The proportion of tests in which 25-OH vitamin D level was <16ng/mL was 33.6% in the AMD group, compared to 0.19% in the controls. The proportion of tests in which 25-OH vitamin D level was >74ng/mL was 32.86% in the AMD group compared to 0.14% in the controls.

Conclusions: Despite the proposed association between vitamin D and AMD, MHS members with AMD had a level of 25-OH vitamin D similar to that of matched controls. The difference between our study and other population based studies investigating this issue can be bias resulting from increased sun exposure in our country including in AMD patients, and high awareness to supplement intake by AMD patients in the population. This will be further investigated and presented.

ASTIGMATISM IN CHILDREN - LONG TERM FOLLOW UP

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Purpose: Astigmatism causes variable refractive errors in different meridians. Previous studies have shown that astigmatic correction before the age of 7 years may prevent meridional amblyopia. The purpose of the study was to find whether the children's age at the beginning of treatment correlates with improvement in visual acuity, and the time until best visual acuity was achieved. We also monitored the amount and axis of the cylinder during the follow up.

Methodes: Retrospective chart review. The charts of 55 children were retrieved. 46 charts (92 eyes) were included in the study. The children had a complete ophthalmological examination. UCVA, BCVA and cycloplegic refraction were noted at the beginning and throughout the follow up. Each child received the best optical correction and was followed by the same team including a pediatric ophthalmologist and an orthoptist.

Results: The average follow up period was 53 months (1-13 years). The average BCVA at the beginning of the treatment was 6/12. The average amount of astigmatism was 1.99D and 80% of the patients had With the Rule astigmatism. At the end of the follow up the average BCVA was 6/6.6, the average amount of astigmatism was 1.89D and 94.5% of the patients had With the Rule astigmatism.

Conclusions: The best VA was achieved the earlier the treatment was given. We also found that the time to improvement of the visual acuity was shorter in the school age children and there was no significant change in the amount of the astigmatism.

PERIOcular STEROID INJECTION FOR THE MANAGEMENT OF UVEITIS IN CHILDREN

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Purpose: To report the outcome of orbital floor corticosteroids injection (OFCl) in the treatment of uveitis in children.

Design: Retrospective interventional case series.

Participants: Nineteen eyes of 15 children.

Intervention: Orbital floor injection of 40 mg/ml methylprednisolone acetate or a combination of 20mg/0.5 ml Triamcinolone and 2mg/0.5ml dexamethasone.

Main Outcome Measures: Best corrected Visual acuity (BCVA), ocular inflammation, time to relapse, systemic corticosteroid and immunosuppressive therapy required, and potential complications of OFCl including cataract progression, high intraocular pressure (IOP), globe perforation and systemic drug toxicity were assessed.

Results: Nineteen eyes of 15 children had OFCl, of them 4 eyes (21%) underwent more than 1 injection. Twelve eyes (63%) improved BCVA by two or more lines. The mean improvement of BCVA after OFCl was 0.18 (from 0.53 to 0.35 logarithm of the minimum angle of resolution) ($P < 0.0005$, paired-samples *t* test), at a mean of 6 weeks (range, 4–20). Fourteen eyes (74%) had significant improvement in inflammation; 4 to 7 weeks post OFCl, with a median of 4 weeks. Anterior uveitis was treated effectively in all eyes, vitritis resolved in all but one case and resolution of cystoid macular edema (CME) was achieved in 6 eyes (55%). Following initial response to OFCl, uveitis relapsed in 7 eyes (50%) after a median time of 4 months (range, 2-5 months). The dosage of oral corticosteroids and/or second-line immunosuppressive medication required to control the inflammation was reduced or able to be stopped in 5 patients (50%). The most common adverse effect was steroid-induced cataract, observed in 4 of 19 eyes (21%), 5 months post OFCl. One patient developed cushingoid features 6 weeks post his second OFCl. There was no raised IOP or globe perforation.

Conclusions: The use of orbital floor injections of corticosteroids resulted in control of the active uveitis and visual acuity improvement in most children. Moreover, it may allow the cessation and/or reduction of immunosuppressive therapy. However, as in adults, OFCl effect is usually short term and cataract formation may occur.

Session II – Cornea

THE EFFECT OF TOPICAL TREATMENT WITH THE MATRIX METALLOPROTEINASE INHIBITOR, DOXYCYCLINE, ON OCULAR CHEMICAL INJURY IN RABBITS

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Introduction: Ocular chemical injury induced by Sulfur Mustard (SM) is characterized by acute lesions, followed by delayed pathology, clinically characterized by epithelial defects and neovascularization (NV). Previously we have shown the involvement of the extracellular matrix remodeling enzymes MMP-2 and MMP-9 in these injuries. Corneal MMP-9 and elevated MMP-2 activities were found during the acute phase, and in vascularized tissue of corneas displaying NV during the delayed pathology. This study aimed to evaluate the beneficial effect of topical treatment with the MMP inhibitor doxycycline.

Patients / Methods: Rabbit eyes were exposed to SM vapor. A clinical follow-up was carried out up to 1 month. Various protocols of post exposure topical doxycycline (2mg/ml) or dexamycinTM were evaluated.

Results: Topical doxycycline was beneficial in ameliorating the ocular injury during the acute phase. NV appeared following treatment cessation, but continual treatment significantly reduced its incidence (1). Similar protocols with dexamycinTM were more efficient. Topical doxycycline, beginning subsequent to the acute phase healing, reduced the extent of the delayed pathology. Postponed doxycycline treatment, beginning at the delayed phase, had no effect on the neovascularized eyes, but was beneficial for non-vascularized eyes. Preliminary results showed no effect of doxycycline on the abnormal MMPs activity in vascularized corneas.

Conclusions: Topical doxycycline was beneficial during the acute phase and in non-vascularized eyes during the delayed pathology. DexamycinTM treatment, despite its known adverse effects, was more efficient. Since doxycycline failed to affect abnormal MMPs activity in vascularized corneas, another therapeutic protocol or different MMP inhibitors might be more efficient against NV.

(1) Kadar et al. Toxicology, 2008

**THE AMINO ACID DERIVATIVE DL-TRIFLUOROLEUCINE
ACTS AS A CHEMOREPELLENT TO PSEUDOMONAS
AERUGINOSA.**

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Introduction: The purpose of the study was to investigate whether microbial chemotaxis can be utilized to prevent and treat infectious keratitis, and specifically to examine the effect of the nonmetabolizable amino acid derivative DL-Trifluoroleucine on *Pseudomonas aeruginosa*.

Patients / Methods: Chemotaxis assays were performed by assessing the directional movement of *Pseudomonas aeruginosa* in response to a given stimulus on minimal media soft agar plates. Cornea penetration assays were performed using whole globes of pig eyes. We compared the number of bacterial colony forming units (CFU) present in pig corneas, preincubated with DL-trifluoroleucine or buffer alone, after *pseudomonas* exposure (ranging 2-8 hours).

Results: *Pseudomonas aeruginosa* was found to consistently migrate away from DL-trifluoroleucine on the soft agar migration assays (mean -13.7 mm, $p < 0.05$, $n=3$). The corneal penetration studies demonstrated that preincubation with DL-trifluoroleucine caused a 70% reduction in the degree of bacterial invasion ($p < 0.0001$, $n=3$).

Conclusions: The data indicate that DL-trifluoroleucine has a chemorepellent influence on *Pseudomonas aeruginosa*. This knowledge suggests that targeting microbial chemotactic behaviors can lead to the development of novel strategies to prevent and treat microbial infections such as infectious keratitis.

ANTI-INFLAMMATORY AND ANTI-VEGF THERAPY FOR NEOVASCULARIZATION ASSOCIATED WITH LIMBAL STEM CELL DEFICIENCY IN RABBITS

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Introduction: Corneal neovascularization (NV) following chemical injury induced by sulfur mustard (SM) was associated with chronic inflammation, limbal damage and increased levels of vascular endothelial growth factor (VEGF). This pathology was diagnosed by impression cytology and histology as part of a limbal stem cell deficiency disorder. Anti-inflammatory treatment with steroids was beneficial in ameliorating the NV, when administered before their appearance and as symptomatic treatment to corneas displaying NV. Yet, corneal deterioration continued when therapy was terminated. The aim of the present study was to compare the efficacy of bevacizumab (Avastin), an anti-VEGF factor, to steroids in ameliorating these lesions.

Patients / Methods: NZW rabbit corneas were chemically damaged using SM vapor. Corneal NV was evaluated by slit lamp examination, combined with morphometric analysis. Corneas displaying NV were topically treated for three weeks with Avastin (6.25 or 25mg/ml, x2/day), steroids (Dexamycin®, x4/day) or a combined therapy. At 3 months post exposure eyes were taken for biochemical and histological evaluations.

Results: Corneal NV developed as early as two weeks following SM exposure and deteriorated with time. A significant regression in NV was observed following Avastin or Dexamycin treatments, however the effect of Dexamycin was more pronounced. Histological evaluation revealed that Dexamycin decreased the inflammatory reaction but had no effect on limbal epithelial damage. The effect of Avastin on the chronic inflammation as well as on limbal damage is still unclear.

Conclusions: Dexamycin was more efficient against NV compared to Avastin. A long term follow-up is needed to elucidate the effect of Avastin on limbal deficiency.

TOPICAL TREATMENT WITH 1% CYCLOSPORINE FOR CHRONIC ADENOVIRAL KERATITIS

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Introduction: To evaluate treatment with topical 1% cyclosporine A (CsA) in patients with subepithelial corneal infiltrates (SEIs) how had been treated with topical corticosteroids before with no improvement or had to stop treatment due to intraocular (IOP) elevation.

Patients / Methods: We retrospectively reviewed the records of 9 patients (12 eyes) before and after treatment with CsA 1% eye drops 2 times a day. The objective data recorded included: best corrected Snellen visual acuity (BCVA), IOP, number of medications in use and evaluation of SEIs severity (improved, stable and worse). For subjective evaluation patients were asked to complete a questionnaire on there last follow-up visit.

Results: 5 (56%) males and 4 (44%) females mean age of 47 ± 13 years were included. mean follow-up on CsA was 13 ± 7 months. The mean BCVA, before and after treatment, was 0.21 ± 0.28 and 0.42 ± 0.4 , respectively with no statistically significant improvement. There was statistically significant reduction in the number of medication in use before and after treatment from 1.88 ± 1.05 to 1.22 ± 0.44 , respectively ($p=0.046$). 6(66%) patients showed clinical improvement and 3(34%) where stable during the treatment period. Patients reported statistically significant reduction in the severity of symptoms before and after the treatment (1=no symptoms, 10=terrible symptoms). The mean score before and after treatment, was 7.25 ± 1.25 and 2.87 ± 0.64 ($p<0.001$), respectively. Most of patient reported no foreign body and glare sensation or side effects with CsA treatment. Overall patients reported improvement in vision (1=none, 10=great) and satisfaction (1=not satisfied, 10=very satisfied) with mean score of 7.5 ± 1.4 and 8 ± 1.3 respectively

Conclusions: Topical CsA 1% is a safe and effective alternative treatment in patients with SEIs who do not respond to other treatment modalities.

BUSIN GUIDE VERSUS FORCEPS FOR THE INSERTION OF THE DONOR LENTICULE IN DESCMET STRIPPING AUTOMATED ENDOTHELIAL KERATOPLASTY

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Introduction: In the last years, Descemet stripping automated endothelial keratoplasty (DSAEK) has replaced penetrating keratoplasty (PKP) as the gold standard surgical treatment for corneal endothelial diseases. The purpose of this study was to compare two insertion methods in DSAEK: Busin guide-assisted versus Forceps-assisted insertion of the corneal lenticule graft.

Patients / Methods: In a prospective, consecutive, comparative, non randomized study, 63 eyes of 63 consecutive patients were included. All patients underwent DSAEK for Fuchs' endothelial dystrophy, pseudophakic bullous keratopathy, aphakic bullous keratopathy, failed graft or iridocorneo endothelial syndrome at the Toronto Western Hospital. Twenty six consecutive donor discs were inserted with the Busin guide and 37 consecutive eyes underwent forceps assisted insertion of the donor. Main outcome measures included: Uncorrected visual acuity (UCVA), best spectacle-corrected visual acuity (BSCVA), manifest refraction, corneal endothelial cell loss, and post-operative complications.

Results: Busin guide-assisted DSAEK group had significantly worse UCVA and lower donor endothelial cell counts preoperatively. No significant differences were noted in the intraoperative or postoperative complications. Six months following surgery, BCVA was not significantly different between groups : 20/37 in the Busin guide-assisted DASEK group versus 20/42 in the Forceps-assisted group (p=0.39). Mean spherical equivalent was -0.02 Diopters and 0.82 Diopters (p=0.06), and mean refractive cylinder was 2.2 Diopters and 1.31 Diopters (p=0.0006), respectively. Endothelial cell loss was significantly lower in the Busin guide-assisted DASEK group: 25% loss versus 34.3 % loss in the Forceps –assisted DSAEK group. (p=0.04)

Conclusions: Although visual outcomes were not different between the groups studied, Busin guide-assisted DSAEK resulted in lower percentage of endothelial cell loss compared with forceps insertion, 6 months following surgery.

PROSPECTIVE EVALUATION OF QUALITY OF CARE AND PATIENT SATISFACTION IN LASIK SURGERY

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Purpose: To evaluate quality of care and patient satisfaction with LASIK surgery for myopia, by objective and subjective parameters.

Methods: A prospective longitudinal study was performed on 95 myopic patients, who underwent LASIK surgery, performed by a single surgeon at Hadassah Optimal. Patients completed four questionnaires: before surgery; one day, one month and three months post-surgery. Questionnaires were based on previously validated tools on patient satisfaction and staff attitudes, published in the literature.

Results: Satisfaction with postoperative uncorrected visual acuity was rated “great” or “very great”, one and three months post-surgery, by 93% and 96% of the patients, respectively. Surgery results meeting expectations were rated “great” or “very great” during the same time period, by 80% and 99% of the patients, respectively. Quality of care was rated “good” or “very good” by 98% of the patients. Patient satisfaction with postoperative uncorrected vision and patient evaluation of quality of care were significantly correlated with objective postoperative uncorrected visual acuity, ($P < 0.001$, $P = 0.016$, respectively). However, there was no significant association with age, fear and pain during surgery. Patient evaluation of quality of care rose significantly with staff's caring attitude ($P < 0.001$) and provision of information on surgery and follow-up ($P = 0.003$).

Conclusions: Final objective uncorrected vision is a good indicator for predicting patient satisfaction and evaluation of quality of care. These parameters improve over time and are not affected by age, fear and pain during surgery. However, they are affected by staff attitude and information provided on the procedure and follow-up.

Session III – Poster presentations 2

EFFECT OF DISINSERTION OF RECTUS EYE MUSCLES ON AQUEOUS HUMOR COMPOSITION IN RABBITS

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Introduction: To evaluate aqueous humor composition and biochemical analysis in a rabbit model for anterior segment ischemia.

Patients / Methods: Six rabbits underwent bilateral 0.2–disinsertion of the 4 rectus eye muscles. One day later, aqueous humor (0.1 ml) was withdrawn from the anterior chamber and the concentrations of electrolytes, glucose, and lactate were determined. Similar assessments were carried out in 8 eyes of 4 non-operated controls.

Results: Sodium and calcium concentrations (mean \pm SD) were significantly higher in the study group than in the controls (142 ± 2.9 compared to 138 ± 2 mmol/L, $P = .002$, and 1.4 ± 0.1 compared to 1.3 ± 0 mmol/L, $P = .005$, respectively). Potassium concentrations were significantly lower in the study group than in the controls (4.1 ± 0.5 compared to 4.5 ± 0.1 mmol/L; $P = .02$). In the study group the mean glucose level was 108.4 mg/dL, which is comparable to the published normal value of 108 mg/dL, and the mean lactate level was 124 mg/dL, significantly higher than the published normal value of 78 mg/dL ($P = .001$).

Conclusions: Bilateral disinsertion of the 4 rectus aqueous barrier, as–muscles in rabbits resulted in a breakdown of the blood reflected and measured by an electrolyte imbalance. These experimental results may be useful in the future in searching methods of manipulating the ischemic response. At present there is no indication to do anterior chamber taps in humans for that purpose.

