Summary of Roundtable Discussion on Clinical Implications of CVD in HIV-infected Patients

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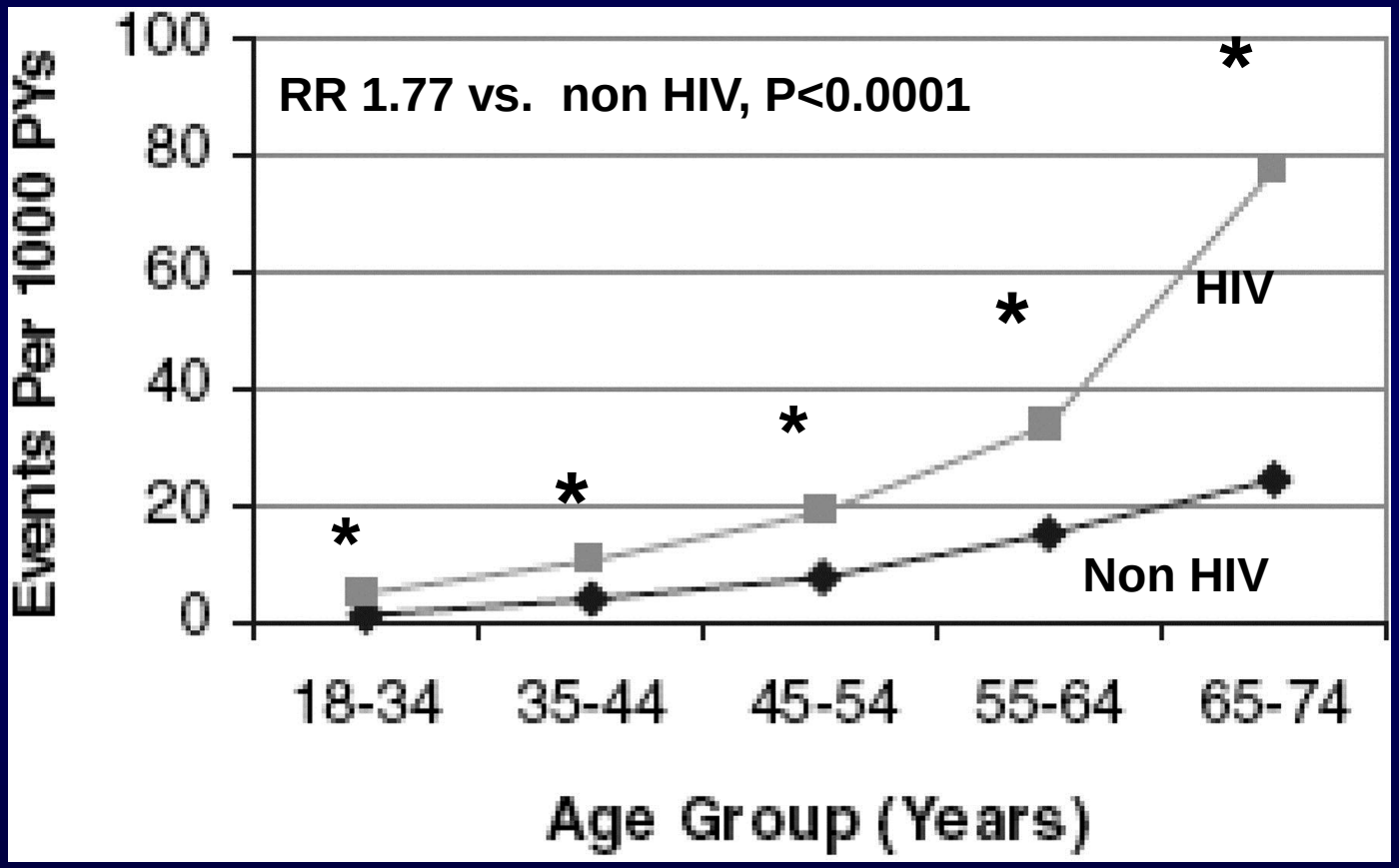
Studies Assessing Relative Risk of CVD in HIV vs. Control

