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TAG
Treatment Action Group

tagline

NEWS ON THE FIGHT FOR BETTER TREATMENT, A VACCINE, AND A CURE FOR AIDS

Martin Delaney, 1945–2009
Life and Work of an AIDS Activist

The world lost one of the pioneers of AIDS activism when Project Inform founder Martin Delaney died of liver cancer in San Rafael, California.

BY MARK HARRINGTON

Martin Delaney

Fighting for people with HIV until the very end of his life, Martin Delaney embodied the qualities that have come to define AIDS activism:

- a willingness to challenge authority
- a belief that science is for people, not for scientists alone
- a belief that ordinary citizens have a right to scientific information, to participate in research (or not), and to access experimental treatments when there are no approved treatment options or when approved treatments are failing
- a willingness to listen to opposing views, and sometimes to change one's mind in light of changing circumstances or emerging new data
- a willingness to compromise where possible without compromising essential rights
- a fierce, dogged, and relentless tenacity that would not rest until the goal was achieved

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Message to the New President:
Join Us in the Fight against AIDS

Recommendations submitted by Treatment Action Group to President Barack Obama on ways to strengthen the struggle to end AIDS.

Demonstrate Strong Domestic and Global Leadership
TAG urges President Obama to make fighting AIDS a national and global priority through personal involvement, public speeches, and swift action. His administration must act immediately to implement a comprehensive national AIDS strategy and preserve the successful PEPFAR (President's Emergency Plan for AIDS Relief) program. The administration must be steadfast in its commitments to reducing HIV transmission, expanding testing and treatment, and protecting the civil and health rights of all individuals affected by HIV.

Put Research for HIV, TB, and Viral Hepatitis Back on Track
The U.S. National Institutes of Health (NIH) leads global research efforts against HIV and is the world's largest government funder of research on HIV and related diseases, such as hepatitis B, hepatitis C, and tuberculosis (TB). In 2008 the NIH spent $2.9 billion on HIV research but only about $160 million on TB, the leading killer

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I first worked with Marty in the spring of 1989. It was the period when ACT UP/New York's Treatment and Data (T+D) Committee was sending monthly delegations to hearings of the National Committee to Review Procedures for the Approval of New Cancer and AIDS Drugs, a panel appointed by President George H. W. Bush and chaired by Louis Lasangna to look at ways to speed up approval of AIDS and cancer drugs. At the same time, Marty and his ally, attorney Jay Lipner from the Lambda Legal Defense and Education Fund, were trying to negotiate with the Food and Drug Administration (FDA) not only how to speed up drug approvals but also how to provide broader access to experimental treatments when they were still in clinical trials.

Jim Eigo from ACT UP's T+D Committee began writing down the precepts of what would become Parallel Track, a proposal originally made by Marty after long conversations with Lipner and Anthony S. Fauci, the director of the National Institute of Allergy and Infectious Diseases (NIAID). Fauci was also seeking ways to reduce the pressure on the NIAID-funded AIDS Clinical Trials Group (ACTG), which could not possibly enroll all the nation's people with AIDS in its small, slow, and almost paralyzed clinical trials network.

Delaney, Lipner, and Eigo were the key community activists in leading the negotiations with the FDA and with NIAID, and later with the drug company Bristol-Myers; these negotiations led to the Parallel Track program for didanosine (ddI), the second AZT-like drug to make its way through clinical trials. The ACTG was unable to enroll more than a couple thousand people with AIDS into its studies of ddI, while around the country, tens of thousands of people with AIDS were failing on or intolerant to AZT, the only approved AIDS drug at that time.

With coordinated pressure from Project Inform, ACT UP, and other activist organizations, as well as from Fauci at NIAID and from Sam Broder, director of the National Cancer Institute, the FDA allowed ddI to be released on a Parallel Track program in the fall of 1989. Eventually over 35,000 people with AIDS received ddI through Parallel Track before the drug was approved by the FDA in late 1991. Since the drug was eventually found effective and approved, in retrospect the Parallel Track program is likely to have saved or extended thousands of lives.

When Treatment Action Group first split off from the T+D Committee and became an independent organization, we collaborated closely with Marty and with Jesse Dobson from Project Inform on what became the Immune Restoration Think Tank (IRTT) series of workshops to expedite research on restoring the immune system damaged by HIV. Jesse died of AIDS in 1993, but Marty, along with Brenda Lein, kept the IRTT workshops alive until the advent of highly active antiretroviral therapy put a temporary eclipse on immune research.