GENE EXPRESSION PATTERNS OF PERIPHERAL BLOOD CELLS SUBPOPULATIONS IN ACUTE OPTIC NEURITIS: THE ROLE OF CD19+ B CELLS

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Introduction: Optic Neuritis (ON) is an inflammatory clinical entity with unclear molecular mechanisms which may herald the diagnosis of multiple sclerosis (MS). We measured gene-expression profiles of peripheral blood mononuclear cells (PBMCs) sub-populations: CD19+ B-cells, CD14+ macrophages, CD4+ , CD8+ T cells in ON patients.

Patients / Methods: Microarray (Affymetrix Inc., 22,215 genes, 60 samples) of PBMC sub-populations from six patients, during 96h of the first episode of ON and 9 matched healthy volunteers were compared. Most informative genes (MIGs) defined as $p < 0.01$ and fold change > 2.0 . Biological pathways were analyzed by Ingenuity software. Gene expression results were verified on samples from additional 5 ON patients and 5 healthy donors using QRT-PCR, Western blot and ELISA.

Results: ON patients had a differentiated gene expression signature of 469, 55 and 55 MIGs respective to CD19+, CD14+, and CD4+ cells. No MIGs were identified for the CD8+ cells. The significant biological findings were related to upregulation of MHC-class-II genes like HLA-DQB1, HLA-DOB and antibody class-switching genes like CD64, IgE in CD19+ B cells in concert of inhibition of anti-inflammatory activity and apoptosis. CD14+ macrophages were characterized by proliferation, CD8+, CD4+ T cells - non-specific activation.

Conclusions: Our findings suggest significant role of B-cells in the pathogenesis of the first episode acute ON. B-cells related genes were involved in the primary immune response on a background of T-cells anergy expression pattern. We propose possible B cell inhibition therapy in a clinical practice of ON treatment.

INCREASED SCOTOPIC AND PHOTOPIC A-WAVE AMPLITUDE ELECTRORETINOGRAM RESPONSES AFTER INTRAVITREAL BEVACIZUMAB INJECTION IN PATIENTS WITH NEOVASCULAR AGE-RELATED MACULAR DEGENERATION

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Introduction: To assess the effect of bevacizumab on the retinal function by full-field electroretinography (ERG) in patients with choroidal neovascular Age-related Macular Degeneration (AMD).

Patients / Methods: Masked, controlled study, included 12 patients with choroidal neovascular AMD, mean age 72 ± 11.5 years, (5 men and 7 women) . The patients were injected with bevacizumab 1.25 mg/0.05 ml (Genentech, San Francisco, CA) unilaterally into the vitreous cavity as part of the standard management. Prior to bevacizumab injection and one month post-injection, full-field ERG (ISCEV; LKC Technologies, Gaithersburg, MD) scans were obtained from both eyes of each patient while the other eye was used as a control group. Scotopic responses were recorded under four incremental light intensities stimuli (0.099 cd-s/m², 2.44 cd-s/m², 23.5 cd-s/m² and 252 cd-s/m²). The photopic responses were evoked by two incremental light intensities (2.44 cd-s/m² and 7.8 cd-s/m²). The differences before and after treatment for the waves amplitude were calculated and the data were analyzed using repeated measure ANOVA model.

Results: The average differences for the incremental light intensities of the scotopic a-wave amplitude in the injected eyes was 15.92 microvolt, compare to 1.33 microvolt in the controlled eyes (P=0.057, ANOVA). The average differences of the photopic a-wave amplitude were 4.97 microvolt in the injected eye and -1.06 microvolt in the control eyes (P=0.01, ANOVA). The average differences of the scotopic b-wave amplitude in the injected and controlled eyes were 13.56 microvolt and 4.11 microvolt respectively (P=0.23, ANOVA). The average differences for the photopic b-wave amplitude in the injected eyes was 5.07 microvolt and -2.70 microvolt in the controlled eyes (P=0.048, ANOVA).

Conclusions: To assess the effect of Bevacizumab (Avastin®), a vascular endothelial growth factor inhibitor on the retinal function by full-field electroretinogram (ERG) in patients with choroidal neovascular Age-related Macular Degeneration (AMD).

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

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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), which to date has no effective treatment. USH1 can be caused by mutations in each of at least seven genes. While truncating mutations of these genes cause USH1, missense mutations of some of the same genes cause nonsyndromic deafness, suggesting that partial or low level activity of the encoded proteins may be sufficient for normal retinal function, although not for normal hearing. Interventions to enable at least some translation of full-length protein, may delay the onset and/or progression of RP in individuals with USH1 due to nonsense mutations. One such possible therapeutic approach is suppression of nonsense mutations by small molecules such as aminoglycoside antibiotics.

Patients / Methods: Suppression of nonsense mutations was initially tested in vitro, using a transcription/ translation assay of a reporter plasmid harboring various nonsense mutations of CDH23 and PCDH15, underlying USH1D and USH1F, respectively. One of these mutations is R245X, which we recently identified as a major cause of USH1 in Ashkenazi Jews. Ex vivo suppression of these mutations is tested using expression constructs transfected into cultured cells. In parallel, we are developing a series of new aminoglycoside-derived compounds, which will maintain their suppressive activity, while having reduced toxicity.

Results: We demonstrated, in vitro, suppression of various PCDH15 and CDH23 nonsense mutations, by commercial aminoglycosides. We also demonstrated ex vivo suppression, by the same aminoglycosides, of the R245X mutation. We are currently generating constructs for ex vivo testing of CDH23 murine nonsense mutations, towards assays in existing mouse models for USH1D. Furthermore, we have designed and synthesized two new promising aminoglycoside derivatives, NB30 and NB54. In vitro and ex vivo assays, together with cellular and acute toxicity experiments proved both a suppressive activity and a significantly reduced toxicity of these compounds in comparison to commercially available aminoglycosides.

Conclusions: The research described here will have important implications for development of targeted interventions that are effective for patients with USH1 and nonsyndromic RP caused by various nonsense mutations.

POLYMORPHISM IN THE WRN GENE IN PATIENTS WITH SENILE CATARACT

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Introduction: The Werner syndrome (WS) is an autosomal recessive disease of premature-aging with early graying of the hair, bilateral cataract formation, type-II diabetes mellitus, osteoporosis and atherosclerosis. The disease is a result of the loss of function of the RecQ helicase, the WRN protein, leading to a breakdown in the maintenance of genome integrity. Polymorphic C1367T in the WRN gene, leads to an amino acid substitution from Cys to Arg. The presence of this polymorphism was in association with higher rate of myocardial infarct, osteoporosis and bilateral cataract. The aim of this study was to investigate the incidence of the C1367T (rs1346044) polymorphism in WRN gene in patients with age-related cataract.

Patients / Methods: Anterior lens capsule material was collected during surgery from 66 patients with senile cataract. All epidemiological data were collected. DNA was extracted, amplified by polymerase chain reaction, and sequenced for WRN gene. DNA samples were screened for polymorphism in rs1346044 WRN gene using restriction enzymes followed by sequencing.

Results: Twenty six males and 40 females were included; mean age was 74.5 ± 9 years. Genotypic frequencies of T/T and T/C were 68% and 32% respectively. No C/C phenotype was found. Eight patients had myocardial infarct; 7 cardiovascular accident and 7 had various tumors, two of them breast cancer.

Conclusions: As the distribution of rs1346044 polymorphism in our cataract population is similar to that in the normal population described in the literature, this polymorphism of the WRN gene is not associated with cataract formation of the elderly. In contrast to findings in progeria, human age-related nuclear cataract is not associated with WRN mutation or dysfunction of the RecQhelicase. Further studies are needed for further clarification.

MOLECULAR CHARACTERIZATION OF CERKL: A GENE UNDERLYING AUTOSOMAL RECESSIVE SEVERE RETINAL DEGENERATION WITH EARLY MACULAR INVOLVEMENT.

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Introduction: CERKL is a novel gene encoding for a ceramide kinase-like protein. CERKL mutations lead to severe retinal degeneration with early macular involvement. Cerkl is expressed in the mouse retina. However, its specific expression pattern within the retina, whether it possesses a kinase activity, and what is the identity of its substrates is currently unclear.

Patients / Methods: In order to generate a specific antibody against CERKL, a 15.4 kDa peptide derived from CERKL unique N-terminal domain was expressed in bacteria. The purified protein was injected to rabbits, the immune-serum was affinity purified and specificity was tested using a western blot. RT-PCR analysis was conducted in order to assess the expression levels of Cerkl in the developing mouse eye as well as to reveal the unique splice variants of Cerkl in the mouse retina. For further investigation of CERKL's protein-protein interactions we are currently employing the RAS recruitment system in yeast.

Results: RT-PCR was performed on total eye RNA to examine the expression levels of Cerkl versus Cerkl in mouse brain comparing to mouse retina. The two tissues showed similar expression levels of Cerkl but differ in the expression of Cerkl, which was highly expressed in the brain but barely detected in the retina. We also found that Cerkl was expressed as early as embryonic day 14 in the mouse eye. Bioinformatic analysis of the human and mouse CERKL genes showed a strong homology between the exons. RT-PCR amplification of mouse retinal RNA revealed four different Cerkl splice-variants. The four isoforms differ in the length of the expected protein products and the existence of the DAGK and CERK domains. The purified antibody that was generated is being further used in western blot and immuno-staining to characterize the expression pattern in the mouse retina.

Conclusions: Cerkl expression begins early in the development of the mouse eye. The four different Cerkl splice-isoforms in the mouse retina may represent various roles of the protein in the eye. Studies of CERKL spatial and temporal expression patterns and characterization of CERKL biochemical properties will advance our knowledge regarding the role of this protein in retinal function and disease.

LAMIN A IS NOT ASSOCIATED WITH AGE-RELATED NUCLEAR CATARACT

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Introduction: Mutations in the LMNA gene encoding lamins A/C are responsible for premature aging syndrome in Hutchinson-Gilford progeria. The most prevalent HGPS mutation, Glycine GGC to Glycine GGT in codon 608 of the lamin A, activates a cryptic splice donor site to produce abnormal lamin A. Cell nuclei from old healthy individuals acquire defects similar to those of HGPS patient cells. Previous studies have shown the presence of Lamin C in both adult and fetal lens cells. However, only Lamin C dimers are observed in fetal lens cells, whereas adult human lens contained dimers, monomers and degraded Lamin C.

Patients / Methods: The purpose of our study is to investigate mutation analysis and level of expression of the truncated LMNA gene in age-related human cataracts.. Methods: Anterior lens capsule material was collected during surgery from 178 patients with senile cataracts. mRNA and DNA were extracted, amplified by polymerase chain reaction, and sequenced for the LMNA gene. DNA (137 samples) and cDNA (43 samples) were screened for polymorphism in LMNA gene G608G, using the novel technique of the sequenom.

Results: One mutation was found in the cDNA sample. No expression of truncated Lamin was noted in mRNA analysis.

Conclusions: In contrast to findings in progeria, the human age-related nuclear cataract is not associated either with LMNA mutations or the truncation of lamin A protein.

FINE MAPPING FOR THE POSTERIOR POLAR CATARACT GENE IN MOROCCAN JEWS

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Background: Posterior polar cataract (PPC) is a clinically distinctive opacity located at the back of the lens. It is commonly acquired in age related cataract, and may infrequently occur in pedigrees with congenital cataract. Previously we assigned the disease gene in Moroccan Jewish families to an 11.3 cM interval on chromosome 14q22-23.

Purpose: To narrow down the disease gene interval and clone the PPC gene.

Methods: We obtained DNA samples from 6 unrelated Moroccan Jewish PPC patients, and genotyped polymorphic markers from the previously identified disease interval.

Results: We were able to narrow down disease interval to 2.0 cM. Gene sequencing is underway.

Conclusions: An as yet unidentified gene associated with posterior polar cataract maps to the long arm of chromosome 14q22.

C3 POLYMORPHISM IS ASSOCIATED WITH INCREASED RISK FOR AGE RELATED MACULAR DEGENERATION IN THE ISRAELI POPULATION

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Introduction: Purpose: Complement activation has been implicated in the pathogenesis of age related macular degeneration (AMD) and variants in genes encoding complement proteins among them complement factor 3 (C3) were associated with AMD. The rs2231099 single nucleotide polymorphism (SNP) in the gene has recently been associated with increased risk for AMD. We aim to evaluate the association between this SNP and AMD in the Israeli population and its association with phenotype and response to therapy of neovascular AMD (NV-AMD).

Patients / Methods: DNA was collected from 250 NV-AMD patients and 162 age-matched controls. Genotyping for rs2231099 SNP was performed by a TaqMan assay and confirmed using sequencing. Genotyping results were correlated with phenotype and response to PDT.

Results: Heterozygosity for rs2231099 (OR = 1.8, 95% CI = 1.1-2.7, P = 0.01) and the rs2231099 risk allele (OR = 2.2, 95% CI = 1.5-4.2, P = 0.00001) were associated with AMD. There was no association between age of onset of NV-AMD, lesion type according to fluorescein angiography, initial visual acuity, or response to PDT and rs2231099 genotypes.

Conclusions: The rs2231099 variant in the C3 gene is associated with AMD in the Israeli population. This finding supports the involvement of C3 protein in AMD. Yet, this C3 variant does not make a major contribution to the variability in phenotype and response to PDT which characterize NV-AMD.

GENETIC ANALYSIS OF FAMILIAL KERATOCONUS IN THE JEWISH POPULATION OF THE NEGEV

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Introduction: Keratoconus is a slowly progressive degenerative disorder of the cornea. It is expressed by thinning and bulging of the cornea, which causes irregular astigmatism with severe reduction of vision. The prevalence of keratoconus is 50-230:100 000 in different ethnic groups.. Most cases of keratoconus are sporadic but several works have showed a genetic basis for this disorder. According to the literature the disease could be transmitted as an autosomal dominant trait with incomplete penetrance or variable expression or autosomal recessive. The rarity of familial cases complicates the genetic mapping of keratoconus. The purpose of this study is to find if there is a genetic basis for keratoconus in the Jewish population of Negev and if the inheritance fashion is similar to that, described in the literature.

Patients / Methods: Thirty seven patients from 4 families participated in the study. All participants had complete ophthalmologic examination, including corneal topography and Eye-Sys. Topographic indices that are used in the diagnosis of keratoconus, including the subclinical cases (form fruste disease), were calculated for each patient. The indices chosen for the diagnosis included: ISI – inferior-superior index and CTI –corneal thickness index. The pedigrees of the studied families were constructed. Blood samples were collected from the study participants and the DNA was extracted. Polymerase chain reaction of DNA of the studied subjects was performed with microsatellite markers, which are located in areas suspected for the keratoconus gene location.. PCR products were separated on polyacrylamid gel by Silver staining method.

Results: In the four examined families keratoconus was diagnosed in all generations, which indicates an autosomal dominant inheritance. In the studied families some loci previously reported in keratoconus were found on chromosomes 2, 16, 20. In Family 4, the largest studied family (included 16 subjects) a specific locus on chromosome 20 was found (chr20:26,014,910-30,040,628). The locus found in our study is narrower, than previously reported (chr20:24,181,755 -43,420,563)

Conclusions: The genetic basis for keratoconus in the Jewish population of the Negev seems to be similar to that described in the Italian population (autosomal dominant with variable expression, 20p11.2 locus). It, however, requires additional studies including genome-wide search with polymorphic DNA markers and further mapping in suspected areas.

THE ROLE OF ABCA4 IN HEREDITARY RETINAL DISEASES IN THE ISRAELI POPULATION

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Introduction: Hereditary retinal diseases exhibit a wide range of retinal phenotypes caused by mutations in over 200 genes. The role of ABCA4 in retinal dystrophies has been studied extensively in Western populations. It is the major, and to date the only, cause of recessive Stargardt. In this study we aim to investigate the role of ABCA4 in Israeli patients with inherited retinal diseases. Patients and Methods: Patients from 126 families with a diagnosis of CD/CRD or Stargardt were recruited. Clinical evaluation included a detailed family history, a full ophthalmologic exam, assessment of refractive error, and electroretinography. Blood samples were collected and genomic DNA was extracted. ABCA4 was screened for mutations by the Asper ABCR chip or by direct sequencing of specific exons.

