

FOIA MARKER

This is not a textual record. This is used as an administrative marker by the William J. Clinton Presidential Library Staff.

Collection/Record Group: Clinton Presidential Records
Subgroup/Office of Origin: National AIDS Policy Office
Series/Staff Member: Patsy Fleming
Subseries: National AIDS Strategy

OA/ID Number: 10181
FolderID:

Folder Title:
Invitations April 1995

Stack:	Row:	Section:	Shelf:	Position:
S	66	5	9	2

3/27

Marcia Hale 456-7060

Dir. Intergovernmental Affairs

Harry McPherson

South Africans

Therapy for Domestic System

April 3 ~~7:00~~ 2pm

~~on~~
~~cancelled by~~
~~4-3-95 by~~
~~Annette~~
yes

Annette 6026
371-

4 PP

Trip to NYC
THE WHITE HOUSE
WASHINGTON

Linnet
~~LENETTE~~ BLA BLA
212-614-0023

A/fare → 300.00
Hotel → 200.00
Taxi → 50.00
Incidentals → 100.00

Hotel Raddison CNE# 4732397
in Empire - Queen Bldg / Non Smoking
44 West 63rd [Broadway & Columbus]
4pm flight to NYC 212-265-7400
Reception 630p

3/24/95

TO Carmena *Rec'd*

DATE _____ TIME 11:45

PHONE CALLS
"WHILE OUT" RECORD

M. Linnet

OF Black Leadership comm.

PHONE 212 614 0023
AREA CODE NUMBER EXTENSION

PF cannot introduce Rev. Jackson

TELEPHONED	☒	PLEASE CALL	☒
CALLED TO SEE YOU	☒	RETURNED CALL	☐
WILL CALL AGAIN	☐	URGENT	☐

MESSAGE
April 25 - 1-230pm

15 mins. Talk

10 Introduce Rev. Jackson

AT will be sent + be with be picked up
at NY Airport & returned.

TAKEN BY: MB

Office of
National AIDS Policy

Correspondence Routing Slip

Tracking Number: G1198
(File name in N:\corres Directory)

Date Rcvd: 3-23-95

From: Debra Fraser-Howe / BLCA

On the Calendar

Staff Action:

Jeff Levi RCVD: _____ DONE: _____

Assigned To: Carmena

RCVD: _____ DONE: _____

Reviewed By Jeff: _____ Reviewed By: Patsy _____

Action for Staff:

Recommendation/Instruction:

① Can you find out if this letter means two different speaking times — a keynote by Patsy and an intro to Jackson? If so, when is PP's speech.

② Once you have that info — walk through w/ PP since we need to reply quickly.

To Support Staff: _____

Date of Response: _____

Copy given to RS
3/27/95
○



AIDS

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Alfonso Wyatt

Executive Director/CEO
Debra Fraser-Howze

April 20, 1995

Ms. Patricia S. Fleming
National AIDS Policy Director
750 17th Street, N.W., Suite 600
Washington, D.C. 20503

Dear Ms. Fleming:

Thank you for agreeing to participate in the National Black Leadership Commission on AIDS nationwide Black Clergy Summit entitled "Turning Pain To Power." The main objective of this invitation-only summit is to bring together some of the clergy leadership from across the country to provide counsel, direction and solutions to the problems facing communities of African descent, particularly the Black church, in the face of the HIV/AIDS epidemic.

The summit will begin on Monday, April 24th with a reception to welcome clergy members and to introduce Ms. Patsy Fleming, National AIDS Czar for the President of the United States of America. The reception will take place from 6:00-8:00 PM in the 50th Floor Tower Room of the Equitable Life Assurance Society, located at 787 Seventh Avenue and 52nd Street. We would like you to attend the reception and you are welcome to bring a guest.

The summit participants consist of 50 selected clergy leaders from across the country, representing the cities of Newark, Detroit, Philadelphia, Chicago, San Diego, Los Angeles, Miami, Tampa, Atlanta and cities across New York State. They represent millions of Black people, many holding elected leadership positions in their denominations in addition to their congregations. Any message to this group affects us worldwide. Thank you again for your participation and please try to make the reception on Monday evening at the Equitable.

Enclosed please find your airline tickets. Please contact Ms. China Bushell at (212) 614-0023 immediately if there are any problems with your travel arrangements. Also attached is a time schedule, contact sheet with important telephone numbers and list of conference hosts along with a summit agenda. We ask that you review the enclosed agenda for the scheduled time and date of your appearance.

Patsy, the city is a buzz with the excitement of your coming. There will be press at the reception, although they will be barred from the actual summit. My press person will call you for necessary information. You can make remarks at the reception in any form you wish. There will be a mix of black leaders -- 100-150 are expected. There will be plenty of refreshments. I am enclosing sixteen invitations for any guests you would like to invite. At the summit you will be introduced by Rev. Herbert Daughtry, from our Board. Other members who will be there with you include: Rev. Calvin Butts III, Rev. Timothy Mitchell, and Darwin N. Davis, my Chairman and Senior Vice President of Equitable Life. Fifty leading clergy from the cities of Newark, Philadelphia, Detroit, San Diego, Tampa, Atlanta, Baltimore, Washington, Chicago, Bel Aire, Los Angeles, Rochester and Syracuse will be participating.

FFC did not receive
e

National Black Clergy Summit

Page 2 of 2

We ask that you speak about President Clinton's commitment, accomplishments, and future goals in AIDS and the role that Black clergy leadership can play in the Administration's efforts. We ask also for your support of our national replication efforts that are presently being initiated through the development of our black clergy project in 17 U.S. cities hardest hit by the epidemic. your presentation will be followed by a personal perspective from a woman who is HIV positive and from the church community. Dr. Beny Primm will follow with a presentation on drug abuse, STDs and AIDS, with a final panel that includes the Ambassador of Uganda, Derrick Bell, Esq., Dr. Andrew Billingsley and Judge Sterling Johnson, who authored the decision on Haitian refugees and is a founding member of the board of BLCA. All of your arrangements are being handled by my Executive Secretary,

Ms. Linet Harley and she can be reached at (212) 614-0023. I look forward to seeing you and hope that you can stay for some of the sessions. We ask that if you have a headshot you bring it with you to the event. We would like to include it in the follow-up journal for the summit.

The National Black Leadership Commission on AIDS will pay all transportation costs incurred in connection with the summit. Please retain and submit all receipts to a member of the BLCA staff for reimbursement.

We look forward with great anticipation to your coming, and may God be with you until we meet again.

Sincerely,



Debra Fraser-Howze

President/CEO

National Black Leadership Commission on AIDS

See you -
😊
God Bless



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Rev. Calvin Rice
Father Robert Seay
Roberta Vogel, Ph.D.
Mark Wade, M.D.
Rev. Preston Washington, D. Min.
Rev. Lee H. Wesley, D.D.
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Dennis Walcott
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Executive Director/CEO
Debra Fraser-Howze

March 22, 1995

Ms. Patricia S. Fleming
National AIDS Policy Director
750 17th Street, N.W., Suite 600
Washington, D.C. 20503

Dear Ms. Fleming:

On behalf of the Black Leadership Commission on AIDS, I want to invite you to be one of our keynote speakers at a special invitation-only national summit on HIV/AIDS for Black Clergy leaders. The summit will be held from Monday, April 24th to Wednesday, April 26th in New York City.

We are asking that you make remarks regarding the President's work and his commitment to HIV/AIDS policy that addresses the needs of the Black community, as well as the need for increased advocacy and service provision in the Black church. We are also asking that you introduce our keynote speaker at a luncheon to be held on April 25, 1995 at the Equitable Life Assurance Society, located at 787 Seventh Avenue and 52nd Street, from 1:00 - 2:30 p.m.; we anticipate a confirmation from Reverend Jesse Lewis Jackson.

The summit has targeted 50 clergy leaders from across the country, and the focus of the summit will be to:

- ◆ Develop a comprehensive national social marketing and business plan for the Black church, to address funding and program needs in response to the HIV/AIDS epidemic in their communities;
- ◆ Develop a framework that will facilitate effective dialogue with the foundation community on the needs of the Black church leadership in their fight against HIV/AIDS;
- ◆ Develop a national initiative that will enable the Black church to become better providers of care and services for people with HIV/AIDS and increase their advocacy and visibility on this issue;

Page 2

Ms. Patricia Fleming
National AIDS Policy Director
March 22, 1995

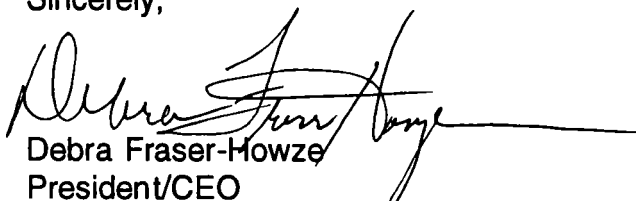
- ◆ Analyze the international import of HIV/AIDS for people of African descent from America to Africa to Brazil and develop an international response to the epidemic;
- ◆ Outline the provisions needed for a sustained education and training curriculum for pastors entering the field from Black seminaries, including urban centers with large numbers of Black seminaries, as well as outline a system that provides continued support for Black leaders.

Other participants include: Rev. Calvin Pressley; Rev. Dr. Franklyn Richardson; Rev. Wyatt T. Walker; Rev. Robert Franklin; along with representatives from the Lilly Foundation; the United Way; Kellogg Foundation; Ford Foundation; the Aaron Diamond Foundation; and AmFAR.

Ms. Linet Harley, Executive Secretary to the President/CEO, will confirm your attendance by telephone. In addition we ask that you have your secretary fax back the enclosed reservation form so we can make all necessary arrangements for your travel and accommodations. All expenses for the summit will be paid for. Please fax the reservation form to the National Black Leadership Commission on AIDS, (212) 614-0057. If you have any questions, please feel free to contact Ms. Harley at (212) 614-0023.

We hope that you will be able to join us for this important summit, and I look forward to seeing you there.

Sincerely,


Debra Fraser-Howze
President/CEO

DFH:sh

Enclosure

*I'm looking forward
to pretty you in this
setting - They will love you!*


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Rev. Timothy Mitchell, D. Min.
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Rev. Calvin Rice
Ruth Robert Seay
Roberta Vogel, Ph.D.
Mark Wade, M.D.
Rev. Preston Washington, D. Min.
Rev. Lee H. Wesley, D.D.
Marjorie Whitburn-Demp
Dennis Wilson
Canon Frederick Williams
Allison Wyatt

Executive Director/CEO
Debra Fraser-Howe

TO: Ms. Carmena Parris

FROM: Linet Harley

DATE: March 29, 1995

RE: **National Black Clergy Project - Ms. Fleming's
availability on April 24, 1995 for opening reception**

There is going to be an opening reception to the National Black Leadership Commission on AIDS Clergy Project to be held on Monday, April 24, from 6-8pm at the Equitable (787 7th Ave, NYC). This reception will also be celebrating Ms. Patricia Fleming in honor of her appointment and we are asking that she be available on April 24th from 6-8pm to attend this reception.

Carmena, please call and let me know if she can do this. Debra really wants her to be there, especially since she wants to introduce her to BLCA's board of commissioners as well as other distinguished guests that will be attending.

Thanks again.

Yes
NP
3/29/95
C

BLACK LEADERSHIP COMMISSION ON AIDS, INC.

105 East 22nd Street
New York, NY 10010
212-614-0023
212-614-0057 - Fax

Here's The Fax!!

Please deliver the following 2 sheet(s) including cover page.

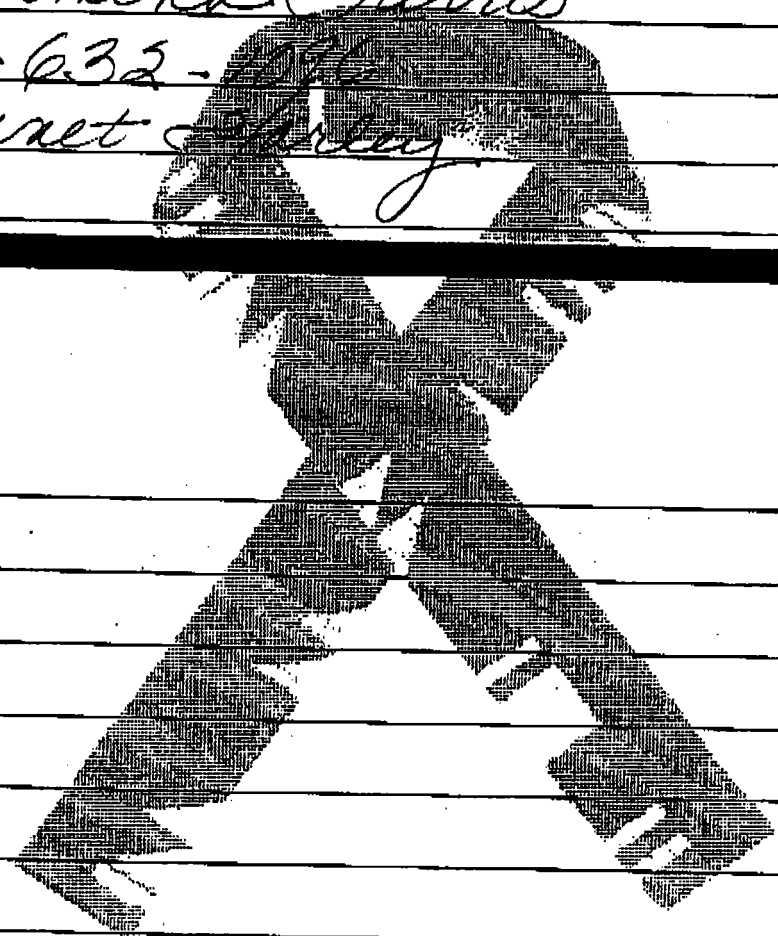
Date: March 29 1995

To: Carmena Harris

Fax: 202-632-1046

From: Lucretia Harris

Fax: _____

Message:

Confirmation via Fax

To: Ms. Linet Harley
National Black Leadership Commission on AIDS

Fax #: (212) 614-0057

From: _____
Attendee's name

Date: _____

_____ I will be unable to attend the National Black Clergy summit
on Monday, April 24, 1995

_____ I will be able to attend the National Black Clergy summit

I will be traveling: _____ via airline

_____ via train

I will require the following travel arrangements: _____

I will require the following hotel accommodations: _____

Please have Ms. Linet Harley contact my secretary, _____,
at the following telephone number: _____

****Please return this fax to the BLCA office. Our fax number is (212)614-0057. If you have any questions,
please call Linet Harley at (212) 614-0023.



AIDS Policy Center *For Children, Youth & Families*

April 19, 1995

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Chicago
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Executive Director

Ms. Patricia Flemming
National AIDS Policy Director
The White House
750 17th Street, NW
Washington, DC 20006

Dear Patsy:

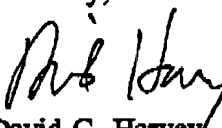
I am very pleased that you will join our community at the APC public policy conference on Monday May 1, 1995 to deliver a plenary address on HIV/AIDS and the Contract with America. On behalf of APC, we remain deeply appreciative of your efforts within the administration and Congress to address pediatric and adolescent HIV public policy issues.

As you are also aware, we are closely monitoring developments related to national HIV testing policy being considered by the United States House of Representatives in the context of reauthorization of the Ryan White CARE Act of 1995 and the Newborn HIV Notification Act of 1995. As an example of our commitment to this issue, we have scheduled a plenary debate on national HIV testing policy at our conference with Congressman Ackerman, New York Assemblywomen Nettie Mayersohn, Keith Krasinski, M.D., Janet Mitchell, M.D., and Gigi Nicks. Mason Essif of CNN and Genevieve Wood of NET Political News Talk Network will facilitate the debate. We are still in need of a spokesperson from the administration and have requested that either Helene Gayle, M.D. or Jim Curran, M.D. from the CDC be a part of the debate.

In order to discuss the most recent developments related to HIV testing policy in Congress and within the administration, I am requesting a meeting with you and representatives of APC for Thursday morning April 27 for some time after 10:30am. We have requested a similar meeting with Phil Lee, M.D.

Thank you for your consideration of this request. We look forward to meeting with you.

Sincerely,


David C. Harvey
Executive Director



WASHINGTON, D.C.

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HOTEL

EXECUTIVE BUSINESS CENTER

999 9th Street, NW
Ballroom Level
Washington, DC 20001
(202) 898-9000 x.3430

Fax (202) 789-2320

DATE: 4/26/95PAGES SENT: 7
(including cover)FAX NUMBER: 632-1096

PHONE NUMBER _____

TO: Ceramea ParrisCOMPANY: Office of the National AIDS CoordinatorFROM: B.J. Harris, Dir. of Gov't Affairs, NASTADMESSAGE: Here is the agenda.IF YOU DO NOT RECEIVE ALL PAGES. PLEASE CALL THE NUMBER
LISTED ABOVE.

Services provided by:

PROJECTION 

**OFFICE OF NATIONAL ADVISORY
EXECUTIVE OFFICE OF THE PRESIDENT**

750 17th Street, N.W.
Washington, DC 20503

Phone: 202-632-1090
Fax: 202-632-1096

FACSIMILE COVER SHEET

TO: Jason Hendrix, NASTAD

FAX NUMBER: 202-434-8092

FROM: Carmona Pam's

DATE: 4-21-95

PAGES INCLUDING COVER SHEET: 2

COMMENTS:

Per your request.

202 434-8092

Jason Hendrix

Fax,

PFs - Bio

**National Alliance of
State and Territorial AIDS Directors****FAX**

444 N. Capitol Street, NW
Suite 617
Washington, DC 20001
Phone (202) 434-8090
Fax (202) 434-8092

*Urgent*TO: Carmen ParrisFROM: Lynne GreathallDATE: 4/20/951 Page(s) to Follow☒ AS REQUESTED☐ REVIEW AND COMMENT☐ AS DISCUSSED☐ PLEASE RESPOND☐ FOR YOUR INFORMATION*Please
disregard
previous
fax.*

IF TRANSMISSION IS NOT COMPLETE, PLEASE CALL (202) 434-8090

COMMENTS:

*+ Please note that Patsy
is scheduled to speak on
Wednesday, April 26
@ 12 noon.*

*(Not on Monday as indicated)
in invitation)*

Please see attached agenda.

By:

Apologies for previous fax

National Alliance of State and Territorial AIDS Directors

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(503) 731-4020

March 4, 1995

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Wayne Sauseda
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(907) 581-4405

Patsy S. Fleming
National AIDS Policy Coordinator
Executive Office of the President
750 17th Street, N.W., Suite 1060
Washington, D.C. 20500

Dear Patsy:

Executive Committee

Both Weinstein
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Florida
Dennis L. Stover
Indiana
Joseph Horman
Maryland
John Auerbach
Massachusetts
Randall Pope
Michigan
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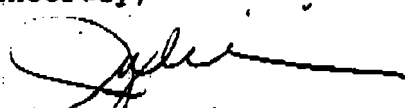
On behalf of the Executive Committee of the National Alliance of State and Territorial AIDS Directors (NASTAD), I would like to extend an invitation to you to participate in our upcoming annual meeting which is scheduled for April 23-26 at the Renaissance Hotel in Washington, D.C. Specifically, we would like you to be our keynote speaker on Monday, April 24 at 8:00 p.m.

