

BOSTON CURE PROJECT'S OFFICIAL KICK-OFF!



BCP staff & Dr. John Copeland with celebrities Miss Waikiki & David "Squiggly" Lander

On November 3, 2001, Boston Cure Project held its inaugural event in the atrium of the Carl J. Shapiro Clinical Center at the Beth Israel Deaconess Medical Center. Our attendees included friends, family members, colleagues and others who were interested in hearing about our mission and learning how they could participate in the effort to cure Multiple Sclerosis. The founders of the Boston Cure Project, President & CEO Art Mellor, and Chief Scientist Dr. Timothy Vartanian, presented an overview of the Boston Cure Project and its mission to cure Multiple Sclerosis by determining the cause of MS. Dr. Vartanian also reviewed the status of research on the cause of MS, describing what is currently known and unknown about the disease.

The evening featured celebrity guest speaker David L. Lander (best known for his role as "Squiggly" in "Laverne & Shirley"). David is a veteran actor with scores of film roles, including "A Bug's Life" and "Scary Movie," as well as dozens of television shows, including "The Drew Carey Show" and "Star Trek: the Next Generation."

David was diagnosed with Multiple Sclerosis in 1984, has written a book about dealing with MS entitled Fall Down

Laughing, and has become a popular and engaging speaker on Multiple Sclerosis. In his remarks, David described the effect MS has had on his career and spoke out about the need to find the cause of the disease.

In addition, we were pleased to present a surprise guest speaker – Liane Mark, Miss Waikiki 2001, who was in Boston with David as part of a speaking tour. Liane was diagnosed with MS in February of 2001 and graciously agreed to say a few words to our audience. She spoke about how her diagnosis came as a surprise to her, especially because she had initially chalked up her symptoms to the effects of wearing high heels so often in pageants. Both of our speakers had our guests laughing one moment and very serious the next, hearing about the difficulties and fears they have personally had to overcome in their battles with MS.

The evening's success exceeded our expectations – approximately 170 people attended the event and we raised almost \$12,000. In addition, 70 new people joined our ranks of volunteers. A complete list of everyone who helped out and more pictures of the event are available at www.bostoncure.org in the News/Events section.



Top Left:
Dr. Tim Vartanian speaking about MS

Top Right:
Art Mellor shakes hands with David Lander in foreground

Bottom:
Volunteer Emily hard at work!

LETTER FROM THE PRESIDENT



Welcome to the first issue of Boston Cure Project Update, our quarterly newsletter. We'll be using it to keep you updated on our work to cure Multiple Sclerosis (MS) by determining the cause.

In each issue we will cover some of the work we've been doing, give overviews of topics relevant to MS that have been in the news recently, provide overviews of people and vendors we are working with, and cover events we have held. We will also publish pictures of people wearing their Boston Cure Project T-shirts if you send them to us. If you're going somewhere interesting let us know your T-shirt size and we'll send you one. If you indicate your T-shirt size when making a contribution of \$25 or more we will also send you a T-shirt.

We've just ended our first calendar year of existence. It's been an extremely exciting time and I feel like we've really gotten a lot done. We have the first draft of our genetics document in review, started work on our pathogens document, and have 2 full-time employees, 2 volunteer staff members and a veritable army of on-call volunteers. A world-class group of researchers has joined our scientific and pharmaceutical advisory boards and we continue to recruit more great people. We have set up our operations and secured our tax exempt status as nonprofit corporation. This newsletter is one of the channels we will use to keep you updated on all the progress we make as we go forward.

We often have more to say about a topic than allowed in the limited space the newsletter provides, so we will augment the material presented here with our email lists and web site. You can sign up for our mailing lists and check out our web site at www.bostoncure.org. If you don't have easy access to the web or just prefer a different form of interaction, you can email me at art@bostoncure.org or call me at 781/788-0880 with any questions or comments you wish to share.

Regards,

Art Mellor President & CEO
Boston Cure Project, Inc.

EMPLOYEE PROFILE: ANDII BRIGGS, OPERATIONS MANAGER

NAME/NICKNAME:

Andrea Briggs/Andii

OCCUPATION:

Operations Manager at Boston Cure Project

REASON FOR JOINING BOSTON CURE PROJECT:

- To help Art
- It was an exciting challenge and a great opportunity
- I love the extra reward of working for a good cause
- I know Art's a force of nature when he decides to do something and he has a solid game plan: he might just do it!