Results: Patients from 35 families were screened for all previously reported ABCA4 mutations. At least one causative mutation was identified in 14 of the families (40%). The most common mutation identified was G1961E and we therefore screened exon 42 (contains the G1961 codon) for mutations in the remaining families. The analysis revealed a high number of G1961E alleles among Israeli families: five patients were homozygous and 19 were heterozygous for G1961E (19%). Interestingly, G1961E was found in families from 6 different ethnic backgrounds (including both Jewish and Muslim patients), with the highest percentage in Ashkenazi Jewish families. Aiming to estimate the carrier frequency in different ethnic groups, we screened 321 healthy individuals and found relatively high carrier frequencies of 1/18 among Ashkenazi, Oriental, and North-African Jewish individuals. Overall, we have identified in this study 13 ABCA4 disease-causing mutations in 38 families.

Conclusions: Our results indicate that ABCA4 is the most common retinal disease gene in the Israeli population. Moreover, a single ABCA4 mutation, G1961E, is common in the Jewish population (about 10 times higher compared with Western populations) with a very high carrier frequency. Is it still unclear whether homozygosity for G1961E leads to retinal disease, since our results indicate that about 10,000 homozygous individuals are expected in Israel, while only 5 have been genetically identified by us so far.

AN ANCIENT FOUNDER SPLICE-SITE RPE65 MUTATION IN NORTH-AFRICAN JEWISH PATIENTS WITH LEBER CONGENITAL AMAUROSIS

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Introduction: Isolated populations are often characterized by the existence of founder mutations that are population-specific. As part of our study of the genetics of inherited retinal diseases in Israel, we identified a relatively large number of retinal degeneration patients from North African Jewish descent. The aim of the current study was to identify the genetic cause of retinal disease in patients from North African Jewish origin who suffer from autosomal recessive (AR) Leber congenital amaurosis (LCA) or early-onset retinitis pigmentosa (RP).

Patients / Methods: Fifty-three families with the diagnosis of AR-LCA or ARRP were recruited for the study. Blood samples were drawn from family members and genomic DNA was analyzed by mutation detection microarray analysis, direct sequencing, and 10K whole genome single nucleotide polymorphism (SNP) markers.

Results: We identified a homozygous splice site mutation, c.95-2A>T (IVS2-2A>T), in the RPE65 gene in seven families, all originated from the North African Jewish population. All affected individuals were homozygous for this mutation and suffered from early-onset and severe retinal degeneration diagnosed clinically as either LCA or early-onset RP. The mutation was not found in North African Jewish patients with ARRP nor in LCA or RP patients from other ethnic origins. These findings suggest a North African specific founder mutation. Haplotype analysis using SNP markers in the vicinity of RPE65 revealed a shared homozygous region in seven patients. Using the DMLE program, the age of the founder mutation was estimated as 100-130 generations ago. To evaluate the carrier frequency of the IVS2-2A>T mutation, we screened 179 origin-matched control individuals and identified two carriers (estimated carrier frequency of ~1.1%).

Conclusions: The age of the RPE65 founder mutation suggests that it originated around the establishment of the Jewish community in North Africa (about 2000-2600 years ago) and is currently the most common identified cause of early retinal degeneration in this population. The results of this study will allow identification of carriers in the community and provide the basis for better genetic counseling and disease prevention. Perhaps more importantly, the 21 patients we have identified who are homozygous for this RPE65 mutation may be candidates for the novel RPE65 gene therapy now available.

PREVALENCE OF CHRONIC DISEASES AMONG PATIENTS UNDERGOING CATARACT SURGERY

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Introduction: It is generally acknowledged that age-related (senile) cataract is a multifactorial disease. Epidemiologic studies of this disease have suggested many risk factors for cataract. The purpose of this study was to evaluate the prevalence of chronic conditions among patients undergoing cataract surgery.

Patients / Methods: We looked at all patients that underwent cataract surgery in the central district of Clalit HMO in Israel that were members in the HMO between 2001-7 (n= 12,984) and compared them to 25,968 age and gender matched controls. All chronic medical conditions that were on the patients' computerized files were collected. We calculated the prevalence of chronic conditions among the cataract patients and the age and gender matched controls.

Results: The most prevalent conditions associated with cataract surgery were: glaucoma (OR=2.1, 95% CI= 1.9-2.2), organ transplant (3.2, 1.8-5.8), amyloidosis (3.4, 1.8-6.6), familial mediteranean fever (2.7, 1.6-4.5), autoimmune diseases (2.0, 1.5-2.5), diabetes (1.5, 1.4-1.58), cardiovascular diseases (1.35, 1.3- 1.4) and smoking (1.3, 1.2- 1.35).

Conclusions: Various systemic conditions are significantly more common among patients undergoing cataract surgery. Detecting such conditions might elucidate the mechanism of cataract creation and prevention.

PROGNOSTIC FACTORS IN POSTERIOR OPEN GLOBE INJURIES (ZONE-III INJURIES).

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Introduction: To describe and identify clinical characteristics, prognostic factors and visual outcome in a group of patients with posterior open globe injuries (zone III injury).

Patients / Methods: A retrospective review was made of all cases of open globe injuries that were examined at the Ophthalmology Department of Soroka Medical Center, Beer-Sheva, Israel, from 1995 to 2005. One hundred and eight consecutive patients suffering open globe injuries were detected. Among them, twenty-one eyes of 21 patients suffering from zone III injury were identified, studied and analyzed statistically. Data recorded included demographic data, cause of injury, initial visual acuity (VA), associated globe morbidity and injuries, Ocular Trauma Score (OTS), surgical procedures, postoperative complications and final VA.

Results: The study group comprised 95% male subjects with a mean age of 35.8 years (range 20–60 years). The median follow-up was 21.2 months (range 6–66 months). In 72.7% of the cases metal was the causative factor. Clinical signs associated with poor visual outcomes included initial VA, eyelid injury, cornea lamellar lacerations or abrasions, iris deformity, lens damage, ocular hypotony, coexisting injuries and low OTS (≤ 2). No cases presented with posttraumatic endophthalmitis.

Conclusions: From this study we conclude that the most important prognostic factor in zone III open globe injury is initial VA. Other prognostic factors associated with worse final outcome are eyelid injury, iris deformity, lens damage, ocular hypotonia, coexisting injuries and low OTS (≤ 2). Posterior open eye injuries were most commonly due to metal entering the eyes of young men.

SEMA-3A DISRUPTS THE REGENERATION PROCESS OF GOLDFISH AND RAT OPTIC NERVES FOLLOWING CONTROLLED INJURY

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Introduction: Retinal ganglion cells (RGC) in mammals degenerate following injury to their axons, as do other CNS neurons. In sharp contrast, RGC of lower vertebrates such as fish do regenerate, and are therefore capable of restoring vision after injury. Regeneration is a complex multi-factorial process that requires a permissive environment. Semaphorin-3A (Sema-3A) is known for its repellent and apoptotic activities in the nervous system and was previously shown to be involved in post-traumatic injuries in the CNS. We investigated its role in degeneration in the fish and in mammals.

Patients / Methods: Western blot and immunohistochemistry were used to evaluate Sema-3A, macrophage invasion to the lesion site, and myelin clearance, in injured optic nerve (ON) in goldfish. Axonal regeneration was examined by retrograde and anterograde labeling with di-Asp and dextran respectively. Following ON axotomy in a rat, anti-Sema3A antibody was applied by intravitreal injection and axonal regeneration was monitored by retrograde labeling with cholera-toxin.

Results: We found a decrease in Sema-3A levels in the retina of goldfish at an early stage after injury but no change in Sema-3A levels in the injured optic nerve. Intravitreal injection of Sema-3A to goldfish shortly after optic nerve injury led to destructive effects on several pathways of the regenerative processes, including the survival of retinal ganglion cells, axonal growth and clearance of myelin debris from the lesion site by macrophages. Injection of anti-Sema3A antibodies to a rat eye following axotomy promoted axonal regeneration.

Conclusions: These data support our previous findings and further validate the notion that Sema-3A is a key factor in the generation of a non-permissive environment following transection of the optic nerve. Neutralization of Sema-3A in injured mammalian optic nerve can enhance axonal regeneration.

HOLOGRAPHIC DYNAMIC CONTROL OF NEURONAL POPULATIONS IN THE RETINA

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Introduction: Retinal prostheses for patients with outer-retinal degenerative diseases could interface directly with surviving retinal neurons using electrode array implants. Direct optical stimulation using light-gated ion channels, has recently been introduced as an alternative method for spatially and temporally precise, minimally-intrusive control of neurons. However, to generate activity patterns that will be translated into a meaningful perception in the brain requires methods that can selectively excite a large population. Here we introduce the development and in-vitro implementation of a novel projection/excitation strategy that can be used to selectively control large retinal neuronal populations, with high temporal precision (msec) and efficient use of light power

Patients / Methods: Of existing display technologies, digital holographic projection ideally meets these constraints, because the use of phase-modulating spatial light modulators (SLMs) and light diffraction allows an efficient use of input light. Our system directs light from Blue, Green and Red DPSS Lasers onto a Ferroelectric liquid crystal SLM that displays binary holograms. Light patterns were coupled into the camera port of an inverted microscope and projected onto retinas, whose responses were measured using a Multi-Electrode Array (MEA).

Results: We demonstrate for the first time responses of a population of retinal ganglion cells to patterns of light holographically projected unto wild-type and ChannerhodopsinII transfected retinas. The neurons exhibit spatially-selective responses and have effective receptive fields. We demonstrate sub-millisecond timescale control over the projected light patterns, multi-wavelength excitation, and computational strategies that eliminate the effect of speckle.

Conclusions: High-rate holographic projection was demonstrated as an enabling photo-stimulation modality towards the development of a retina neuro-prosthetic, theoretically capable of eliciting over 1 million spikes per second, with millisecond timing precision. Our system can also be applied in experimental studies of the visual system requiring ultra-high-rate stimulus control.

INTRAVITREAL INJECTION OF CILIARY NEUROTROPHIC FACTOR TO ENHANCE IN SITU DIFFERENTIATION OF STEM-CELL-FACTOR-MOBILIZED BONE-MARROW-DERIVED STEM CELLS

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Introduction: We previously observed the efficient incorporation of intravenously or intravitreally injected adult bone-marrow-derived stem cells (aBMSCs) in the retina following anterior ischemic optic neuropathy (AION). Furthermore, independent homing of green-fluorescent-protein (GFP)-labeled cells from the bone marrow was enhanced with granulocyte macrophage colony stimulating factor (GM-CSF). However, the cells differentiated before homing and incorporation and expressed microglial markers. In the present study, we assessed the effect of stem cell factor, a mobilizing but not a differentiating agent, in this setting. In situ differentiation was further enhanced by intravitreal injection of ciliary neurotrophic factor (CNTF).

Patients / Methods: Syngeneic bone marrow cells (BMCs) expressing constitutive GFP were transplanted into mice. Three months after hematopoietic reconstitution, and one day before induction of AION, the BMCs were mobilized with SCF (5 consecutive daily doses). Immediately after AION induction, CNTF was administered to the vitreous of the injured eyes.

Results: GFP-positive BMCs were incorporated preferentially in the injured eye, adjacent to the retinal ganglion cell layer. The incorporated cells adopted mainly glial and endothelial phenotypes. Intravitreal injection of CNTF somewhat enhanced glial differentiation, as detected by immunostaining for the astrocyte marker, glial fibrillary acidic protein.

Conclusions: SCF, a potent hematopoietic mobilization agent, enhances BMC homing and incorporation without the microglial differentiation noted with our earlier use of GM-CSF mobilization. The cells adopted glial and endothelial phenotypes. Intravitreal injection of CNTF had a minor effect on differentiation.

EXPRESSION AND LOCALIZATION OF CLAUDINS, TIGHT JUNCTION MEMBRANE PROTEINS, IN THE MOUSE EYE

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Introduction: Tight junctions (TJs) are circumferential strands around cells that selectively modulate paracellular permeability between extracellular compartments and are also involved in maintaining cellular polarity. The main components of TJ strands are members of the claudin family which include more than 20 highly conserved proteins. Claudins are expressed in various tissue including the eye. The mammalian eye is a complex organ containing several distinct tissues (e.g., lens, cornea, retina, and iris), each performing specialized functions to detect a visual image. This kind of structure obligates restrict barriers, and therefore, the expression of a wide variety of claudins. The importance of claudins in the eye was demonstrated in people with severe ocular abnormalities caused by mutations in CLDN19.

Patients / Methods: The expression and distribution of claudins in the eye was examined by RT-PCR and immunostaining techniques.

Results: RT-PCR analysis of the mouse eye revealed changes in claudin expression pattern in the adult eye comparing to the embryo (E14). Claudin 1, -2, -3, -4, -5, -7, -10, -11, -12, -13, -19 and -23 are expressed in the adult, while in the embryo, claudins 6, -8 and -16 were also detectable. By immunofluorescence staining we determined the localization of the various claudins in the adult mouse eye. Claudin 1 and -10 are expressed in the retinal pigmented epithelium and the ciliary body. In the cornea claudin 2 and -4 were detectable. The lens epithelium was positive only for claudin 5, which also localized in the choroid and the optic nerve. The optic nerve was also positive for claudin 11, which was detectable also in the retina. At least 3 other claudins are expressed in the retina. These include claudin 3, -7 and -12.

Conclusions: Determination of claudin expression profile in the eye will lead to a better understanding of the mechanisms underlying the eye function and physiology and reflect the importance and complexity of the barrier function of TJs in the eye.

THE EFFECT OF TOPICAL STEROIDS ON BLOOD GLUCOSE PROFILE IN DIABETIC PATIENTS

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Introduction: Our previous study evaluated the effect of dexamethasone eyedrops on blood glucose profile following cataract operations and demonstrated that postoperative dexamethasone eye drops have a greater effect on the blood glucose profile of diabetic versus nondiabetic patients. The purpose of the present study was to investigate the effect of topical steroidal eye drops on blood glucose levels and glycemic control among diabetic patients.

Patients / Methods: A retrospective observational cohort study was performed. We reviewed the electronic medical records of all the diabetic members in the district of the largest health maintenance organization in Israel (the Central District of Clalit Health Services). We documented all steroidal eye drops prescriptions (n=44,118) filled by diabetic patients in the district between 1/1/98 and 31/7/06. We included only those patients that filled at least 3 consecutive prescriptions (n=2697 patients). Main Outcome measures included the relationship of topical steroidal eye drops prescription use on blood glucose levels and Hemoglobin A1C levels among diabetic patients.

Results: The baseline fasting glucose level was 145.8 ± 3.7 (SEM) mg% and HbA1c $7.5 \pm 0.16\%$. Fasting blood glucose levels increased up to 160 ± 5.7 mg% on day 10 following topical steroid treatment. HbA1c increased to $8.1 \pm 0.2\%$ around day 40 under topical steroids treatment.

Conclusions: The use of topical steroids by diabetic patients appear to increase blood glucose levels and interfere with glycemic control. Clinicians should be alerted to this risk and may initiate appropriate follow-up in this patient subgroup.

MODULATION OF VEGF EXPRESSION BY RPE CELLS DEPENDS ON THE MODE OF α V INTEGRIN INHIBITION

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Introduction: Alpha v integrins, α v β 3 and α v β 5, are implicated in both normal angiogenesis and neovascular ocular diseases. Inhibition of these integrins is a useful approach to modulation of pathological angiogenesis. We investigated the role of α v integrins in the regulation of expression of proangiogenic and antiangiogenic factors by RPE cells.

Methods: Cell cultures: ARPE-19 and EAhy.926 endothelial cells (EC). mRNA levels were examined by real-time PCR arrays and validated by real-time-PCR (RT-PCR). VEGF and α v protein expression were tested by ELISA and flow cytometry, respectively.

