Decolonizing Penn's Preclinical Curriculum

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Background

- Race is a social construct with no biological basis
- Biological construction of race dates back to slavery
- A lot of these race-based standards in medicine have been debunked

Primary Objective

- Evaluate Perelman's preclinical curriculum to determine best practice for education of current and future medical students

Aims

Identify and characterize	Identify and characterize the misuse of race/ethnicity in each course of the curriculum
Conduct	Conduct review of literature to determine best practice/evidence for use of race/ethnicity
Develop	Develop specific evidence-based recommendations for course directors

Methods - Team Members

- Dr. Jaya Aysola, Christina Amutah, Adjoa Mante, Sanjna Surya and Kaliya Greenidge

Methods - Curating Examples

- Careful review of courses
- Listened to video recordings to garner any additional context
- Examples were reviewed and flagged if they did not meet evidence-based standards for the use of race/ethnicity

Methods - Literature Search

- Each course example and mention of race was reviewed by research team to identify challenges
- Any challenge identified was then researched in the following ways:
  - Conducted a literature search specific to the condition
  - Literature search on how best to use race specific to each example

Methods - Developing Recommendations

- Team met weekly to review each course case and the literature search findings

1

Harmful diagnostic bias

2

Pathologizing race

3

Race-based clinical guidelines

4

Inaccurate semantics

5

Attributing disease burden to only genetic susceptibility

Findings – 5 conceptual themes

Theme 1: Harmful Diagnostic Bias

Cell and Tissue Biology

Cystic fibrosis (CF) is a common recessive genetic disease that affects the entire body, causing progressive disability and, often, early death. CF is most common among Caucasians; one in 25 people of European descent carry one allele for CF. One in ~650 people has CF.

Current Curricula

- Describes prevalence of CF in Caucasians without discussing prevalence in other racial or ethnic populations
- Leads learners to see CF as a white disease
- Neglects other racial/ethnic groups

Up-to-Date Evidence

- Leads to under-diagnosis in non-white populations
  - Delayed and missed diagnoses in Africa

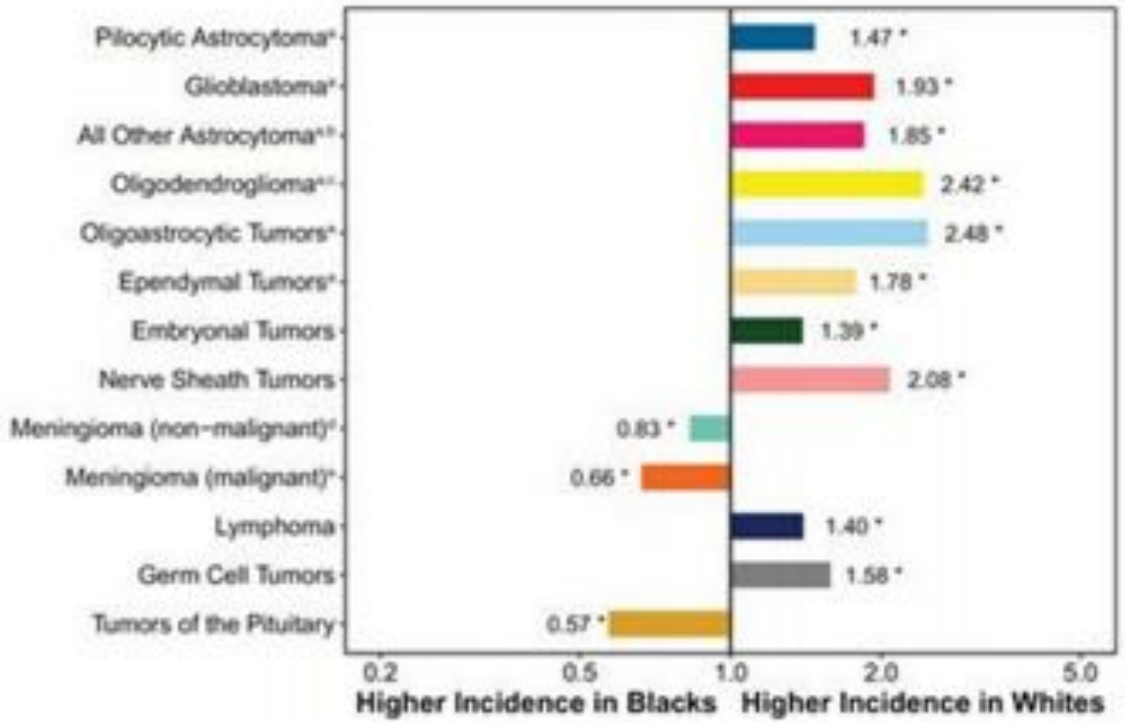
Evidence-based Recommendations

- Differences by race/ethnicity should be contextualized

Theme 2: Pathologizing Race

Brain & Behavior

Incidence



Incidence rates for non-malignant and malignant meningioma and tumors of the pituitary are higher among Blacks than Whites