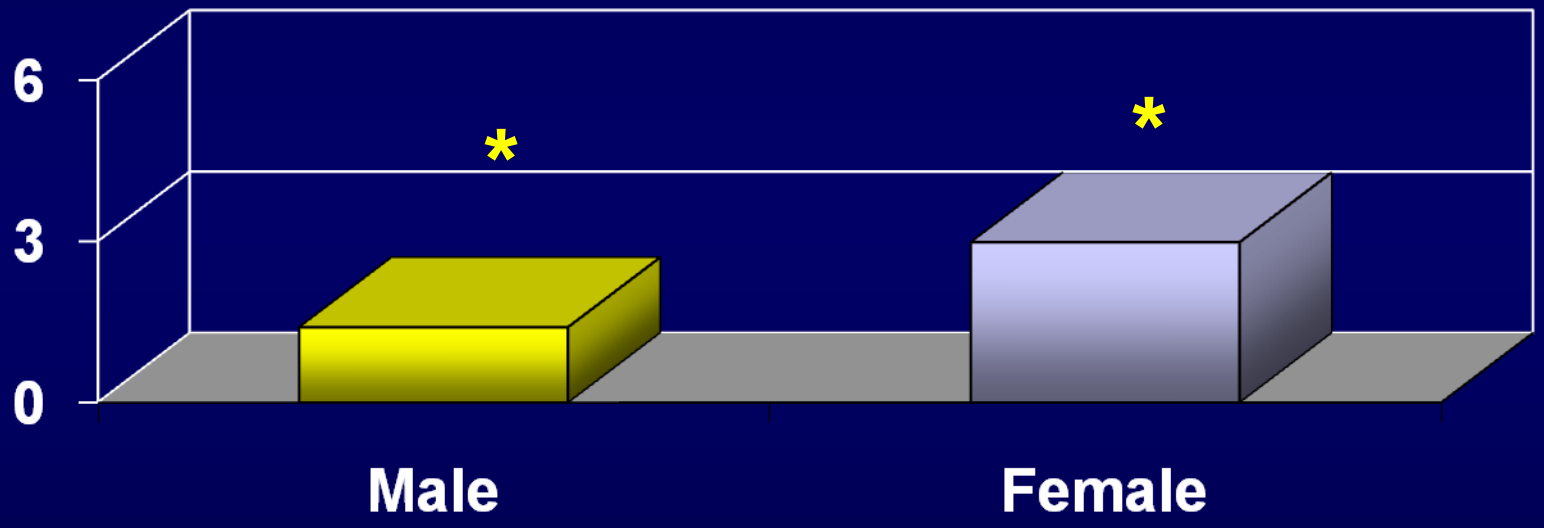
Study	Year	Population	N (HIV)	% Female	Primary Result	Effect SIze	Control Group
Klein	2002	Kaiser	4159	0	Inc MI Inc CHD	1.5 RR 1.7 RR	Yes
Currier	2003	Medicaid	28513	27	Inc CHD 18-33	2.06 RR	Yes
Triant	2007	Partners	3851	30	Inc MI	1.75 RR	Yes
Obel	2007	Danish Cohort	3953	24	Inc CHD on ART	2.12 RR	Yes
Lang	2010	FHDH	74958	-	Inc MI	1.5 SMR	Yes (ext.)

MI rates in HIV+ (3800) and non-HIV-infected patients (1,000,000) between 1996-2004



Triant et al JCEM 2007

Cardiovascular Risk
Women vs. Men
RR vs. Controls



*** P< 0.001 vs Control**

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Importance of Traditional and Nontraditional Risk Factors

	HIV (N=3851)		Non-HIV (N=1044589)	
	Number	%	Number	%
Hypertension	818	21.24†	165665	15.86
Diabetes	443	11.50†	68565	6.56
Dyslipidemia	896	23.27†	184291	17.64

Attenuation of Risk When Adjusting for:

- HTN 4%
- Dyslipidemia 10%
- Diabetes 10%

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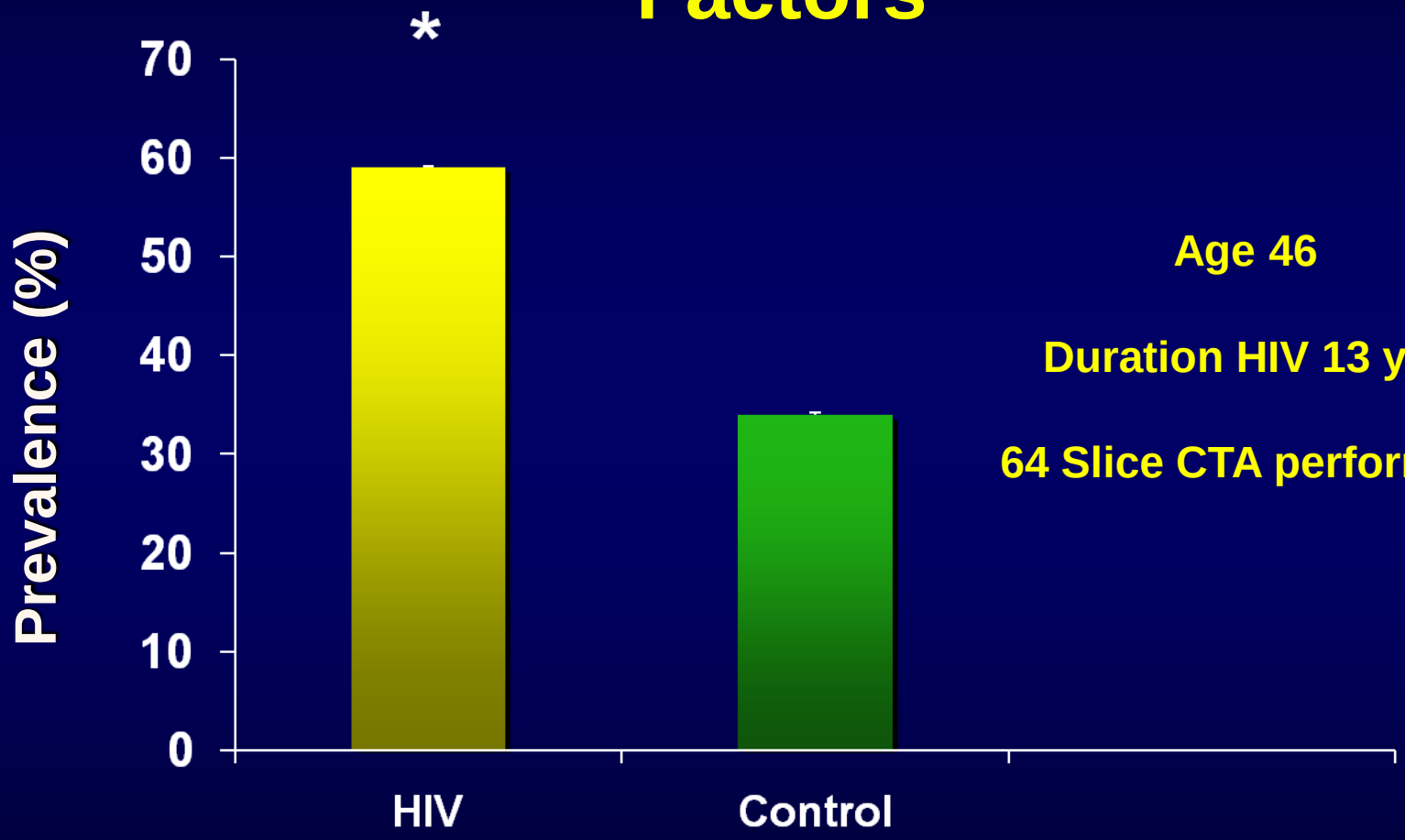
Presence of Plaque in Young Asymptomatic HIV vs. Non HIV Matched for Traditional Risk Factors



****P*=0.02 compared to Controls**

Lo et al. AIDS 2010

Consensus

- There does appear to be emerging evidence for increased CVD risk in HIV.
- Increased traditional risks which are more prevalent in HIV, such as smoking, diabetes, hypertension, and dyslipidemia should be diagnosed and addressed, using existing recommendations .
- All HIV patients should have these issues addressed early in their care and regularly readdressed, eg when going on new medications.
- Lifestyle intervention is critical for modifiable risks.

Questions (I)

1. Is HIV a CVD risk factor equivalent?
2. How should evidence for increased CVD risk in HIV be factored in to current screening and detection algorithms?
3. Do we have enough evidence to assume increased risk and treat as such?
4. Should all HIV patients be risk stratified with a risk prediction algorithm, eg Framingham? How does the Framingham equation perform in the HIV population? How should it be used?
5. What additional information do we need?

Questions (II)

1. Does the pathophysiology of CVD in HIV differ such that we should use different tests to detect it and different strategies to treat it?
2. Does the evidence of the risk justify treating with ASA, earlier intervention with lipid lowering therapy?
3. What additional information and studies do we need to make these decisions?

Future Directions

- Obtain further evidence for the degree of risk association with HIV, to determine if we can use HIV status as a risk equivalent and how we might optimally incorporate this information into current practice patterns
- Obtain further data on the relative mix of traditional and nontraditional risk factors that contribute to increased CVD in HIV, and in particular, learn more about managing nontradtional risk factors, eg through control of virus, inflammation, etc