Results: Comparison between RPE and EC revealed much higher VEGF levels in RPE cells, as compared to EC: 1000 fold and 5 fold higher at mRNA by a specific antibody and protein levels, respectively. Inhibition of α v β 3 (LM609) resulted in a 10 fold downregulation of VEGF mRNA level. mRNA levels of other major proangiogenic factors, as well as that of PEDF, the major antiangiogenic factor of RPE, remained unchanged. We further studied the role of RPE α v integrins by silencing the α v chain using siRNA. Using this approach, we obtained 50-80% suppression of α v mRNA, and about 90% suppression of α v protein. v resulted in a significant α synthesis. Surprisingly, genetic suppression of upregulation of mRNA levels of the major proangiogenic factors VEGF (1.8 fold) and angiopoietin (2 fold), and downregulation of PEDF (1.7 fold). VEGF protein level was also upregulated (1.9 fold) in α v suppressed RPE cells.

Conclusions: Our results demonstrate that in RPE cells, VEGF expression is reduced by pharmacological inhibition of α v β 3, and enhanced by siRNA suppression of α v integrins. Thus, modulation of retinal VEGF levels by blocking α v integrins by specific inhibitors might prove an efficient approach to treatment of neovascular diseases, as opposed to genetic ablation of these integrins.

RETINAL NERVE FIBER LAYER THICKNESS IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA SYNDROME (OSAS).

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Introduction: To study retinal nerve fiber layer (RNFL) thickness in patients with obstructive sleep apnea syndrome (OSAS).

Patients / Methods: 120 patients with OSAS were analyzed and compared with an age-matched control group of 104 subjects. Patients with OSAS underwent sleep studies to determine the respiratory disturbance index (RDI) during night sleep and were classified as having moderate (RDI, 20-39), and severe (RDI > 40). The RNFL thickness was assessed by optical coherence tomography (StratusOCT) using the fast RNFL thickness (3.4) scan acquisition protocol.

Results: Compared with the control group, eyes with OSAS showed a thinner RNFL in the 360 degrees average measurement ($101.52 \pm 11.3 \mu$, $101.38 \pm 11.3 \mu$ for control right eye and left eye respectively and $97.86 \pm 12.03 \mu$, $96.5 \pm 12.13 \mu$ for OSAS in the right eye and left eye respectively, $p=0.02$) and in the superior ($p=0.034$), inferior ($p=0.03$) quadrants in the right eye and superior ($p=0.03$), temporal ($p=0.02$) quadrants in the left eye. There are no correlations between the RDI and any of the RNFL measurements on the SAS eyes.

Conclusions: RNFL thickness was not correlated with the severity of OSAS however the thickness of RNFL was reduced in patients with OSAS compared to controls by less than 5μ (5%). The clinical significance of this change needs to be determined.

Session IV - Genetics

CONE DYSFUNCTION AND CONGENITAL DAY BLINDNESS IN AWASSI SHEEP IS CAUSED BY A MUTATION IN THE CNGA3 GENE

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Introduction: To study a flock of Improved Awassi sheep in which lambs with congenital impaired day vision were reported.

Patients / Methods: Epidemiology of the flock was studied through breeding records. Six affected lambs and six age-matched normal controls were selected for detailed functional investigation. Behavioral changes were quantified by measuring maze passage time under scotopic and photopic conditions. Flash electroretinography (ERG) was used to investigate rod and cone function. Light microscopy and immunohistochemistry were used to study retinal structure. The coding regions of the CNGA3, CNGB3, and GNAT2 genes, known to cause achromatopsia in humans, were sequenced using RNA extracted from the retinas of 4 affected and 8 non-affected lambs; design of the PCR primers was based on the orthologous bovine genes.

Results: Out of 7,099 lambs that were born in the flock between 11/2004-6/2008, 124 lambs (1.7%) were diagnosed with impaired vision. The genealogy of the affected lambs suggested an autosomal recessive mode of inheritance. Daytime maze passage time of affected animals was significantly longer than normal controls, but there were no differences between groups in scotopic maze passage time. Electroretinography showed normal rod function and diminished, but not abolished, cone function in affected animals. Histological and immunohistochemical evaluation of affected retinas revealed the physical presence of both red-green and blue cones in large numbers at least up to the age of 7 months, suggesting that the behavioral day blindness and reduced cone ERGs reflect cone dysfunction rather than severe cone photoreceptor loss. Affected lambs were found to be homozygous for a single nucleotide substitution in the cone photoreceptor cGMP-gated channel alpha-subunit (CNGA3) gene, changing amino acid R236 to a stop codon.

Conclusions: Our results show that a mutation in the ovine CNGA3 gene causes impaired daytime vision and diminished cone function in sheep while cone photoreceptors are histologically present in large numbers at least until the age of 7 months. We propose that affected sheep can serve as a naturally-occurring large animal model for human achromatopsia, including the study of gene therapy for this disease.

GENETIC ANALYSIS OF BEST DISEASE IN THE ISRAELI AND PALESTINIAN POPULATIONS

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Introduction: Vitelliform macular dystrophy (Best disease) is a hereditary form of macular degeneration that usually begins in childhood or adolescence. The disease is characterized by typical "egg-yolk" macular lesions caused by abnormal accumulation of lipofuscin within and beneath the retinal pigment epithelium (RPE) and by a severely reduced Arden ration on electro-oculography. Progression of the disease leads to destruction of the RPE and loss of central vision. Best disease was considered to be inherited in an exclusively autosomal dominant (AD) manner, but lately autosomal recessive (AR) inheritance has been reported in a few families. Mutations in the BEST1 (VMD2) gene are associated with this disorder. The goal of the current study was to characterize the phenotype and genotype in Israeli and Palestinian families with Best disease.

Patients / Methods: Clinical evaluation included detailed family history, a full ophthalmologic exam, electrooculography, full-field electroretinography, and optical coherence tomography. Blood samples were obtained from patients and unaffected family members and direct sequencing of PCR products covering the 11 exons of BEST1 has been performed.

Results: Among the 710 families we recruited so far with inherited retinal disease, 14 carried the clinical diagnosis of Best disease. Sequencing analysis of the BEST1 open reading frame revealed eight different missense mutations in eight of the 14 families. Five of the mutations are novel and inherited in an AD pattern. The remaining three mutations have been previously described in families with AD Best disease. However, interestingly, we identified two of these mutations in families with an AR inheritance pattern: carriers had a normal phenotype while the affected patients were homozygous for the causative mutation. One of them had an atypical early-onset and severe form of Best disease. A search for possible modifier alleles is in progress.

Conclusions: Five novel BEST1 mutations were identified in our populations and the results suggest that a number of heterozygous individuals also carry as yet unidentified modifier/protective alleles. Future studies of BEST1 as well as other genes may lead to the identification of these alleles, which could serve as good candidates for "gene protection therapy" in the context of an AD disease.

THE SPECTRUM OF RETINAL DISEASE CAUSED BY NR2E3 MUTATIONS IN ISRAELI AND PALESTINIAN PATIENTS

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Introduction: The NR2E3 gene encodes a nuclear receptor transcription factor that is involved in photoreceptor differentiation. NR2E3 mutations were shown to be the cause of different clinical diagnoses such as enhanced S-cone syndrome (ESCS), Goldmann-Favre syndrome (GFS), and clumped pigmentary retinal degeneration (CPRD). Our purpose was to evaluate the involvement of NR2E3 in retinal degenerative diseases in the Israeli and Palestinian populations.

Patients / Methods: Patients from 35 families with various autosomal recessive retinal degenerative diseases were recruited. Clinical evaluation included a full ophthalmologic exam and full-field electroretinography (ERG). Haplotype analysis was performed by studying single nucleotide polymorphisms within the NR2E3 gene. A screen for mutations was performed by direct sequencing of PCR products.

Results: We recruited six consanguineous Muslim families and two Jewish families with a clinical diagnosis of ESCS, GFS or CPRD. Patients from four of the Muslim families were homozygous for the same NR2E3 mutation, c.119-2A>C, but presented considerable variability in fundus appearance and retinal function even among patients of comparable ages. Funduscopic findings ranged from mild changes which included elongated retinal flecks with preserved foveal structure through the presence of multiple spots of deep chorioretinal atrophy with cystoid macular edema and culminating in a young patient with perimacular atrophy, classic pigment clumps beyond the arcades and severe cystoid macular edema/schisis. Two additional patients further expand the clinical spectrum associated with this same mutation, and were diagnosed with GFS in view of significant vitreous involvement. The ERG findings in these two patients differed markedly from the other patients with the same mutation and from the classic ESCS findings, with severely reduced responses under all stimulus conditions. Interestingly, homozygosity analysis in 27 additional consanguineous families with a clinical diagnosis of retinitis pigmentosa (RP) revealed a different homozygous NR2E3 mutation, c.932G>A, in two families. The ERG responses in these patients were indeed compatible with RP and also did not show the characteristic ESCS pattern.

Conclusions: Our results indicate that patients with NR2E3 mutations may manifest variable phenotypes ranging from ESCS to RP. Moreover, patients who are homozygote for the same NR2E3 mutation can have variable expression of retinal disease, suggesting the involvement of modifier genes.

INTRAFAMILIAL GENETIC HETEROGENEITY IN ISRAELI CONSANGUINEOUS FAMILIES WITH RETINAL DEGENERATION

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Introduction: Hereditary retinal dystrophies (HRDs) are a heterogeneous group of diseases, which cause visual loss due to the premature death of rod and cone photoreceptors in the retina. Retinitis pigmentosa (RP), characterized by night blindness followed by visual-field loss, is the most common form of HRD. In Leber congenital amaurosis (LCA), the most severe form of HRD, both rods and cones have degenerated or are nonfunctional at birth, or are lost within the first years of life. Besides being clinically heterogeneous, HRDs are also genetically heterogeneous. At least 37 genes and loci have been implicated in nonsyndromic autosomal recessive RP and/or LCA.

Patients / Methods: Several consanguineous Muslim Arab families from Northern Israel segregating early-onset HRD were analyzed by haplotype analysis for all known genes and loci underlying autosomal recessive nonsyndromic RP and LCA, followed by whole-genome homozygosity mapping approach, with the Affymetrix GeneChip Human Mapping 250K SNP Array. Mutation screening of the underlying genes was performed by direct sequencing.

Results: Despite the high degree of consanguinity in each family, we found allelic heterogeneity in one of them, in which affected individuals were compound heterozygotes for two different mutations of the *CRB1* gene. In two additional families we found evidence for locus heterogeneity. In one of these families a novel homozygous mutation of *RDH12* was found in 14 of 17 affected individuals. Our data indicate that in the other affected individuals the disease is caused by a different gene/s.

Conclusions: Our findings demonstrate that while homozygosity mapping is an efficient tool for identification of the underlying mutated genes in inbred families, both locus and allelic heterogeneity may occur even within the same consanguineous family. These observations should be taken into account, especially when studying relatively common and highly heterogeneous conditions, such as HRD.

RELATIVE IMPORTANCE OF MAJOR RISK SINGLE NUCLEOTIDE POLYMORPHISMS (SNPS) FOR THE DEVELOPMENT OF AGE RELATED MACULAR DEGENERATION (AMD) IN ISRAEL

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Introduction: To evaluate the contribution of risk SNPs for the development of AMD in the different ethnic groups composing the Israeli population and to assess its discriminatory accuracy.

Patients / Methods: Genotyping for rs10490924 in LOC387715, Tyr402His variant in complement factor H (CFH), and rs2231099 in complement component 3 (C3) was previously performed in 255 AMD patients and 119 controls. For the purpose of this study, the Population Attributable Risk (PAR) for each SNP and its association with AMD in the major ethnic groups composing the Israeli population was calculated. Discriminatory accuracy was evaluated using Receiver Operating Characteristic (ROC) curve analysis.

Results: PAR was 24.5%, 44.9%, and 47.9% for the C3, CFH, and LOC387715 SNPs, respectively. The SNP in CFH was associated with AMD among Ashkenazi Jews [Odds Ratio (OR) = 2; 95% confidence interval (CI) = 1.3-3.1; $p = 0.003$]. There was a trend towards such an association among Sephardic Jews. The LOC387715 SNP was associated with AMD among Ashkenazi [OR (95% CI) = 2.9 (1.8-4.7); $p < 0.0001$] and Sephardic Jews [OR (95% CI) = 3.5 (1.9-6.6); $p < 0.0001$], and among Arabs [OR (95% CI) = 6 (1.5-24.7); $p = 0.022$]. ROC analysis showed area under the curve (AUC) of 0.6, 0.68, and 0.63 for the variants in C3, CFH, and LOC387715, respectively.

Conclusions: Majority of cases of AMD in Israel are attributable to the SNPs in CFH, LOC387715, and C3. The LOC387715 is a major risk factor for the disease in the ethnic groups composing the Israeli population while the CFH variant shows stronger association with the disease among Ashkenazi Jews. By itself, none of the SNPs may serve as a diagnostic test for AMD.

GENOTYPE AND ETHNICITY CORRELATIONS IN CONGENITAL GLAUCOMA IN ISRAEL

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Introduction: Mutations in the CYP1B1 gene are frequently found in Primary Congenital Glaucoma (PCG) patients. We examined the role of CYP1B1 mutation in Muslim Arabs, Druse and Jewish patients with PCG in Israel.

Patients / Methods: The entire coding regions of CYP1B1 gene was screened in 86 individuals from 22 families of 29 patients with PCG including 7 Muslim Arabs families (12 patients), 7 Druse families (9 patients) and 8 Jewish families (8 patients). First line screening was done by the DHPLC apparatus followed by sequencing of the DNA. CYP1B1 mutations were correlated with the severity of disease (age at diagnosis, difficulty in achieving intraocular pressure control).

Results: A mutation in CYP1B1 was found in 12(54%) families (18 patients): 71% (5/7) Muslim Arabs, 71% (5/7) Druse, 25% (2/ 8) Jews. The most frequent mutation was Arg469Trp found in 4/9 Muslim Arabs and 5/7 Druse patients with CYP1B1 mutation but not in the Jews. Three patients were compound heterozygous. Muslim Arab with two missense mutations (E229K, paternal and R368H, maternal); Druse with one missense mutations (MIT, maternal) and one frame-shift (Pro289 ins C, paternal); Druse with one missense mutations (MIT, maternal) and Arg469Trp (paternal). The phenotype of the disease was severe in 75% (9/12) of the Muslim Arabs and 89% (8/9) of Druse patients while Jews had a mild disease (75%, 6/8). Disease severity closely correlated with the type of the mutations in the CYP1B1 gene.

Conclusions: The majority of Muslim Arabs and Druse with PCG in Israel had mutations in the CYP1B1 gene and had a severe disease. Jews had a mild disease and may have mutations in genes yet to be identified. The variation of PCG phenotype among different ethnic populations may be explained by the variability in genotype.

Session V – Retina I

EFFECT OF INTRAVITREAL BEVACIZUMAB (AVASTIN) ON THE GROWING RABBIT EYE

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Introduction: Diode laser photocoagulation is currently the preferred treatment for ROP, with an anatomic success rate of up to 92%. However, some premature infants of very low gestational age have shown a failure to respond, with the development of aggressive posterior ROP and consequent retinal detachment. Several groups have conducted preliminary trials of intravitreal bevacizumab (Avastin®) in small series of patients with ROP. Good anatomic outcomes were reported, with no local or systemic side effects. The possible systemic and ocular effects of intravitreal bevacizumab in premature neonates are not known, and there are no studies of ocular growth following intravitreal bevacizumab injection. The purpose of the present study was to examine the effect of intravitreal bevacizumab injections on the structure and growth of the young rabbit eye.

Patients / Methods: Twelve 6-week-old rabbits underwent intravitreal injection of bevacizumab (1.25 mg/0.05 ml) in one eye and no injection in the other (study group). Six additional rabbits served as controls. Slit-lamp examination, fundus evaluation, and measurements of corneal power, central corneal thickness, corneal astigmatism, and axial length were performed before treatment and 1, 2, and 3 months after. At 3 months, the rabbits were sacrificed for histological study.