Current Curricula

- No useful diagnostic information
- Unnecessarily pathologizes Black patients
- Binary neglects other racial/ethnic groups

Up-to-Date Evidence

- Perpetuates that "blackness" is associated with disease although race is a social construct
- Negative consequences to pathologizing race

Evidence-based Recommendations

- Remove the slide
- Contextualize disease burden

Theme 3: Race-based clinical guidelines

Renal

MDRD and CKD-EPI GFR Estimating Equations

- Can calculate an estimate of GFR using just serum creatinine, gender, and race (with computer/app)

- **MDRD**
  - **GFR (mL/min/1.73 m^2) = 175 × $(S_{cr})^{-1.154}$ × $(Age)^{-0.203}$ × (0.742 if female) × (1.212 if African American)**

- **CKD-EPI**
  - **GFR = 141 × min $(S_{cr}/\kappa, 1)^{\alpha}$ × max$(S_{cr}/\kappa, 1)^{-1.209}$ × 0.993^{Age} × 1.018 [if female] × 1.159 [if black]**

κ is 0.7 for females and 0.9 for males,
α is -0.329 for females and -0.411 for males,
min indicates the minimum of S_{cr}/κ or 1, and
max indicates the maximum of S_{cr}/κ or 1.

Current Curricula

- Teaches the race correction factor in the GFR equation
- Confuses Black and "African-American"

Up-to-Date Evidence

- Race correction factor exacerbates racial/ethnic disparities
- Proved unnecessary by a study on Brazilians
- Recent study uses the example of a mixed race patient to demonstrate inaccuracies

Evidence-based Recommendations

- Present evidence on both sides

Theme 4: Inaccurate semantics

Biochemistry

2.17 Clinical Case

A 21-year-old exchange student from Nigeria was brought to the hospital with a high fever. The day before admission, he was vomiting and had severe headaches and a backache.

The ED doc suspected malaria and ordered a blood smear, which was positive - treatment with primaquine was started immediately.

Four days later, the patient noticed that his urine was almost black. A complete blood analysis showed a low RBC and an elevated reticulocyte. The RBC contained Heinz bodies, the Hb levels were low, and serum bilirubin levels elevated.

Glucose-6-phosphate dehydrogenase (G6PD) deficiency is a genetic disorder that is most common in African American males in the United States. G6PD deficiency mainly affects red cells,. The most common medical problem it can cause is hemolytic anemia. That happens when red blood cells are destroyed faster than the body can replace them. When the flux through the pentose pathway is slow, Heinz bodies are produced as are ROS and the concentration of methemoglobin. The elimination of ROS and the reduction of methemoglobin both require NADPH. So basically there is a competition between these two processes. When the flux through the pentose pathway is reduced, due to a G6PD deficiency, methemoglobin levels increases as due Heinz bodies Fe^{2+} along with many other metals can serve as single electron donors in the Fenton reaction to produce reactive oxygen. Redox-active metal ions, such as iron, copper, and sometimes manganese, can also convert O_2 to ROS, which is responsible for oxidative stress (OxS).

Current Curricula

- Highlights the inconsistencies in perception of patients' races by implicitly grouping a "21-year-old exchange student from Nigeria" with "African-American males"

Up-to-Date Evidence

- Brazilian investigation found no correlation between ethnic origin and G6PD deficiency
- "African-American" is imprecise

Evidence-based Recommendations

- Focus on disease pathology rather than race
- Contextualize disease burden

Theme 5: Attributing disease burden to only genetic susceptibility

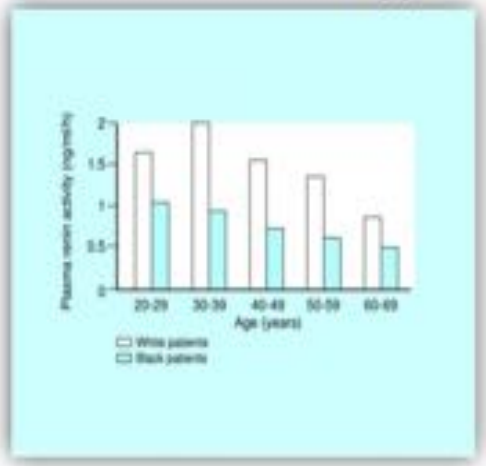
Cardiology

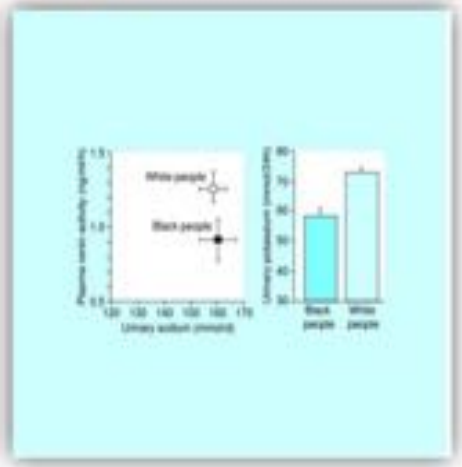
Epidemiologic Factors Known to Influence the Prevalence of Hypertension