As you know, NASTAD is a three year old alliance of directors of state and territorial based HIV/AIDS prevention and care programs. Over the last year, we have focussed on activities designed to strengthen HIV/AIDS programs administered by state and territorial health departments including the implementation of HIV prevention community planning and the reauthorization of the Ryan White CARE. Your perspective on these issues and other Administration initiatives would be very valuable. In addition, this would be an excellent opportunity to provide an update on your activities since your appointment and to share your priorities for the future.

We are very hopeful that you will be able to join us on April 24. If you are unavailable at this time, we are happy to discuss other times that fit your schedule. I will follow-up with your office in the next week or so to discuss your availability. In the meantime, I can be reached at (202) 434-8090 if you or your staff have any questions.

Thank you for your consideration. We look forward to hearing from you.

Sincerely,


Julie M. Scofield
Executive Director

Executive Director

Julie M. Scofield

DRAFT**Wednesday, April 26****8:30 am Continental Breakfast Grand Ballroom - Central****8:30 am - 9:30 am Plenary Session Grand Ballroom - Central**

Update from HRSA

❖ Terje Anderson, Vermont

❖ Anita Eichler, Director, Division of HIV Services, HRSA

9:30 am - 12 noon General Session Grand Ballroom - Central

Conclusion of NASTAD business, nominations & elections, consideration of resolutions, and vote on by-laws.

❖ Randy Pope, Michigan

❖ Beth Weinstein, Connecticut

12 Noon Working Lunch Meeting Rooms 8 & 9

❖ Wayne Sauseda, Chair, NASTAD

❖ Patricia Fleming, National AIDS Policy Coordinator

1:30 pm - 3:00 pm 4 Concurrent Sessions III**Strategic Planning Work Group meeting Meeting Room 02**

❖ Sally Finney Brazier, Kansas, Chair, Strategic Planning Working Group

Housing (HOPWA) Meeting Room 03

❖ Aimee Berenson, AIDS Action Council

❖ Lynda Kettinger, South Carolina

❖ Casey Blass, Texas

❖ Craig Thompson, Mississippi

New Technologies for Testing for HIV Meeting Room 04

❖ Sandra Schoenfisch, Florida

❖ Doug Morgan, New Jersey

OPEN SESSION Meeting Room 11

Time to address issues raised during the meeting.

3:15 pm - 4:15 pm Closing Session & Remarks Meeting Rooms 8 & 9❖ David Mulligan, Commissioner, Massachusetts Department of Health,
Chair, ASTHO HIV Committee, ASTHO Liason to NASTAD

❖ Wayne Sauseda, Chair, NASTAD

❖ Julie Scofield, Executive Director, NASTAD

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National AIDS Policy

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Tracking Number: 61290
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Date Rcvd: 4-11-95

From: ABA

Staff Action:

Jeff Levi RCVD: _____ DONE: _____

Assigned To: Churn

RCVD: _____ DONE: _____

Reviewed By Jeff: _____ Reviewed By: Patsy _____

Action for Staff:

Recommendation/Instruction:

To Support Staff: _____

Date of Response: _____

~~Decline per SE~~
yes
Reception
Friday, April 21
4-6 pm
On Calendar
Accepted w/ Keith White
4/13/95
2

Office of
National AIDS Policy

Correspondence Routing slip

on Calendar

Tracking Number: 1224
(File name in N:\corres Directory)

Date Rcvd: 3-27-94

From: ASTHO

Staff Action:

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Assigned To: Casmena - scheduling

RCVD: _____ DONE: _____

Reviewed By Jeff: _____ Reviewed By: Patsy _____

Action for Staff:

Recommendation/Instruction:

To Support Staff: _____

Date of Response: _____

Reception
April 26 (Wash)
5-7 PM
yes tentatively
Accepted 3/29/95
D



The Association of State and Territorial Health Officials

requests the pleasure of your company

at

a reception with state health officers.

Sequoia Restaurant, Washington Harbour

3000 K Street, NW

Washington, D.C.

Wednesday, April 26, 1995

5:00 p.m. - 7:00 p.m.

R.S.V.P. 202/546-5400 by April 19, 1995.

Feb 16
Erin Bush
202-543-9129
American Social
Health Assoc.

Peggy Clark, President

April ~~16~~ 7

2 pm 1/2 Hr

on calendar

area <u>4-3</u> TIME <u>12:00</u>		TELEPHONED	PLEASE CALL
PHONE CALLS		CALLED TO SEE YOU	RETURNED CALL
"WHILE OUT" RECORD		WILL CALL AGAIN	URGENT
<u>Erin Bush</u>		MESSAGE <u>Cancel Apr 7 meeting at</u>	
<u>Amer. Social Health</u>		<u>2:00 pm. Neither Peggy or</u>	
<u>543 9129</u>		<u>& Lynn can make it. How about</u>	
AREA CODE	NUMBER	EXTENSION	
543	9129		
TAKEN BY: <u>MA</u>			

February 16, 1995



Ms. Patsy Flemming
 Director
 Office of National AIDS Policy
 750 Seventeenth Street, NW
 Suite 600
 Washington, DC 20503

Post-It™ brand fax transmittal memo 7671

of pages > 2

To	Camina	From	Earl
Co.	PARAS	Co.	Bush
Dept.		Phone #	543-9129
Fax #	632-1096	Fax #	543-5327

American
 Social
 Health
 Association

11 Massachusetts Avenue, NE
 Washington, DC 20002
 (202) 543-9129
 (202) 543-5327

Dear Ms. Flemming:

On behalf of the American Social Health Association (ASHA), I would like to request a meeting with you to discuss the role of sexually transmitted disease (STD) prevention in curbing the spread of HIV/AIDS. As coordinator of the Coalition to Fight Sexually Transmitted Diseases (STD Coalition) and operator of the national AIDS hotline, ASHA has been a leader in the fight against all STDs for the last eighty years.

As you know, pre-existing STDs facilitate HIV transmission. STD prevention and treatment programs are a particularly effective strategy for AIDS prevention.

Enclosed are documents which offer compelling evidence that STDs increase susceptibility to HIV transmission. Of considerable note:

- The NIH report that prospective and case control studies with controls for behavior have shown that a women with gonorrhea is placed at a 3 to 8-fold increased risk of HIV transmission. Men with syphilis experience a 3 to 9-fold increased risk.
- Areas of the nation that experience the highest incidence of STDs, also exhibit the highest rate of HIV infection in women of childbearing age.

Twelve million new STD infections occur in the United States every year. When one considers that each of these persons faces an increased risk of acquiring HIV, it is clear that STD prevention and control should be an integral part of the national AIDS program.



Ms. Flemming
February 16, 1995
Page Two

Yet the materials developed by your office describing the President's FY 1996 AIDS initiative do not mention STD initiatives. We are concerned that this omission may indicate that the Administration has not recognized STD prevention as a key aspect of the fight against AIDS. Certainly, the FY 1996 budget request for the CDC's STD/HIV division and the proposed consolidation with TB and HIV programs suggests that the Administration may not be giving STD control high priority.

I would appreciate the opportunity to discuss these issues in person. Ms. Erin Bush of ASHA's Washington office will contact your office next week in order to determine if your schedule would allow for a brief meeting.

Thank you for considering this request.

Sincerely,

Peggy Clarke
President

enclosures

Carmina -

add to files on
meeting with
Peggy Clark

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STD NEWS

A QUARTERLY NEWSLETTER OF THE AMERICAN SOCIAL HEALTH ASSOCIATION

ASHA Study Assesses STD Prevention Behavior Among College Women

A new study from the American Social Health Association explores behavior among college women that is related to their risk for sexually transmitted diseases. Results of the study will be announced in late March in conjunction with National STD Awareness Month, which ASHA sponsors in April.

STDs are considered to be epidemic among college-age people, says Dr. Linda Alexander, ASHA director of women's health and survey coordinator. "Our survey focused on women because they bear a disproportionate burden from STDs," she explained. "Women—especially young women—are more susceptible to infection, less likely to experience STD symptoms and more difficult to diagnose. And in women, STDs can lead to consequences like pelvic inflammatory disease, infertility and ectopic pregnancy."

The most startling findings are the number of college women who have had unprotected oral sex, the number who have never had a pelvic exam and the number who have been forced to have sex.

In order to better understand some of the underlying issues associated with the STD epidemic, ASHA distributed self-reporting questionnaires to 1,000 women students at two major universities in the mid-Atlantic area.

Alexander says the college population was chosen because it provides an accessible sample, whereas other women in the same age group are more difficult to target. Also, Alexander explained, college students are more easily targeted with prevention messages that can be developed based on survey results.

According to Alexander, the most startling findings from the study are the number of college women who have had unprotected oral sex, the number who have never had a pelvic exam and the number who have been forced to have sex.

The study indicates that one in four women students, and one in five sexually active women students, have never had a pelvic exam. Oral sex was the most frequently reported form of sexual activity (96 percent), followed by vaginal intercourse (92 percent) and anal sex (17.5 percent), but more than three-fourths of the respondents never use condoms for oral sex. Almost one-fourth of the women reported having been forced to have sex at least once.

"The findings related to oral sex reflect a common misperception in our society that STDs are spread only by vaginal or anal sex," Alexander commented. "It was also significant that so many women in this group—typically more educated and affluent than other women their age—have been subjected to forced sex."

Other findings revealed that most women students have had vaginal sex with more than one partner during their *(continued on page 5)*

volume 2
number 3
Spring 1995

The American Social Health Association is the only nongovernmental organization dedicated solely to the prevention and control of all sexually transmitted diseases.

April is STD Awareness Month

For the fourth year, ASHA will sponsor National STD Awareness Month in April as an opportunity to focus national attention on the epidemic of STDs. This year's STD Awareness Month observance will emphasize the impact of STDs on young people, with ASHA's survey of college women as a centerpiece. ASHA encourages other organizations to develop activities during April that will heighten public awareness of STDs and their impact on people under 25. For information, call the public relations department at 919/361-8400.



Office of
National AIDS Policy

Correspondence Routing Slip

Tracking Number: G1086
(File name in N:\corres Directory)

Date Rcvd: 2-27-95

From: Peggy Clarke - ASHA

Staff Action:

Jeff Levi RCVD: _____ DONE: _____

Assigned To: Shelley

RCVD: _____ DONE: _____

Reviewed By Jeff: _____ Reviewed By: Patsy _____

Action for Staff:

Recommendation/Instruction:

HA! If you do this,
I want to be there - see comments
re Consolidation.

To Support Staff: _____

Date of Response: _____

Open

*May 6 in
Sept
is call
off with
discovery*

February 16, 1995



Ms. Patsy Flemming
Director
Office of National AIDS Policy
750 Seventeenth Street, NW
Suite 600
Washington, DC 20503

*American
Social
Health
Association*

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Sincerely,

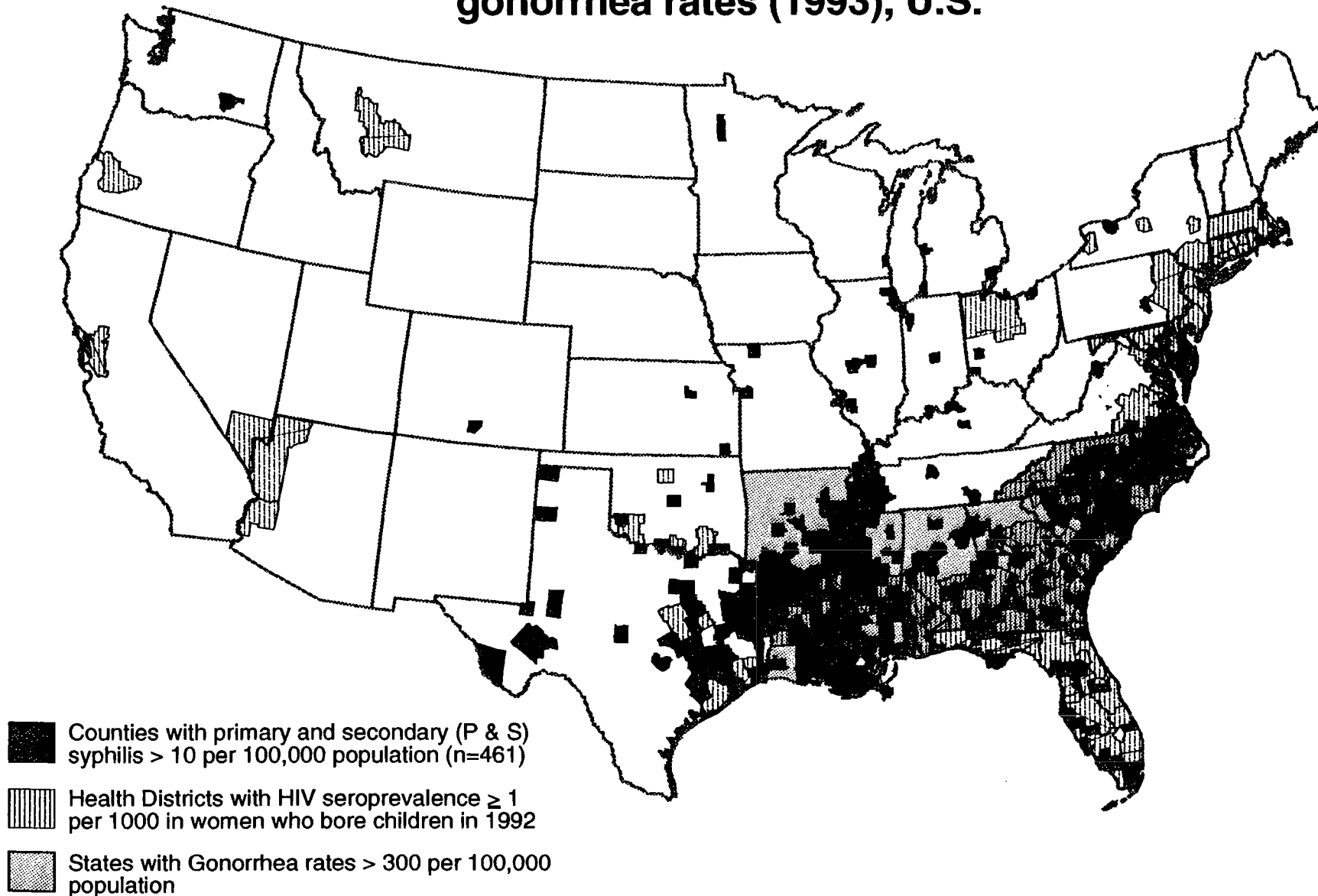
Peggy Clarke

Peggy Clarke
President

enclosures

↑ They are
afraid of being
held accountable!
JL

Health Districts with the highest HIV seroprevalence in women who bore children in 1992, counties reporting the highest primary and secondary syphilis rates (1993), and states reporting the highest gonorrhea rates (1993), U.S.



Epidemiological Synergy

Interrelationships between Human Immunodeficiency Virus Infection and Other Sexually Transmitted Diseases

JUDITH N. WASSERHEIT, MD, MPH

*From the Sexually Transmitted Diseases Branch,
National Institute of Allergy and Infectious Diseases,
National Institutes of Health, Bethesda, Maryland*

Understanding the role of other sexually transmitted diseases (STDs) in the transmission of human immunodeficiency virus (HIV), the role of STDs in progression of HIV disease, and the role of HIV infection in alterations of natural history, diagnosis, or response to therapy of STDs is critical to the development of optimal strategies for HIV control. One hundred sixty-three studies on the interrelationships between HIV infection and other STDs were examined. Of 75 studies on the role of STDs in HIV transmission, the 15 analyses of examination or laboratory evidence of STDs adjusted for sexual behavior showed that both ulcerative and nonulcerative STDs increase the risk of HIV transmission approximately 3- to 5-fold. Due to limited data, the role of STDs in progression of disease remains unclear. Preliminary data from 83 reports on the impact of HIV infection on STDs suggest that, at a community level, HIV infection may increase the prevalence of some STDs (e.g., genital ulcers). If coinfection with HIV prolongs or augments the infectiousness of individuals with STDs, and if the same STDs facilitate transmission of HIV, these infections may greatly amplify one another. This "epidemiological synergy" may be responsible for the explosive growth of the HIV pandemic in some populations. Effective STD control programs will be essential to HIV prevention in these communities.

THE INTERRELATIONSHIPS between human immunodeficiency virus (HIV) infection and other sexually transmitted diseases (STDs)* are unique, complex, and intriguing. They probably explain, in part, the heterogeneous faces of the HIV epidemic around the globe.¹⁻³

Reprinted with permission from Chen LC, Sepulveda J, Segal SJ, eds. *AIDS and Women's Reproductive Health*. New York: Plenum Press, 1992. Chapter 5.

The author thanks Willard Cates, Jr., Jeffrey Harris, and Penelope Hitchcock for their valuable input to this article, and Susan Emelio, Aretha Hankinson, and Ernestine McNeill for their excellent secretarial assistance.

Reprint requests: Judith N. Wasserheit, MD, MPH, Sexually Transmitted Diseases Branch, National Institute of Allergy and Infectious Diseases, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD 20892.

Received for publication February 13, 1991, revised June 25, 1991, and accepted July 10, 1991.

* Over twenty syndromes are now recognized as sexually transmitted diseases. These include infections caused by hepatitis B virus, several ectoparasites, and a number of enteric pathogens. However, as a reflection of available data and to limit the length of this review, we will focus exclusively on the classical STDs that result in primary manifestations in the genital tract.

and may well provide insights into the pathogenesis of all STDs, including HIV infection. Furthermore, these interrelationships have compelling implications for HIV control efforts.

Three relationships between HIV infection and other STDs have been postulated: 1) increased transmission of HIV in the presence of other STDs; 2) accelerated progression of HIV disease in the presence of other STDs; and 3) alteration in the natural history, diagnosis, or response to therapy of other STDs in the presence of HIV infection.

What has a decade of research on HIV infection taught us about these interrelationships? This review examines available data on the impact of STDs on HIV transmission and progression, and the reciprocal impact of HIV infection on other STDs. It also summarizes possible mechanisms for each relationship. It considers the program and policy implications of these relationships as we currently understand them, and emphasizes that for some STDs, the "epidemiologic synergy" between the relationships may be as important as the individual relationships themselves. Finally, the chapter highlights persistent research needs.

The Impact of Sexually Transmitted Diseases on Human Immunodeficiency Virus Transmission

Methodologic Considerations

Several methodologic problems must be considered in a critical analysis of studies of the impact of STDs on HIV transmission. First, such studies are complicated by the fact that HIV infection is, itself, an STD. Because HIV infection and other STDs share a common dominant mode of transmission, common human reservoirs, and common behavioral risk factors, STDs could merely

be surrogates for factors such as regular contact with prostitutes or other multiply-infected persons, rather than causal links in HIV transmission. Analyses that control for sexual behavior are, therefore, essential to establishing whether STDs are independent, biologic risk factors for HIV transmission. Unfortunately, even when such analyses are performed, measures of sexual behavior are often imperfect for the index subject, unobtainable for partners, and difficult to validate for both.