Results: Within the study group, there were no significant differences in clinical parameters between the bevacizumab-treated and fellow eyes at any time point or overall. The estimated confidence intervals extended to about one standard deviation of their respective values. Comparison of the injected eyes in the study group with the uninjected eyes in the control group yielded similar findings. There were no between-group differences in ocular growth and no abnormalities of the anterior and posterior segments or the retina and retinal vasculature.

Conclusions: Intravitreal bevacizumab apparently has no adverse effect on the growth and development of the young rabbit eye, in biologically or statistically significant way. Intravitreal bevacizumab is a promising agent for the treatment of ROP. However, caution is necessary in generalizing our results in a growing animal eye to the developing eye of premature neonates with ROP. More data in humans are needed to rule out possible local and systemic effects.

EVALUATION OF THE METABOLIC EFFECTS OF INTRAVITREAL KENALOG IN RABBITS

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Introduction: Purpose: To evaluate and quantify the metabolic effects of a single intravitreal Kenalog (commercial triamcinolone acetonide) injection on blood glucose levels in rabbits.

Patients / Methods: Ten NZW rabbits of the same age were divided into 2 groups: Five rabbits were treated with intravitreal Kenalog (4mg/0.1ml) injected into their right eye (Study group), and five rabbits were treated with intravitreal injection of 0.1ml saline to the right eye (Control group). Serial blood glucose measurements were performed 3 and 4 day's before the injection and at 1, 2, 3, 4, 7 and 10 days after intravitreal injection.

Results: Glucose levels were higher in the rabbits injected with Kenalog compared to the control group injected with saline. A statistically significant difference in glucose level between treatment and control groups was found on day 1 (171.00 ± 14.37 vs 148.40 ± 8.47 ; $P= 0.02$) and day 2 (166.20 ± 5.26 vs 156.00 ± 5.57 ; $P=0.02$) after injection and borderline statistically significant differences in day 3 after injection (162.80 ± 8.70 vs 154.40 ± 2.79 ; $P=0.07$). At longer periods of follow-up (up to 10 days) glucose levels were similar in both groups ($p>0.1$).

Conclusions: A single intravitreal Kenalog (4mg/0.1ml) injection induces an elevation of the blood glucose during the first 3 days after injection. This supports the assumption of a systemic absorption of kenalog in rabbits.

INFLUENCE OF NON-TOXIC DOSES OF BEVACIZUMAB AND RANIBIZUMAB

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Introduction: Ranibizumab is highly effective but relatively expensive anti-vascular endothelial growth factor (VEGF). Bevacizumab, an anti-VEGF antibody approved for use in colon cancer, costs about 1/40th the price of ranibizumab. The purpose of this study was to compare the influence of bevacizumab and ranibizumab on angiogenesis in an in vitro model.

Patients / Methods: A model consisting of H5V cells derived from murine hearts and bovine aortic endothelial cells (ECs) was used. The H5V cells were treated with three concentrations of bevacizumab and ranibizumab (0.125 mg/mL, 0.25 mg/mL, and 0.50 mg/mL) for 24 hours before all experiments. The effects of bevacizumab and ranibizumab on EC proliferation were compared by ³H-thymidine incorporation assay. Toxic effects and the safety of each drug in clinical concentrations were assessed by annexin 5 staining. The effects of the drugs on EC functions were assessed by their ability to adhere to fibronectin and their tube formation capacity on matrigel.

Results: . Both bevacizumab and ranibizumab equally suppressed the adhesive properties of ECs to fibronectin, and similarly inhibited EC proliferation capacity in a dose-dependent manner. Both inhibited the ECs' tube formation capacity on matrigel, and were equally safe.

Conclusions: Ranibizumab and bevacizumab at low, non-toxic doses similarly inhibit several properties of the angiogenesis process. Inhibition of EC adhesion to fibronectin and formation capacity do not seem to be directly related to the anti-angiogenic effects as indicated by inhibition of VEGF. Further studies for delineating the exact mechanism of action of ranibizumab and bevacizumab in angiogenesis are warranted.

RISK ASSESSMENT AFTER MORE THAN 2500 AVASTIN INJECTIONS

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Introduction: To determine the incidence of complications following intravitreal injection of bevacizumab.

Patients / Methods: A total of 2536 intravitreal bevacizumab injections (2.0 mg/0.08 ml) were performed from February 2007 to march 2008. The number of the injections was determined from the injection log books. The injections were performed as an office based procedure with use of povidone-iodine, sterile drape and lid speculum as a part of preinjection preparation. Conjunctival displacement with a cotton tip was done prior to the injection. Postoperative topical antibiotics were given for 1 week. The main outcome measures were the incidence of complications. Follow-up after each injection was at least 6 weeks.

Results: Small subconjunctival haemorrhages were noted in 15 % of the eyes. No patient experienced a subtotal subconjunctival or vitreous haemorrhage. Four patients presented with uveitis. Three patients presented with iritis and one patient with vitritis, several days following intravitreal injection of bevacizumab. The patients were given topical corticosteroid therapy. The inflammation resolved in all cases within 1 to 2 weeks. There was one case of suspected endophthalmitis, this patient had an episode of BE vitritis prior to the injection. There were two cases of retinal detachment one needed pars plana vitrectomy and one had localized retinal detachment and was treated with laser barrage.

Conclusions: Although there is no consensus regarding the intravitreal injection procedure technique, the incidence of complications was very low in a large series of injected patients in an office setting and the incidence compares favorably with that reported in clinical trials. Perhaps displacing the conjunctiva before injection can lead to a lower rate of endophthalmitis.

THE EXPRESSION AND POSSIBLE ROLE OF ACETYLCHLINESTERASE (ACHE) FOLLOWING PHOTIC-STRESS IN THE MOUSE RETINA

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Introduction: Increasing evidence has shown that AChE may be involved in stress-induced retinal cells' apoptosis. Here, we tested the involvement of AChE in stress response of the retina and try to elucidate the signal transduction pathway involved.

Patients / Methods: Adult albino and pigmented mice were exposed for 2, 10, 24 hours to bright, damaging light after being two weeks in continuous darkness. The electroretinogram (ERG) was recorded to assess retinal function. AChE mRNA expression was monitored by in situ hybridization and by Real-Time PCR. Immunohistochemistry was performed to check ATF3 protein expression, and Tunnel was used to determine apoptosis. Reporter assay analysis was used for testing AChE-promoter activity.

Results: Mice kept for 2 weeks in darkness and then exposed to bright fluorescence light showed reduction of ERG responses depending upon the period of exposure. Augmented expression of AChE mRNA was found after 2 and 10 hours of exposure to light in photoreceptor inner segments, bipolar cells, and ganglion cells, compared to control mice kept in darkness and not exposed to bright light. The light-exposed mice exhibited up-regulation of ATF3 (an immediate early gene, which its mRNA and protein levels are highly induced following a variety of cellular insults), in the inner nuclear layer (INL). Apoptosis was observed only in the ONL of light exposed mice. Interestingly, ATF3 repressed AChE promoter activity in PC12 transfected cells.

Conclusions: Light-stress induces AChE mRNA expression followed by apoptosis. The significant inhibitory effect of ATF3 on AChE-promoter activity in PC12 cells, could explain our in vivo observations in which apoptosis was restricted to the outer retina, while the inner retina was probably protected by ATF3 from the AChE-induced apoptosis.

PROANGIOGENIC POTENTIAL OF MICROVASCULAR ENDOTHELIAL CELLS (EC) IS ENHANCED BY COCULTURE WITH RETINAL PIGMENT EPITHELIAL (RPE) CELLS

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Introduction: RPE cells and microvascular EC play a major role in the development of choroidal neovascularization (CNV). Our aim was to investigate the interaction between RPE and EC cells in coculture, and to explore the influence of RPE on the EC angiogenic activity

Methods: RPE and EC were grown alone or in coculture for 3-14 days, under normoxia or hypoxic conditions. Separation of EC and RPE from coculture was achieved by using antibodies specific to each cell type. EC pro- and antiangiogenic gene expression was analyzed using a Real-time PCR array containing 88 genes associated with either positive or negative regulation of angiogenesis. Tube formation assay on ECM was used to study the functional effect of EC coculture with RPE.

Results: Coculture of EC with RPE under normoxic conditions induced markedly upregulated EC mRNA expression of 10 genes involved in positive regulation of angiogenesis, among them VEGF, HIF-1 alpha, as well as collagen IV, The expression of EC VEGF was further enhanced by coculture with RPE under hypoxic conditions. Exposure of EC to hypoxia alone induced significant upregulation in the expression of the 10 genes upregulated by coculture with RPE (see Table below). Following coculture with RPE, EC demonstrated enhanced tube formation on ECM.

Table: EC genes upregulated by hypoxia alone or coculture with RPE (fold enhancement)

Gene	Hypoxia	Coculture with RPE
Angiopoietin-like protein 3	67.5	50.8
FGF-1	6	35
FGF-2	11	20
FGF receptor 3	35	82
MMP-2	2.7	2.5
HIF-1 alpha	2.5	5.4
VEGF-A	147	184
VEGF-C	6.5	4.4
TNF-alpha	9.2	4.3
Collagen IV	86.8	150

Conclusions: Coculture with RPE enhances the proangiogenic potential of EC under normoxia, which is further enhanced by hypoxia. These data suggest that EC residing in close proximity to RPE cells might be more prone to hypoxia-induced angiogenesis involved in CNV.

TRAUMATIC BRAIN INJURY INDUCED NEUROPROTECTION OF RETINAL GANGLION CELLS TO OPTIC NERVE CRUSH

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Introduction: To assess the effect of traumatic brain injury (TBI) on retinal ganglion cells (RGCs) survival after optic nerve crush in rats, and evaluate the role of retinal brain-derived neurotrophic factor (BDNF) as a neuroprotective agent in that model.

Patients / Methods: Unilateral TBI was induced in anesthetized Lewis rats by controlled cortical impact. At different times thereafter, the contralateral optic nerve was mildly crushed. RGCs survival was assessed 2 weeks later by counting RGCs labeled retrogradely with 4-Di-10-Asp. After TBI, retinal BDNF was assayed at intervals by RT-PCR.

Results: Optic nerve crush resulted in death of 50% of RGCs within 2 weeks. TBI inflicted 11 days before crush injury, but not concomitantly with it, prevented 50% of that RGCs death. This neuroprotective effect was obliterated by intra-peritoneal injection of mega-dose (30 mg/kg) methylprednisolone (MP) adjacent to TBI, but not by low dose (1 mg/kg) of MP. Retinal BDNF mRNA was increased ipsilateral to TBI, but decreased in the contralateral retina. BDNF peaked 1 day after TBI, decreased on day 11, and rose again on day 21.

Conclusions: Mild optic nerve crush in rats causes death of RGCs, at least 50% of which is prevented by an unrelated distant CNS injury. A mega-dose of methylprednisolone, commonly used to treat traumatic optic neuropathy in humans, inhibits the neuroprotective effect. Retinal BDNF may not participate in TBI-induced neuroprotection since one would expect mRNA levels for this neurotrophin to be upregulated. These findings might be applicable in a clinical situation of traumatic optic neuropathy.

Session VI – Retina II

VISION IMPROVEMENT IN TREATMENT TRIAL WITH 9-CIS BETA-CAROTENE FOR RETINITIS PIGMENTOSA PATIENTS

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Introduction: A previous study demonstrated that high oral doses of 9-cis beta carotene improve retinal functions significantly as measured by electroretinography and the perimetry in patients with a specific type of congenital stationary night blindness. The purpose of this study is to determine whether the same therapy is efficacious for treating retinitis pigmentosa patients who were genetically evaluated. .

Patients / Methods: In a double-masked, placebo-control cross-over trial, patients with retinitis pigmentosa were given daily four commercially available 15mg capsules containing powder rich in 9-cis Beta Carotene for 90 days. The patients were tested for best corrected visual acuity and underwent electroretinography using an ISCEV compliant protocol, mesopic and scotopic Goldmann perimetry and fundus imaging.

Results: No complications were recorded to be associated with the treatment. Patients reported visual improvement which was confirmed by a significant improvement in retinal functions as measured by visual acuity tests, perimetry and electroretinography.

Conclusions: The outcomes of the initial subjects suggest that 9-cis beta carotene is safe and effective in human patients with retinitis pigmentosa. The extent of visual function improvement and the optimal dose have to be further evaluated.

TREATMENT OF CONGENITAL STATIONARY NIGHT BLINDNESS WITH AN ALGA CONTAINING HIGH DOSE OF 9-CIS BETA CAROTENE

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Introduction: To assess the effect of oral administration of the alga *Dunaliella bardawil* which contains 50% all-trans beta-carotene and 50% 9-cis beta-carotene isomers on the visual functions of patients with Congenital Stationary Night Blindness (CSNB; Fundus albipunctatus).

Patients / Methods: Six patients, average age 37 ± 11 years old, with clinically and genetically diagnosed Fundus albipunctatus were treated with four capsules daily of *Dunaliella bardawil* for 90 days. The subjects were tested bilaterally before and after treatment by ERG in both eyes using an ISCEV compliant protocol (LKC Technologies, Inc., Gaithersburg, MD). Light adaptation for 10 minutes of white background light (0.023cd-s/m^2) followed by white single flash stimulus (2.44cd-s/m^2) and white 2.44cd-s/m^2 30Hz flicker. The scotopic responses were recorded for dim single flash stimulus (0.023cd-s/m^2) and bright single flash stimulus (2.44cd-s/m^2). The patients were dark adapted for additional 90 minutes after recording the ERG scotopic 30 minutes dark adaptation (total 120 minutes dark adaptation) and thereafter they were light adapted. The b-wave amplitudes were measured and the percentages of change were calculated by subtraction of baseline ERG responses from post treatment responses and divide by the baseline responses for each eye.

Results: The average mean deviation of visual field showed significant improvement from -4.99 ± 2.1 pre-treatment to -3.63 ± 2.88 post-treatment (t-test $p=0.034$). Although the scotopic ERG responses for 30 minutes dark adaptation did not show significant change from baseline (b-waves maximal rod responses percentage of change were $89\% \pm 109\%$, few of the patients did improve), the 120 minutes dark adaptation ERG b-wave responses was doubled. Averaged b-waves isolated rod responses improved from $79 \mu\text{V} \pm 40 \mu\text{V}$ to $178 \mu\text{V} \pm 79 \mu\text{V}$ ($p < 0.001$, T-test, average percentage of change $141\% \pm 61\%$) and b-waves maximal rod responses increased from $192 \pm 51 \mu\text{V}$ and post-treatment was $291 \pm 52 \mu\text{V}$ ($p < 0.001$, T-test, average percentage of change was $63\% \pm 63\%$). All patients demonstrated a clinically significant improvement in the b-wave maximal response amplitude which was found to be similar in both eyes of each patient

Conclusions: Oral treatment with the Alga *Dunaliella bardawil* significantly improved dark adaptation function in Fundus albipunctatus patients.

COMPARISON BETWEEN THE FORESEE HOME PERIMETER AND THE AMSLER GRID, IN PATIENTS WITH AGE RELATED MACULAR DEGENERATION

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Purpose: Self monitoring is an essential part in the management of intermediate AMD to assist in the detection of neovascular AMD. To this day, the only available self monitoring tool is the Amsler grid. Recently, a new home device that runs a computerized perimetry test to track changes in the visual field of intermediate AMD patients, the ForeseeHome (Notal Vision, Israel), was developed.

Objective: To compare the ability of the ForeseeHome and the Amsler grid in detecting visual field abnormalities among patients with CNV and intermediate AMD

Methods: Patients with newly diagnosed (up to 6 months) non-treated CNV patients secondary to AMD or intermediate AMD patients; age ≥ 50 , VA 20/200 or better, and mental ability to perform a ForeseeHome test were recruited to the study. Prior to treatment or routine ophthalmic examination, each participant underwent an Amsler grid examination. Lack or existence of visual field abnormalities (including: distortions, holes or local blurring) were defined as Amsler negative or Amsler positive, respectively. This was followed by ForeseeHome test, which classified the examination as ForeseeHome negative or positive. Both examinations were done in an unsupervised manner. Each participant was tested on one eye only.