LAST JOB/OCCUPATION:

Executive Assistant at THINQ Learning Solutions

CURRENT RESIDENCE:

Wonderful, fun, multi-cultural, beautiful Lowell

HOMETOWN:

King of Prussia, PA; Ventor, NJ; Haddonfield, NJ; Somer's Point, NJ; Ardmore, PA; Villanova, PA; Lompoc, CA... (we moved around a lot!)

PERSON (DEAD OR ALIVE) YOU WOULD MOST WANT TO HAVE DINNER WITH:

God. I have a lot of questions I want answered!

FAVORITE MOVIE:

I LOVE movies! Some of my favorites: Fight Club, Willie Wonka & the Chocolate Factory, Lord of the Rings, Silence of the Lambs, Alfred Hitchcock's Lifeboat, Evil Dead 2, Chocolat, The Matrix, Young Frankenstein, The Lion in Winter, Tampopo, Rocky Horror, The Shawshank Redemption, Star Wars, Raiders of the Lost Ark, Pulp Fiction, Priscilla Queen of the Desert, Miracle on 34th Street (the original, not colorized version, of course!), The Sixth Sense, Aliens, Blade Runner, The Hitcher, The Princess Bride, Terminator, The Exorcist, This Is Spinal Tap, Monty Python & the Holy Grail, Pleasantville, The Man Who Would Be King, and A Christmas Story

LEAST FAVORITE THING:

People who don't try.



OVERVIEW OF BOSTON CURE PROJECT'S CURE MAP

TO CURE MULTIPLE SCLEROSIS, WE MUST FIRST LEARN WHAT CAUSES IT

The mission of the Boston Cure Project is to cure Multiple Sclerosis. We believe that the fastest route to a cure will come from determining the cause or causes of Multiple Sclerosis (MS), because then we will know exactly where to focus our therapeutic research. We further believe that a clearly defined, comprehensive, and continuously refined plan of research is vital to finding the causes of MS in the shortest time. The development and implementation of this plan, which we call the Cure Map, is the main program at the Boston Cure Project.

MULTIPLE SCLEROSIS IS A COMPLEX DISEASE WITH MULTIPLE APPARENT ROOT CAUSES

All diseases are the effects of one or more root causes. Such primary causal factors can be grouped into five categories:

1. Genetics – dysfunction caused by abnormal genes or genetic function
2. Pathogens – bacteria, viruses, and other infectious agents
3. Toxins – poisonous substances
4. Nutrition – improper balance of necessary nutrients
5. Trauma – physical injury or mental stress

Many diseases are caused by a single, often obvious, factor such as a single defective gene, a particular type of bacterium, a polluting poison, lack of a certain nutrient, or an explicit injury. The individual causes of these diseases are usually identified relatively easily and the development of a cure can progress in a straightforward manner. However, Multiple Sclerosis appears to be one of the many complex diseases that has multiple causal factors acting in concert. Conventional methods to isolate an individual root cause have been attempted but with no conclusive results. Instead of a single cause for MS, it appears that both a genetic pre-disposition and a triggering or aggravating environmental component are necessary to cause symptoms. The fact that the causes of MS are complex and therefore more time-consuming to discover makes it all the more critical for research to proceed according to a logical and comprehensive strategy.

THE CURE MAP STRATEGY IS BASED ON SYSTEMATICALLY LISTING AND INVESTIGATING ALL POSSIBLE CAUSES OF MULTIPLE SCLEROSIS

In order to determine the causes of a complex disease, it is best to have a comprehensive list of all possible causes of disease, along with an investigative protocol to determine if a particular cause is playing a role in the disease. It would then be possible to systematically test the role of each possible cause for a given disease and either include or exclude it as a contributing factor. After testing every possible cause (or at least a sufficient number to be able to definitively explain the disease), the causes would be known and methods of prevention, repair, and treatment could be developed.

Unfortunately, though, such a list does not exist. The number of possible disease causes is enormous and not all of them are known, so to list them all individually would be a practical impossibility. Also, the means of determining the roles of the causes we do know about are often technically difficult, resource intensive, or not possible for ethical reasons.

Yet, despite such limitations, this basic approach of listing and then checking each possible cause is still the soundest way we currently know to systematically determine the causes of a complex disease. Our plan is to categorize the known root causes of the disease and then analyze previous MS research results to determine what is already known about each possible cause for MS. From this point, we will develop a strategy for further research in the most promising areas based on our recognition of encouraging patterns and relationships, as well as identifying holes in our knowledge that need to be filled.