Additional potential for confounding arises because of the frequency of multiple co-infection among individuals infected with STDs. Comprehensive STD testing and analyses that adjust for the presence of multiple STDs are necessary to ensure that spurious associations between one STD and HIV infection do not result from underlying associations between another STD and both HIV and the STD in question.

A third problem springs from the potential impact of HIV infection on other STDs. Without prospective studies documenting temporal sequence, it is impossible to determine whether STDs facilitate HIV transmission, or whether they are markers for HIV-related immunosuppression. Furthermore, in case-control studies, detection bias may occur if, due to HIV infection, STDs are more prominent, more persistent, or more readily identifiable in laboratory testing.

Finally, both STDs and the populations that acquire them are heterogeneous. Relationships between specific STDs and HIV infection may not be uniform. In addition, the importance of STDs in HIV transmission probably depends upon competing risk factors (e.g., receptive anal intercourse or circumcision), the prevalence of which varies with gender and sexual preference. Therefore, studies that use aggregate variables such as "any STD," or that provide risk estimates that are not stratified by gender and sexual preference, are difficult to interpret.

Studies of Genital Ulcer Disease and Human Immunodeficiency Virus Transmission

Including conference abstracts, there are over 70 publications in the international literature linking STDs with an increased risk of HIV infection.⁴⁻⁷⁸ Data from at least one study support associations between HIV infection and every STD that has been examined, i.e. chancroid, syphilis, genital herpes, gonorrhea, chlamydia, trichomoniasis, genital warts, and donovanosis. Only 25 reports, however, present multivariate analyses that incorporate at least some measure of sexual behavior. These include 3 analyses of prospective or nested case-control studies^{7,18,33,39,40,44,58,76} and 17 analyses of cross-sectional case-control studies.^{3-10,13,21,22,26,29,35,41,42,54,60,62,66-68} They represent 15 study populations or da-

taset. For the methodologic reasons discussed above, this paper will focus on these studies in weighing the evidence that STDs are risk factors for HIV transmission.

Data supporting the concept that genital ulcer disease (GUD) is a risk factor for HIV transmission are currently most compelling (Table 1). Genital ulcer disease, either as a clinical syndrome or as an etiologic diagnosis such as syphilis or herpes, was investigated in each of the 15 study populations. The 10 reports linking syndromic GUD with HIV infection come from studies performed in two sites, Nairobi, Kenya and Kinshasa, Zaire, in heterosexual populations in which the majority of ulcers are due to chancroid. Controlling for sexual behavior, two of the three prospective studies found a significantly increased risk of HIV seroconversion among subjects with GUD (odds ratio [OR] 3.7 for women, 4.7 for men).^{7,58} The third prospective study failed to demonstrate an association, but the incidence of GUD in the cohort was only 5%.⁴⁴

Four of the seven case-control studies listed also found significant associations between GUD and HIV seropositivity.^{26,42,62,66} Risk estimates for women were similar to those detected in the prospective studies, but for men, estimates based on physical diagnosis of GUD ranged from an OR of 8.2 to an OR of 18.2. The latter value was for circumcised men, and reflects an interaction between GUD and circumcision with respect to risk of HIV infection.⁶⁶ It should be noted that the three case-control studies in which no significant association was found between GUD and HIV seropositivity^{13,29,67} were all performed in women, and two relied upon subjects' history of GUD without any clinical or laboratory verification. The fact that all STDs (including chancroid) are frequently asymptomatic in women, and that diagnosis of GUD may be more difficult in women than in men, may explain these divergent results. Not surprisingly, a significant association between the presence of IgG antibody to *Hemophilus ducreyi* and HIV seropositivity was found among both men and women (OR 3.0, 95% confidence interval [CI] 1.8-5.1 for men; OR 3.3, 95% CI 2.2-4.8 for women), but these risk estimates were not controlled for sexual behavior or other STDs.³⁶

The relationship between syphilis, per se, and HIV infection was examined in two prospective^{18,76} and nine case-control^{8,9,13,21,29,35,42,60,68} studies, which simultaneously considered behavioral risk factors (Table 1). Significant associations between syphilis and HIV infection were found in nine of the eleven analyses. As was the case with studies of GUD, risk estimates based on serodiagnosis of syphilis were slightly higher for heterosexual men (median OR 6.0, range 3.3-8.7) than for women (median OR 4.0, range 2.5-5.4). This may be due to a more limited misclassification bias in men than in

TABLE 1. Summary of Multivariate Analyses of Sexually Transmitted Diseases and Human Immunodeficiency Virus Infection by Study Design and by Gender and Sexual Preference of Subjects

	Prospective or Nested Case/Control Studies*	Cross-sectional Case/Control Studies*
Genital ulcers (mainly chancroid)		
Number of studies	3	7
Number of studies detecting significant association	2	4
Risk estimate median (range)		
Women	3.7 Px	3.3 Px
Heterosexual men	4.7 Px	2.4 Hx; 13.2 (8.2–18.2) Px
Homosexual men	NA	NA
Syphilis		
Number of studies	2	9
Number of studies detecting significant association	2	7
Risk estimate median (range)		
Women	NA	1.8 Hx; 4.0 (2.5–5.4) Lab
Heterosexual men	NA	2.0 Hx; 6.0 (3.3–8.7) Lab
Homosexual men	1.5 Hx; 2.2 Lab	3.0 Hx; 8.4 (2.0–9.9) Lab
Genital herpes		
Number of studies	3	3
Number of studies detecting significant association	1	2
Risk estimate median (range)		
Women	NA	1.9 (women and men) Lab
Heterosexual men	NA	2.3 Hx; 5.9 (3.3–8.5) Lab
Homosexual men	3.5 (2.5–4.4) Lab	
Chlamydial infection		
Number of studies	2	2
Number of studies detecting significant association	2	0
Risk estimate median (range)		
Women	4.5 (3.2–5.7) Lab	NS Lab
Heterosexual men	NA	NA
Homosexual men	NA	NA
Gonorrhea		
Number of studies	4	6
Number of studies detecting significant association	2	3
Risk estimate median (range)		
Women	3.5 Lab	5.6 (3.8–8.9) Lab
Heterosexual men	NA	NS Hx
Homosexual men	1.5 Hx	NA
Trichomoniasis		
Number of studies	1	1
Number of studies detecting significant association	1	0
Risk estimate median		
Women	2.7 Lab	NS Lab
Heterosexual men	NA	NA
Homosexual men	NA	NA
Anogenital warts		
Number of studies	0	5
Number of studies detecting significant association	0	3
Risk estimate median (range)		
Women	NA	3.5 (3.1–4.1) Hx
Heterosexual men	NA	NS
Homosexual men	NA	3.3 Hx; 3.7 Px

* Px = by physical examination; Hx = by history; NA = not available; Lab = by laboratory studies; NS = not significant.

women for the reasons mentioned above. In homosexual men, the single prospective study yielded low risk estimates ranging from an OR of 1.5 for history of syphilis to an OR of 2.2 for serodiagnosis. Case-control analyses resulted in higher values (range 2.0–9.9). Since the higher odds ratios in these reports were based upon syphilis serologies rather than historical data, it is unlikely that recall bias is responsible for these differences. One of the

two studies that did not demonstrate an association relied upon personal histories of syphilis from the women studied,³⁵ and the other was conducted among prostitutes in whom no syphilis was detected.³⁹

Six studies specifically addressed the relationship between genital herpes or herpes simplex virus type 2 (HSV-2) infection and HIV infection. Three prospective studies were performed in homosexual male co-

horts,^{33,39,76} and one of these documented a stronger association between HSV-2 seroconversion and HIV seroconversion (OR 4.4) than between HSV-2 seropositivity and HIV seroconversion (OR 2.5).³³ This may reflect the more fulminant course and more persistent lesions observed in primary genital herpes in comparison to recurrent disease. The other two prospective studies, one of which also measured HSV-2 serologies³⁹ and the other of which relied upon history alone,⁷⁶ failed to demonstrate a significant association. Risk estimates from a case-control study of homosexual men that included serology⁶⁸ were slightly higher than those from the prospective study described above (median OR 5.9, range 3.3–8.5). A study conducted in female prostitutes did not find an association.²⁹

Finally, it is noteworthy that one study of Zulu STD clinic patients has investigated the role of donovanosis in HIV infection.⁵² Unfortunately, the methods for diagnosing donovanosis were not stated, and potential confounding by behavioral risk factors was not considered. Nevertheless, among men, the presence of donovanosis of at least six weeks duration was clearly associated with HIV seropositivity ($P = 0.001$).

Studies of Nonulcerative Sexually Transmitted Diseases and Human Immunodeficiency Virus Transmission

Data on nonulcerative STDs and HIV infection are far more limited than data for GUD. Yet if, indeed, these syndromes do facilitate HIV transmission, the proportion of HIV infection attributable to the nonulcerative STDs (attributable risk) will far outweigh that due to GUD, because, in most populations, diseases such as chlamydial infection, gonorrhea, and trichomoniasis are far more common than genital ulcers.

Four studies examined chlamydial infection (Table 1). The two prospective studies,^{44,58} performed in African prostitute cohorts, each found a significant association between chlamydial cervicitis and HIV seroconversion (median OR 4.5, range 3.2–5.7). In contrast, neither of the case-control studies^{29,67} identified chlamydial infection as a risk factor for HIV seropositivity. The latter studies were also conducted in female prostitutes and used chlamydial culture or direct antigen detection methods. Their results are not surprising. Whereas the serologic tests employed in the case-control studies of GUD detect past infection, these methods detect current infection, which is likely to be linked to prior HIV acquisition primarily through persistent risky sexual behavior patterns.

Gonorrhea was significantly associated with HIV infection in two of four prospective^{44,76} and three of six case-control^{35,42,54} analyses that attempted to adjust for sexual behavior (Table 1). Among women, risk estimates

ranged from a relative risk (RR) of 3.5 to an OR of 8.9, with the lower figure being generated by the single prospective study. In homosexual men, gonorrhea was associated with less than a two-fold increase in risk of HIV infection. The two prospective studies that did not detect a significant association between gonorrhea and HIV infection were performed in gay male cohorts, and relied upon histories of gonorrhea rather than on clinical or microbiologic evidence of infection.^{18,40} As with studies of chlamydial infection, the frequency with which the case-control studies failed to detect an association may be related to the temporal mismatch of using laboratory tests for current gonococcal infection to examine the role of gonorrhea in HIV acquisition that occurred in the past. In at least one of these studies,²⁹ however, an additional problem was that swabs for gonococcal cultures were obtained from the vagina, rather than from the cervix.

With respect to HIV transmission, trichomoniasis remains the least well studied of the common STDs (Table 1). A prospective study⁴⁴ detected almost a three-fold increase in risk of HIV seroconversion among female prostitutes with trichomoniasis in comparison to those without infection, while a case-control analysis²⁹ found no association.

Lastly, an association between anogenital warts and HIV infection has been suggested in three of five case-control studies.^{13,41,60} Risk estimates were similar for women (median OR 3.5, range 3.1–4.1) and for homosexual men (OR 3.3 for history of genital warts, 3.7 for evidence of lesions upon examination). In homosexual men, an even stronger association was noted between the presence of human papillomavirus (HPV) DNA in anal specimens and HIV seropositivity (OR 10.4, 95% CI 1.9–56.6).⁴¹ Among HIV-positive men in this study, the mean number of CD4 cells was lower in the HPV-infected group than in the HPV-uninfected group ($P = 0.10$). Although these differences were not significant, they are consistent with augmented expression of HPV infection as a result of HIV-induced immunosuppression. Unfortunately, no prospective studies that include data both on genital warts and on sexual behavior are yet available to clarify this issue.

The Impact of Sexually Transmitted Diseases on Human Immunodeficiency Virus Progression

Methodologic Considerations

One of the most challenging problems in studies of the impact of STDs on progression of HIV disease is the need to control for duration of HIV infection. In cross-sectional case-control studies the period between diagnosis of HIV infection and onset of acquired immunodeficiency syndrome (AIDS) or AIDS-related complex (ARC) may correlate poorly with true duration of infec-

tion. Even in prospective studies, the timing of HIV seroconversion can be established only to the level of precision permitted by the interval at which HIV testing is performed.

Adjustment for baseline immunologic function and for age are also important. More marked immunosuppression at enrollment may, for example, be associated both with accelerated progression of HIV disease and with increased frequency or severity of recurrences of viral STDs. Age-related cohort effects in STD incidence may give rise to spurious associations between STDs and rapid progression to advanced stages of HIV infection.

Finally, number of exposures to an STD pathogen, rather than categorical presence or absence of any exposure, may be a risk factor for accelerated HIV progression. If this is the case, the frequency of asymptomatic infection and financial and compliance constraints on repeated STD testing may make demonstrating such an association exceedingly difficult.

Studies of Sexually Transmitted Diseases and Progression of Human Immunodeficiency Virus Disease

Only a handful of studies have addressed the role of STDs in HIV progression.⁷⁹⁻⁸³ Most are univariate analyses that do not control for baseline immunologic status or for age. Roughly half examined cohorts in which the approximate date of HIV seroconversion could be determined. Overall, the results are inconclusive.

Of three studies focusing on herpesvirus infections, two^{79,80} evaluated HIV seroconverters and adjusted for age. Subjects were homosexual males in one study, and persons with hemophilia in the other. Neither analysis controlled for baseline immunologic function. The third study⁸¹ analyzed seroprevalent cases of HIV infection in homosexual males and stratified them by initial CD4 level, but did not consider age. No significant associations between genital herpes and rate of progression of HIV disease were noted in any of these studies, regardless of whether HSV infection was documented serologically or by clinical history, and regardless of whether HIV progression was measured by p24 antigen level, by CD4 deficit, or by clinical outcomes, such as development of AIDS, ARC, or persistent generalized lymphadenopathy (PGL). Interestingly, in these studies, companion analyses of infection due to other herpesviruses, such as varicella zoster (VZV), cytomegalovirus (CMV), and Epstein-Barr virus (EBV), also did not support an association between these viral infections and an increased risk of progression of HIV disease.

The impact of bacterial STDs on HIV progression has received little attention. In asymptomatic, HIV-positive patients enrolled in a study of neurologic complications of HIV infection,³² a significant association was noted

between the presence of p24 antigen and historical or serologic evidence of syphilis ($P = 0.03$). These data were not, however, controlled for duration of HIV infection, immunologic status, or age. Data from homosexual men in the Multicenter AIDS Cohort Study (MACS) are also equivocal. In univariate analysis, subjects who developed AIDS within 30 months of HIV seroconversion were 3.4 times as likely to report a history of syphilis at some time prior to HIV infection as were controls matched for duration of HIV infection who did not develop AIDS within 30 months (Phair J. Personal communication). This difference was borderline significant. Parallel univariate analyses of gonococcal infection also suggest a link with progression. Progression to AIDS within 30 months of seroconversion was associated with a history of rectal gonorrhea both prior to (OR 3.3, $P < 0.05$) and after (OR 9.2, $P < 0.05$) seroconversion. No significant association was found between a history of urethritis before or after seroconversion and development of AIDS within 30 months.

Finally, one study³³ of steady male partners of male AIDS patients reported that PGL was significantly more common among HIV-positive subjects with a history of an STD since last intercourse with the AIDS patient, than among HIV-positive subjects without such a history (univariate OR 30, $P < 0.001$). Similar tantalizing data are provided by the MACS. In univariate analysis, subjects reporting any STD in the six months prior to HIV seroconversion were more than six times as likely to develop AIDS within 30 months than were subjects who denied having any STDs during this period ($P < 0.05$) (Phair J. Personal communication).

The Impact of Human Immunodeficiency Virus Infection on Other Sexually Transmitted Diseases

Methodologic Considerations

Because of its impact on other pathogens or on the host response, HIV infection could, theoretically, affect other STDs in a number of ways. HIV infection might alter STD incidence or frequency of recurrences. It might give rise to atypical presentations, including larger, more numerous, or more persistent lesions. Alternatively, HIV infection might modify the natural history of STDs, resulting in more frequent or more rapid development of complications. Performance of laboratory tests and response to standard therapy might also be affected.

Several of the methodologic considerations in studies of these issues are identical to those discussed for studies of the impact of STDs on HIV transmission. For the reasons stated above, sexual behaviors and coinfection with other STDs are potential confounders in the rela-

tionship between HIV infection and STD transmission, and prospective studies are essential to establish whether STDs facilitate HIV transmission, HIV facilitates STD transmission, or both.

Additional considerations are, however, important in studies of natural history, diagnosis, and response to therapy. Health care behaviors, such as rapidity of response to STD symptoms, self-medication, or compliance with STD therapy may influence each of these, and may also be related to HIV infection status. It should also be recognized that, by increasing the duration of an STD, HIV infection could increase the incidence of the STD in a community without directly facilitating its transmission through altered susceptibility or altered infectiousness in individuals.

Perhaps the most critical (and to date most often ignored) factor in assessing the impact of HIV infection on other STDs is the need for an HIV-negative comparison group. Our overall understanding of the natural history of STDs such as syphilis, chancroid, or HPV infection is still relatively embryonic. Furthermore, some of these syndromes are infamous for their protean clinical manifestations. In this context, case reports and case series of purported HIV/STD interactions are uninterpretable at best, and misleading at worst.

Studies of Genital Ulcer Disease In Human Immunodeficiency Virus Infection

At the current time, few definitive statements can be made about the impact of HIV infection on other STDs. Although much more has been published on this topic than on the role of STDs in progression of HIV disease, the majority of this literature consists of case reports and case series. Table 2 summarizes the strength of existing data linking HIV infection with alterations in STD clinical presentation, natural history, diagnosis, and response to therapy.

Decreased responsiveness to standard therapy is the most convincingly documented effect of HIV infection on chancroid. Two randomized clinical trials of single dose therapy with a trimethoprim-sulfonamide or quinolone antibiotic^{34,85} indicate that treatment failures are at least six times as common among HIV-positive patients than among HIV-negative patients. In one study,³⁴ in fact, treatment failure was a useful predictor of underlying HIV infection (positive predictive value = 71%). A case report (which lacks culture confirmation of *H. ducreyi* infection) and anecdotal information suggest that HIV infection may also result in atypical presentations of chancroid, including larger, multicentric, or extragenital lesions, which may be accompanied by systemic symptoms.^{36,87} Lymphadenitis may be less prominent and lesions may be more persistent in cases of

chancroid accompanied by HIV infection than in HIV-negative cases of chancroid. No data are available with respect to the impact of HIV infection on diagnosis of *H. ducreyi* infection.