Results: The study included 26 CNV patients and 31 intermediate patients (average age: 78; range: 60-91). Among the 31 intermediate AMD patients, 26 were Amsler negative and 26 were ForeseeHome negative, yielding an identical specificity of 83.8% for both the Amsler grid and the ForeseeHome. Among the 26 CNV patients, 23 were ForeseeHome positive, compared to only 15 Amsler positive, yielding a sensitivity of 88.4% and 57.6% for ForeseeHome and Amsler grid, respectively ($p < 0.05$).

Conclusion: Consistent with previous studies comparing the Amsler grid with the PHP perimeter (a professional device based on a technology similar to the ForeseeHome), these results show that the ForeseeHome is statistically superior to the Amsler grid in terms of sensitivity. The ForeseeHome may therefore be a promising, reliable and more accurate tool for self monitoring the visual field of intermediate AMD patients.

CYSTOID FOVEAL OEDEMA IN SYMPTOMATIC INNER LAMELLAR MACULAR HOLES

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Introduction: Inner lamellar macular hole (LMH) was considered a relatively risk-free condition that rarely progresses or worsens. Nowadays, at the optical coherence tomography (OCT) era, increasing evidence seems to position it differently. The aim of the study was to describe morphologic abnormalities associated with symptomatic LMH using the OCT that may explain reduced visual acuity and the benefit of vitrectomy with peeling of the inner limiting membrane following surgery.

Patients / Methods: In a retrospective study on consecutive symptomatic patients with LMH, OCT scans were compared with normal controls. Analysis was referred to LMH-associated abnormalities at the residual fovea, mainly cystoid spaces that manifested as cystoid foveal oedema, intraretinal split and epiretinal membranes (ERM).

Results: Twenty two eyes of 20 patients (mean age, 68 years; range, 22-94) were analyzed. Best corrected visual acuity ranged from 6/9 to 6/120. Cystoid foveal oedema that contained cystoid spaces of various sizes was found in 21 (95%) eyes, intraretinal split in 18 eyes (82%) and ERM in 16 eyes (73%).

Conclusions: The appearance of cystoid oedema at the residual fovea in symptomatic LMHs may explain in part, A) reduced visual acuity and/ or metamorphopsia, B) reversibility and frequent benefit of pars plana vitrectomy with peeling of the internal limiting membrane (ILM). The old notion on the low incidence of LMH progression may probably be related in part to, A) lower diagnostic accuracy before OCT was available and, B) the already spontaneously peeled ILM. Further studies are required to verify these observations, which may merit surgical considerations.

THE RATES OF THROMBOEMBOLIC EVENTS IN MACCABI HEALTHCARE ADULT MACULAR DEGENERATION MEMBERS.

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Introduction: AMD is a multifactorial disease. It is well accepted that advanced age, smoking, obesity, high content of blood lipids and atherosclerosis are risk factors for the development of the disease. These same factors are known to increase the risk of arterial thromboembolic events (ATEs) as well. A potential link between AMD and vascular disease would have therapeutic implications given current concern that some intravitreal anti-vascular endothelial growth factor (VEGF) treatments for AMD could increase stroke risk. The aim of this study is therefore to prospectively assess the link between AMD and ATEs.

Patients / Methods: Retrospective cohort study carried out at Maccabi Healthcare Services (MHS). A total of 9045 MHS members diagnosed with AMD between 18/4/1996 to 12/6/2008 were identified. A total of 90450 MHS members without diagnosis of AMD were frequency matched for birth date. Patients with a primary MI prior to date of AMD diagnosis and January 1st 1998, as well as deceased patients were excluded. Therefore, the total study cohort was comprised of 6546 AMD patients and 61672 non-AMD controls. Incident primary MI cases were identified from MHS registry of cardiovascular diseases, according to the International Classification of Diseases (ICD-9) codes.

Results: AMD patients were more likely to be older (70.36 vs. 68.31 years), women (61.2% vs. 58.2%) and have a higher proportion of patients with hypertension (82.3% vs. 78.1%). During 4.75 and 11.65 years of mean follow-up, there were 159 (5.1 per 1000 person-years) and 2997 MI's (4.2 per 1000 person-years) among AMD and non-AMD patients, respectively. The age and gender adjusted HR of MI among AMD patients was 1.01 (95%CI: 0.85-1.20). Baseline medical characteristics that were associated with increased risk of mortality included diabetes mellitus (1.50; 1.39-1.61), hypertension (1.98; 1.77-2.22), older age (1.03 per year; 1.02-1.04), and male gender (2.41; 2.24-2.59). The fully- adjusted HR associated with AMD was 1.03 (95%CI: 0.87-1.22).

Conclusions: despite the shared risk factors associated with AMD and ATEs, MHS members with AMD had a rate of ATEs similar to that of matched controls. Rates of ATEs increased in patients with comorbidities.

THE ROLE OF MICROPARTICLES (MPS) IN DIABETIC RETINOPATHY

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Introduction: Diabetic retinopathy (DR) is one of the leading causes of blindness in the western world. The advanced proliferative stage of DR is characterized by ocular neovascularization. Microparticles (MPs), which are shed from cell membranes, are associated with thrombosis and play a key role in vascular dysfunction and angiogenesis. The aim of the study was to evaluate the role of MPs in DR.

Patients / Methods: Microparticles were obtained from DR patients and healthy controls (HC) and characterized by defining their cell origin, thrombogenicity and growth factors content. Additionally, MPs thrombogenic and an angiogenic effects on endothelial cells were evaluated by FACS, ELISA, quantitative-PCR and time-laps microscopy.

Results: DR patients presented higher number of MPs (Annexin V -positive MPs) and higher level of platelet origin MPs (CD62 positive MPs) than healthy controls, as measured by FACS. In addition, MPs obtained from DR patients expressed higher level of vascular endothelial growth factor (VEGF) than MPs of healthy controls, as measured by ELISA. Exposure of human umbilical vein endothelial cells (HUVEC) to DR patients' MPs resulted in fivefold increase of mRNA expression of tissue factor (TF) - the main activator of the coagulation cascade - compared to cells stimulated by healthy controls' MPs. Furthermore, TF antigen level was low in cells stimulated by healthy controls' MPs ($3.7 \pm 1.48\%$ labeled cells) and significantly higher in cells stimulated by DR MPs ($8.6 \pm 5.2\%$ labeled cells, $p=0.012$). Time-laps microscopy demonstrated that MPs of DR patients affect cell angiogenesis as measured by tube formation assay. Adding DR MPs to HUVEC maintains tubes, reduces their tube collapsing and keeps the tube-net stable over time.

Conclusions: These findings may clarify the role of MPs in vascular dysfunction and angiogenesis which characterize diabetic retinopathy. MPs could play an important role in diabetic retinopathy etiology.

Session VII – Cataract

THE POTENTIAL OF DESFERRIOXAMINE AND ZINC-DESFERRIOXAMINE FOR REDUCTION OF LENS OXIDATIVE DAMAGE.

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Introduction: One of the major insults that can promote aging of the eye lens and cataract formation is oxidative damage. The “Free Radical Theory of Aging” proposes that aging results from the accumulation of damage caused by reactive oxygen species (ROS) known as “free radicals”. Free radicals can damage membranes, modify proteins and cause protein aggregation. We investigated the damage to the eye lens by high oxygen load and determined whether antioxidants reduce the oxidative damage.

Patients / Methods: We incubated bovine lenses in culture conditions. The lenses were divided to 4 groups; 1. Control group. 2. Oxygen-treated lenses, 3. Oxygen-treated lenses in the presence of Desferrioxamine (DFO) 4. Oxygen-treated lenses in the presence of Zinc-desferrioxamine (Zn-DFO). Each group contained at least 20 lenses. Treated lenses were exposed to 100% oxygen 4 times with 24 hours interval between exposures on days 2, 3, 4, 5, of the culture period, each time for 2 hours. During exposure to oxygen the medium of all the samples (including controls) was changed to PBS. During culture progressive changes in lens optical quality were followed. At the end of the culture, lens morphology, changes in lens structural proteins and epithelial enzymes were analyzed.

Results: Exposing lenses in culture to oxidative insults caused optical damage accompanied by morphological changes, changes in lens epithelial enzymes, and aggregation of lens proteins. The presence of antioxidants in the culture medium reduced the oxidation damage.

Conclusions: Our study shows that antioxidants can protect the lenses from oxidative damage. Zinc-desferrioxamine (Zn-DFO) showed better protection than Desferrioxamine (DFO) alone.

THE PROTECTIVE EFFECT OF DIFFERENT OPHTHALMIC VISCOELASTIC DEVICES ON CORNEAL ENDOTHELIAL CELLS DURING PHACOEMULSIFICATION IN A RABBIT MODEL – PART I

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Introduction: Purpose: To evaluate the protective effect of different Ophthalmic Viscoelastic Devices (OVDs) on corneal endothelial cells during phacoemulsification in an extreme rabbit model.

Patients / Methods: 24 rabbits eyes were randomly assigned to 4 equally sized groups. Endothelial cell counts were performed in all eyes prior to study initiation. In Group A the aqueous humor was replaced with Visiol® (TRB CHEMEDICA); In group B the aqueous humor was replaced with Biolon™ (Bio-Technology General Ltd.); In group C the aqueous humor was replaced with Viscoat® (Alcon®). In group D the aqueous humor was not replaced with any OVD; this group served as the control group. All the eyes were exposed to 5 minutes phacoemulsification. Endothelial cell counts were repeated 4 days post-surgery.

Results: Group C (Viscoat®) showed the highest endothelial cell loss - 30%, followed by group B (Biolon™) - 25%, group A (Visiol®) - 22%, and the control group - 19%. None of the differences between the groups were found to be statistically significant ($p>0.05$). The differences within each group were found to be statistically significant, $p=0.028$.

Conclusions: None of the tested OVDs showed better protective effect in comparison to the control group. However, it should be noted that due to the extremity of the model applied in the current study further examinations will be performed in Part II using a softer model.

THE PROTECTIVE EFFECT OF DIFFERENT OPHTHALMIC VISCOELASTIC DEVICES ON CORNEAL ENDOTHELIAL CELLS DURING PHACOEMULSIFICATION IN A RABBIT MODEL – PART II

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Introduction: Purpose: To evaluate the protective effect of different Ophthalmic Viscoelastic Devices (OVDs) on corneal endothelial cells during phacoemulsification in a rabbit model.

Patients / Methods: 18 rabbit eyes were randomly assigned to 3 equally sized groups. Endothelial cell count and pachymetry were performed in all eyes prior to study initiation. In Group A the aqueous humor was replaced with Biolon™ (Bio-Technology General Ltd.); In group B the aqueous humor was replaced with a combination of Viscoat® (Alcon) and Provisc® (Alcon) using the soft shell technique; In group C the aqueous humor was replaced with a combination of Visiol® (TRB CHEMEDICA) and Biolon™ using the soft shell technique. The eyes were exposed to alternating 10 seconds of phacoemulsification and a 10-second pause until a total exposure time of 2.5 minutes was reached. Endothelial cell count and pachymetry were repeated 3 days post-surgery.

Results: Group A (Biolon™) showed the highest endothelial cell loss - 13%, followed by group B (Viscoat® and Provisc®) - 7%, and group C (Visiol® and Biolon™) - 4%. The difference between groups C (Visiol® and Biolon™) and A (Biolon™) was found to be statistically significant, $p=0.037$. Accordingly, Group A (Biolon™) demonstrated the largest increase in corneal thickness - 8%, followed by group B (Viscoat® and Provisc®) - 7%, and group C (Visiol® and Biolon™) - 2%.

Conclusions: The soft shell technique using a combination of Visiol® and Biolon™ demonstrated better protective effect on the corneal endothelial cell during phacoemulsification comparing to Biolon™ alone.

CEFUROXIME- IS IT SAFE ENOUGH TO BE USED ON A LARGE SCALE BASIS IN CATARACT SURGERY?

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Introduction: Intracameral Cefuroxime (a second generation cephalosporin) prophylaxis was recently found to significantly lower the risk of endophthalmitis following cataract surgery, and is now the common practice used for post operative endophthalmitis prevention. Our aim was to evaluate retinal toxicity of Cefuroxime in a rabbit model.

Patients / Methods: Two groups of albino rabbits were used. A low dose group (1mg/0.1ml, the clinically used dose, n=9) and a high dose group (10mg/0.1ml, n=20). The Right eye of each rabbit was injected with 0.1 ml Cefuroxime solution (Experimental eye) and the left eye was injected with 0.1 ml saline (control eye). Electroretinogram (ERG) and Visual Evoked Potential (VEP) were recorded at 3 hr, 4 days, 1, 2 and 4 weeks. Histological preparations and GFAP immunostaining were made throughout the follow-up period.

Results: No functional (ERG and VEP) or morphological damage was found in the low dose group, but GFAP, a marker for retinal damage, was over-expressed in the experimental compared to the control retinas. In the high dose group, we found a significant decrease in the ERG responses of the experimental eyes as early as 3 hr after injection, followed by partial recovery during 4-weeks of follow-up. The VEP did not differ significantly between the two eyes. Morphology of retinas from the experimental eyes was markedly changed, mainly involving the outer segments of the photoreceptors, localized in the region of injection. GFAP expression was markedly elevated in Muller cells of the experimental eyes and not in the control eyes.

Conclusions: Cefuroxime is toxic to the rabbit retina when it is injected intravitreally at a dose 10 times higher than the clinically used dose. The toxicity pattern showed a transient reduction in the ERG responses that was followed by gradual partial recovery. A dose of 1mg/0.1ml of Cefuroxime, the clinically used dose, did not cause measurable toxic effects to the rabbit retina. These data indicate that caution should be exercised when Cefuroxime is used in ophthalmic surgery.

CORNEAL ENDOTHELIAL MORPHOLOGIC FEATURES IN TOXIC ANTERIOR SYNDROME IN AN ISRAELY POPULATION

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Introduction: The aim of the present study was to document clinical and laboratory findings of 21 cases of toxic anterior segment syndrome (TASS) following cataract surgery and intraocular lens (IOL) implantation with emphasize on morphologic features of the corneal endothelium.

Patients / Methods: This single, observational retrospective-based case-control Study included 21 eyes of twenty one patients presented TASS following cataract surgery with intraocular lens (IOL) implantation. Corneal endothelial morphologic features: endothelial cell density (ECD), coefficient of variation (CV) of cell area, and percentage of hexagonal cells were obtained from patient's files. The values obtained from the 21 eyes with TASS (TASS group) were compared with those in fellow eyes(21 eyes=control group). The Student t test for unpaired groups was used to compare the results at a 5% significance level.

Results: Eyes in the TASS group had a low mean ECD (2302 ± 220 cells/mm² vs 2853 ± 145 cells/mm²), high CV of cell area, and high mean percentage of hexagonal cells compared with those in the control group.

Conclusions: There is a significant corneal endothelial cell loss in the toxic anterior segment syndrome.

Session VIII - Glaucoma

INTERACTIONS BETWEEN TRABECULAR MESHWORK CELLS AND LENS EPITHELIAL CELLS – A POSSIBLE MECHANISM IN INFANTILE APHAKIC GLAUCOMA

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Introduction: Infantile aphakic glaucoma may develop as a postoperative complication of early childhood cataract surgery. Its causes and mechanisms to date are poorly understood. Our goal is to study the mechanisms leading to trabecular meshwork (TM) dysfunction and glaucoma following the cataract removal. We focus on deciphering the interactions between TM cells and lens tissue or conditioned medium by analyzing changes in TM cells co-cultured with lens epithelial cells (LECs), or cultured in the presence of factors found to be secreted by LECs.

Patients / Methods: These interactions were studied by analyzing for morphological alternations, and differential gene and protein expression. The presence of factors found to be secreted by LECs was analyzed using cytokines antibody array membranes.

Results: TM cells grown in the presence of LECs exhibited structural changes (mainly volume and size enlargement and decreased cell-cell interactions), as well as altered protein expression (mainly cytoskeletal), and gene expression (such as genes related to cell morphogenesis and inflammatory response). Several cytokines were found to be elevated in the medium of LECs, and of the co-culture, but not in the medium of TM cells, suggesting their role in the changes observed in TM cells co cultured with LECs.