THE CURE MAP EXECUTES THIS METHODOLOGY IN FOUR PHASES

The Boston Cure Project Cure Map will be developed and executed in five tracks: one for each causal category (genetics, pathogens, toxins, nutrition, and trauma). Each track is implemented in four phases:

Phase 1: Develop a systematic description of each root cause category

Phase 2: Review the MS literature as it applies to the systems developed in Phase 1

Phase 3: Determine promising areas of research and how to address them

NEURAL STEM CELLS:

POTENTIAL TOOLS FOR PROMOTING REPAIR AND REGENERATION IN MULTIPLE SCLEROSIS

Stem cell research has attracted an enormous amount of attention in recent years because of its therapeutic potential in human diseases. Stem cell research may lead to effective new treatments for multiple sclerosis and thus it is prudent to describe what stem cells do, how these therapies might work and what further research is required in this area.

STEM CELLS GENERATE OTHER CELLS

Most cells in the body are specialized, having distinctive characteristics to perform specific functions. A neuron must be and is different from a skin cell, which is different from a liver cell, a muscle cell, and so on. Every specialized cell in the body, from neurons to skin cells, is ultimately derived from a progenitor or stem cell, that is less specialized. When a progenitor can give rise to two or more types of cells (e.g., neurons and oligodendrocytes), it is said to be multipotent. If the progenitor cell also can divide into more progenitor cells as well, then it is said to be self-renewing. A *stem cell* is simply a cell with both of these capabilities – it can divide to form more than one differentiated cell type and it also can divide to create more stem cells.

Stem cells are defined both by the tissue they belong to and the number and types of different cells they can produce. In general, the earlier the stage of development from which the stem cell is derived, the greater the number of cell types that it can form. For example, early in embryonic development, embryonic stem cells are formed that can contribute to every tissue and cell type in the body. As development proceeds and organs form, the earliest cells that compose each organ are, in general, capable of giving rise to the various cell types within that organ but not those of other organs (these are known as tissue restricted stem cells). Similarly, in adults, stem cells or progenitor cells exist in body tissues that can give rise to multiple cell types for those tissues. For instance, in the central nervous system (CNS), neural progenitor cells or neural stem-like cells are cells derived from the nervous system that may generate the various CNS cell types. In the earliest stages of CNS development, neural tube stem cells can give rise to all of the major CNS cell types (neurons, astrocytes and oligodendrocytes). As development proceeds, the stem/progenitor cells become more restricted in the types of cells they can produce.

PROPOSED THERAPIES FOR MS INVOLVE REGENERATING DAMAGED STRUCTURES AND DELIVERING SIGNALING MOLECULES

In scientific investigation, we study stem cells not only to further our understanding of how living beings develop but also to see how they could be used as potential therapeutic

agents. Two types of therapeutic uses of stem cells have been identified for diseases such as multiple sclerosis.

The first is using stem cells to regenerate structures that were injured in the disease. In MS, for instance, stem cells might be used to replenish absent or diminished numbers of oligodendrocytes, which are the myelin-forming cell of the CNS. The goal would be that these newly integrated oligodendrocytes would then regenerate the myelin sheath that surrounds axons and that is critical to neural function but is damaged or destroyed in MS lesions. Similarly, stem cells that differentiate into neurons could conceivably be induced to bridge severed axons in MS lesions, thereby filling in the gaps that the disease has caused in the central nervous system. The fact that stem cells are able to integrate into neuronal circuitry after injury makes this approach feasible.