A great deal of attention has been focused on the impact of HIV infection on syphilis.³⁸⁻⁹³ Numerous case reports suggest that, in the setting of HIV infection, the clinical presentation of syphilis may be atypical;⁹⁴⁻⁹⁹ progression to neurosyphilis or other tertiary disease may be fulminant;¹⁰⁰⁻¹⁰⁶ serologic tests may be both falsely positive and falsely negative;¹⁰⁷⁻¹¹¹ and standard therapy for early infection may be inadequate.^{100,112-114} Unfortunately, few studies provide the denominator data or comparison groups necessary to assess the importance of these reports either in terms of their absolute frequency among HIV-infected syphilis patients or in terms of their relative frequency in comparison to that among HIV-negative syphilis patients. The pleomorphic presentations and the well-recognized limitations in serodiagnosis of syphilis in otherwise healthy individuals compound this problem. Furthermore, in weighing these data, it is worth highlighting several points that are often forgotten: even in the absence of HIV infection, central nervous system involvement is not uncommon in early syphilis; neurologic symptoms may occur during secondary infection; meningovascular syphilis may follow on the heels of secondary syphilis by as little as a few months; and standard benzathine penicillin therapy of early syphilis does not reliably achieve treponemicidal levels in the central nervous system.¹¹⁵⁻¹¹⁹

Alterations in the natural history of primary syphilis are suggested by one controlled study performed in rabbits.¹²⁰ Compared with rabbits infected with *Treponema pallidum* alone, rabbits latently infected with HIV and then coinfecting with *T. pallidum* exhibited more extensive and more persistent cutaneous lesions.

The issue of accelerated progression to neurosyphilis has been examined in a study that compared 15 HIV-positive and 25 HIV-negative patients with untreated primary or secondary syphilis.¹²¹ Using rabbit inoculation, *T. pallidum* was isolated from cerebrospinal fluid (CSF) in a similar proportion of HIV-positive and HIV-negative subjects (27% and 32%, respectively, $P = 1.00$). The prevalence of reactive CSF serologic tests for syphilis, and of elevated CSF protein were also similar in the two groups. Only CSF leukocytosis was significantly associated with HIV seropositivity ($P = 0.02$).

Although HIV infection was not associated with an increased risk of progression to neurosyphilis, an increased incidence of treatment failure occurred in HIV-infected patients with infectious syphilis.¹²¹ *T. pallidum* was reisolated from three of four patients treated with CDC-recommended single-dose benzathine penicillin in whom *T. pallidum* had been recovered from CSF prior

TABLE 2. Possible Impact of Human Immunodeficiency Virus Infection on the Presentation, Natural History, Diagnosis, and Therapy of Other Sexually Transmitted Diseases

	Clinical Presentation	Natural History	Performance of Standard Laboratory Tests	Response to Standard Therapy
Chancroid	Multicentric, extragenital lesions; systemic symptoms; less lymphadenitis (?)	Larger, more persistent lesions (?)	Altered accuracy of culture or serology for <i>H. ducreyi</i> *	Increased risk of treatment failure with single dose regimens(++)
Syphilis	Atypical presentations (?)	More persistent 1° lesions(+) Accelerated progression to CNS or tertiary disease (?/-)	Delayed or persistently negative serologies (?/-) Abnormally high titers (?) Persistent elevation of titers following therapy(-) Seroreversion in advancing HIV disease(+)	Increased risk of treatment failure with single dose bicillin for 2° syphilis(+)
Herpes	Atypical sites, multicentric disease (?)	Increased frequency of recurrences (?) Increased size and duration of lesions (?) Increased incidence of disseminated disease*	Altered accuracy of culture or serology for HSV*	Increased incidence of acyclovir resistance(+)
Genital warts/Human papillomavirus infection	Multicentric disease, larger lesions (?) Increased frequency of infection with multiple HPV types(+)	Increased incidence recurrences(+) Increased incidence progression to dysplasia or neoplasia(++)	Increased HPV viral load(+) Increased HPV detection with advancing HIV disease(++)	Decreased responsiveness to topical, laser, or surgical therapy(++)
Gonorrhea	Atypical presentation*	Increased incidence gonococcal PID(+) Increased incidence DGI (?)	Altered accuracy of culture for <i>N. gonorrhoeae</i> *	Increased incidence of PPNG(+) Increased incidence of treatment failure*
Pelvic inflammatory disease	Lower prevalence of leukocytosis(+) Altered prevalence of fever(-) Atypical presentations*	Increased severity or duration (?) Increased incidence of sepsis* Altered incidence of infertility or ectopic pregnancy*	Altered accuracy of laparoscopic or histopathologic diagnosis*	Decreased responsiveness to CDC regimens(+)
Chlamydia and trichomoniasis	No published data currently support or refute associations in any of the four categories.			

Adapted from Table 2, Report of the Research Subcommittee, AIDS/STD Task Force, World Health Organization, 1990.
 CNS = central nervous system; HIV = human immunodeficiency virus; HSV = herpes simplex virus; HPV = human papillomavirus; PID = pelvic inflammatory disease; DGI = disseminated gonococcal infection; PPNG = penicillinase-producing *Neisseria gonorrhoeae*.

(++) = Likely (supported by multiple studies in humans that include a comparison group).

(+) = Probable (supported by a single study in animals or humans that includes a comparison group).

(?) = Possible (supported by anecdotal information or case reports).

(-) = Unlikely (refuted by at least one study in humans that includes a comparison group).

* = Unknown (supported by no published data).

to therapy. All three treatment failures occurred in HIV-infected subjects, while the single patient in whom therapy was effective was HIV-negative. In light of the extremely small sample size, these data must, of course, await confirmation.

The few available studies of the performance of serologic tests for syphilis give the impression that accuracy is similar for active disease in HIV-positive and HIV-negative individuals, but that following syphilis therapy, reactive treponemal tests (FTA-ABS or MHA-TP) may become negative with advancing HIV disease.¹²²⁻¹²⁴ In-

vestigators in Zaire, for example, found no significant differences between 40 HIV-positive and 50 HIV-negative female prostitutes with syphilis in terms of the proportion with initial RPR titers of 1:16 or greater or in terms of the change in RPR titer 9 to 12 months after therapy.¹²³ However, in San Francisco, in a study of 109 homosexual men treated 2 months to 26 years previously for syphilis, loss of reactivity of treponemal tests occurred in 13 patients: 36% of those with AIDS, 43% of those with ARC, 7% of asymptomatic HIV-positive subjects, and in none of the HIV-negative subjects.¹²⁴ In

multivariate analysis, seroreversion was independently associated both with the presence of symptomatic HIV infection (OR 7.3, 95% CI 1.4–38.2) and with a T4/T8 ratio of 0.6 or less (OR 7.4, 95% CI 1.2–46.4).

Both case reports and clinical impressions suggest that the severity of herpes simplex infections may be increased in HIV-positive patients. In these patients, herpes may present as multiple necrotic ulcers in atypical locations and appears to progress to more extensive and more persistent lesions than in HIV-negative individuals.^{125–129} Anecdotal information also indicates that the frequency of recurrences may increase as HIV-related immunosuppression progresses.^{129–131} Unfortunately, to date no controlled studies address any of these issues. Data are also lacking on the impact of HIV infection on the incidence of visceral dissemination and on the accuracy of HSV detection by culture or serology.

More convincing data exist to suggest that the incidence of resistant HSV infection is increased among HIV-infected patients. Although with dosage adjustment acyclovir remains effective in the majority of individuals coinfecting with HIV and HSV,^{130–132} a number of reports document evolution of acyclovir-resistant HSV isolates, most of which are susceptible to other antivirals.^{133–141} In addition, one study evaluated sensitivity patterns of 25 HSV isolates from 16 HIV-infected patients; 40 isolates from 38 cancer patients; and 55 isolates from 54 immunocompetent individuals.¹⁴² Acyclovir-resistance was found in five isolates from a single patient with AIDS who had been on escalating doses of acyclovir for almost a year and a half. Prior acyclovir therapy had been administered to 62% of the HIV-infected patients, in comparison to 8% of the cancer patients, and 7% of the immunocompetent group. These data suggest that the increased incidence of acyclovir-resistance in HIV infection is due to the super-position of immunosuppression on a population with high levels of exposure to this medication.

Studies of Nonulcerative Sexually Transmitted Diseases in Human Immunodeficiency Virus Infection

Human immunodeficiency virus infection appears to affect many aspects of HPV infection (Table 2). Several case reports, involving both men and women, emphasize that in HIV-infected individuals HPV infections are often multicentric,^{143–146} and anecdotal information suggests that genital lesions are frequently large in such cases. Furthermore, the incidence of infection with multiple HPV types may be increased in the presence of HIV infection. In one study, 23% of 52 HPV-infected men with CDC Group IV HIV disease had multiple HPV types.¹⁴⁷ A second, comparative study put these results

into perspective by identifying multiple HPV types in 35% of 17 women infected with both HIV and HPV, but in none of the 8 women infected with HPV but not HIV.¹⁴⁸

Both the incidence of recurrences of genital HPV lesions and their responsiveness to standard therapy are probably altered in the setting of HIV infection. Among 116 homosexual men treated with laser surgery for anal warts,¹⁴⁹ for example, the average number of recurrences of genital lesions was significantly greater among HIV-infected than among HIV-negative patients ($P < 0.005$). Other investigators^{150,151} have reported that the median number of treatments required for cure and the median duration of anogenital warts following therapy were greater among HIV-infected homosexual men than among HIV-negative patients ($P < 0.01$ for each), but comparable analyses of heterosexual men did not yield significant differences. These findings are probably due to the more advanced level of immunosuppression among the homosexual than among the heterosexual HIV-infected men, since peripheral CD4 counts were significantly lower and intralesional CD8 levels were significantly higher in the former group. Recurrent disease in the face of podophyllin, cryosurgery, electrocautery, and systemic alpha-2-interferon has also been noted.^{146,150}

Human immunodeficiency virus infection also appears to facilitate development of anogenital dysplasia or neoplasia. A number of case series highlight the high prevalence of premalignant or malignant anogenital lesions among HIV-positive patients.^{143,147,152–156} Studies of both men and women incorporating HIV-negative comparison groups^{148,157–162} have also consistently demonstrated a significant association between HIV infection and cytologic or histopathologic abnormalities in cervical or anal specimens (OR 4.1–7.1 for men; 3.1–10.1 for women). Four of these investigations^{148,158,160,161} indicate that the frequency and severity of anogenital abnormalities is directly related to level of immunosuppression. Furthermore, three of four studies that specifically examined the relationship between squamous intraepithelial lesions and HPV infection found a stronger association in the presence of HIV infection than in its absence.^{148,159,163,164} In the single multivariate analysis that controlled for sociodemographic factors and for other STDs, odds ratios for the association between squamous intraepithelial lesions and HPV infection increased from 2.3 among 60 HIV-negative women, to 8.8 and 29 among 36 asymptomatic and 36 symptomatic HIV-positive women, respectively.¹⁶³

These results must, however, be viewed with caution. Data from more than one study suggest that detection of HPV DNA may be facilitated in the setting of HIV infec-

tion, particularly in the presence of advanced disease.^{41,148} The load of HPV genomes per unit amount of cellular DNA may, for example, be increased in HIV-infected individuals.¹⁴⁸ Until the functional significance of such findings is clarified, the role of HIV infection in the relationship between HPV and anogenital neoplasia will be difficult to assess.

Few data speak to the effect of HIV infection on other STDs. There are currently no data to suggest that HIV infection alters the clinical presentation or diagnosis of lower tract gonococcal or chlamydial infections, although the incidence of gonococcal infection may be increased. A prospective study of Nairobi prostitutes found a significantly greater incidence of gonococcal cervicitis among HIV-positive than among HIV-negative women.¹⁶⁵ Unfortunately, these data were not controlled for factors such as condom use or other incident STDs. The data are particularly difficult to interpret because a subsequent analysis in the same population and similar analyses in Kinshasa prostitutes did not confirm this association.

The risk of complications of gonococcal disease may also be increased in the setting of HIV infection. Investigators in Nairobi⁵³ found that HIV-infected patients with gonorrhea were more than 3.5 times as likely to harbor penicillinase-producing gonococci (PPNG) than HIV-negative patients ($P < 0.002$). In addition, a prospective study conducted among Nairobi prostitutes documented a four-fold increase in the incidence of gonococcal pelvic inflammatory disease (PID) among 151 HIV-positive subjects in comparison to that among the 81 patients not infected with HIV, but no increase in the incidence of chlamydial PID (Brunham R, Plummer F. Personal communication). Disseminated gonococcal infection may also be particularly problematic among HIV-infected individuals. In contrast to the highly penicillin-sensitive arginine, hypoxanthine, and uracil-requiring (AHU) strains that are usually associated with disseminated gonococcal infection, investigators in London¹⁶⁶ reported a case of disseminated gonococcal infection in an AIDS patient caused by a moderately resistant isolate (auxotype *P. serovar* 1B7).

Finally, anecdotal information suggests that HIV infection may affect the clinical course and response to standard therapy of PID. Currently, only one published study has examined this issue.¹⁶⁷ Among 110 women hospitalized in New York City for acute PID, surgical intervention due to persistent fevers despite antibiotic therapy was almost four times as common among the 15 HIV-infected patients than among the 95 HIV-negative patients ($P = 0.058$), and admission leukocytosis was substantially less common in the former group ($P = 0.001$). These differences appeared to be unrelated to

microbial etiology. Unfortunately, neither the duration of symptoms of PID prior to admission nor the findings during surgery were discussed in this report.

Potential Mechanisms

A detailed discussion of the pathophysiologic mechanisms mediating the interrelationships between HIV infection and other STDs is beyond the scope of both this review and our current knowledge. A short summary of potential mechanisms due either to the host response or to pathogen interactions is, however, useful in emphasizing the biologic plausibility of the relationships described above.

One aspect of the host response to STDs that may contribute to HIV transmission is the recruitment of HIV-susceptible or HIV-infected cells, such as T lymphocytes or macrophages. A number of investigators have identified HIV in genital secretions¹⁶⁸⁻¹⁷⁷ or tissue^{178,179} and several have documented localization of the virus to mononuclear cells.^{173,177-179} Immunohistologic studies indicate that while lymphocytes and macrophages are normally present in the tissues and secretions of the male and female genital tract¹⁸⁰⁻¹⁸² and in the rectal mucosa of homosexual men,¹⁸³ these HIV target cells increase in the setting of genital tract inflammation.^{184,185} such as that associated with STDs.¹⁸⁶⁻¹⁸⁸ Thus STDs could potentially augment the susceptibility of HIV-negative partners by increasing the number of cells available for invasion by the virus, and could augment the infectiousness of HIV-positive partners by increasing the number of virus-laden cells being inoculated.

Another mechanism by which some STDs might facilitate HIV transmission is disruption of epithelial barriers in the genital tract. The integrity of the genital mucosa appears to be an important host defense against infection,¹⁸⁹ and is clearly breached in cases of GUD. As with STD-related genital tract inflammation, GUD-induced breaks in the lining of the genital tract could affect both susceptibility and infectiousness. In fact, HIV has been isolated directly from genital ulcers in both men and women.^{190,191} The consistency with which STDs appear to promote HIV transmission suggests that disruption of epithelial barriers is not the primary mechanism involved. The efficiency with which GUD increases HIV transmission, however, may be due in part to the simultaneous operation of both of these mechanisms.

Both stimulation and suppression of the systemic immune response have been invoked as possible mechanisms for various aspects of the interrelationships between HIV and other STDs. It has been postulated that repeated STD-related activation of T lymphocytes plays a role in susceptibility to HIV infection¹⁹² and in progression of HIV disease, and HIV-related polyclonal B cell

activation has been raised as possible explanation for false positive results to serologic tests for STDs. On the other hand, the role of STD-induced immunosuppression has been considered in susceptibility to HIV infection or in progression of HIV disease,¹⁹³ and HIV-induced immunosuppression almost certainly is a factor in alterations in the natural history and response to therapy of some STDs. With the exception of the last of these points, however, few data support these hypotheses.

Finally, interactions between HIV and STD pathogens, may be responsible for increased transmission or accelerated progression of these diseases. Viral STDs might potentiate HIV infection by stimulating expression of cell surface receptors that facilitate HIV attachment, by coding for gene products that are necessary for HIV growth, or by temporarily or permanently altering HIV expression through phenotypic mixing or genetic recombination.^{193,194} *In vitro* data suggest that the herpesviruses (HSV, EBV, and CMV) can, for example, transactivate HIV,¹⁹⁴⁻¹⁹⁸ although the clinical significance of these observations remains to be determined. It is also possible that HIV gene products, such as the TAT protein, could play a role in HPV-associated neoplasia, much as it appears to promote development of Kaposi's sarcoma.

Conclusions

As we enter the second decade of the HIV pandemic, many questions about the interrelationships between HIV infection and other STDs remain unanswered. Data on interactions between HIV and STDs are still limited in quantity and quality, and preliminary conclusions drawn today are likely to demand revision in the near future. Yet patterns are emerging on which to base a conceptual framework for understanding these interrelationships (Figure 1). Such a framework may be useful in generating testable hypotheses for additional research.

These patterns are summarized below.

Several prospective studies that control for behavioral risk factors and for other STDs support the hypothesis that the risk of HIV transmission is increased in the presence of each of the genital ulcer diseases and in the presence of nonulcerative STDs, such as chlamydial infection, gonorrhea, and trichomoniasis. HPV infection stands out as the one STD for which no well-designed, prospective studies of HIV transmission are yet available. Further studies may well demonstrate that HPV infection and anogenital warts do not facilitate HIV transmission, but instead are themselves promoted by HIV-related immunosuppression.

In general, the role of STDs in HIV transmission as it relates to gender and specific STD syndromes is beginning to emerge. The role of STDs may be somewhat greater among heterosexual men than women, but data are still too sparse to be conclusive. It is also noteworthy that risk estimates from studies of heterosexual populations are largely similar for ulcerative and nonulcerative STDs, and cluster in the range of a three to five-fold increase in the risk of developing these STDs. Limited data currently preclude comparisons between the role of STDs among heterosexual and homosexual populations. Finally, although specific mechanisms must be elucidated, both biologically and epidemiologically it is highly plausible that other STDs would facilitate transmission of HIV.