Conclusions: Many of the changes observed in TM cells after exposure to LECs were reported in primary open-angle glaucoma. This strengthens the suspected role of LECs in the development of aphakic glaucoma. Co-culture of TM cells in the presence of the suspected cytokines will be further performed.

THE EFFECT OF AGING ON RGCS SUSCEPTIBILITY TO ELEVATED INTRAOCULAR PRESSURE

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Introduction: Epidemiologic data suggest that progression in glaucoma is faster in old patients compared to young. In this study we evaluated the effect of aging on retinal ganglion cells (RGCs) susceptibility to elevated intraocular pressure (IOP) using a glaucoma model in rats.

Patients / Methods: Experimental glaucoma was induced in rats with different age groups: 3, 6, 13 and 18 months old (n= 48). Two months after the induction of glaucoma, RGCs were labeled by Fluorogold injection into the superior colliculus. Ten days later, eyes were enucleated and retinas were prepared as whole mount. Labeled RGC were counted in the glaucomatous and control-fellow eyes.

Results: IOP was elevated in all rats. Peak IOP ranged between 27- 35 mmHg in the control-non-glaucomatous eyes to 41- 48 mmHg in glaucomatous eyes. The total number of RGCs in control eyes decline depending on age, from 7832±195 at 5 months to 7131±430 at 8 months and 5412±236 at 20 months old (p =0.001647). A faster decline was demonstrated in eyes exposed to experimental glaucoma 2 months earlier, from 4255±1055 RGCs at 5 months old to 3165±908 at 8 months old and 1418±347 at 20 months old.

Glaucomatous damage increased depending on age, from 45.4%±13.4% at 5 months old to 55.1%±12.6% at 8 months and 74%±6% in 20 months old rats

Conclusions: In control eyes RGCs degeneration increased with age. In glaucomatous eyes, RGCs degeneration in old rats is enhanced compared to young rats, beyond the effect of age. We suggest that aging increase the susceptibility of RGCs to elevated IOP.

SYSTEMIC TREATMENT WITH RASAGILINE, A SELECTIVE MONOAMINE OXIDASE INHIBITOR, IS NEUROPROTECTIVE IN GLAUCOMATOUS RAT EYES

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Introduction: In this study we evaluated the neuroprotective effect of Rasagiline (N-propargyl-1 (R)-aminoindan) a selective monoamine oxidase inhibitor, on the survival of retinal ganglion cells (RGCs) in glaucomatous rat eyes. Rasagiline belongs to the propargyl amines family. This family includes a large number of neuroprotective substances that were investigated in many animal models of injury. Rasagiline neuroprotective effect was demonstrated in vitro and in vivo: prolonging survival of cells in hypoxia and in the presence of different excitotoxic substances. Rasagiline protected rodents from brain damage due to ischemia, anoxia, and Parkinson.

Patients / Methods: The neuroprotective effect of intraperitoneal (IP) daily injections of Rasagiline (0.5 mg/kg and 3 mg/kg) was evaluated and compared to saline injections in Wistar rats using the translimbal photocoagulation model for induction of glaucoma (n = 29). Intraocular pressure was measured before the laser treatment, immediately after, and then weekly. Ten days before enucleation Fluoro-gold was injected to the Superior colliculus. Seven weeks after the induction of glaucoma, eyes were enucleated and retinas were prepared as whole mounts. Evaluating RGC survival was achieved by counting surviving labeled RGCs.

Results: Seven weeks after induction of glaucoma, the mean number of surviving RGCs was significantly higher in both Rasagiline groups. The mean RGC survival in the Rasagiline 3mg/kg was $43\pm 8\%$ and in the Rasagiline 0.5mg/kg $43\pm 9\%$ compared to $23\pm 4\%$ survival of RGCs in control-saline group, ($p=0.03$, $p=0.04$, respectively).

Conclusions: Systemic treatment with different dosages of Rasagiline appears to significantly enhance the survival of RGCs in experimental glaucoma.

TOPICAL GLAUCOMA THERAPY AS A RISK FACTOR FOR NASOLACRIMAL DUCT OBSTRUCTION

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Introduction: The tearing patient presents the ophthalmologist with a complex diagnostic and therapeutic challenge. Epiphora may be caused by excretory lacrimal apparatus pathology, of which nasolacrimal duct obstruction (NLDO) plays a significant role. NLDO might occur secondary to various etiologies. The most common cause for NLDO is idiopathic primary acquired nasolacrimal duct obstruction (PANDO). The aim of the present study was to investigate a possible association between PANDO and primary open angle glaucoma (POAG).

Patients / Methods: This is a retrospective comparative study. The study group consisted of all 209 consecutive eyes (178 patients) whose lacrimal system had PANDO in patients over 50 years of age during the 10 year study period. The control group consisted of consecutive 183 eyes (183 patients) who presented for cataract surgery, during the same time period. The main outcome measures were: Prevalence of POAG in study and control groups; Effect of topical glaucoma therapy use profile on PANDO prevalence. Medical records of all patients included in the study were reviewed. Data collected included demographic details and history and characteristics of POAG treatment.

Results: The prevalence of POAG in PANDO group (23%) was significantly higher than that of control group (6%, $P < 0.0001$). The average history of POAG was longer in PANDO group (14.10 \pm 5.59 years), compared to control group (9.55 \pm 7.23 years, $P = 0.025$). The average number of topical glaucoma therapy drugs per glaucomatous eye in PANDO group (1.58 \pm 0.92) was significantly higher than that of control group (0.73 \pm 0.90, $P = 0.002$). Bilateral nasolacrimal duct obstruction (NLDO) was more common among glaucoma patients in PANDO group (38.23%), compared to non glaucoma patients in the same group (11.80%, $P = 0.0002$). Significantly higher percentage of glaucoma patients in PANDO group (69%) were treated with timolol, compared to glaucoma patients in control group (18%, $P = 0.005$).

Conclusions: Chronic use of timolol containing topical glaucoma therapy preparations in glaucoma patients is associated with an increased risk for the development of nasolacrimal duct obstruction. Large scale prospective studies are needed in order to ascertain this association.

CONJUNCTIVAL WOUND HEALING PROBLEMS RELATED TO AHMED GLAUCOMA VALVE SURGERY

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Introduction: There is a long list of possible post operative conjunctival complications related to Ahmed glaucoma valve (AGV) including next three: A. Conjunctival dehiscence and retraction leading to exposure of underlying donor patch. B. Device exposure. C. Device extrusion.

Purpose: To analyze the risk factors, frequencies, management and outcomes of conjunctival complications.

Methods: Retrospective chart review of 268 subjects who had undergone AGV insertion at Toronto Western Hospital from September 1, 1999 to March 31, 2007. Only cases with regular follow-up for at least 1 year were included.

Results: Mean follow-up was 43.17 months (± 26). The mean age was 64 years (24-88). The mean pre-op IOP was 23 mmHg using 3.86 hypotensive medications which dropped to 13 mmHg at 1 year ($p < .0001$) and remained stable at the following annual visits using 1.8 ($p < .0001$) and 3.5 hypotensive meds ($p = 0.14$) at first and 7th year respectively. Conjunctival dehiscence (group A) was recorded in 55 subjects (20.52%) at a mean of 31 (2-180) days after surgery, and 11 (4.1%) developed tube or plate exposure (group B) after a mean of 35.7 (4.5-78) months, and 3 had both (1.1%). No extrusion was recorded (group C). 44.5% had fornix based flaps and non-absorbable conjunctival closing sutures and 55.5% had limbal base flaps and absorbable sutures. 89% of group A required no further intervention whereas 7.37% and 3.63% required 1 or 2 repairs respectively. 7 exposed AGV (group B) were removed and 4 (group B) were repaired utilizing a donor lamellar corneal patch graft and conjunctival auto-graft. Buccal membrane was used in one subject. Endophthalmitis developed in 2 subjects with exposed AGV (group B) leading to 1 enucleation.

Discussion: AGV surgery is frequently complicated by conjunctival dehiscence which usually resolves with conservative treatment. AGV exposure on the other hand, is much less frequent and requires surgical revision.

AUTOLOGOUS PATCH GRAFT IN AQUEOUS GLAUCOMA SURGERY

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Introduction: To evaluate the efficacy and safety of autologous scleral patch graft for covering the subconjunctival tube in aqueous shunt surgery.

Patients / Methods: A retrospective study of 30 patients (30eyes) who underwent Ahmed glaucoma shunt with autologous scleral patch graft at the Carmel Medical Center and had at least 6 months of follow-up. Glaucoma diagnosis included open angle glaucoma (n=18), neovascular glaucoma (n= 4) and congenital glaucoma (n=8). A free rectangular scleral lamellar flap was created from the operated eye and was used to cover the subconjunctival part of the tube.

Results: The mean follow-up was 13.3 ± 8.3 (range 6- 36 months). The mean intraocular pressure before surgery was 37.4 ± 4.6 and was reduced to 16.8 ± 4.8 at last follow-up. The mean number of medications was 2.7 ± 0.9 before surgery and 1.2 ± 1 at the last follow-up. All eyes tolerated the autologous graft well with no clinical evidence of tube erosion or any intraoperative or postoperative complications.

Conclusions: Autologous patch graft is effective and safe for covering the subconjunctival tube in Ahmed glaucoma Shunt surgery. This technique has the advantage of ease of availability and no cost.

COMPUTER-GUIDED PATTERNED LASER TRABECULOPLASTY

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Purpose: Argon laser trabeculoplasty (ALT) with 100 ms pulses is an effective therapy for lowering intraocular pressure (IOP). Similar reductions in IOP have been achieved using ns (SLT) and microsecond (MLT) pulses, which produce less thermal damage to trabecular meshwork (TM). Lack of clinically visible changes may make the accurate alignment of subsequent pulses difficult. We describe a novel computer-guided treatment technique – Patterned Laser Trabeculoplasty (PLT) using the PASCAL® Photocoagulator system, and its preliminary evaluation in patients with open angle glaucoma.

Methods: In this prospective case series 50 eyes of 26 patients with uncontrolled and untreated open angle glaucoma received patterned laser treatment (360 degrees, 532nm, 5ms, 100 µm spot). The laser was first titrated for TM blanching at 10 ms, and using the same power sub-visible treatment was applied with 5 ms pulses. The arc patterns of 75 spots covering 22.5° of TM rotated automatically after each laser application by 22.5°, so that the new pattern is applied at the untreated position, and no visibility of the previously treated area is required for alignment. Pre- and post-treatment IOP was monitored with a Goldmann tonometer.

Results: 1200 laser spots were placed per eye in 16 steps. The IOP decreased from pre-treatment level of 21.9 mmHg to 15.9 at 1 month (n=44) and maintained stable at 15.8 at 6 months (n=23). This highly statistically significant result ($p < 2 \times 10^{-7}$) corresponds to a 24% IOP reduction. There were no complications or adverse events.

Conclusions: Single administration of PLT with 5 ms exposures provided a 24% reduction in IOP during the follow-up interval of 6 months. Pulse energy with PLT protocol decreased by a factor of 10 on average, compared to ALT. PLT provides for rapid and precise computer-guided treatment with exact abutment of the patterns, and no visible damage to trabecular meshwork.

Session IX – Oncology

TREATMENT OF UVEAL MELANOMA BY IRREVERSIBLE ELECTROPORATION - FINITE ELEMENT MODEL STUDY

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Introduction: Irreversible electroporation (IRE) is a novel tool for treating solid tumors by applying high electrical field pulses on the target tissue. Some of the advantages of this technique are its specificity for affecting cells and preserving connective tissue (like the sclera, corneal stroma). However, since it is difficult to focus the electrical energy on the tumor, it is crucial to choose optimal treatment parameters. The goal of this study was to develop a mathematical model for evaluation of optimal electrode configuration and pulse parameters that will induce maximal tumor eradication using IRE.

Patients / Methods: A geometrical model of an eye with a 6 mm height uveal melanoma tumor was constructed. A 3D mesh (~ 8,000 elements) was created using Comsol Multiphysics Version 3.3. We simulated a treatment plan of electrical pulse while changing the position of electrodes (external, internal, adjacent), and the electrical conductance of the tumor, sclera and vitreous. Treatment protocol goal was to achieve an electrical field of 800 V/cm in at least 50 percent of tumor while monitoring for minimal electrical field other areas.

Results: External surface electrode combined with an intravitreal ground electrode was found to achieve maximal tumor coverage with minimal pulse amplitude. Assuming an estimated tumor conductance of 0.5 S/m, most of the tumor volume achieved the threshold using a pulse of less than 100 V. However, in tumor with higher conductance, a significantly higher electrical pulse was needed in order to achieve treatment goal. When using only adjacent external electrodes placed on the sclera, achievement of treatment goals could be reached by using higher electrical pulse. The safety data depicted that the electrical field in the macular area were low (less than 10 V/cm).

Conclusions: Electrical field thresholds for irreversible electroporation can theoretically be reached in uveal melanoma using external as well as internal electrode configuration. Tumor conductance has a significant effect on treatment efficiency. The conductances of the sclera above the tumor and of the aqueous humor are of secondary importance. Human as well as animal studies are needed in order to explore the potential of IRE in treating uveal melanoma.

THE IMPACT OF TREATING PRIMARY POSTERIOR UVEAL MELANOMA ON SERUM BIOMARKER LEVELS

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Introduction: The serum biomarkers osteopontin (OPN), S-100, melanoma-inhibitory activity (MIA), and tissue polypeptide-specific antigen (TPS) were found to be significantly elevated in patients with metastatic uveal melanoma, as compared to disease-free patients. The purpose of this study was to examine the impact of treating the primary uveal melanoma by enucleation or brachytherapy on the levels of serum biomarkers.

Patients / Methods: Levels of serum biomarkers were analyzed for 75 uveal melanoma patients. The serum biomarker levels were measured using the ELISA method. A matched-pairs analysis was used to compare baseline (pre-treatment) marker levels with those measured at 1, 4, and 10 months after treatment. Differences in biomarker levels were analyzed over the entire group and for each treatment group separately.

Results: Of the 75 patients, 62 underwent brachytherapy and 13 were enucleated. One month following treatment S-100, MIA, and TPS, levels did not change, while OPN increased (11.51 to 13.11 ng/ml, $p = 0.0430$). Four months after treatment there were no changes in marker levels from baseline, except for a decrease in S-100 levels in the enucleated patients (0.09 to 0.06 $\mu\text{g/L}$, $p = 0.0492$). S-100 levels decreased in the brachytherapy patient group (0.11 to 0.08 $\mu\text{g/L}$, $p = 0.0251$) after 10 months. No other changes in marker levels were detected at the 10-month time point.

Conclusions: The statistically significant changes in OPN and S-100 levels bear no clinical significance in comparison with the previously published magnitude of increase in serum marker levels upon the appearance of metastatic disease in the liver. Since treatment of primary uveal melanoma does not have a considerable impact on serum biomarkers, these results emphasize the usefulness of these biomarkers in the early detection of metastatic uveal melanoma in the liver.

RUTHENIUM-106 PLAQUE BRACHYTHERAPY FOR THICK POSTERIOR UVEAL MELANOMAS

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Introduction: Ruthenium-106 brachytherapy is considered to be an effective method of treatment for small to medium uveal melanomas, up to a thickness of 8 mm. The purpose of this study was to examine the effectiveness and safety of Ruthenium-106 plaque brachytherapy in the management of thick posterior uveal melanoma.

Patients / Methods: In a non-randomized comparative case-series, 126 consecutive patients who were diagnosed from 1989 to 2007 with thick (apical height ≥ 8.0 mm) uveal melanoma were included. 63 patients primarily treated with Ruthenium-106 plaque brachytherapy were compared with 63 patients primarily treated with enucleation. The main outcome measures were visual acuity, eye retention, local recurrence, metastases, all-cause mortality and melanoma-related mortality.