Marty and Project Inform were also key players in supporting what became the FDA's program to expedite drug approvals based on changes in surrogate markers such as CD4 counts and, later, HIV RNA (viral load), thus shortening the duration of pivotal clinical trials and hastening FDA approval of a new wave of highly active antiretroviral drugs.

Soon, the treatment landscape changed radically in the United States and other developed countries; AIDS death rates plummeted by over two-thirds and a new era of care for HIV was inaugurated.

In the late 1990s TAG worked closely with Marty and his colleagues on getting the FDA to hold hearings to emphasize the importance of studying new drugs in salvage (e.g., multidrug-experienced) populations. As a result, most of the new anti-HIV drugs approved in the past decade—tenofovir, FTC, T-20, tipranavir, darunavir, raltegravir, and maraviroc among them—were first studied and first approved in salvage therapy populations. The result was, again, the prolongation or saving of thousands of lives.

TAG also worked closely with Marty, with Project Inform, and with Linda Grinberg and her Foundation for AIDS and Immune Research (FAIR) on a series of workshops on the role of treatment interruptions in HIV therapy. Clinical trials stimulated by these meetings later demonstrated that treatment interruptions were unsafe.

The last time I saw Marty in person was in February 2008 at the 15th Annual Retrovirus Conference in Boston, where we met to plan a workshop on eliminating HIV persistence and eradicating HIV infection, which TAG cosponsored with amfAR, FAIR, and Project Inform in November 2008.

By the fall, Marty was too sick to travel. After beating hepatitis B infection in the 1980s and surviving a quadruple bypass earlier in this decade, his two previous conditions had conspired to produce a virtually untreatable hepatocellular carcinoma that was only finally diagnosed in late 2008. The location of the tumor, its spread, and the prior cardiovascular disease made getting a new liver difficult if not impossible. Marty called me in early December and reported on his condition. He said, "I've survived two life-threatening diseases already, but I'm not sure I'll be able to make it this time."

As always, he faced the situation with courage and honesty. This time there were not a lot of treatment options. In his last weeks he was surrounded by his family and by longtime colleagues and friends such as Brenda Lein, Anne Donnelly, and David Evans. Over Christmas he even made it to Hawaii for a vacation. Upon his return to California he entered the hospital but soon returned home. I flew out to visit him on Friday, January 23, but it was too late. By the time I landed, Marty was gone. I spent that evening with his friends and colleagues at the memorial wall at Eighteenth and Castro Streets, where people left flowers, lit candles, hung Buddhist prayer flags, and posted tributes to Marty.

It is too soon to comprehend our loss. ●

Looking for a Cure for AIDS
What are the Mechanisms of HIV Latency and Persistence?

The government has an extensive plan for conducting AIDS research, but finding a cure is not part of it.

BY BOB HUFF

Two of the great quests in science are to develop a vaccine to prevent HIV infection and a treatment to cure it.

The 200-page National Institutes of Health (NIH) *Plan for HIV-Related Research** for 2010 sets out the U.S. government's priority areas for AIDS research and serves as a road map to coordinate its investment in AIDS science. The plan establishes two major priorities for NIH AIDS research: the prevention of HIV transmission and the prevention and treatment of HIV-associated illnesses and coinfections.

A glance at the plan makes it clear that finding a vaccine is a key goal of the NIH AIDS science effort. This is certainly justified given the enormous impact that a preventive vaccine would have on the course of the epidemic. However, if you search for mention of a cure, you will come up short. Attempting to eradicate HIV infection is not a priority, an objective, or even a strategy mentioned in the NIH plan. This gap in the national research effort raises a troubling question: How likely are we to find a cure for AIDS if it is not on the official road map?

One reason why curing HIV infection receives insufficient attention from the scientific establishment is because it seems like an extraordinarily difficult—perhaps impossible—goal to achieve; research money tends to flow to problems that people believe can be solved.

There are two issues that will make curing HIV infection so tough. The first is that HIV inserts its DNA into the DNA of an infected person's immune cells, and in some of these cells, the viral genes go to sleep, giving no sign they are there until they are activated at some future point. This is called *latency*. Because modern antiretroviral (ARV) drugs can effectively prevent circulating virus particles from infecting fresh cells, latency on its own wouldn't be such a problem if all of the infected cells died off fairly quickly (cells that actively produce virus tend to self-destruct after a short while; uninfected and latently infected cells survive longer). But some of the infected immune cells go into a "resting state" of dormancy, and may stay that way for ten years or longer. Others may divide and give rise to fresh daughter cells that carry a latent copy of HIV. This means that HIV infection is *persistent*.