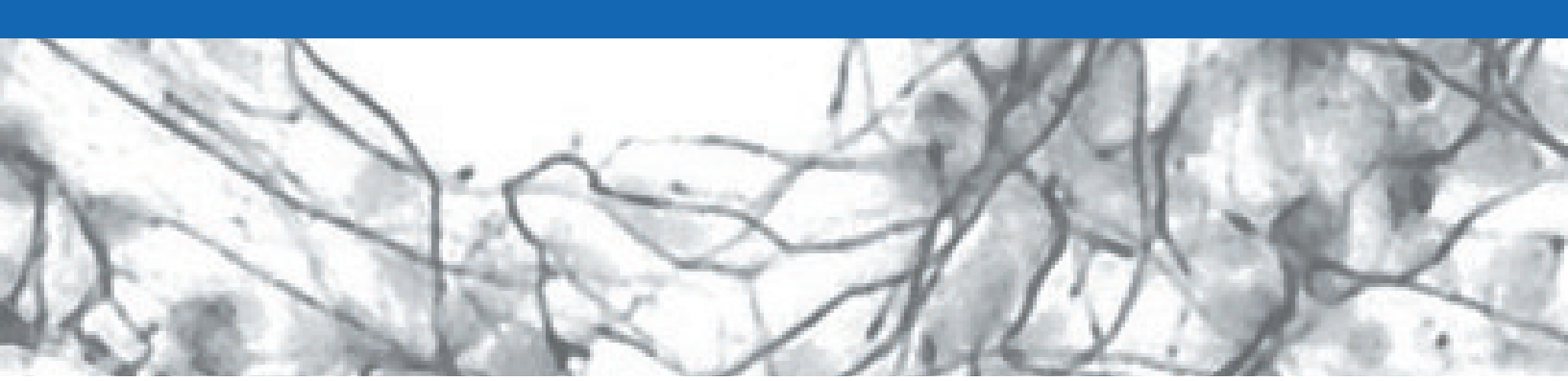
The second potential therapeutic use of stem cells is to deliver one or more therapeutic molecules into the CNS. For example, if the absence of remyelination in MS is due to the absence of needed chemical signals to the oligodendrocyte, then a genetically engineered stem cell could potentially deliver the appropriate signaling molecules, inducing remyelination of damaged nerves.

On the surface, these therapeutic strategies may seem straightforward, but in reality the complexities of the injured nervous system pose several difficult obstacles between introduction of the stem cell into the CNS and repair of damaged cell structures. Before discussing those obstacles, we need to understand how stem cells differentiate into the appropriate cell types at the right time and in the right place.

RECONSTRUCTION (REGENERATION) OF THE INJURED CENTRAL NERVOUS SYSTEM IS A FOUR-STEP PROCESS

In normal development, stem cells respond to local cues that direct their migration and differentiation such that the correct cells form in the relevant environment at a relevant time. In the central nervous system this is important both for normal neuronal circuitry to form and also to make sure that major developmental epochs follow the correct sequence – for example, cells that produce myelin are needed only after axons and synapses are already formed and in place. If myelinating cells formed prior to generation of axons, axonal growth would likely be inhibited.

In general, there are four major steps that a stem cell must accomplish in order for regeneration to occur. (Other events are necessary as well but these are the most important to understand.)



The stem cell must develop or differentiate into the proper cell type(s). For example, in Parkinson's disease the goal is for stem cells to regenerate into the dopamine-producing neurons in the part of the brain (the substantia nigra) that is degenerated in this disease. In multiple sclerosis, the goal is for stem cells to differentiate into oligodendrocytes and neurons.

The stem cells themselves or the cells they differentiate into must migrate to a location relevant for repair. Even if cells are injected directly into a damaged region as in stroke, they must remain localized to that site to do their work. In multiple sclerosis, cells must migrate to multiple lesion sites throughout the central nervous system.

Once localized to the site of injury or lesion, cells must then further differentiate or mature to form the specialized cell structures that allow the system to function normally. In spinal cord injury, for example, damaged axons and neurons need to be replaced by regenerated neurons and axons from the inserted stem cells. Furthermore, it is not enough for grafted cells to differentiate into just any neuron, they must also differentiate into the correct neuron for the job. A neuron that recognizes dopamine as a neurotransmitter would be useless in cellular environments that use acetylcholine. In multiple sclerosis, where remyelination is one of the major tasks, cells that have migrated to lesions must differentiate into oligodendrocytes and begin the process of forming myelin.

In addition to correctly differentiating, migrating, and forming structures, these cells must also find their appropriate targets. Using spinal cord injury as an example again, it is clear that destruction of motor pathways from the brain to the spinal cord have to be reconstructed with proper left/right symmetry and organization of the neurons controlling upper and lower limbs and the torso (somatotopic organization). It would be potentially disastrous if the rewiring of the central nervous system was inaccurate.