The role of STDs in the progression of HIV disease, on the other hand, is still unknown. The few available data come from univariate and bivariate analyses of a limited number of STDs in homosexual or hemophilic populations. Half of the studies focus on seroprevalent cases of HIV infection in which the duration of infection can not be ascertained. It is, therefore, difficult to interpret the results of these studies, which suggest that HSV infection plays no role in progression, while syphilis and rectal

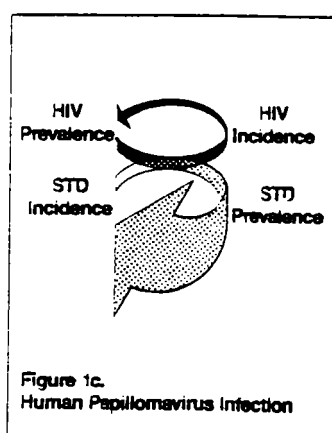
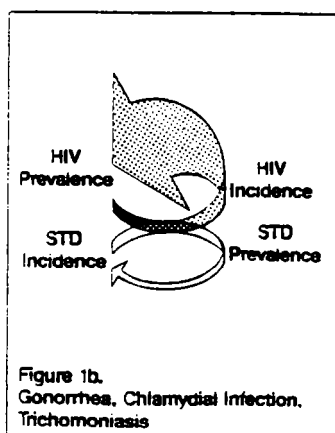
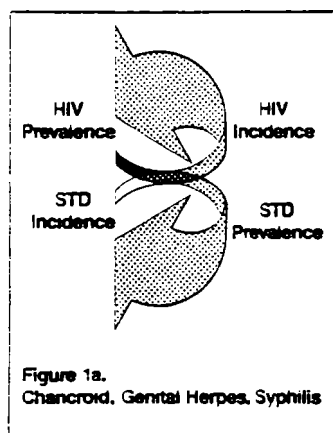


Fig. 1. The relationships between human immunodeficiency virus (HIV) infection and other STDs may differ for different STDs. Genital ulcer diseases (GUD) and HIV infection appear to reinforce each other, because these STDs facilitate HIV transmission, while HIV infection may simultaneously prolong or augment the infectiousness of individuals with GUD (1a). The discharge syndromes probably interact with HIV infection in a unidirectional fashion by promoting HIV

transmission without a synergistic increase in their own prevalence or incidence (1b). Human papillomavirus infection may represent a traditional opportunistic infection, the expression and progression of which are augmented by HIV (1c).

gonorrhea may possibly accelerate the pace of HIV disease. One can only emphasize the need for additional research on this issue.

Data on the impact of HIV infection on other STDs are also compromised by several methodologic problems, the most critical of which is the dearth of studies that include comparison groups. Nevertheless, the information that is available suggests that HIV infection may have clinically significant effects on a number of STDs. In order to understand HIV-STD interactions, it may, in fact, be useful to think about STDs in four categories: those in which HIV alters the course of lower tract, communicable stages of disease; those in which HIV affects the development of noncommunicable sequelae of lower tract disease; those in which HIV impacts upon both aspects of disease; and those in which HIV seems to have little effect.

Genital ulcer diseases appear to fall into the first category. Available data suggest that HIV prolongs the duration of GUD through effects such as more persistent lesions, more frequent lesion recurrences, more common treatment failures, and even through atypical presentation of the disease resulting in delayed diagnosis. At present, it is not clear whether the infectiousness as well as the prevalence of GUD is increased in the setting of HIV infection. No comparative studies currently indicate that HIV promotes the development of sequelae of GUD.

Nonulcerative STDs can be divided among the remaining categories. Gonococcal infections may represent the second category. Little evidence suggests clinically significant alterations in lower tract gonococcal infection, but the development of complications, such as PID or disseminated gonococcal infection, may be affected by concomitant HIV infection. In contrast, HPV infections and genital warts should be considered to represent the third category. HIV probably increases the prevalence (and perhaps the infectiousness) of anogenital lesions, as well as the risk of HPV-associated neoplasia. HIV seems to have little effect on chlamydial infections and on trichomoniasis, although to date, these STDs have received minimal attention.

What do these patterns tell us about the HIV pandemic and approaches to its control? The pieces of this puzzle may be most informative when the role STDs play in HIV transmission and the impact of HIV infection on other STDs are considered together. In the past, these have usually been examined as separate questions. Attention has focused primarily on the former, in the hope that identification of modifiable risk factors for transmission would lead to development of effective interventions for primary prevention.¹⁹⁹⁻²⁰¹ While the latter issue has concerned many involved in STD research and control efforts, it has generated little interest among HIV investigators or care providers.

Because of our compartmentalized approach, the potential significance of a bidirectional interplay between HIV infection and other STDs has not been fully appreciated. If coinfection with HIV prolongs or augments the infectiousness of individuals with specific STDs, and if the same STDs facilitate transmission of HIV, at a community level, the two infections could greatly amplify one another (Figure 1A). Mutually reinforcing increases in HIV and STD incidence may well underpin the explosive growth of the HIV pandemic in some heterosexual populations^{1-3,202} and may represent a uniquely effective epidemiological synergy between disease syndromes that are linked by common behaviors.²⁰² The limited data that are available suggest that such synergy may well occur between HIV infection and GUDs, such as chancroid, herpes, and syphilis. Other STDs, such as gonorrhea, chlamydia, and trichomoniasis appear to interact with HIV infection in a unidirectional fashion: they may promote HIV transmission, but HIV infection does not seem to result in a synergistic increase in the prevalence (and hence the incidence) of this group of STDs (Figure 1B). HPV infection may represent a different, unidirectional model that is closer to that of traditional opportunistic infections. It probably does not facilitate HIV transmission, but HIV may well augment expression and progression of HPV disease (Figure 1C).

Program and Policy Implications

The program and policy implications of the interrelationships between HIV and other STDs are clearcut and urgent. They need not and should not await additional research. As has been highlighted by other researchers,^{199,200,204-206} HIV and STD control efforts must go hand in hand in terms of clinical services, research, and training. Programmatically, this means that STD services must be strengthened or established *de novo*, and that they must be integrated as cornerstones of HIV prevention programs. On a policy level, countries that hope to minimize the impact of the HIV epidemic must recognize that STDs are one of the most readily modifiable risk factors for spread of HIV within a community. They must, therefore, make STD control a major health priority.

Both individual programs and national policies should focus on the full spectrum of locally prevalent STDs, rather than exclusively on genital ulcers. The epidemiological synergy described above implies that high levels of GUD are likely to result in more rapid increase in HIV incidence than nonulcerative STDs. However, most nonulcerative STDs also appear to increase HIV incidence, and, because of their higher prevalence in many populations, these STDs may be responsible for a larger proportion (attributable risk) of HIV transmission than genital ulcers.

Because of their complementary role both in HIV and in STD prevention, behavioral interventions, such as condom promotion, should be emphasized. Furthermore, in patients coinfectd with HIV and other STDs, condoms may work where antibiotics fail. Counselling of STD patients should include information on their high risk of HIV infection.

Finally, research and training are essential components of any effective, longterm public health program. Coordination of HIV and STD activities should extend to the research and training arenas.

Research Needs and Opportunities

The complex and unique interrelationships between HIV infection and other STDs provide some of the most exciting challenges in science today. Research questions cross-cut a number of disciplines and demand a marriage of basic science, epidemiological, clinical, and behavioral research approaches.

Future studies of the role of STDs in HIV transmission should be prospective in design, and must control, where possible, for behavioral risk factors and for the presence of other STDs. Sexually transmitted diseases should be documented microbiologically, rather than historically. Investigators should focus on STDs and populations that, to date, have received little attention in prospective studies. As is apparent from Table 2, these include syphilis and herpes in heterosexuals; chlamydia, gonorrhea, and trichomoniasis in males; and HPV infections in all populations. The importance of HPV and genital warts in HIV transmission remains particularly ill-defined. As has been highlighted by others, definition of the attributable risk of individual STDs in specific populations will be important to the rational development of program priorities.

Additional studies of the role of STDs in HIV progression are urgently needed. To minimize recall bias and to permit adjustment for duration of infection, these studies should follow HIV seroconverters prospectively, controlling for factors such as age and baseline immunologic status. They should be conducted in nonhemophilic heterosexual populations as well as homosexual cohorts. Investigators should consider number of episodes of STDs, as well as categorical STD exposure variables.

Future studies of the impact of HIV infection on other STDs must include comparison groups. Among the highest research priorities are improved definition of HIV-induced alterations in the natural history of STDs, both as they affect the prevalence of STDs in the community and as they affect the incidence of severe sequelae, such as tertiary syphilis, anogenital malignancy, or pelvic inflammatory disease, infertility, and ectopic pregnancy. Development and testing of STD regimens that

are effective in HIV-infected individuals is equally important.

Complementary research efforts should focus on the mechanisms that mediate each of the interrelationships between HIV and STDs. In clinical studies, comparison of partners of HIV-infected individuals with and without STDs will be needed to determine whether STDs contribute to HIV transmission by increasing infectiousness as well as by increasing susceptibility to HIV. Elucidation of the molecular pathogenesis of interrelationships between HIV and STDs will contribute substantially to our understanding of each of these diseases individually, and more broadly, will offer insights into ways in which pathogens interact to promote disease.

Ultimately, the goal of medical research is to provide the knowledge with which to control disease. Research on the interrelationships between HIV infection and other STDs must converge in the development and testing of sustainable interventions that will help contain the spread of the most devastating pandemic in the era of modern medicine. Intervention trials that conclusively demonstrate the importance of STD control in HIV prevention will be complex, expensive, and time-consuming. They may, however, be critical to informing health policy makers of the interrelationships discussed in this review, and to convincing them of the necessity of allocating greater resources to STD control as part of integrated HIV and STD prevention programs.

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Sexually Transmitted Diseases in the AIDS Era

Development of STD Diagnostics for Resource-Limited Settings is a Global Priority

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THE HUMAN immunodeficiency virus (HIV) pandemic has focused attention on sexually transmitted diseases (STDs), both because HIV infection is a fatal STD, and because other STDs such as chancroid, genital herpes, syphilis, trichomoniasis, gonorrhea, and chlamydial infection have been repeatedly implicated as risk factors for sexual transmission of HIV. Increased attention has also increased awareness that many STDs disproportionately affect the female, the fetus, and the newborn. Gonococcal and chlamydial infections cause pelvic inflammatory disease, infertility, and ectopic pregnancy. Several common STDs adversely affect pregnancy and result in spontaneous abortion, stillbirth, chorioamnionitis, premature rupture of membranes, pre-term delivery, and post-partum endometritis. Neonatal infections include gonococcal and chlamydial conjunctivitis, which may lead to blindness, and chlamydial pneumonia, which may lead to chronic respiratory disease. Human papillomavirus (HPV) genital infections are closely correlated with cervical cancer, which is one of the most common cancers in women throughout the developing world.¹

Despite recent global efforts in health education aimed at preventing sexual transmission of HIV, STDs remain hyperendemic in many developing countries and in the inner-city populations of industrialized countries.^{2,3} Throughout the world, the majority of STDs are clustered in the resource-limited settings of urban and peri-urban areas where increasing numbers of adolescents and young adults, poverty, unemployment, lack of education, inferior status of women, and social disintegration fuel the epidemic spread of STDs.

A consensus has emerged that the prevention of sexually transmitted HIV infection, as well as the prevention of the major sequelae of STDs in women and infants, mandate a global initiative for the prevention and

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control of STDs. This initiative will depend on the development of inexpensive, simple, rapid diagnostic tests that are appropriate for resource-limited settings. Such tests are particularly important for diagnosis of STDs in women. In resource-limited settings, clinical algorithms based on recognition of symptoms and signs of STDs have been useful for diagnosis and treatment of urethritis and genital ulcer disease in men, but are far less useful for diagnosis of STDs in women. Similarly, microscopic examination of Gram-stained smears of exudate is approximately 90% sensitive for diagnosis of gonorrhea in symptomatic men, but is less sensitive (50–70%) in women.⁴

In a recent workshop* co-sponsored by the National Institute of Allergy and Infectious Diseases and the

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United States Agency for International Development, STD researchers and representatives from funding agencies, health-care delivery agencies and industry convened to examine the potential for and constraints on developing STD diagnostics for resource-limited settings.

The participants considered the special requirements of resource-limited settings and concluded that useful diagnostics tests should be:

- inexpensive: provider cost less than \$1.00 (U. S.) per patient
- simple: no equipment and minimal training required
- rapid: results available before the patient leaves the clinic
- performed on convenient specimens: simple to collect, socioculturally acceptable, no separation or preparation needed
- able to use stable reagents: long shelf-life, no refrigeration required
- packaged simply: functional, low cost
- appropriately sensitive and specific: dependent, in part, on the potential morbidity and cost for undetected infection, and the cost of the resulting treatment

Examples of diagnostic tests with the appropriate format are the occult-fecal blood card and the urine-glucose dipstick. Except for the leukocyte-esterase dipstick (used in some settings to detect urethritis in men), currently no such STD diagnostic tests exist.

The workshop reviewed the available biotechnology, as well as the pressing needs of a global STD prevention program. The group consensus was that the principle obstacle for the development of these tests was no longer biotechnologic. Recent advances, and possible modifications of older diagnostic approaches, provide a number of alternatives for resource-limited settings:

- Inexpensive biochemical tests for enzymes and metabolites are the most promising.
- Immunodiagnostics, including antigen detection tests and serologic assays, using newer, simpler formats, may be inexpensive and practical enough to be useful.
- Culture of some microorganisms (e.g., *Neisseria gonorrhoeae*) remains appropriate and useful in central laboratory facilities.

Nucleic acid probes, despite their extraordinary potential as sensitive and specific diagnostic tools, are not yet applicable for resource-limited settings.

Given the availability of appropriate biotechnology for development of simple, rapid, easy-to-use, inexpensive diagnostic tests, a list of priorities for test development (table 1) was established based on an assessment of the global STD problem.

(1) Because complications of chlamydial and gonococcal cervicitis are devastating for women and neonates, and because these diseases and associated sequelae account for a high proportion of STD-related health-care costs, diagnosis of cervical and urethral infection was given top priority. Specifically a chlamydial diagnostic test for use in women and men, and a gonococcal test in women, are urgently needed. Considerable interest was expressed in the development of multispecific diagnostics to test simultaneously both for gonococcal and chlamydial infection.

(2) Tests to identify the etiologic agents of genital ulcer disease (GUD) were designated a second major priority, especially in light of associated increased risk of HIV Transmission. Development of specific tests for early syphilis, using finger-stick blood specimens, and for detection of *Haemophilus ducreyi* in genital ulcers was recommended. A multispecific format would also be beneficial in this setting.

(3) Prevention and early detection of cervical cancer was the third priority. To prevent cervical cancer, the participants of the workshop highlighted the need for training programs for cytopathologists, and for developing appropriate, cost-effective schedules for obtaining

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TABLE 1. Priorities for the Development of STD Diagnostics for Resource-Limited Settings

1. Tests for chlamydial infection of the cervix and the male urethra and for gonococcal infection of the cervix
2. Tests for primary and secondary syphilis using finger-stick whole blood and tests for detection of *haemophilus ducreyi* in genital ulcers
3. Practical systems for implementation of cervical pap-smear screening.
4. Serologic tests for herpes simplexvirus 2 and *h. ducreyi* (for purposes of assisting surveillance and evaluation of STD/AIDS control programs)

and reading Papanicolaou (PAP) smears at least once during the lifetime of adult women, as currently recommended by the World Health Organization. Because neither the natural history of HPV infections nor the pathogenesis of cervical neoplasia has been completely defined, and because considerable biotechnologic effort is already underway to develop improved diagnostics for HPV infection, the allocation of substantial resources for new diagnostic tests for HPV in resource-limited settings was believed to be premature.

(4) The fourth priority was the development of serologic tests that would provide an index of aggregate risk of STDs in populations. These tests would be important research tools to assess the impact of STD prevention programs, particularly in adolescents and young adults. The development of Herpes simplexvirus 2 and *Haemophilus ducreyi* serologic assays should be considered.

Because the absence of effective distribution networks is a major barrier to STD control efforts in many resource-limited settings, the workshop participants considered the distribution and use of appropriate diagnostic tests. In industrialized countries, national health agencies, such as the Centers for Disease Control in the United States, and the existing health-care infrastructure could facilitate distribution and use of tests in STD, antenatal, and family-planning clinics, as well as public-health laboratories.

Within developing countries, distribution problems are compounded because laboratory facilities are limited and often are not well integrated into the clinical system or into a national laboratory network. Here, it will be necessary to define where the patient seeks care. In many African, Latin American and Asian countries, large numbers of men seek care for STDs from private physicians, pharmacists, or from unauthorized drug distributors. Therefore, it will be important to explore innovative distribution approaches including social marketing of diagnostic tests within the existing infrastructure, while strengthening the health-care delivery and laboratory infrastructure for STDs. The World Health Organization and other international agencies could facilitate distribu-

tion and use of STD diagnostics in appropriate settings in the developing world.

Finally, to expedite development of STD diagnostic tests for resource-limited settings and to provide a sustained effort towards this goal, workshop participants agreed that an interagency working group should be established. Within this group, representatives of industry, funding agencies, and domestic and international governmental agencies, as well as health-care providers, will work together to stimulate and coordinate research and development of specific STD diagnostic tests for the evolving needs of resource-limited settings. This working group will also stimulate development of marketing and distribution strategies, including strategies to provide existing tests at lower cost to resource-poor settings.

Globally, STDs, including HIV infection, present an extraordinary challenge for the current generation of health-care professionals, researchers and policy makers. A successful response to this challenge will require commitment of energy and resources to a global STD prevention program that is closely coordinated with national AIDS control programs, as well as with maternal-child health, family-planning, prenatal-care, adolescent-health and health-education programs. The development of STD diagnostic tests appropriate for use in resource-limited settings will be only one aspect of an effective global STD prevention program. Without these essential tools, however, we cannot begin to control STDs or their devastating sequelae.

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Sexually Transmitted Diseases in the AIDS Era

Gonorrhea, syphilis and other infections still exact a terrible toll. Social conditions help to fuel the new epidemics—and only a combination of social and health programs can defeat them

by Sevgi O. Aral and King K. Holmes

In almost all of the industrialized countries the three classic venereal diseases—gonorrhea, syphilis and chancroid—have nearly disappeared. Throughout Europe, Australia, New Zealand and Japan the incidence of gonorrhea has been declining steadily for 15 to 20 years. In Sweden alone the gonorrhea rate dropped by more than 95 percent between 1970 and 1989. These improvements probably reflect the effectiveness of public health measures taken in those countries.

In shocking contrast, those three sexually transmitted diseases (STDs) have actually been increasing at epidemic rates among urban minority populations in the U.S. Urban poverty and so-

cial disintegration, along with prostitution and the relatively new phenomenon of sex in exchange for drugs, seem to underlie this epidemic.

Further compounding the STD problem in the U.S. are the rise of drug-resistant strains of sexually transmitted bacterial infections and the rapid spread of incurable viral STDs. The deteriorating STD situation of the U.S. urban underclass increasingly resembles that seen in the slums of the least developed countries, where acquired immunodeficiency syndrome (AIDS), caused by the human immunodeficiency virus (HIV), has been spreading at epidemic rates among heterosexuals.

We have reviewed the available data on the incidence of STDs as well as on the social, economic and political forces that seem to be fueling the epidemic. Our conclusion is that certain policies must be implemented if the U.S. is to control STDs in general and to prevent a heterosexual HIV epidemic in the urban underclass.

Data on the incidence of gonorrhea and syphilis, which are perhaps the best known of the STDs, demonstrate the trends we have observed. Traditionally the reported incidence of gonorrhea in the U.S. has been higher among men than among women and higher among blacks than among whites. Since 1975 intensified public health efforts, along with the aging of the "baby boom" generation, have

brought about a decline in the overall incidence of gonorrhea in the U.S.