- Heredity:
  - Population level:
    - US African Americans, Africans in the UK
    - Asians (especially when living in Asia)
  - Family level:
    - Familial aggregation
    - Aggregation in monozygotic twins (50%)
- Dietary sodium intake:
  - Population level link between dietary sodium intake and hypertension prevalence

3F-Hypertension Overview
Dr. F. Silvestry
11:00 AM
09/05/2018

Racial Differences in RAAS in Hypertension





Current Curricula

- Does not discuss environmental context, suggesting genetic basis

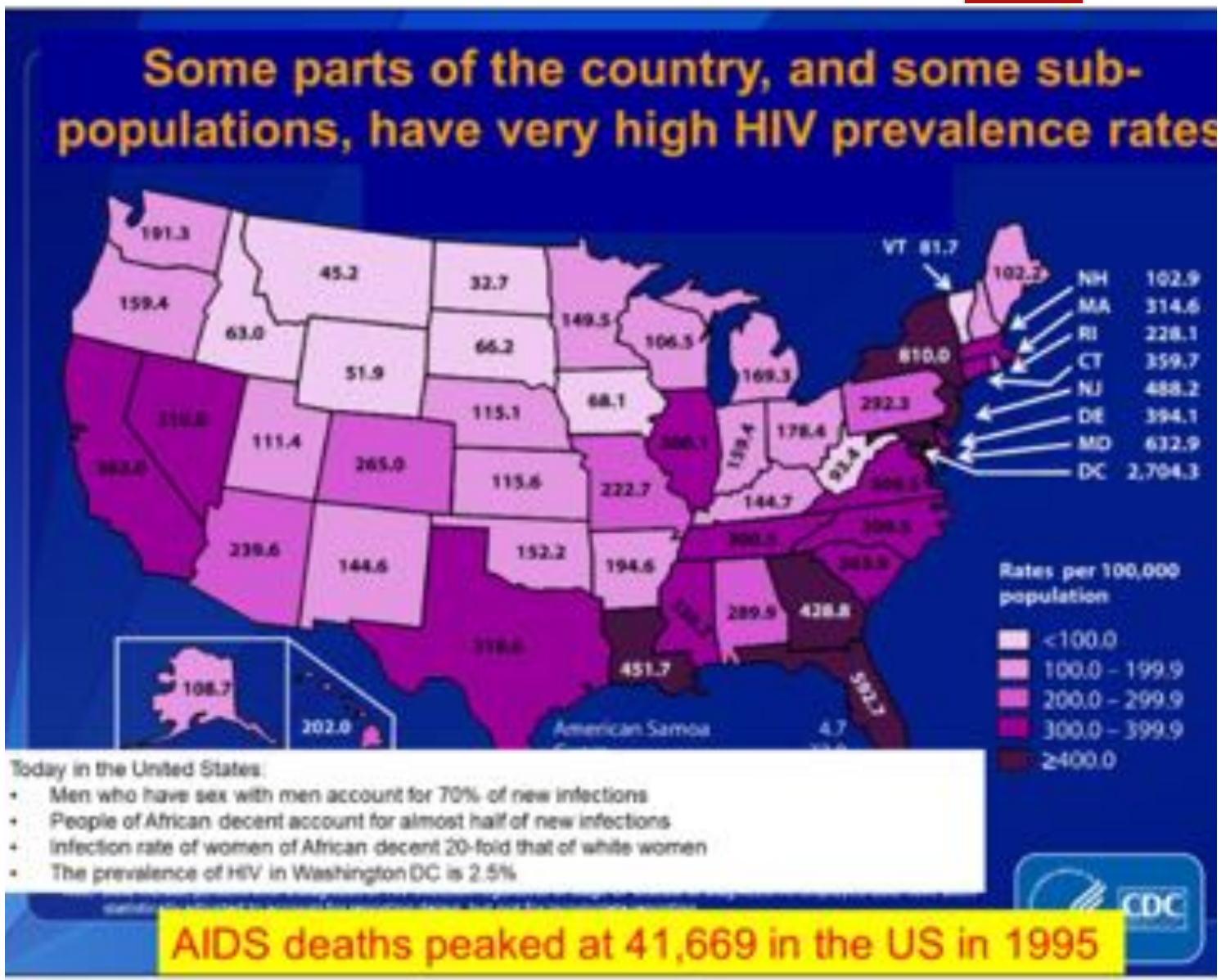
Up-to-Date Evidence

- No risk factors unique to Black people
- Stress is a predictor of health outcomes
  - Residential segregation
  - Structural racism

Evidence-based Recommendations

- Highlight significant role of social environment among populations

Microbiology



Some parts of the country and some sub-populations have very high HIV prevalence rates. Men who have sex with men account more than 70% of new infections in the US; people of African decent account for nearly half of all new infections, and the infection ra women of African decent is 20-fold above that of white women and 5-fold above that of Hispanic women.