Because HIV can establish a persistent and latent infection in very long-lived resting immune cells, a *reservoir* of HIV is created within the body that could take decades to disappear—and that's if the reservoir was never replenished by virus that managed to escape the antiretroviral drugs. One of the lingering questions for scientists is whether reservoirs are replenished by active viral replication or not. In any event, if the drugs are stopped before the reservoir is fully depleted, then HIV will likely resume infecting new cells, and levels of virus in the body will surge.

The investigation of HIV latency and persistence does not appear in the short list of topics that the NIH says will receive the highest priority under its plan for HIV research. Only a few lines in the plan call for studying factors that enable HIV to establish a persistent infection or for understanding the reservoirs that permit HIV persistence. It's no wonder that scientists who apply for funding to study HIV latency are so often turned down: the term does not appear in the NIH plan.

There are many good reasons to be skeptical about the chances for actually curing HIV. The mechanisms that permit latency are still not fully understood, and there are competing theories for how the viral genes are silenced in certain cells. In fact, there are likely multiple mechanisms at work, which means that any single approach to a cure could be insufficient.

In brief, the main theoretical strategies for eliminating HIV from the body involve:

1. waking up every single latently infected cell in the reservoir then letting each die off—all while keeping any new cells from becoming infected
2. finding a way to identify latently infected cells then specifically killing them
3. sending molecular robots into cells to search for HIV DNA sequences then deleting or scrambling them

The last of these strategies is the sci-fi approach and (despite a recent report**) may not be possible for many decades. Identifying latently infected cells and killing them sounds ideal, but how to do it remains a puzzle since, by definition, such cells look exactly like uninfected cells. Thus, a strategy of waking up the cells of the latent reservoir and getting them to start making HIV copies seems like a plausible first step. Once awake, the infected cells would self-destruct or be eliminated by the immune system; antiretroviral drugs would protect new cells from becoming infected; and, theoretically, the body would soon be free of HIV. This approach is thought of as "purging the reservoir" and a few early, though so far unsuccessful, trials have been attempted in people.

Research into how the reservoir of latently infected cells can be flushed out is proceeding slowly in a few laboratories around the world. Support from the NIH is needed to invigorate this research and put finding a cure for AIDS back on the map. ●

** www.oar.nih.gov/strategicplan/fy2010*

*** Sarkar I, Hauber I, Hauber J, Buchholz F. HIV-1 proviral DNA excision using an evolved recombinase. Science. 2007 Jun 29;316(5833):1912-5.*

Quest for a Cure

A Conversation with Doug Richman and Celsa Spina

There are only a handful scientists working on finding a cure for HIV infection. Doug Richman and Celsa Spina, from the University of California–San Diego, are among them. We asked about the scientific and funding barriers they and their colleagues are facing and about the outlook for new and better HIV drugs.

BY BOB HUFF

Bob: When will we know if curing HIV infection in a person is possible?

Doug: When we succeed. There are two major potential accomplishments in HIV: a vaccine to prevent the disease and an intervention to cure it. One is for the uninfected and one is for the infected. It isn't clear that either is possible, but the potential benefits of both are so great that it's absurd to give up without trying.

Lots of people are working on vaccines, but the people who are working on eradicating the latent reservoir of HIV is a pretty small group—there are only about a half dozen investigators' groups working on eradication. This is partially because some people may not think eradication is an achievable objective; it's been hard to figure out how to address the problem; and it's very complicated: we're not sure what the mechanisms are. And because the reviewers that evaluate NIH grant proposals have been so skeptical, even the few groups that are interested have had a hard time getting funded. With the vaccine there is a lot of successful experience with other viruses, and people know what the two arms of the immune system are. But we don't even know what the mechanisms of eradication would be. And if we identify the mechanisms then we still have to find small molecules that work. So I think we need a lot of basic science research.

Bob: What would you be screening for right now if you were looking for molecules? Do you have an assay?

Doug: I think Celsa has got the best in vitro model. A lot of the cell line models are very artificial, but she's got a model using primary human lymphocytes. I think the SIV macaque model reflects latency, but working with macaques is very difficult and expensive and you can't screen things in macaques; you have to have something you think would work before you start testing it.