REGENERATING DAMAGED TISSUE IN MS IS COMPLICATED BY THE NATURE OF THE DISEASE AND THE FUNCTION OF STEM CELLS

In addition to performing these important development steps, regeneration of damaged tissue requires a stem cell to also overcome significant obstacles. Most research efforts investigating the usefulness of stem cells in regeneration take advantage of receptive tissue environments such as newborn

animals or lesions with no scarring or destructive inflammation. This is routine and appropriate in a line of scientific investigation that is relatively new because it allows scientists to assess the utility of an intervention under the best of circumstances. In multiple sclerosis, however, the environment is not as ideal. The evolution of a lesion involves not only destruction of myelin and some axons but also the formation of an astroglial scar. Such scars inhibit both the migration of new cells into the lesion and the elongation of axons or myelin membranes from cells within the lesion. So one major question is whether the tissue environment in MS is receptive to the growth and development of neurons and their axons, and oligodendrocytes and their myelin. Relatively little research is currently being performed on scar formation in the CNS; therefore, this question is far from being answered.

A second important obstacle relevant to MS is that inserting stem cells into multiple lesions located throughout the brain and spinal cord may prove technically difficult. Targeting stem cells to lesions requires making use of both the migratory potential of stem cells and a relevant attractant signal within a lesion. If either the signal to attract stem cells is weak or the migra-

tory potential of the particular stem cells used is limited, then successful engraftment is unlikely. Physically targeting stem cells to lesions through microinjection has limitations due to invasiveness and tissue injury, but this may change with further development of micro-device technology.

A third limitation of stem cell technology is the potential for creating tumors. Because stem cells have an enormous capacity for cell division, introducing these cells in a time and place where the normal limits on their growth are absent may lead to uncontrolled division, thus creating abnormal new growths or even malignant tumors. In addition, stem cells are cultured in dishes (in vitro) and are thus subject to chromosomal alterations that many cells undergo after multiple divisions in a foreign environment; these alterations may themselves produce harmful effects in recipients.

The fourth obstacle relevant to MS is that introduction of stem cells does not (presumably) alter the underlying mechanism of injury to myelin, oligodendrocytes and axons in MS. Therefore regeneration of damaged cells may not be a permanent solution because the newly regenerated cells may be subject to the same type of damage as the original cells.

It is clear that due to their regenerative role, flexibility and power, stem cells hold great potential for treating diseases involving cell and tissue damage such as MS.

NEURAL STEM CELLS:

POTENTIAL TOOLS FOR PROMOTING REPAIR AND REGENERATION IN MULTIPLE SCLEROSIS

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THERAPIES INVOLVING DELIVERY OF MOLECULES VIA STEM CELLS MAY BE EFFECTIVE IN TREATING MS

A possibly more effective use of stem cells in MS would be the introduction of molecules that promote the survival of neurons and oligodendrocytes and the regeneration of myelin and axons. This notion was highlighted by a recent report that many MS lesions have premyelinating oligodendrocytes in contact with axons that apparently halted their repair work at the stage between contact with axons and formation of myelin (see Chang et al., 2002, below). This suggests that the relevant molecular signals for myelination are absent. Once these signals are identified, genetically engineered stem or other cells could be used to deliver these molecules.

STEM CELLS SHOW GREAT PROMISE, BUT MUCH WORK IS STILL NEEDED

It is clear that due to their regenerative role, flexibility and power, stem cells hold great potential for treating diseases involving cell and tissue damage such as MS. However, many questions still need to be resolved before this potential can be realized. *For more information on the concepts discussed here, we invite you to refer to the resources listed below, or go to www.bostoncure.org for a more extensive list of resources.*

FURTHER READING

Chang, A., W.W. Tourtellotte, R. Rudick, and B.D. Trapp. 2002. Premyelinating oligodendrocytes in chronic lesions of multiple sclerosis. *N Engl J Med.* 346:165-173.

Duncan, I.D., W.E. Grever, and S.C. Zhang. 1997. Repair of myelin disease: strategies and progress in animal models. *Mol Med Today.* 3:554-561.

Herrera, J., H. Yang, S.C. Zhang, C. Proschel, P. Tresco, I.D. Duncan, M. Luskin, and M. Mayer-Proschel. 2001. Embryonic-derived glial-restricted precursor cells (GRP cells) can differentiate into astrocytes and oligodendrocytes in vivo. *Exp Neurol.* 171:11-21.

Macklis, J.D. 2001. Neurobiology: New memories from new neurons. *Nature.* 410:314-315, 317.

Snyder, E.Y., and J.D. Macklis. 1995. Multipotent neural progenitor or stem-like cells may be uniquely suited for therapy for some neurodegenerative conditions. *Clin Neurosci.* 3:310-316.