Current Curricula

- Does not provide environmental or social context
- Highlights incidence rates in Black people

Up-to-Date Evidence

- Societal-level factors
- Widespread distrust
- Mass incarceration and ineffective drug policies

Evidence-based Recommendations

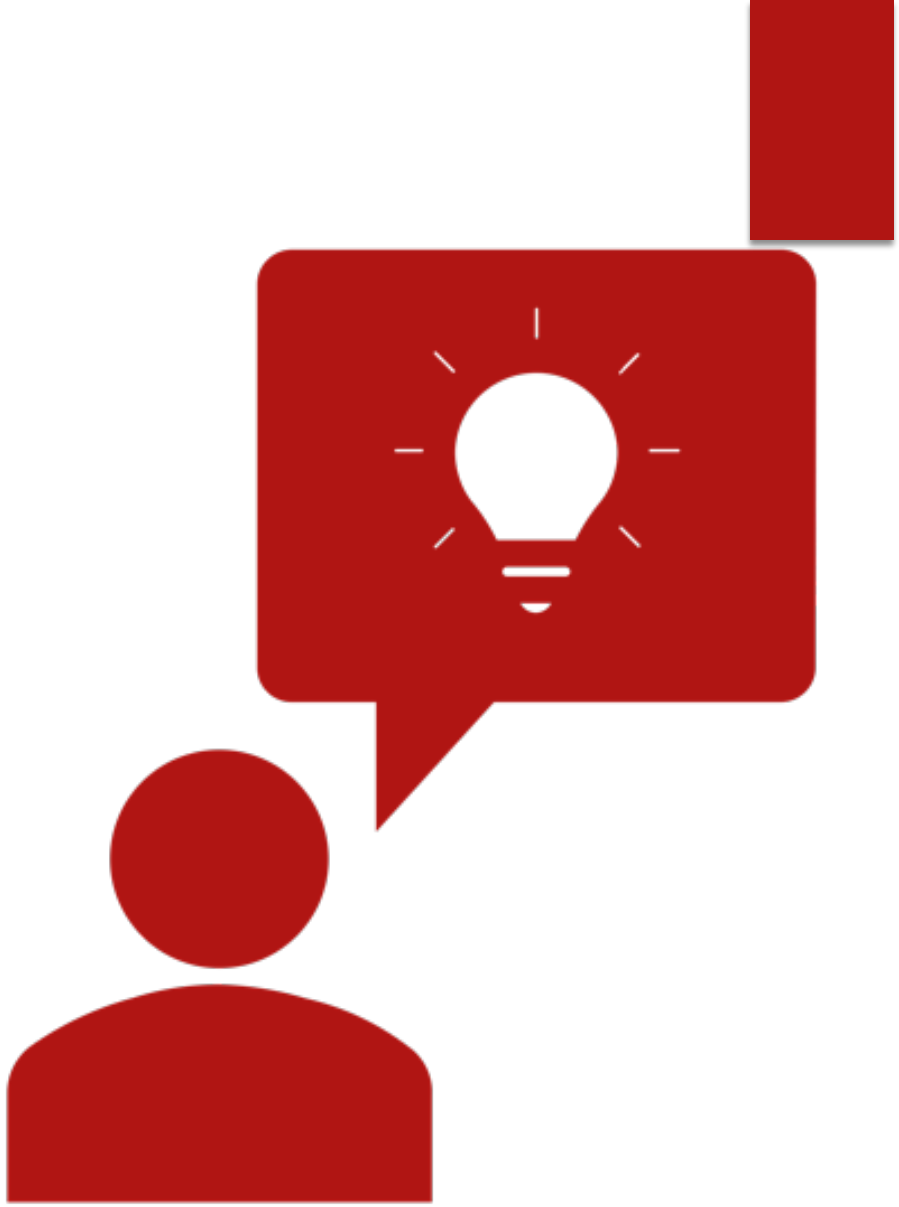
- Highlight significant role of social environment among populations

Next Steps

- Scholarship
  - SUMR presentation
  - Manuscript of archetypes with recommendations to change
- Operations
  - Course director resource guide
  - Curriculum committee mandate

Lessons Learned

- If this were any other realm of medicine, this would not be necessary
- Evidence is everything



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