I think what a number of us think needs to be done is to set up some sort of a collaboration among the NIH, industry, and some investigators. It begins with discovery. Work on mechanisms is done by different investigators; high-throughput screening of molecules can only be done in the pharmaceutical industry; and developing applications requires a lot more collaboration.

Anything too innovative or too risky is not being funded.

Bob: Can the site where HIV integrates in the host genome be a factor in latency?

Doug: I think the data are pretty convincing that the probability of integration is related to the chromatin structure and activity of transcription of the gene. But those are probabilities. The question is, if it integrates in some other places in much lower frequencies, then do you have to deal with different locations or with different strategies? Are there multiple mechanisms you have to deal with? This is why science is slow and steady and it will take a decade if not longer [to understand this]. There's not going to be a magic bullet discovered on the first experiment. . . .

Celsa: We've been trying to do things with in vitro models in primary cells. We'd like to look at an individual cell and correlate where the virus integrated with whether or not that virus is replication competent. But at this point we don't know how to do that.

Doug: We know there's a lot more integrated DNA than there is replication competent DNA in infected cells. And the questions are: Is it the integration site? Is it whether the provirus is competent or not? There are a lot of details.

Bob: If the integrated DNA can't make a fully competent virus, can it still make HIV proteins that have toxic effects on their own?

Doug: You know, because a third of our genome is integrated retrovirus, and, the more people look, the more they see that even some of the integrated retrovirus that entered our genome ten million years ago is producing some transcription or translational products—and some of them have consequences. So it is probable that HIV may do that to some extent, too. In fact some of the RNA we're seeing may be the result of those things being produced, but we're not confident.

Bob: It seems like the coming of raltegravir really revitalized this field of eradication.

Doug: I have no idea why. That makes no sense to me whatsoever. The thing that turned on a lot of people was the more rapid clearance of RNA. It hasn't been absolutely proven, but I think, logically—and Bob Siliciano has a paper with modeling—it's just a function of the mechanism of action. It's at a step after reverse transcription. So if you have reverse transcripts already, efavirenz is no longer going to be effective until the next round of replication, while raltegravir will, so you clear the RNA faster. But it has no long-term difference in terms of activity; at six months they're identical. This is mythical thinking rather than scientific thinking, but that happens a lot in the field.

Bob: It was dramatic when we saw it.

Doug: It is a dramatic observation. It's an incredible drug, but it isn't going to eradicate, that's all. In terms of treating people, it's remarkable.

Bob: Is the fragility of its resistance profile a problem?

Doug: I don't get the fragility thing. Two of the most fragile drugs we have are the NNRTIs and 3TC or FTC, and they're both components of one of the best regimens we have. But if they are potent, and people take their drugs, there will be no fragility. Those drugs have a low genetic barrier, so if people don't take their drugs properly, and you get replication in the presence of suboptimal concentrations of drug, you get failure with resistance. The number of people who failed with resistance to raltegravir in the study of initiating treatment is very small. Even in the salvage study it was people who had nothing to add to it. That shows what a great drug it is.

Bob: In a clinical trial, why does it take many weeks or months to get, say, 90% of the trial population below 50 copies? What is going on during that period? Is replication continuing?

Doug: I think all new infections are blocked. But you've got a lot of cells that are already infected; that are producing virus; that don't get killed off right away. So the activated T lymphocyte has a half-life of a day or day and a half, but macrophages and nonactivated T cells that are infected are going to get activated and produce virus; the macrophages are constantly producing virus until they die off. But I don't think you're getting new infections because we really don't see much evolution once you start therapy.

Bob: Do you think we will see another generation of ARVs?

Doug: The number of people looking has diminished. And the need is, to some extent, less now that it has ever been; we have more at our disposal that works than we ever had. Could we do even better? Yes. Are there people for whom the drugs are not enough? Sure. But the impetus to find more drugs is diminished in the pharmaceutical industry. The number of companies actively looking is about half of what it was five or ten years ago. But there are some really good companies that are trying to find new drugs.

I think the NNRTI from Idenix looks promising. But I don't see anything else in clinical trials that I'm aware of that is that exciting.

Bob: Do you think you could get more bang for your buck by developing new formulations like a patch or a depot for people who have trouble taking pills consistently?

Doug: There aren't many of those people.

Bob: Even in a clinical trial there's that last 10% who fail.