Yet this reduction has not been consistent throughout the entire population. The racial difference in the gonorrhea rates has sharply widened since 1984, when the incidence among blacks began rising rapidly. (Some of the difference between the races may reflect underreporting of the disease in whites: whites more frequently rely on personal physicians and private clinics, which tend to report gonorrhea cases less thoroughly than do public clinics.)

Much has been learned about the spread of gonorrhea by studying antibiotic-resistant strains of gonococcal bacteria. Until 1976, all cases of gonorrhea could be cured by penicillin. In that year, however, a man traveling to the U.S. from the Philippines was found to have a resistant strain. Such strains began to spread globally, and by 1987 they accounted for 2 percent of all the cases reported in the U.S. That proportion reached 5 percent by 1989.

SEXUALLY TRANSMITTED viruses and bacteria are responsible for epidemics in the developing world and in certain U.S. populations. These micrographs show an isolated hepatitis B virus (a), herpes simplex virus type 2 (yellow and green) inside an infected cell (b), intracellular bacteria called *Chlamydia trachomatis* (orange) that cause chlamydial infection (c) and the human immunodeficiency virus (HIV) (d) that causes AIDS.

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Because penicillin-resistant strains are easily identified, they can be traced as they move through a community. In Seattle during 1986 and 1987, for example, an outbreak of approximately 200 cases of gonorrhea was caused by a unique resistant strain. The first cases predominantly involved white and Hispanic men who often had a history of prostitute contact. After several months, however, nearly all the cases involved heterosexual black men and women, more than 80 percent of whom had histories of recent prostitution or sex with prostitutes or recent use of intravenous drugs or "crack" cocaine.

This patient profile was distinctly different from that of the 1960s and 1970s, when cases of gonorrhea in

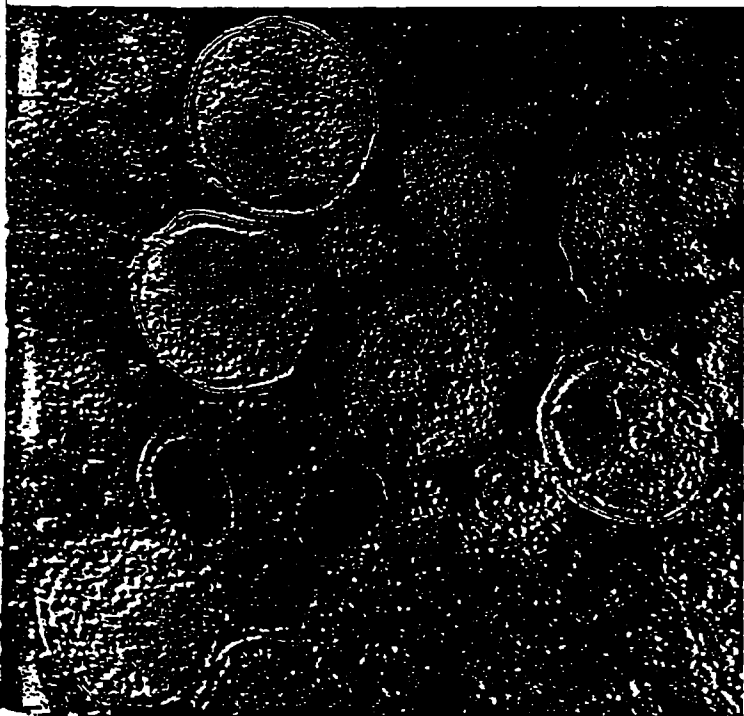
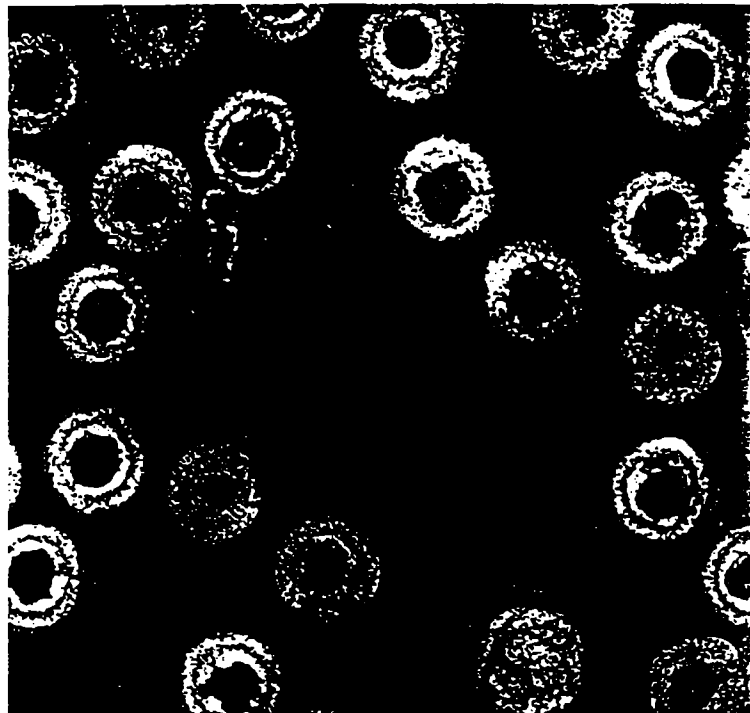
Seattle usually involved whites and were rarely linked to prostitution or illegal drug use. It heralded a national pattern that would also characterize the emerging epidemiology of other STDs such as syphilis.

In the U.S. the incidence of infectious syphilis cases peaked at 76 cases per 100,000 citizens after World War II and then fell to only four per 100,000 citizens for 1955-1958. In 1959 the trend reversed, and the incidence rose rapidly to 12 per 100,000 in 1965—triple the level of just a decade earlier. The increase has often been attributed to a decline in federal funding for syphilis control.

Between 1965 and 1982 the total in-

cidence of infectious syphilis changed very little in women but more than doubled in men because of an epidemic of syphilis among homosexual men. From 1982 through 1986 the reported national rate of infectious syphilis dropped steadily, largely because of a steep decline in the incidence among homosexual men, whereas the rates in women changed very little. Between the last quarter of 1986 and the end of 1989, however, the incidence rose sharply in men and women.

This overall increase is actually attributable to a very steep rise in infections among black men and women since 1985. The pattern of resurgent syphilis has been further documented in local syphilis outbreaks in New York



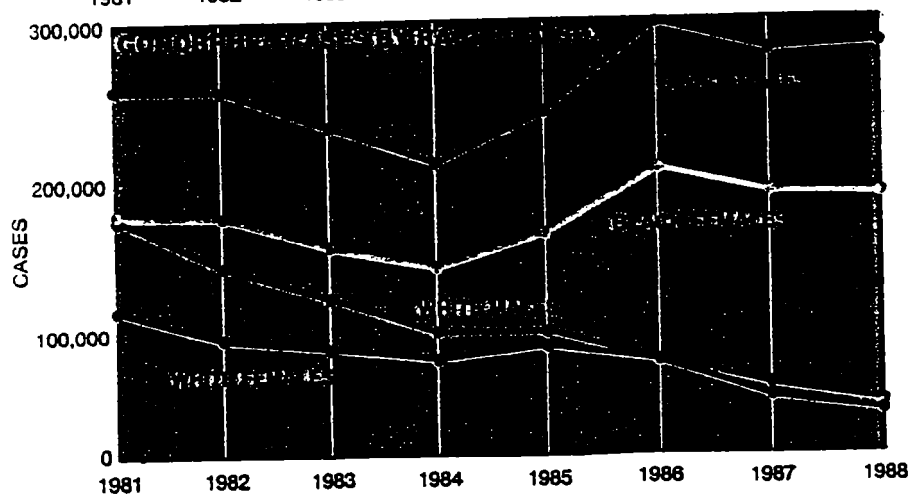
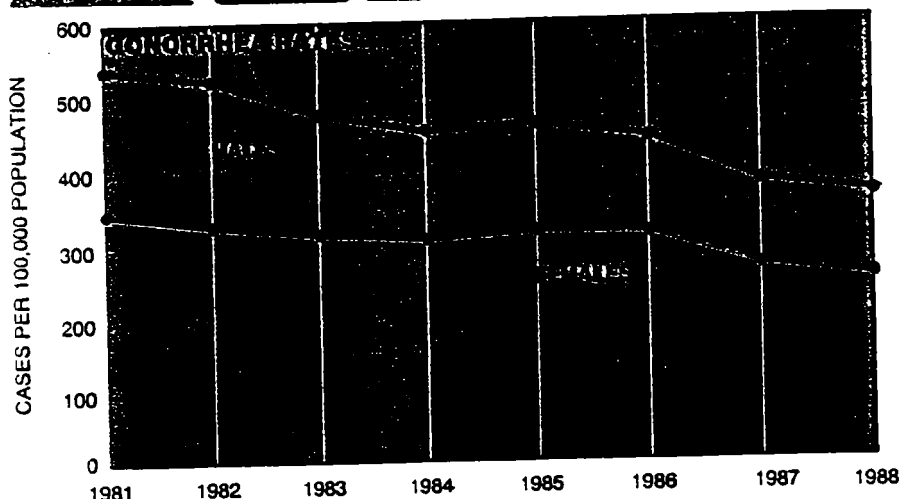
City, Philadelphia, Baltimore and Los Angeles and some areas in Texas and South Florida. In Philadelphia and New York, the use of crack cocaine and the exchange of sex for it have been closely linked to a higher incidence of syphilis.

The distribution of syphilis in the U.S. population is also reflected by blood test data collected in a national health and nutrition examination survey of the U.S. population (known as NHANES II) conducted between 1976 and 1980. Youth, single marital status, lack of education, low income and urban residence were all found to be associated with positive blood tests for syphilis, but the greatest difference

was seen between races: controlling for other factors, the rate of positive blood tests was almost fivefold higher among blacks than among whites.

Another STD of growing importance in the U.S. is chancroid. In Africa this infection is the most common cause of genital sores, and it has always been strongly linked to prostitution and to sex with prostitutes. In 1985 a multinational research group found genital ulcers, most of which were chancroid, in 42 percent of the prostitutes from a slum area of Nairobi. Subsequent studies showed that genital ulcers in prostitutes and in male STD clinic patients were associated with a greatly increased risk of sexually acquired HIV infection.

In the U.S. chancroid had been rare since World War II. Beginning in 1984, however, a series of outbreaks began to appear in the inner-city and migrant-labor populations of Los Angeles, Dallas, Boston, New York City, New Orleans and several Florida cities. The total number of chancroid cases reported in the U.S. rose from 665 in 1984 to 4,714 in 1989.



This increase could have profound public health consequences because chancroid may facilitate HIV transmission. Worse still, the bacterium that causes chancroid has developed resistance to many antimicrobial drugs. In persons who have been exposed to HIV, chancroid often fails to respond to some therapies that are otherwise highly effective. Thus, HIV infection may help the spread of a bacterial STD that in turn helps to spread HIV.

A fourth bacterial infection—one caused by intracellular bacteria called chlamydia—has now become far more common than gonorrhea, syphilis or chancroid in the U.S. It is also a much more important cause of reproductive health problems in women. Like the gonococcus, chlamydia usually causes infection of the urethra in men and of the cervix in women. If the infection ascends into the uterus and fallopian tubes, it can cause abnormal bleeding, vaginal discharge or lower abdominal pain. Even in the absence of those symptoms, scarring of the fallopian tubes can cause blockages and sterility.

Because silent but destructive disease is the hallmark of chronic chlamydial infection, we feel screening for it should be part of routine pelvic examinations in women. Unlike gonorrhea, chlamydial infection commonly occurs in all racial and ethnic groups and in all socioeconomic classes. Young women and women using oral contraceptives seem more susceptible than others to acquiring chlamydial infection of the cervix if they are exposed.

Because laboratory testing for chlamydia is not yet widely used and chlamydial infection is not reported in most states, its incidence cannot be well documented. Still, it is clear that asymptomatic chlamydial infection is much more common in men and women than asymptomatic gonorrhea. It is common to find silent chlamydial infection of the cervix in up to 5 percent of middle-class pregnant women and female college students, whereas gonococcal infection is typically found in less than 1 percent. In two military studies, silent chlamydial infections of the urethra were found in about 10 percent of healthy, young men.

Chlamydial infection seems to be prevalent among the U.S. middle class because programs recommended for controlling the spread of chlamydia have not been widely implemented. Such programs have been very effective, at least in the middle class, at controlling gonorrhea, syphilis and chan-

GONORRHEA is caused by the bacterium *Neisseria gonorrhoeae* (top). In the U.S. the

the U.S., where public health measures for controlling chlamydia have been in place for several years, the number of infections has declined.

Comprehensive programs to control chlamydia have been formulated by experts at the national level but have rarely been implemented by local health departments and practicing physicians. In the U.S. the estimated direct medical costs and indirect costs associated with chlamydial infection came to \$1.5 billion a year in the mid-1980s. Research has shown that chlamydial infection is the primary preventable cause of sterility in women and that the transfer of a mother's infection to her infant at birth can lead to a common form of infant pneumonia. A recently completed major multicenter study in the U.S. suggests that chlamydial infection in pregnant women is associated with premature birth.

Furthermore, chlamydial infection of the cervix causes inflammation resulting in microscopic cervical ulcerations. (This consequence is also probably true for gonococcal infection of the cervix.) Other STDs that cause obvious ulcerations of the genital skin seem to increase the risk of acquiring or transmitting HIV infection sexually. It is not surprising, then, that two studies in Africa have suggested that women with cervical chlamydial infection are at greater risk of acquiring HIV infections sexually.

In view of the overwhelming evidence that chlamydial infection is the most common curable STD and a major cause of infertility in women and that it increases the susceptibility of women to HIV, it is difficult to understand why many programs for STD control, family planning and maternal-child health do not provide diagnostic testing for this disease.

Unfortunately, not all STDs are caused by bacteria, and therefore not all are curable with antimicrobial agents. Viruses cause STDs as well, and these represent an especially difficult public health challenge because they cannot yet be cured. Lesions caused by the human papillomavirus can be eliminated or suppressed with toxic chemicals, freezing, electrocautery or laser therapy, but these procedures cannot rid a patient of the virus.

During the late 1970s and 1980s, new tests were developed that could detect the proteins and DNA of viruses that cause STDs. Epidemiological studies based on these tests are now disclosing the vast scope of the viral STD

problem as well as the distribution and determinants of these infections.

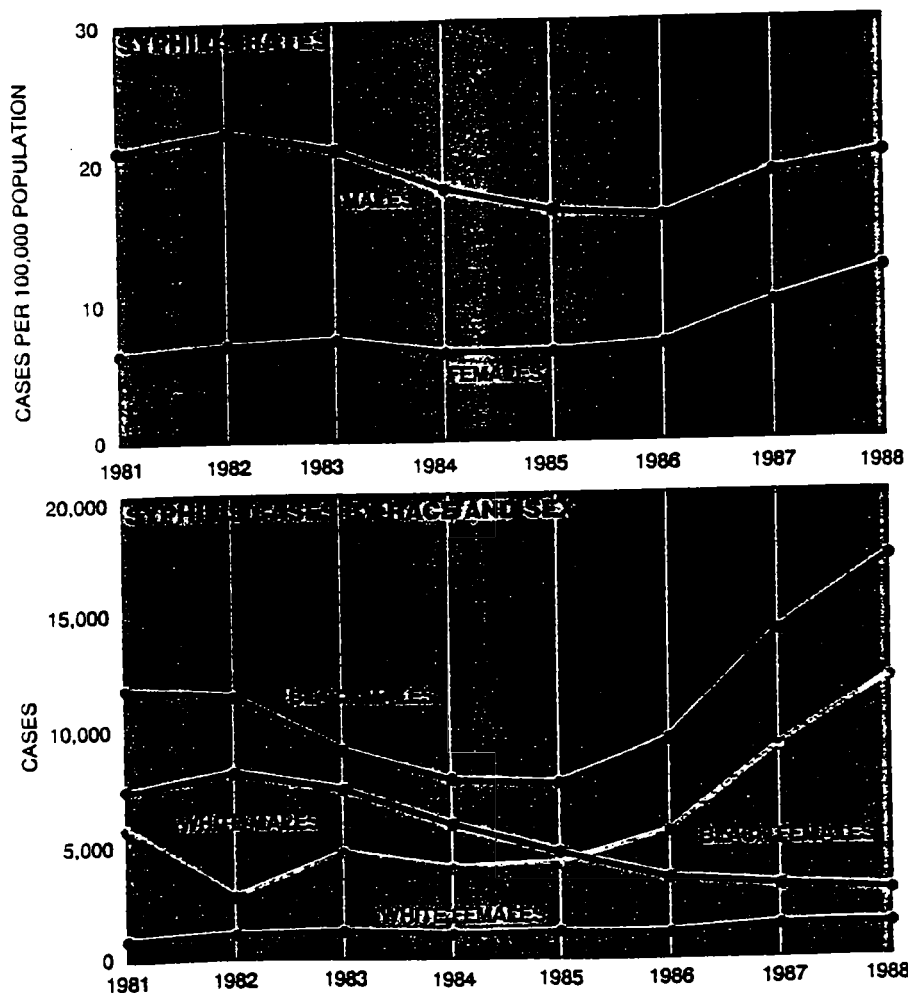
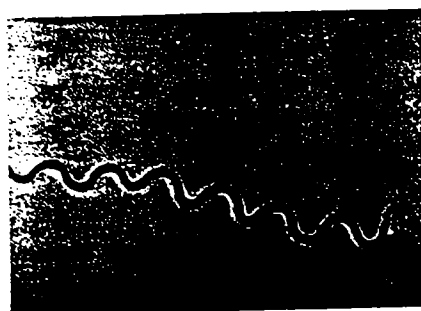
Genital herpes and genital warts, both of which are caused by viruses, seemed to be the most rapidly spreading STDs from the mid-1960s until the onset of the AIDS epidemic. The full extent of the problem was not clear, however, until random samples of blood serum collected during the late 1970s were tested for the presence of antibodies against herpes simplex virus type 2 (HSV-2). The presence of such antibodies almost always reflects previous genital infection with the virus.

The results suggested that approximately 25 million U.S. civilians had been infected with HSV-2 as of 1978—about 10 times more than might have been expected from the reported number of physician consultations for genital herpes. The data also showed that HSV-2 antibody is approximately two to three times more common among blacks than whites and that, at least among blacks, women are more likely to have HSV-2 antibodies than men. Antibodies to HSV-2 are more prevalent

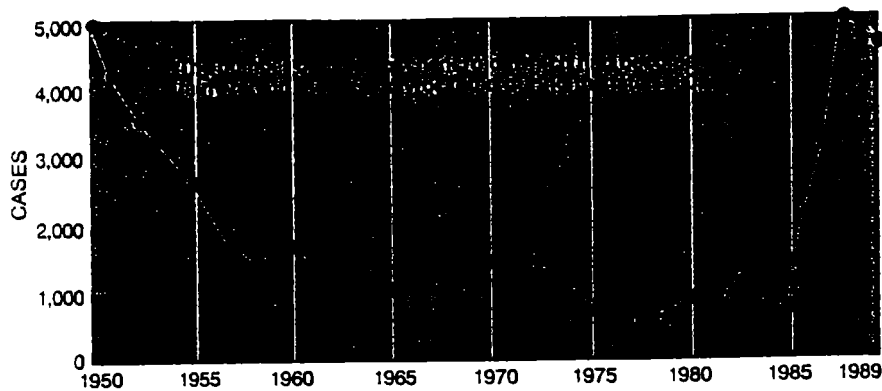
among persons of lower socioeconomic class and inner-city residents.

