I. Epidemiology of Myopia

A. Definition
1. Physiologic <25.5 mm axial length 0 – 3D
2. Intermediate <32.5 mm axial length –3 – 5D
3. High >32.5 mm axial length –5 - -8D
4. Pathologic >32.5 mm axial length >-8D

B. Associated Pathology
1. Posterior staphyloma
2. Macular hemorrhage
3. Macular degeneration
4. Subretinal neovascularization
5. Cataract
6. Glaucoma
7. Retinal detachment
8. Strabismus

C. Limitations of Myopia Research
1. Few longitudinal studies – prevalence not incidence
2. Not all ocular components of refraction measured
   a. Corneal Curvature
   b. Cycloplegic refraction
   c. Axial length
   d. Lens thickness/curvature
   e. Anterior chamber/vitreous chamber depth
3. Intervention studies limited by
   a. Retrospective nature
   b. Lack of control group
   c. High dropout rate
d. Selection bias  
e. Poor sample size  
f. Inadequate follow up  
g. Lack of randomization  
h. Unmasked subjects/observers  

D. Prevalence of myopia – dependent on Ethnicity/Age/Socioeconomic Status  

1. Ethnic variability  

<table>
<thead>
<tr>
<th>Country</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Solomon Islands</td>
<td>0.8</td>
</tr>
<tr>
<td>U.S.</td>
<td>25 – 43</td>
</tr>
<tr>
<td>Taiwan</td>
<td>75</td>
</tr>
<tr>
<td>Singapore</td>
<td>65 – 90</td>
</tr>
<tr>
<td>China</td>
<td>37</td>
</tr>
<tr>
<td>Nepal</td>
<td>3</td>
</tr>
<tr>
<td>Chile</td>
<td>19</td>
</tr>
<tr>
<td>India</td>
<td>10-20</td>
</tr>
</tbody>
</table>

2. Is prevalence increasing  

a. Higher prevalence of myopia in younger cohorts  
b. Increased incidence vs. hyperopization of population  

E. Cost of myopia  

1. Morbidity  

a. Refractive errors – a leading cause of vision loss throughout the world.  
b. In U.S. – 5th leading cause of vision impairment, 8th leading cause of legal blindness  
c. Economic cost $2-4 billion/year spent in the treatment of myopia  
d. Quality of life decreased in patients with pathologic myopia  

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II. Cause of Myopia: Nature vs. Nurture

A. Emmetropization
   1. Shift in refraction form hyperopia to emmetropia
   2. Process directed by genes or environment.

B. Nature – our genes determine refractive status
   1. Twin Studies
      Heretibility 90%
   2. Ethnic variations suggest genetic predisposition
   3. Linkage Analysis – autosomal dominant pedigrees – locus chromosome 8, 12 and 18
   4. Zadnik Study – Children of myopic parents have longer eyes than cohort

C. Nurture – how we use our eyes determines refractive status
   1. Status of aboriginal peoples
      a. North American Eskimo
   2. Correlation of study habits with myopia
   3. Progression of myopia accelerated during school year
   4. Correlation of level of education attainment with myopia
   5. Correlation of occupation demands

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III. Animal Models of Myopia

A. Mammal/Avian
   1. Monkey – Raviola/Weisel/Hung/Smith
   2. Chicken – Wallman/Stone/Schaeffel
   3. Tree Shrew – Norton
   4. Marmoset – Judge

B. Form deprivation leads to axial elongation
   1. Independent of neurofeedback
   2. Time dependent but reversible
   3. Focal ocular growth may occur
   4. Affect mediated by neurochemical factors at level of retinal/choroid/sclera
      a. Dopamine
      b. Cholinergics
      c. Vasoactive intestinal polypeptide
d. Diurnal signals – melatonin
   5. Signal for change in production of growth mediators
      a. Messenger RNA
      b. Glycosaminoglycan synthesis
      c. Matrix metalloproteinase synthesis

C. Optical defocus can affect ocular growth so that eye compensates for defocus
   1. Monkey – spectacle
   2. Tree shrew – spectacle
   3. Marmoset – contact lens
   4. Chicken – spectacle

D. Human correlates of deprivation myopia
   1. Congenital cataract
   2. Vitreous hemorrhage
   3. Keratitis
   4. Hemangioma
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IV. Model of myopia

A. Inherited predisposition for myopia
   1. Ethnic background
   2. Parental history
   3. Accommodative problems
      a. Poor facility of accommodation
      b. Near point esophoria

B. Near work
   1. Accommodation
      a. Mechanical affect on globe/lens
      b. Retinal blur
         i. compounded by accommodative problems

C. Retinal blur initiates biochemical signal for axial growth as form of compensation for retinal defocus

D. Emmetropization gone awry leads to myopia
V. Interventions to prevent myopia

A. Bifocals
   1. Prevent accommodation
   2. Prevent retinal blur
   3. Houston myopia control study
   4. COMET Study – no significant decrease in myopia progression.

B. Contact lenses
   1. Rigid lenses
   2. Not orthokeratology
   3. Singapore study (Katz) – no slowing of the progression after 2 mos.
   4. CLAMP Study – no significant slowing

C. Anticholinergics
   1. Atropine
      a. Slows rate of progression to 0.05-0.20 d/yr compared to control group
         (0.24 – 0.91 d/yr)
      b. Rebound after discontinuation
      c. Statistical limitations
         i. anecdotes/single cases long term follow up inadequate
   2. Pirenzepine – selective antimuscarinic
      a. Efficacy studies ongoing – 50% reduction in progression short term study

D. Alternative therapies - unproven
   1. Bates exercises
   2. Anti-accommodative exercises
   3. Accupuncture
   4. Herbals
   5. Scleral augmentation
   6. Ocular hypotensives
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VI. Future directions and patient recommendations

A. Research directions
   1. Genetic
      a. Find additional loci
   2. Epidemiologic
      a. Natural history studies
      b. Identify risk factors
   3. Biochemical
      a. Utilize animal models to determine signal for eye growth then manipulate signal
   4. Interventions – controlled/masked/long term/prospective

B. When faced with young myope
   1. Cycloplegic refraction
   2. Consider progressive bifocals
   3. Consider use of atropine
   4. Early use of RGPs
   5. Encourage enrollment in clinical trials