Snyder, E.Y., and A.L. Vescovi. 2000. The possibilities/perplexities of stem cells. *Nat Biotechnol.* 18:827-828. [taf?file=/nbt/journal/v818/n828/full/nbt0800_0827.html](http://www.nature.com/taf/DynaPage.taf?file=/nbt/journal/v818/n828/full/nbt0800_0827.html)

OVERVIEW OF BOSTON CURE PROJECT'S CURE MAP

Continued from Pg. 3

Phase 4: Execute experiments to include/exclude specific causes

This 4-step process can be applied to any disease with unknown and possibly multiple causes. Because the focus of the Boston Cure Project is MS, our efforts will target the causes of MS. However, the work we do in Phase 1 and in developing our methodology can be used for any disorder.

THIS IS A BIG TASK, BUT IT CAN BE DONE PIECE-BY-PIECE

The Boston Cure Project is taking on a significant task. However, this task can be accomplished by structuring the solution correctly. The key is to be driven by a clearly defined goal: to cure MS through determining the cause. This ultimate objective is motivated by the personal desire of the founders – one who has MS and one whose brother has MS – and the personal desires of our volunteers, many of whom are affected by MS either directly or through a loved one.

We at the Boston Cure Project derive inspiration from other similarly goal-based efforts that have accomplished tasks considered impossible when they were first considered: the Apollo Program, the Manhattan Project, and the Human Genome Project, to name a few. They all share the characteristic of having a specific goal to be achieved, in spite of that goal's difficulty.

In tackling the problem of determining the causes of MS, we will lay the groundwork for determining the causes of many other diseases. Our processes, methodologies, and infrastructure can be used by other efforts to determine the causes of other diseases and we will make our research, expertise, technology, and results available to anyone working on curing disease.

This is a digest version of the original article, which can be viewed in its entirety on www.bostoncure.org, at the bottom of the "What We Do" section.

T-SHIRT PICTURES

WANT A BOSTON CURE PROJECT T-SHIRT?

Visiting an exotic (or not so exotic) locale? If you offer to take a picture at your destination with a Boston Cure Project T-shirt on, we'll send you one for free!



Sandra in beautiful Paris!



Art & Deb on their honeymoon in Iceland



Andii & husband John Copeland volunteer at local church

VENDOR PROFILE: CLOCKWORK DESIGN GROUP, INC.

Founded in 1994, Clockwork Design Group is a graphic design and advertising agency that emphasizes personal service, quality work and creative solutions. At Clockwork, we help businesses grow by enhancing and unifying their image. To do this, we create a visual identity that reflects the unique benefits and features of our client's products and services.

Our clients span a broad range of industries, including high-tech, legal, financial, real estate, construction, publishing, consulting, telecommunications, and retail. Typical projects include corporate brochures, direct mail, catalogs, ad campaigns, annual reports, trade show signage, logo design and corporate collateral, and website design.

We are pleased to have volunteered our services to Boston Cure to create their logo design and the design and layout of this newsletter.

For more information, please visit www.cdgi.com, or call Vanessa or Sandra at 781-938-0006.



From the left: Mike, Vanessa (with Zora), Chad and Sandra



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Have you moved?
Changed your email
address? Let us know!
Send changes in
contact information to
info@bostoncure.org
or give us a call at
781-788-0880!

We've Moved! Boston Cure Project moved in December, 2001 to the Gold Wire Technology offices in Waltham, MA. Our mailing address is unchanged (13 Belton St., Arlington, MA 02474). Our most up-to-date phone numbers are:
Phone: 781-788-0880 * Fax: 781-788-8118

CONTRIBUTE TO BOSTON CURE PROJECT:

By Check: make checks payable to Boston Cure Project, Inc., and mail to:

Boston Cure Project,
13 Belton St.
Arlington, MA 02474

By Credit Card: on www.bostoncure.org, click "Donate/Volunteer," click "Donate Money," and follow instructions under the heading "Donations by Credit Card."

Volunteer Today: See www.bostoncure.org for volunteer opportunities or contact our volunteer coordinator Debbie Burrage (781-788-0880 * dab@bostoncure.org).

Want a Boston Cure Project T-Shirt? For any donation of \$25 or more, we will send you a t-shirt upon request. If you offer to send us a picture of yourself in one of our t-shirts at some unusual locale, we'll send you one for free! **Please remember to indicate t-shirt size when making your request.**

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Have you moved? Changed your email address? Let us know! Send changes in contact information to info@bostoncure.org or give us a call at 781-788-0880!

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This newsletter available online at www.bostoncure.org, in the "News & Events" section.