Doug: There's 10% failure, but it's usually 7 or 8% who are lost to follow up or withdraw from the study. You can't treat those people. In most studies during the last five years, at most 1–2% have drug resistance. A person who fails with drug resistance is a nonadherer. That's a pretty small failure rate. Getting nine out of ten to comply is good. There are enough people with emotional or substance abuse problems for whom antiretrovirals are not the most important issues; those are the limitations in our clinic.

Bob: How do you see HIV treatment guidelines evolving in the United States?

Doug: The guidelines are changing. The data that's accumulating about the earlier initiation of treatment having an impact on non-AIDS-related morbidity and mortality have convinced the leaders about starting earlier. The question is whether you treat everybody or start at 500 instead of 350; it's gone up to that level. The practical issue is that since nobody's been able to implement the CDC guidelines [on testing and referral to care], we're still identifying new patients with low CD4 counts, so it's not a realistic argument for me.

Celsa: What about the developing countries?

Doug: That's a disaster. We're treating three million people with regimens we wouldn't give our own patients. People are getting neuropathy; getting lipodystrophy; lactic acidosis; all the problems you get with thymidine analogs. Resistance is accumulating. We've got great drugs for the United States. If you're going to make a big difference, you take the drugs that are really good and figure out how to get them to the people who need them.

Bob: Under the current conditions for science funding, what is the outlook for a young investigator?

Doug: We have already lost a significant portion of a generation in this country. The young people who are still here, for example, amaze me. I'm not sure that I would have persisted in the face of the obstacles that they have dealt with. It was easier for me as an assistant professor to get a grant, but I don't think I would have had the strength to do it otherwise. And we have all lost a lot of time. I have never been as unproductive as in the last several years . . . because I've been writing grants instead of doing science. A combination of the increased grant writing plus the incredible amount of paperwork that's being generated by bureaucracies that have never existed before has really inhibited creativity. We're losing a generation . . . and some of us need to be replaced. When I go to Europe or Australia or India or China, I can see where the future is. Hopefully there will be a change.

Celsa: But it will take a while to recover. The great thing about science in the U.S. was that it allowed off-the-wall thinking. In other countries where they didn't have the resources, you didn't have that creative thinking.

Doug: That's what's happened here, so that latency research has basically been stymied.

Celsa: Anything that is too innovative or too risky is not being funded. ●

TAG to NIAID: Recommendations to Stimulate Research on HIV Persistence

Treatment Action Group's response to the National Institute of Immunology and Infectious Diseases (NIAID) request for comments on the development of a research funding opportunity on HIV latency and persistence.

BY TREATMENT ACTION GROUP

Treatment Action Group (TAG) strongly supports reinvigorating the research effort to cure HIV infection. As total eradication of HIV from the body would be essentially impossible to prove, at least with the current tools available, we believe that cure should be defined as the long-term absence of detectable HIV replication without the need for ongoing treatment. TAG encourages NIAID to request proposals for conducting goal-oriented, multidisciplinary research that aims to definitively answer key outstanding questions facing scientists attempting to maximize the chance for elimination of HIV from the human body.

It is possible to envision mutually complementary roles being played by academia, the private sector, and the government in this area of research. Academia has generated a number of lines of investigation and continues to do so, while pharmaceutical companies have the appropriate resources to screen compounds when investigators identify potential targets. The government needs to examine the process for approving translational research in humans, as the current process has been blamed for slowing clinical testing of promising leads and inhibiting progress in the field.

TAG is seriously concerned about the need to increase the funding available for the traditional, investigator-initiated (RO1) grant program. This mechanism has a rich history of supporting innovative, breakthrough research and—at least from the perspective of an organization outside of the NIH—it is disheartening to see large sums of money being used to support directed, multicenter research projects at the same time that young investigators are leaving the field due to ever-diminishing RO1 funding. It is vital that study sections responsible for reviewing RO1s be up to date on the importance and current status of research aiming to cure HIV infection, as increasing funding would be a Pyrrhic victory in the absence of mechanisms that can ensure that grant submissions in this area are appropriately reviewed and prioritized.

Cure should be defined as the long-term absence of detectable HIV replication without the need for ongoing treatment.