Two groups of researchers, one led by Andre Nahmias of Emory University, the other by Lawrence Corey of the University of Washington, found that only one quarter to one third of persons with serum antibody to HSV-2 were aware of having previously had symptoms consistent with genital herpes. Only a few genital HSV-2 infections ever seem to cause recognizable symptoms. New cases of genital herpes often arise after persons are exposed to the virus by a sex partner with an asymptomatic infection.

During the 1970s, researchers ob-



SYPHILIS is caused by the bacterium *Treponema pallidum* (top). The incidence of syphilis decreased among men during the early 1980s but later rose in both sexes (middle), primarily because of increases among black men and women (bottom).



served an association between cancer of the cervix and genital herpes. Many women infected with HSV-2 were counseled that they might be at serious risk for cervical cancer. Yet some medical authorities argued that genital herpes might only be a marker for high-risk sexual behaviors that could also transmit other STDs—perhaps even an unrecognized STD that was the true cause of cervical cancer. Many researchers therefore began to look more closely at genital warts, which are benign growths caused by the human papillomavirus (HPV).

A major advance in the study of HPV came with the application of recombinant DNA technology, which could isolate and clone, or copy, strands of DNA from papillomaviruses in warts. The cloned HPV-DNA could be labeled radioactively and used to probe for complementary DNA strands in genital lesions, including cancerous and precancerous lesions. Harold zur Hausen and his colleagues from West Germany led in identifying new types of HPV by this method. Today more than 60 types have been identified, several of which cause genital infections.

Like herpes infections, few of which produce sores, only a minority of HPV genital infections seem to produce obvious genital warts. Those types of HPV that most commonly cause genital warts are only rarely found in cervical cancer tissue. On the other hand, certain types that do not usually cause visible warts—especially types 16, 18 and 31—are very strongly associated with precancerous lesions or invasive cancers of the cervix, vagina, vulva, penis and anus.

Consultations for genital warts increased from 169,000 in 1966 to more than two million in 1988. It seems likely that the incidence of subclinical HPV infections, including those associated with genital cancers, is rising at a similar rate.

At the moment, genital and anal HPV

portion of sexually active adults seem to be infected. In a recent survey, HPV type 16 was detected in the Pap test specimens of 22 percent of female college students and 44 percent of female STD clinic patients in Seattle. The implications of finding a cancer-associated virus in such a high proportion of young women is obviously of great concern.

Hepatitis B infection is another important viral STD. There are an estimated 200 million carriers of hepatitis B virus in the world, and the number of new cases a year in the U.S. is estimated to be 300,000. It is discouraging to note that the overall measured incidence of hepatitis B has not declined much since 1982, despite a steep decline in high-risk homosexual contacts among men and the availability of an effective hepatitis B vaccine. Of all the new hepatitis B infections attributed to a known risk factor, the percent attributed to homosexual contacts declined from 21 percent in 1982 to 9 percent in 1987. During that same period, however, the proportions attributed to heterosexual contact and intravenous drug use both rose, from 15 to 22 percent and from 15 to 28 percent, respectively. Hepatitis B virus infection attributable to heterosexual contact is most common among persons of lower socioeconomic status.

During the past decade, the viral STD attracting the most attention from both the medical authorities and the general public has been AIDS, which is caused by HIV infection. On a global basis, the overwhelming majority of HIV infections are sexually transmitted. The World Health Organization projects that by the year 2000, up to 90 percent of all HIV infections in the world will be transmitted heterosexually.

Of the sexually transmitted HIV infections, most of those occurring in North America have been transmitted

most of those occurring in Africa and parts of the Caribbean have been transmitted heterosexually to women and men during vaginal intercourse. Heterosexual transmission also accounts for a growing proportion of the cases of AIDS and HIV infection in Europe and Latin America, and heterosexual HIV infections are now proliferating at explosive rates in parts of Thailand and India. Homosexual transmission predominates among men in North America, but heterosexual transmission is increasing: it accounted for approximately 5 percent of the newly diagnosed cases of AIDS in the U.S. in 1988 and 1989, including nearly 30 percent of the cases in adult women.

In the U.S., cases of AIDS ascribed to heterosexual transmission have disproportionately involved urban minority populations. For example, among U.S. men the incidence of heterosexually acquired AIDS is approximately 10 times greater for blacks and four times greater for Hispanics than for whites. There are several possible explanations for this pattern. The incidence of HIV infection transmitted by intravenous drug abuse is higher among blacks and Hispanics, providing a reservoir for heterosexual transmission. The incidence of other STDs is also higher among blacks and Hispanics. To the extent that certain STDs are risk factors for sexual transmission of HIV, the higher incidence of such STDs in racial minorities also may contribute to the higher incidence of HIV in these groups.

The evidence that other STDs increase the sexual transmission of HIV can be summarized as follows. The STDs that cause genital ulcer disease—chancroid, syphilis and genital herpes—have been associated with an increased risk of acquiring HIV infection in heterosexual men and women in Africa. Syphilis and herpes have also been associated with HIV infection in heterosexual men and women and in homosexual men in the U.S. In African women the risk of heterosexual acquisition of HIV has been elevated in those with gonorrhea or chlamydial infection of the cervix or those with a form of vaginal discharge caused by *Trichomonas*, a common parasite.

Conversely, HIV infection leads to altered manifestations of other STDs and thereby probably promotes their spread. Genital and anorectal herpes ulcers normally heal within one to three weeks, but they may persist for months as highly infectious ulcers in persons with HIV infection. As previously noted, HIV infection also raises the risk of treatment failure for chancroid ulcers.

ure of syphilis treatments and for altered manifestations of syphilis and gonorrhea in HIV-positive persons. We can therefore postulate that HIV and other STDs may promote one another's spread. That vicious circle can best be interrupted by more aggressive control of STDs other than HIV.

STDs and AIDS constitute exceptions to the shifting disease patterns in the modern world. Economic development has been marked by a demographic transition: declining fertility rates and mortality (especially childhood mortality) change the age distribution of the population. The rising average age of the population creates what has been termed the epidemiologic transition, in which the infectious diseases of childhood become less important and the chronic diseases of adulthood become more important.

An extremely important consequence of these demographic and epidemiologic transitions has received little attention, however. When childhood mortality falls but birthrates do not, a huge increase in the number of teenagers and young adults precedes the increase in the number of adults. This trend can be pictured as a slowly ascending bulge in the age pyramid. Because sexually transmitted infections are concentrated among adolescents and young adults, these infectious diseases can be expected to rise in incidence as the population bulge passes through the teen and young adult years.

When the number of young people is disproportionately large, they may also be less influenced by the social norms of the older generation. Consequently, more of them may engage in behaviors that raise their STD risks. It is quite likely that the demographic shift has played a part in the epidemic of STDs and the increases in other phenomena of adolescence such as juvenile drug use and related crime.

Today the black and Hispanic populations of the U.S. have age pyramids that closely resemble those of developing countries, with large proportions of teens and young adults and relatively few older adults. The proportions of the younger age groups in the white population, meanwhile, are declining.

Furthermore, the poor inner-city minority populations of the industrialized countries (particularly the U.S.) resemble the populations of developing countries (particularly in their urban areas) in many other demographic, sociopolitical and economic characteristics that are conducive to the high incidence and rapid spread of STDs. First,

both underdeveloped countries and inner-city minority populations are characterized by rapid demographic changes. Developing countries generally have high population growth rates, high rates of urbanization and population movements of all types. Inner-city populations in the industrialized countries, too, are highly mobile and change rapidly through middle-class flight and the increasing concentration of young, poor minority populations.

Rapid, drastic economic changes also mark both developing countries and industrialized inner cities. Developing countries fundamentally restructure themselves as they make the transition from agricultural self-sufficiency to dependence on wage labor. Similarly wrenching changes occur in some U.S. inner-city minority communities because of the sale of drugs, especially crack cocaine. Large sums of money change hands very quickly in the drug trade: this money is introduced suddenly into the daily life of inner-city communities, often through very young teenagers, thus radically altering the social structure.

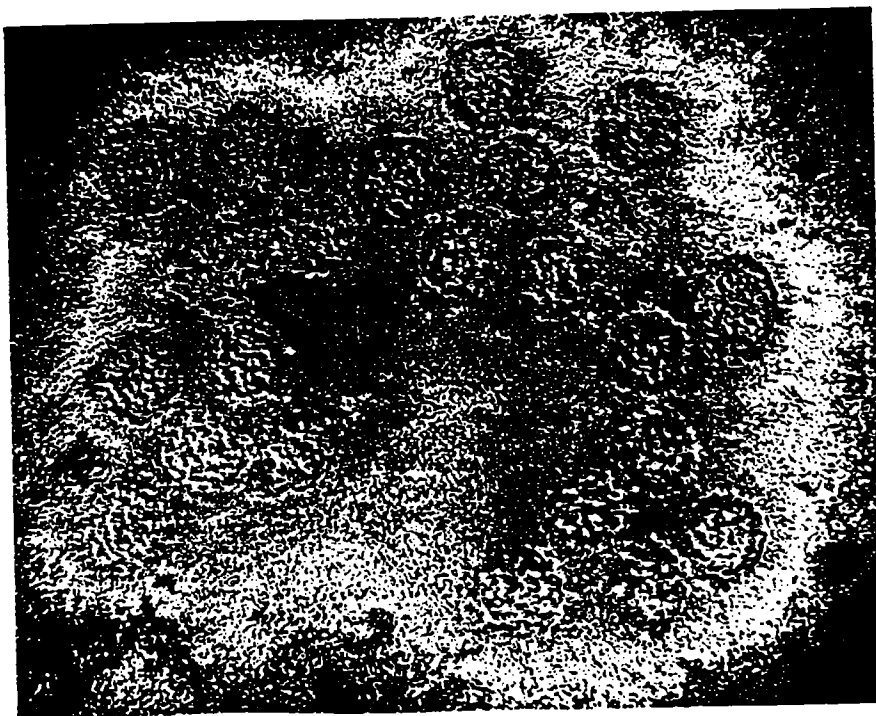
Underdeveloped countries and inner-city minority populations both have unstable power hierarchies. In many parts of the Third World, frequent civil wars, coups and border disturbances and the need for a strong police and military presence to assure peace in daily life testify to the lack of political stability. Similarly frequent violence in the inner cities of the U.S. points to the tentative

nature of power hierarchies in those communities.

The swift demographic, economic and political changes in these populations result in a social situation in which levels of transience and marginality are high. These conditions reinforce exchanges based on socioeconomic inequality, such as prostitution and illegal drug use. Prostitution has always been most common in settings characterized by poverty, social disintegration and a double standard of sexual behavior. Indeed, prostitution and the drug trade emerge as adaptive responses that help people acquire money, power or pleasure that would otherwise be unattainable. Yet those activities can enhance the spread of STDs.

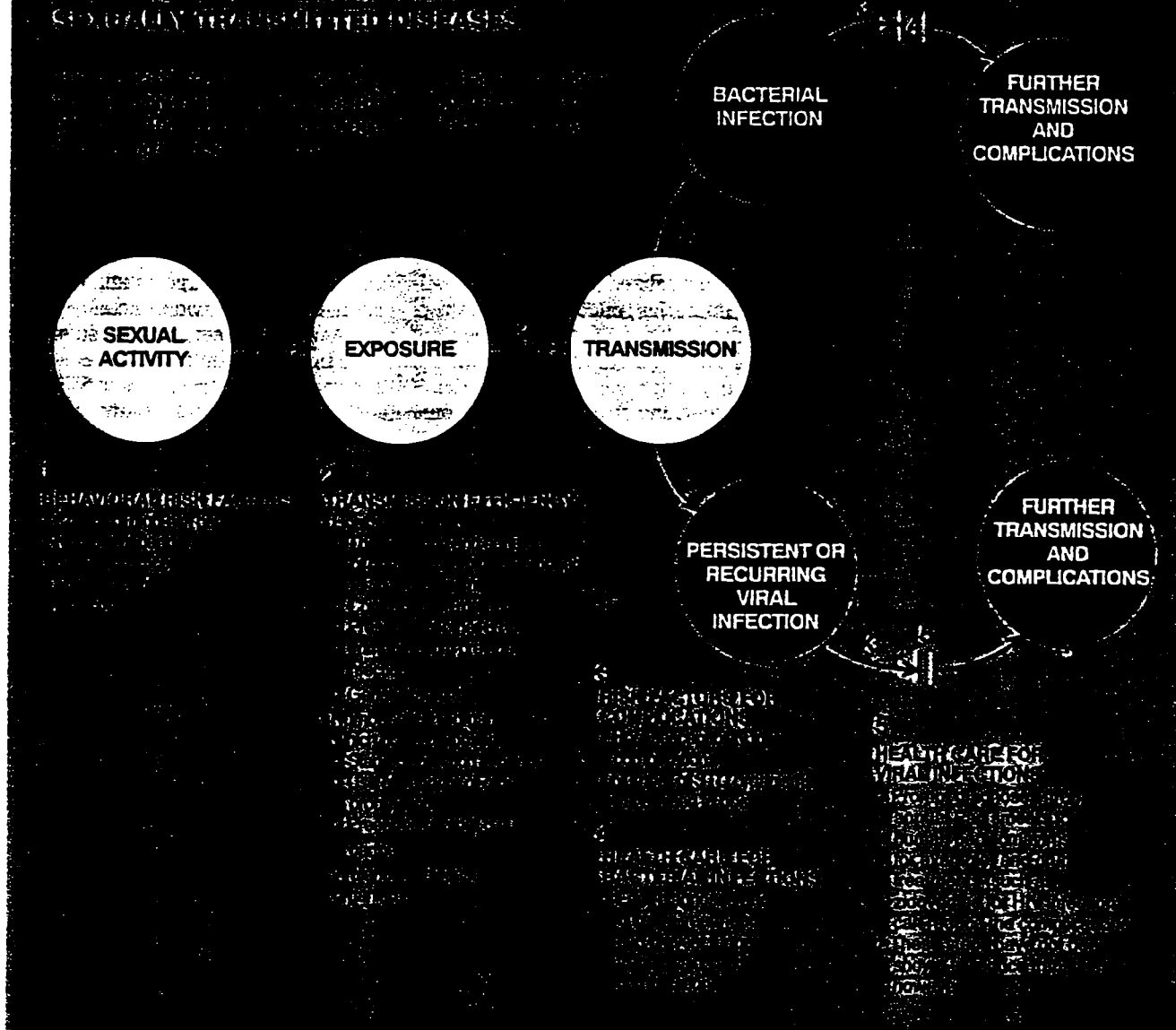
During the sexual revolution of the 1960s, prostitution was not considered a prominent factor in the STD epidemic in the U.S. During the 1980s, however, prostitute contact has reemerged as an important risk factor for STDs. Its increased importance has partly to do with the growing tendency toward transiency among the U.S. inner-city urban population.

The men involved in STD outbreaks are often (but not always) transients of some kind: foreigners, military servicemen, truck drivers, migrant laborers and so on. Their only sexual, and sometimes social, interactions with the main community may be through prostitute contact. The male-specific nature of most international migration (most markedly in its initial stages) and of



HUMAN PAPILLOMAVIRUSES cause the benign growths called genital warts. Some types that do not usually cause warts are associated with a variety of cancers.

RISK FACTORS AND INTERVENTIONS FOR SEXUALLY TRANSMITTED DISEASES



most temporary unskilled and semi-skilled labor tends to reinforce and foster prostitute contact.

Recent studies in urban areas of the U.S. have also found that transmission of gonorrhea, syphilis, chancroid and HIV infections has been closely associated with the exchange of sex for drugs such as crack. Women, particularly adolescent women, sometimes engage in very large numbers of sexual contacts to support their addiction. The epidemic increase in crack cocaine use, which began in the U.S. around 1985, therefore seems to be a major contributor to the simultaneous epidemic increase in syphilis, chancroid and gonorrhea among inner-city minority groups.

What can be done to interrupt the self-reinforcing relationships between

and health care behaviors both strongly determine the incidence of bacterial STDs and their complications. Sexual behaviors influence the spread of the diseases, and good health care behaviors—which lead to prompt medical consultation and notification of exposed sex partners—result in curative treatments and the interruption of the chain of transmission.

For the spread of viral STDs, sexual behaviors are much more important than health care behaviors because the diseases are not curable. Still, health care can be important for individuals: regular Pap tests enable early detection of precancerous conditions in the cervix, and early detection of HIV infection now leads to life-prolonging medical interventions.

the face of the trends we have discussed. Yet the evidence of changing sexual behaviors among homosexual men and the increasing use of condoms by customers of prostitutes in many societies stand as testimony to the power of public health efforts.

The steady decrease in the incidence of gonorrhea and syphilis among adult white heterosexuals in the U.S. and other countries is consistent with the institution of effective public health control measures. Yet the reemergence of bacterial STDs in young, black and Hispanic inner-city poor populations, coupled with the rising incidence of AIDS and other viral STDs, has created a demand for public health care and preventive interventions that exceeds the capacity of many systems to provide diagnostic

countries concentrate within specific high-risk heterosexual populations and population subgroups—the so-called core groups of frequent transmitters for these diseases—innovative programs are needed to focus STD control efforts. Core groups can be defined as those in which the rate of transmission is high enough to sustain an STD within the population: on average, every infected person infects at least one other person. Preventing or curing an STD in a member of a core group has much more impact than doing so in a person who is unlikely to transmit the infection.

A public health focus on the determinants of STDs in individual cases and prevention approaches that identify persons most at risk are most appropriate when exposure to the cause of a disease is heterogeneous in the population. Yet as Geoffrey Rose of the London School of Hygiene and Tropical Medicine asserted in a paper in 1985, the population approach becomes more important as a group's exposure to a disease becomes more homogeneous and widespread. Therefore, choosing the most appropriate disease-control strategy depends on determining the relative heterogeneity of exposure to causal factors within and across population subgroups.

At present, U.S. society is marked by increasing homogeneity within social groups and a widening gap between them. The formation of a growing underclass reflects these tendencies. Such social problems as lack of education, joblessness, homelessness, welfare dependency, family dissolution, drug abuse, homicide and other crimes concentrate more and more in inner-city neighborhoods.

STDs and many other health problems also follow this pattern. The incidence rates and morbidity for many STDs differ greatly among socioeconomic, demographic and behavioral subgroups. Within these subgroups, the risk factors that determine what causes individual cases of STDs may be far less important than the overriding social forces.

Consequently, the strategies for preventing STDs should target not only susceptible high-risk persons but also entire communities and subgroups with high STD rates. Such strategies, ranging from improved birth-control services to better educational and job opportunities, should address the full scope of factors underlying the high rates.