Results: Patients treated with brachytherapy were significantly younger and had significantly smaller tumors, compared to patients treated with enucleation. The mean tumor thickness in the brachytherapy group was 9.29 ± 0.90 mm (mean \pm SD; range 8.00 - 11.81 mm), compared to 12.23 ± 1.89 (8.00 -19.40 mm) in the enucleation group. The 5 and 10 years melanoma-specific mortality was 18.7% and 30.0% for brachytherapy patients and 27% and 39.9% for enucleation patients ($p=0.37$ and $p=0.33$). When comparing the brachytherapy patients with enucleation patients who had tumors not thicker than 12 mm (32 patients, 10.84 ± 0.87 mm, range 8.00 -11.72 mm), the 5- and 10-year metastases rates were 31.8% and 38.6% ($p = 0.35$ and $p = 0.91$), and the 5- and 10-year melanoma-related mortality rates were 25% and 32.5% ($p = 0.65$ and $p = 0.99$). No significant difference in survival was noted between patients who did and did not develop local recurrence. Of the eyes that were initially treated with brachytherapy, 71.4% were saved from enucleation, which was performed for tumor regrowth and/or complications (neovascular glaucoma). Of the brachytherapy patients, 70.8% had best corrected visual acuity of 20/200 or worse at their last follow-up.

Conclusions: Ruthenium-106 brachytherapy is an effective and safe alternative to enucleation in treating thick posterior uveal melanomas.

THE EFFICACY OF COLOR DOPPLER ULTRASOUND FOR DIAGNOSIS AND FOLLOW-UP OF ORBITAL HEMANGIOMA IN CHILDREN

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Introduction: Capillary hemangioma is the most common orbital vascular tumor in children. Diagnosis is based on the clinical findings, and supported by imaging studies like CT and MRI. Color doppler ultrasound was recently described as reliable, non-invasive, non-radiating and cheap imaging tool for differentiating orbital processes in children. On ultrasound, hemangiomas appear as encapsulated lesion with a smooth contour and a low internal echogenicity. Color doppler imaging shows marked intralesional flow. The objective of this study is to evaluate the efficacy of color doppler ultrasound as a single imaging modality for the diagnosis and follow-up of children with a clinical diagnosis of orbital hemangioma.

Patients / Methods: This study included all children with intraorbital and periorbital tumors that were clinically and echographically diagnosed as hemangiomas and who were evaluated in our clinic between 2004 and 2008. Data collected included demographic details, clinical findings, treatment, and outcome.

Results: We reviewed the medical records of 13 children (9 boys, 4 girls). Mean age at diagnosis was 21 weeks (range: 5-40 wks). Mean follow-up period was 19 months (range: 4-31 mos). Complete resolution of the tumor occurred in 4 children (31%), partial resolution in 7 children (54%) and in 2 children (15%) there was no change in tumor size. Only 3 of the children were treated – 2 (15%) with systemic corticosteroids and 1 (8%) with intralesional corticosteroids injection. Ten children (77%) were not treated. In all cases, the clinical course during the follow-up period supported the initial diagnosis of hemangioma, without any need of using other imaging modalities.

Conclusions: Color doppler ultrasound is a non-invasive imaging modality than can be reliably used for the initial diagnosis and continuing follow-up of children with orbital hemangioma.

OUR EXPERIENCE WITH ANTERIOR APPROACH AND CRYOEXTRACTION FOR SURGERY OF ORBITAL CAVERNOUS HEMANGIOMA

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Introduction: In the oculoplastic unit of the Goldschleger eye institute, transconjunctival or transcutaneous anterior orbitotomy using surgical microscope and cryoextraction is routinely employed for the removal of most orbital tumors. The purpose of this presentation is to summarize our experience with this cryo-assisted, minimally invasive, anterior approach used for extraction of orbital cavernous hemangiomas.

Patients / Methods: Retrospective, noncomparative, consecutive, interventional case series, of patients with orbital cavernous hemangioma operated on in the Goldschleger Eye Institute over the last 5 years. The medical records were reviewed and full data regarding the gender, age, location and size of the hemangiomas, the surgical procedure and the result of short follow-up examination were retrieved.

Results: During the last five years from October 2003 to September 2008, 73 patients underwent extraction of orbital tumors in the Goldschleger Eye Institute. In 24 of them (32.9%) the diagnosis was cavernous hemangioma. These patients included 12 males and 12 females, and their age at surgery was 47.3 ± 10.3 years (mean + SD). Intraconal location was found in 20 (83.3%). The longest axis of the tumor was 10 to 40 mm (mean: 18.1 mm). Cryo-assisted extraction was employed in all operations. In all except one case the tumor was extracted using the minimally invasive anterior approach. The one case that underwent lateral orbitotomy had a very small, deeply located apical lesion. The operations with anterior approach lasted 57.9 ± 15.0 minutes (mean + SD). In none of them there was need for transformation into lateral orbitotomy and there were no intra-operative complications.

Conclusions: The anterior orbitotomy approach using surgical microscope and cryoprobe for extraction of orbital cavernous hemangioma is safe, relatively short and straightforward. This minimally invasive approach avoids, in most cases, the need for temporal or frontal bone flaps, and is suggest as the first choice for extraction of orbital cavernous hemangioma as well as other solid, well demarcated tumors. Large size of the tumor and location medial to the optic nerve, are not contra-indications for this surgical approach

Session X – Free Papers

CESSATION OF THE OCULOCEPHALIC REFLEX IN NORMAL HEALTHY BABIES

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Introduction: To evaluate the cessation of the oculocephalic reflex in normal healthy babies.

Patients / Methods: A prospective, longitudinal, comparative case series of 325 babies aged 1-32 weeks were examined. The oculocephalic reflex was examined by two ophthalmologists by placing the babies' head at 30 degrees above horizontally and rapidly rotating the head from side-to-side in the horizontal and vertical plane while watching the conjugate eye movement. The presence and cessation of the oculocephalic reflex was analyzed in relation to gestational age (GA), postpartum age (PPA), postconceptional age, birth weight and current weight (CW). The logistic regression analysis was used to determine the probability of the disappearance of the reflex in relation to previous variables.

Results: The rate of babies without oculocephalic reflex was calculated in a linear logistic model. In 75% of the babies younger than 11.5 weeks of age, the reflex was present and in >95% of the babies aged 11.5 to 20 weeks, the reflex was missing. The PPA and CW were highly correlated ($r=0.7%$) with the cessation of the reflex. PPA had a greater influence than GA for the development of the reflex (although both were significant for the group without it; $p=0.01$ and $p=0.04$, respectively).

Conclusions: The oculocephalic reflex starts to diminish during the first weeks of life and gradually decreases in prevalence at 11.5 weeks. This process has a longitudinal development and is part of normal maturation of the visual system.

MACULAR VOLUME AND RNFL THICKNESS ARE CORRELATED WITH CLINICAL OCULAR EXAMINATION AND DISEASE STATUS IN PATIENTS WITH MULTIPLE SCLEROSIS

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Introduction: PURPOSE: To identify the best OCT parameter which correlates with clinical ocular examination and disease status as determined by Expanded Disability Status Scale (EDSS).

Patients / Methods: PATIENTS: 75 patients with multiple sclerosis including 44 patients (88 eyes, group 1) without a history of optic neuritis, and 31 patients (37 eyes, group 2) with a history of optic neuritis. METHODS: All patients underwent complete ocular examination including: best corrected visual acuity (BCVA), contrast sensitivity (CS), color vision (CV), visual fields (VF) and optic nerve morphology assessments. OCT Fast Macular Thickness scan was used to assess macular volume and average RNFL thickness was measured with Fast RNFL 3.4 mm scan.

Results: In both groups there was high correlation between macular volume and each of the ocular examined parameters: BCVA (group1: $p=0.03$, group 2: $p=0.03$), contrast sensitivity (group1: $p<0.0001$, group 2: $p<0.0001$), color vision (group1: $p<0.0001$, group 2: $p=0.004$), visual field mean deviation (group1: $p=0.001$, group 2: $p=0.03$) and optic nerve morphology (group1: $p<0.0001$, group 2: $p=0.03$). Average RNFL thickness positively correlated with each of the ocular clinical parameters only in patients with a history of optic neuritis (BCVA: $p=0.004$, CS: $p<0.0001$, CV: $p=0.002$, VF MD: $p=0.001$ and optic nerve morphology: $p<0.0001$). Both OCT parameters had negative correlation with disease duration and EDSS only in patients who didn't experience optic neuritis (macular volume: $p<0.0001$, $p<0.0001$ respectively; RNFL average thickness: $p<0.0001$, $p=0.04$ respectively).

Conclusions: In all multiple sclerosis patients macular volume correlated with visual function. Macular volume and RNFL thickness correlated with disease status only in patients who didn't experience optic neuritis.

"HIDE AND SEE(K)"- A CHAMELEON'S VISUALLY GUIDED RESPONSE TO THREAT.

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Introduction: Chameleons exhibit independent, large amplitude eye movements that scan their surroundings while keeping relatively motionless. Under threat, they keep their body on the side of the perch opposite the threat, while fixating it binocularly or monocularly. This position is maintained, by counter-rotating their body at the angular magnitude of the threat's motion. Differential eye use (laterality) in vertebrates has been previously established. Do chameleons display laterality of body orientation or eye movements? These questions are especially interesting in view of their full optic decussation and unique patterns of eye movements.

For chameleons, presented with a localized visual threat, we determined (1) patterns of monocular and binocular eye movements (2) the effects of visual obstruction on the monocular and binocular eye use, and (3) differential eye use and motor responses (laterality).

Methods: The test apparatus comprises (1) a modular vertical pole (of a given diameter) that can be smoothly rotated to any angle ($\leq 180^\circ$) and on which the tested animal perches (2) two video cameras: one on the chameleon-threat axis, viewing the chameleon, and one viewing the chameleon from above 90° to the former) (3) a localized visual threat.

Results: Within two days of hatching the chameleons showed clear hiding responses. No clear evidence for laterality in eye use was detected yet motor adjustments were more precise when relative threat motion was to the right.

Conclusions: The eye tracking the threat was predominately that towards which the threat was relatively moving.

UTILIZING FUNCTIONAL MAGNETIC RESONANCE IMAGING OF THE VISUAL CORTEX TO EVALUATE MACULAR FUNCTION IN RESPONSE TO A FLASH STIMULUS

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Introduction: The aim of the study was to design a test which integrates a robust visual stimulus with functional MRI (fMRI) of the visual cortex, which will allow specific assessment of macular function in cases of opaque media. To examine validity of the stimulus paradigm, it was initially examined in normal subjects and age-related macular degeneration (AMD) patients with clear media.

Patients / Methods: Functional MRI activation patterns in the visual cortex were assessed in six healthy volunteers and three AMD patients. First, in order to identify the regions in the brain responding to macular activation versus peripheral retinal activation, conventional retinotopic mapping using an expanding annular stimulus was performed. Subsequently, the visual system was evoked by a series of flashing light stimuli at different frequencies using a grid pattern (Espion® system, Diagnosys LLC, Littleton, USA). This type of stimulus can penetrate even severe opacities in the media. The fMRI data in response to this novel stimulus paradigm was analyzed and compared with the response to the conventional clear media-based retinotopic stimulus.

Results: The foveal and parafoveal regions of interest (ROI) were individually defined for each subject according to their activation by the expanding annular stimulus. In all healthy subjects, a flashing light stimulus at a frequency of 16Hz robustly activated the foveal and parafoveal (mean \pm SEM; averaged percent signal change: 1.86 ± 0.14 ; extending 6387 ± 1689 mm³). In AMD patients with macular dysfunction, a correlation between visual acuity and volume activation was observed ($r_6 = 0.839$, $P < 0.05$) in addition to a correlation between visual acuity and percent blood oxygen level dependent (BOLD) signal change from fovea ROI ($r_6 = 0.851$, $P < 0.05$).

Conclusions: fMRI activation patterns of the visual cortex in response to a flashing stimulus presented at 16Hz allow assessment of macular function in healthy subjects and AMD patients. This method may be used in the future to assess macular function in patients with opaque media, as a pre-operative screening tool to predict visual outcome.

IN VITRO SYNERGY OF MOXIFLOXACIN AND CEFUROXIME AGAINST OCULAR CLINICAL ISOLATES

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Introduction: Postoperative endophthalmitis is a potentially devastating complication of cataract surgery. Efficient perioperative prophylaxis is widely studied, especially due to the continuous emergence of resistant strains. Cefuroxime (Zinacef) is lately used for intracameral injection at the conclusion of the surgery. Topical antibiotics are frequently instilled perioperatively in order to penetrate into the anterior chamber and provide additional antibacterial coverage. The combination of two antibiotic drugs broadens the antibacterial coverage, however Interactions between different antibiotic agents may affect their potency. The purpose of this study was to examine the interaction between moxifloxacin (Vigamox) and cefuroxime towards several ophthalmic pathogens.

Patients / Methods: Clinical isolates of Streptococcus pneumonia (x2 strains), Coagulase-negative Staphylococcus (x2 strains), Methicillin-sensitive Staphylococcus aureus, Methicillin-resistant Staphylococcus aureus, and Pseudomonas aeruginosa were used. Minimum inhibitory concentrations (MICs) were determined for each of the clinical isolates. Testing for synergy was determined by the checkerboard technique. Combinations of moxifloxacin and cefuroxime were tested against the clinical isolates in BHI broth. The fractional inhibitory concentration (FIC) index for each antibiotic in every combination was calculated. A synergistic effect was determined when FIC index was >2 .

Results: The FIC index was 2 and 2.5 for each of the two S. pneumonia strains respectively, 1.5 for each of the staphylococci strains, and 2.5 for P. aeruginosa. These results suggest a synergistic effect of this antibiotic combination for S. pneumonia and P. aeruginosa and an additive effect of staphylococci. No antagonism was detected

Conclusions: The Checkerboard method showed evidence of either synergy or additive effect between moxifloxacin and cefuroxime with no evidence of antagonism. This data provides additional support for advantage of combining the two antibiotics for enhanced prophylaxis in cataract surgery.

INTRAVITREAL INJECTIONS OF NEUROTROPHIC FACTORS SECRETING MESENCHYMAL STEM CELLS ARE NEUROPROTECTIVE IN POST OPTIC NERVE TRANSECTED RAT EYES

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Introduction: To evaluate the neuroprotective effect of intravitreal injections of neurotrophic factors secreting mesenchymal stem cells on the survival of retinal ganglion cells (RGCs) in post optic nerve transected rat eyes. The injected cells are bone marrow derived mesenchymal stem cells (MSC) that were induced into neurotrophic factors secreting cells (NTF-SC) to combine stem cell-based therapy with neurotrophic factors-based neuroprotection. NTF-SC produce and secrete high levels of BDNF, GDNF, VEGF and IGF1.

Patients / Methods: A novel protocol was used to induce MSC of rat and human bone-marrow origin into neurotrophic factors secreting cells. The neuroprotective effect of intravitreal injections of either untreated MSC or NTF-SC was evaluated and compared to PBS injections using optic nerve transection model (n = 132). Systemic Cyclosporine was daily injected to some of the rats to assess the influence of immunosuppression. RGCs were labeled by applying Rhodamine Dextran to the optic nerve or Fluorogold into the superior colliculus. Cells-treated and PBS-treated eyes were compared in a masked way 8 days after optic nerve transection. For cell tracking purposes, MSC were labeled with PKH26 and analyzed at 3 time points using immunohistochemistry and PCR.

Results: Eight days after optic nerve transection, the mean RGCs damage was significantly reduced after intravitreal injections of NTF-SC of human origin ($31\% \pm 3\%$, $p = 0.0005$) or un-treated MSC ($34\% \pm 5\%$, $p = 0.07$) compared to PBS ($54\% \pm 3\%$). When systemic immunosuppression was applied the mean RGCs damage was still significantly reduced after intravitreal injections of NTF-SC of human origin ($55\% \pm 3\%$ RGCs damage, compared to $62 \pm 4\%$ damage for un-treated MSC and $62 \pm 3\%$ for PBS injected eyes). Immunohistochemistry for transplanted NTF-SC demonstrated that stem cells survive in the retina and vitreous at least 9 days after injection. However, the number of cells was reduced on the 9th day compared to the day of injection.

Conclusions: Cell-based delivery of neurotrophic factors is significantly neuroprotective in optic nerve injury. This novel approach is a safe and efficacious method of generating NTFs in the eye and may be potentially neuroprotective in glaucoma. Similar experiments with glaucoma model are in progress.