In terms of specific issues in the field, TAG previously noted the problems associated with relying excessively on highly artificial cell line models ("pristine, beautiful, irrelevant systems," as they have been described), and this remains a significant problem in studies of HIV latency and persistence. The NIH should explicitly encourage the development of models involving primary human CD4 T cells (and other cells that support HIV infection) in order to recapitulate in vivo cell behavior as closely as possible. Another immediate priority should be ensuring that key assays and techniques are standardized in order to ensure that research results can be compared. Examples include assays for the measurement of integrated HIV proviral DNA and techniques for measuring HIV DNA and RNA in tissues. Imaging studies of CD4 T cells, antigen-presenting cells, and human host tissue dynamics in vivo are also a promising and underfunded research area of interest.

Animal models can play an important role in the study of HIV persistence, but NIAID should request that they be used strategically to address the specific questions for which they are best suited. For example, the current simian immunodeficiency virus (SIV) model is not ideal for studying antiretroviral therapy (ART), but can shed light on issues such as the in vivo tissue distribution of virus-infected cells. Priority should also be given to evaluating candidate simian/HIV hybrid viruses (SHIVs), such as those containing HIV reverse transcriptase, which may allow investigators to model suppressive ART in macaques. It would be an enormous benefit to the field if an appropriate SHIV could be developed and made available as a reagent.

Perhaps the holy grail of persistence research is the identification of unique markers that might allow cells containing transcriptionally silent HIV provirus to be identified and targeted. The search for such markers needs to be part of the research effort toward a cure, and comprehensive analyses of gene expression, protein production, and signaling pathways in latently infected versus uninfected cells are needed. Investigators in the burgeoning field of systems biology might be able to provide insights and should be considered for inclusion in multidisciplinary efforts to address HIV persistence.

Since several studies of treatment intensification are ongoing through both investigator-initiated efforts and the AIDS Clinical Trials Group (ACTG), the question of whether traditional ART can reduce HIV reservoirs is therefore being addressed and does not require an additional mechanism of support. ●

Obama, continued from page 1

of people with HIV globally. Even less was spent on hepatitis B and C, two devastating viral infections among people with HIV in the United States. New resources must be committed to the scientific struggle against these diseases.

Funding for the NIH has stagnated during the past five years—a period during which biomedical inflation shrank the value of each research dollar by 20%. The funding drought has hurt all biomedical research, including AIDS research. This puts an entire generation of young scientists at risk of being unable to fulfill their ambitions to make the medical breakthroughs needed to develop better treatments, cures, and vaccines.

TAG recommends that the Obama administration commit to at least five years of 15% year-on-year growth of the overall NIH budget (for fiscal years 2010–2014) in order to regain the momentum achieved between 1998 and 2003, when the NIH budget doubled.

The administration should protect and support a strong Office of AIDS Research at the NIH to ensure that the nation's investment in AIDS and AIDS-related research responds to scientific opportunities and is well-coordinated across the 27 NIH institutes and centers.

Our nation's AIDS research enterprise must balance efforts to achieve the long-term goal of a cure and a vaccine for HIV with research efforts on shorter-term goals, such as better treatment and preventive interventions appropriate for a broad range of settings—including resource-poor developing countries. Intensified research is also needed to understand the impact that mass testing campaigns and earlier treatment could have on the transmission and prevalence of HIV worldwide. Despite the achievement of bringing antiretroviral therapy to over 3 million people with HIV who live in developing countries, 30 million others still lack treatment—and more than half of those do not know they have HIV. We still lack treatment regimens cheap enough, powerful enough, and safe enough to be taken for decades without the need for expensive monitoring to prevent the emergence of drug resistance or dangerous side effects.

Strengthen and Integrate Domestic HIV Prevention and Treatment

According to U.S. Centers for Disease Control statistics, in 2006 about 1.1 million Americans were living with HIV; 56,300 people became newly infected. In the same year, 37,852 people were diagnosed with AIDS and more than 14,000 people who had AIDS died. These stark numbers grimly illustrate that support for HIV prevention, testing, and treatment in this country remains insufficient.

The Obama administration must reverse the growing number of new HIV infections in the United States by implementing a fully funded and comprehensive HIV prevention plan as part of a national AIDS strategy. The prevention plan must include safer-sex education for all ages; distribution of condoms and other barrier methods to prevent sexual transmission; full funding for needle exchange, harm reduction, and addiction treatment programs for drug users; and continuing efforts to protect the nation's blood and organ transplant supplies from HIV and other transmissible diseases.