During the 1980s, public STD services in the U.S. were overwhelmed and

for public health care has exceeded the capacity to provide diagnosis and treatment. STD clinics are closing earlier in the day, patients are waiting longer hours to receive care and greater numbers of people are being turned away without receiving care. Although people are invited to return the next day or after the weekend, the close association of STD with drug use and with sex for drugs ensures that many potential patients will continue to spread infection after being turned away.

In a survey we conducted in May 1989, 19 out of 23 public STD clinics throughout the U.S. reported one or more features of the pattern of delayed treatment. It is ironic that public health officials lament the difficulty of finding the sex partners of crack-using patients with STDs and emphasize the need for more workers to trace sexual contacts, while people who do show up at the clinics are being turned away because of a lack of services.

Clinical services for diagnosing and treating STDs must be greatly expanded until all patients can be examined and treated on the day that they present themselves. That goal will require expanded clinic facilities, recruitment of trained clinicians and substantially increased funding for diagnostic testing of STDs, especially chlamydial infections. STD services also can be increased by closer integration with programs for family planning, prenatal care and AIDS prevention. In this way, persons who seek such care can also receive routine screening and treatment for STDs. Family-planning services should also be available through STD clinics, but that step will require even further expansions of clinical staffing.

Control of STDs in the inner-city underclass also calls for new approaches, such as satellite clinics in inner-city neighborhoods, that reach out to the affected communities. Mobile clinics or vans could provide services to the growing homeless population. In addition, innovative behavioral interventions must be expanded to prevent the incurable STDs. Health care education and other interventions must be offered in terms that are appropriate but explicit to youths in and out of schools in the communities at risk. It is essential that the inner-city communities and the health sector work as partners in developing and administering the clinical services and behavioral intervention programs.

Because diverse health problems often overlap many social and economic problems, public health policies should be coordinated with other programs

active in the community, such as Job Corps and Head Start. Low-cost housing and drug treatment and public service and prison programs could also be used to forge health initiatives. It seems that local governments serving the urban poor are no longer able to provide the levels of support essential for STD control services. Leadership and expanded support from national and state levels are needed.

Innovative approaches are needed to increase the availability of health care workers, particularly physicians, and services for urban poor populations. For instance, National Health Service Corps physicians could be deployed to treat AIDS and STDs among the urban poor. Without such help, it is unlikely that there will ever be enough physicians and nurses in the inner cities to meet the desperate need for services.

In the 1990s the solution to the STD crisis cannot rest solely on preventing high-risk behaviors in individuals. The factors that determine the high incidence of AIDS and other STDs in specific groups and in entire communities must also be identified and addressed. A balanced public health STD program for our cities in the AIDS era must include expanded clinical and health education services. Clinical services for STD control should grow to meet the demand, integrate with family-planning and related programs, and adopt the technological advances of the past decades in STD prevention and control. Prevention activities ought to include population and group approaches as well as individual counseling. By themselves, the medical solutions to the prevention and control of STDs and AIDS are not enough: they must be coupled with the identification and correction of the societal factors responsible for the global pandemic.

FURTHER READING

- SICK INDIVIDUALS AND SICK POPULATIONS. Geoffrey Rose in *International Journal of Epidemiology*, Vol. 14, No. 1, pages 32-38; March 1985.
- EXTENDING EPIDEMIOLOGIC TRANSITION THEORY: A NEW STAGE. Richard G. Rogers and Robert Hackenberg in *Social Biology*, Vol. 34, Nos. 3-4, pages 234-243; Fall/Winter 1987.
- QUIET RIOTS: RACE AND POVERTY IN THE UNITED STATES. Fred R. Harris and Roger W. Wilkins. Pantheon Books, 1988.
- EPIDEMIOLOGY OF SEXUALLY TRANSMITTED DISEASES. Sevgi O. Aral and King K. Holmes in *Sexually Transmitted Diseases*. Edited by K. K. Holmes, P. A. Mårdh, P. F. Sparling and P. J. Wiesner. McGraw-Hill, 1990.

TO <u>~~Barry~~</u>		TELEPHONED	PLEASE CALL	
DATE _____ TIME _____		CALLED TO SEE YOU	RETURNED CALL	
PHONE CALLS		WILL CALL AGAIN	URGENT	
"WHILE OUT" RECORD		MESSAGE _____		
M. <u>Barry Lottan</u>	/ 3-9-95 fax			
OF <u>Whitman-Walker</u>	yes			
PHONE <u>202</u> <u>797</u> <u>3521</u>	TAKEN BY: <u>Michael</u>			
AREA CODE	NUMBER	EXTENSION		

**Office of
National AIDS Policy**
Correspondence Routing slip

on Calendar

Tracking Number: 1209
(File name in N:\corres Directory)

Date Rcvd: 3-24-95

From: Jim Graham

Staff Action:

Jeff Levi RCVD: _____ DONE: _____

Assigned To: AF

RCVD: _____ DONE: _____

Reviewed By Jeff: _____ Reviewed By: Patsy ☒

Action for Staff:

Recommendation/Instruction:

*Carmena -
Please call to
confirm. Would
it be possible
to get Hts before
that night to
avoid the crash?
P*

*Jessica Montgomery will messenger
the tickets on 4/17*

To Support Staff: _____

Date of Response: _____

Wilmington1407 S Street, N.W.
Washington, DC 20009202/ 797-3500
FAX: 202/ 797-3504

Richard

4/22

TO:

Ken Barrett

7⁰⁰/₉

FROM:

Barry Laffon

Number of Pages (Including Cover): 3

Date:

3/9/95

Message:

Please give me a call when you
receive this fax.

This is a copy of letter sent last month.

Thanks for your assistance
(202) 797-3521

This facsimile is intended only for use of the addressee named above and may contain legally privileged and/or confidential information. If you are not the intended recipient of this facsimile, you are hereby notified that any dissemination, distribution or copying of this facsimile is strictly prohibited. If you have received this facsimile in error, please immediately notify us by telephone and destroy.

Questions or Problems.
Please Call me at 202/ 797-3521
Thank you.

ELIZABETH TAYLOR
MEDICAL CENTER

WHITMAN-WALKER CLINIC

February 6, 1995

Ms. Patricia S. Fleming
National AIDS Policy Director
750 17th St., NW
Washington, D.C. 20503

Dear Ms. Fleming:

I hope this letter finds you in the best of health and spirit. As you will remember from last year, with all the terrific assistance you provided us, spring is the time for our annual fund raiser to benefit the Elizabeth Taylor Medical Center. On this occasion we also recognize a leader in the battle for AIDS awareness and support.

On Saturday, April 22, 1995, Whitman-Walker Clinic will present it's annual "*Friends for Life*," Spring Gala. This event, chaired by Miss Elizabeth Taylor, will take place at the Warner Theatre and will feature a performance by one of America's treasures, the Alvin Ailey American Dance Theater. We would appreciate having your participation on the Gala Honorary Committee and share in what we know will be a great demonstration of support for the nation's first stand-alone facility for the treatment of HIV/AIDS.

The "*Friends for Life*" Gala was one of the most successful events in the Washington area, for AIDS support. Prior to last year's performance, by Mikhail Baryshnikov and the White Oak Dance Project, the evening began with a presentation of the "*Friend for Life*" Award to The Honorable Donna E. Shalala, U.S. Secretary of Health and Human Services. This year, the Board of Directors has voted to recognize Mrs. Tipper Gore, who has shown such an immense and longstanding commitment to the work of Whitman-Walker Clinic.

Following the evening's performance, there will be a festive party for the members of the Dance Company, our Gala Chair, Miss Taylor, our Honoree, Mrs. Gore, and our guests and supporters. If you agree to become a Gala Honorary Committee Member, please respond by February 17, 1995, and enclose in your reply a black and white photograph, which will appear in the event program.

With your support we can make the 1995 "*Friends for Life*" Gala more successful than last year's; moreover, it will provide vital funding for the services provided at the Elizabeth Taylor Medical Center and the Robert N. Schwartz Housing Services. There is a great need in our community, and with you working side-by-side with us, we will stand tall in this challenge.

page two

I know that Miss Taylor joins me in thanking you for your efforts on behalf of those living with HIV and AIDS. I hope you feel as I do, that this battle is among the most important we face. Please consider taking this important step in the pursuit of victory.

With every good wish,

Sincerely,

Jim Graham
Executive Director



MEMORANDUM

April 6, 1995

*American
Social
Health
Association*

TO: Camina Paris
FROM: Erin Bush
SUBJECT: Meeting with Peggy Clarke

311 Massachusetts Avenue, NE
Washington, DC 20002
(202) 543-9129
Fax (202) 543-5327

I tried to call you this AM, but it seems that your phone system is suffering from fits. I hope it is not causing havoc!

I spoke with ASHA's President, Peggy Clarke, and our Washington Representative, ~~Lynn Morrison~~, and have discovered that **the morning of Monday, April 17th** would be perfect. I promise that if you schedule a ~~meeting with Patsy~~ at this time I will not allow Peggy or Lynn to cancel.

Please give me a call (if you can) to let me know how this looks from your end. I can be reached at 202/543-9129.

Thank you for all of your help.

NO
*If she is in town
some other time - try then*
Done

New Jersey Medical School 40th Anniversary Symposium:

*Communities in Jeopardy: The Maturation of
the HIV/AIDS Epidemic*

April 19, 1995

Medical Science Building Room 556B

NO
RS has to
write letter to
be read at mtg.

8:00 a.m.

Welcome Address

Ray Lourenco, M.D.

Dean of the New Jersey Medical School

Introduction

James M. Oleske, M.D., M.P.H.

Francois-Xavier Bagnoud Professor of Pediatrics

Clinical Issues - Moderator: *Patricia Kloser, M.D., Department of Medicine*

9:00 a.m.

Care of the Pregnant HIV Positive Women: New Breakthroughs and
Future Challenges

Arlene Bardeguet, M.D.

Department of Obstetrics and Gynecology

9:30 a.m.

Tuberculosis in Communities with a High Prevalence of HIV/AIDS

Lee Reichman, M.D., M.P.H.

NJMS-National Tuberculosis Center

10:00 a.m.

Prospective Studies of Women and Drug Users in New Jersey:
Epidemiologic & Immunologic Insights

Stanley H. Weiss, M.D.

Department of Preventive Medicine & Community Health

10:30 a.m.

Break - Visit Posters & Exhibits

Psychosocial Issues - Moderator: *Sandra Lewis, Psy.D., Departments of Psychiatry & Pediatrics*

10:45 a.m.

AIDS Orphans: Extending the African American Family

Cynthia Paige, M.D.

Department of Family Medicine

11:15 a.m.

The Intersection of Nutrition with HIV/AIDS: The Potential of
Supplementation Therapies

Donald Louria, M.D.

Department of Preventive Medicine & Community Health

11:45 a.m.

Panel Discussion - Faces of the Epidemic: Supporting Caretakers

Mary Boland, R.N., M.S.N. - Moderator

Department of Pediatrics

12:15 p.m. Lunch - will be served on C-Level

Decision-making and Patients, Families and Health Care Workers: The Ethical Challenge of HIV/AIDS

Patricia Fleming, Ph.D. (Invited)

National AIDS Policy Director for The White House

Basic Science - Moderator: Frederick Coffman, Ph.D., Department of Pathology

1:30 p.m. Pathogenic Mechanisms in HIV-1 Associated Progressive Encephalopathy in Children

Leroy Sharer, M.D.

Department of Laboratory Medicine & Pathology

2:00 p.m. Three-Dimensional Structure of HIV Reverse Transcriptase

Mukund Modak, Ph.D.

Department of Biochemistry & Molecular Biology

2:20 p.m. Designer Vaccines: The Challenges of HIV Vaccines & Immunotherapy

Raphael Mannino, Jr., Ph.D.

Department of Laboratory Medicine & Pathology

2:40 p.m. HIV Diagnostics in the Developing World

Thomas Denny, B.S.

Center for Laboratory Investigations, Department of Pediatrics

3:10 p.m. Break - Visit Posters & Exhibits

Education - Moderator: Robert L. Johnson, M.D., Department of Pediatrics

3:30 p.m. Spreading the Word: The Role of a National Resource Center

Carolyn Burr, R.N., M.S.

National Pediatric & Family HIV Resource Center

Department of Pediatrics

3:45 p.m. Transforming Practice: Educating Health Care Professionals

Debra Bartelli, M.P.H.

Division of AIDS Education

Center for Continuing Education in the Health Professions

Closing Remarks

4:00 p.m. Living with HIV: A Vision of Hope

James M. Oleske, M.D., M.P.H.

Department of Pediatrics

National Pediatric
HIV Resource Center

15 South Ninth Street
Newark, NJ 07107
(201) 268-8251
FAX (201) 485-2752

Ken
get
Carol Levine's
to
F Julie +

talk to her
about the
A letter

X

Date: 3/1/95

No. of pages (excluding this one) 2

To: Ken Barnett

FAX: 202.632.1096

From: Julie Plunkett

Please call if FAX transmission incomplete or interrupted.

Message

Hi Ken!

as discussed, here

is a copy of the

agenda for the

April 19, 1995

HIV/AIDS Symposium

at UMONT - NJMS.

Thanks for your help!

Regards -

Julie Plunkett

Street Address:

1601 N Kent Street, Room 1200
Arlington, VA 22209
Phone: 703-875-4714/4726
FAX: 703-875-4686

**Mailing Address:**

Room 1200, SA-18
Washington, DC 20523-1817

Agency for International Development
Office of Health and Nutrition
Bureau for Global Programs, Field Support and Research

To: Patsy Fleming
attn: Carmena - Paris

From: Jeanine Buzzy

Phone: 202-632-1090

FAX: 202-632-1096

Number of pages (including cover sheet): 2

MESSAGE:

April 11-24 PF - Has original
Carmena -
Hold this one on
PP's calendar
until she decides if

On calendar

Hi Patsy -
How are you? I am faxing you an invitation to attend an NGO Meeting with USAID concerning the draft policy guidance document that Nils Daulaire is in the process of completing. I will courier you a copy of the document. The meeting is April 11th at 2:00pm at the State Dept. Rm. 1205. Carol Lancaster will chair. I would love it if you could come.

Please let us know -

J. Jeanine

P.S. Hill Staffers have also been invited.



March 24, 1995

U.S. AGENCY FOR
INTERNATIONAL
DEVELOPMENT

Dear Colleague,

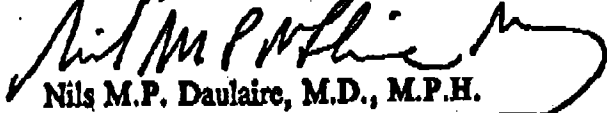
USAID is currently finalizing an updated draft of the Agency's *HIV/AIDS Policy Guidance* document. This document is intended to provide USAID staff and our cooperating partners a description of the priorities, strategies and activities that USAID will support to address the challenges posed by the HIV/AIDS epidemic. You will find a copy of this draft enclosed for your review and comments. In July of 1994, a previous draft was circulated and input solicited from various sources. The enclosed document represents a revision based on the comments received.

As part of the process of finalizing this guidance document, USAID would like to invite you to participate in a PVO/NGO meeting with USAID senior officers on April 11 from 2:00 to 4:00PM in Room 1205, Department of State, Washington, DC 20523.

We sincerely appreciate your time and effort concerning the development of USAID's *HIV/AIDS Policy Guidance* and look forward to hearing your suggestions. Please RSVP to Sandy Jenkins (Telephone: 703 875 4494; FAX: 703 875 4686) and provide a date of birth and social security number to allow preclearance for security access. On the day of the meeting come with a picture ID card to the Diplomatic Entrance of the Department of State, which is at the intersection of C and 22nd streets, N.W.

If you are unable to attend this meeting, we would still appreciate any written comments you may have. Please send these to: Dr. Paul De Lay, G/PHN/HN/HIV-AIDS, SA-18, Room 1200, USAID, Washington, DC 20523-1817 (telephone 703 875 4616).

Sincerely,



Nils M.P. Daulaire, M.D., M.P.H.

Chief Policy Advisor on
Population, Health and Human Development
Bureau for Policy and Program Coordination



Jacob A. Gayle, Ph.D.

Chief, HIV-AIDS Division
Office of Health and Nutrition

CURE OF ACQUIRED IMMUNE DEFICIENCY SYNDROME (A. I. D. S.)

The background and significance; would relate to the memory of house flies; blowing (life) of carcass (death) and feces (death); feeding (life) from feces (death); noticing the different opposites (life/death) in these components: Similar to; semen (life) being mixed with feces (death), from sodomy: And good blood (life) mixed with contaminated blood (death) by dirty syringe: Inventing a living - death bacteria formulating off of life, or A. I. D. S.

DESCRIPTION

The specific aim of this research is for the cure of Acquired Immune Deficiency Syndrome (A. I. D. S.), would be a neutralizing process. Since, A. I. D. S., is a; living - death bacteria formulating off of life, then, the cure is the opposite of this symbolic-equation which is a; death - living anti - bacteria formulating off of death (corpse). If necessary toward the missing - link of A. I. D. S., which would require exercising the mind, body and psyche according to the natural design of the A. I. D. S. recipient.

SAFETY

The health relatedness of this research; must proceed with extreme precaution, in health safety for all applicants involved, in this research, so as not to be infected with A. I. D. S.

RESEARCH PLANS

Would relate to blood type of the corpse (unprocessed) matched with A. I. D. S. recipient's blood type, being given:

(1.) The solvent from the corpse, after, being blown by a house fly producing a; ' protective life - sustaining solvent, ' for the fly larva, amidst the multiplicity of bacteria of the corpse. (2.) The mole which grow from the corpse, similar, to the way penicillin is produced. (3.) Similar, biological structure of the solvent.

POSSIBILITY

For more advance form of A. I. D. S. infection, an identical blood type fly - larva treated A. I. D. S. corpse should suffice.

Respectfully,

Donald C. Crockett

Donald C. Crockett

115828

Turney Center Industrial Prison
and Farm

Route 1

Only, Tennessee, 37140 - 9709

Update: 12/16/94
Sworn & Subscribed
before me this 16th day
of December, 1994
Notary Public
My Commission Expires;
7-17-96

This is where your money should be going.

Sincerely,

Donald C. Crockett

Donald C. Crockett

115828

Special Needs Facility,
9-2-S204

7575 Cockrill Bend Industrial
Road

Nashville, Tennessee 37243-
0475

Date; 4/1/95

TO <u>Carmena</u>			TELEPHONED ☐		PLEASE CALL ☐	
DATE <u>3-27</u> TIME <u>5:36</u>			CALLED TO SEE YOU ☐		RETURNED CALL ☐	
PHONE CALLS			WILL CALL AGAIN ☐		URGENT ☐	
"WHILE OUT" RECORD			MESSAGE <u>11:00 Fri. Apr. 7</u>			
M. <u>~~Col.~~ Donald Burke</u>						
OF _____						
PHONE <u>301</u> <u>217</u> <u>9410</u>			TAKEN BY: <u>mg</u>			
AREA CODE NUMBER EXTENSION						