HIV testing must become a routine and voluntary part of medical care; referrals to affordable treatment must be available wherever HIV testing is carried out. Tighter integration of prevention, testing, and treatment in this country will result in fewer new infections and fewer deaths among those who have HIV.

Special emphasis must be placed on reaching individuals disproportionately affected by HIV in the United States, especially African Americans, gay and bisexual men, Latinos and Latinas, drug users, and the incarcerated. HIV prevention and treatment services are critically lacking for prisoners, detainees, and others incarcerated in state and federal facilities. These services include provision of condoms, clean syringes, and harm reduction education; and care and treatment for HIV, viral hepatitis, and TB.

Strengthen Community Involvement in the Fight against AIDS

President Obama has said that real change comes from the bottom up. We urge the administration to increase support for community-based HIV prevention and service organizations that strengthen community responses to the epidemic—both in the United States and internationally—through mechanisms such as the Ryan White CARE Act and the PEPFAR program. Until there is a cure for AIDS and a vaccine for HIV, the best way to fight the epidemic is by strengthening communities with the information, resources, tools, and treatments they need to prevent HIV transmission and to guarantee the health and rights of all people living with HIV.

Support the Global Fund and Other Key International Organizations

The Obama administration must champion fully funding the Global Fund to Fight AIDS, Tuberculosis, and Malaria—currently facing a $5 billion gap between now and 2010—and keep its promise to contribute our nation's full share of resources needed by the Fund. The administration should also strengthen its collaboration with the World Health Organization; support incoming leadership at UNAIDS; and join UNITAID, a multilateral funding mechanism established to purchase second-line HIV and TB drugs and diagnostics.

Support Reforms at the IMF

The Obama administration must support International Monetary Fund (IMF) reform of debt- and inflation-reduction policies that limit public sector spending for health and education in resource constrained countries. The administration should urge the IMF to improve transparency and access to information about its decision making and include a greater range of stakeholders in developing policies.

TAG is committed to working with the new administration to ensure that the health and human rights of all people are protected and upheld to the highest possible standards. ●

TAG NEW WAYS TO CONTRIBUTE

Supporting TAG is a wise investment in AIDS treatment advocacy. With a small but well-organized and highly respected staff of professionals, every donation to TAG brings us one step closer toward better treatments, a vaccine, and a cure for AIDS.

There are several ways you can support TAG today!

Make a tax deductible gift now
by credit card using our secure website (www.treatmentactiongroup.org) or by calling Joe McConnell at 212.253.7922 to request a donation envelope.

Celebrate!
Expand your support for TAG by asking your friends and family to make a donation in your honor to celebrate your birthday, anniversary, or the holidays. An acknowledgment will be sent to donors, and you will be informed of gifts made in your honor. Please call Joe McConnell at 212.253.7922 to request that materials be sent to friends and family.

Support TAG's Research in Action Awards
Each December, TAG's Research in Action Awards event honors some of the most important scientists, artists, celebrities, and activists working for better treatments, a vaccine, and a cure for AIDS. Past honorees and presenters have included New York State Senator Tom Duane, director and artist John Waters, award-winning playwright Terrence McNally, actor Nathan Lane, and stage and screen actress Kathleen Turner, among many other scientists and dedicated AIDS activists. Join us this December!

Does your company have a matching gifts program?
If so, you can double or even triple the donation you make to TAG. If your company offers a matching gifts program, please complete its matching gift form and send it in with your donation to TAG.

Make a gift of stock to TAG
Gifts of stock benefit TAG and the donor. The donor who purchased the stock at a lower price receives the tax deductible benefit of the stock's price on the day it is transferred to TAG.

For more ways to support TAG, please visit our website at www.treatmentactiongroup.org or contact Joe McConnell at 212.253.7922.

TAG BE INVOLVED

About TAG

Treatment Action Group is an independent AIDS research and policy think tank fighting for better treatment, a vaccine, and a cure for AIDS. TAG works to ensure that all people with HIV receive lifesaving treatment, care, and information. We are science-based treatment activists working to expand and accelerate vital research and effective community engagement with research and policy institutions. TAG catalyzes open collective action by all affected communities, scientists, and policy makers to end AIDS.

Program areas include antiretroviral treatments, basic science, vaccines, prevention, hepatitis, and tuberculosis.

Join TAG's Board

TAG is always seeking new board members. If you are looking for a great place to invest your time and talents, please call Barbara Hughes, TAG board president, to learn more about board opportunities with TAG.

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