Industrial lithium–sulphur firms are also bullish. At PolyPlus, Visco says that the firm's protective lithium material is advancing batteries that cycle lithium ions through sea water — although not for many cycles. Other companies working hard include Sion Power in Tuczon, Arizona (now partly owned by German chemical firm BASF), and Oxis Energy, at the Culham Science Centre in Abingdon, UK.

Last year, these three firms separately announced a total of US$90 million in new investment, of which some $15 million came from US federal grants. Another funding boost will come from a new US Department of Energy hub — the Joint Center for Energy Storage, based at Argonne — which aims to make batteries five times more powerful and 80% cheaper within five years. It has $120 million to give out in research grants, but is not specifically focused on lithium–sulphur.

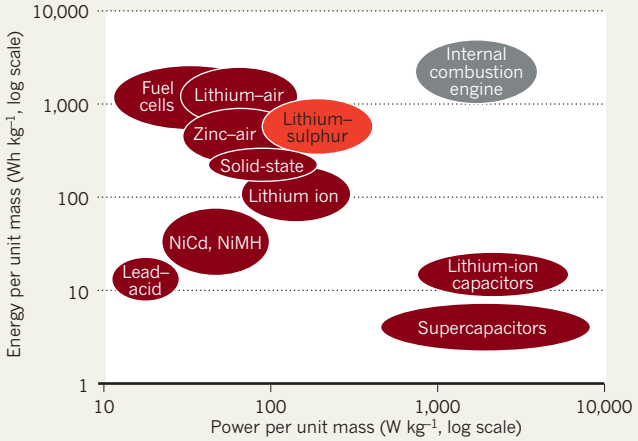
First sales of prototype batteries are likely to be to military and defence markets, for use in such applications as military drones, where the high energy densities and relative safety are more important than the thousands of charge cycles a commercial electric car requires. Researchers do not expect to see a commercial lithium–sulphur battery before the end of the decade. Clever nanostructuring that works in the lab may be too expensive as an industrial proposition. And the field suffers from hype — with academic discoveries often blurring key details, such as battery currents, to highlight one or two impressive numbers. "We need a concerted effort to make sure cells are standardized, so that we are comparing the same things when reporting data," says Nazar.

Nonetheless, sulphur is edging ahead of other futuristic battery designs, such as the high-energy-density lithium–air battery — in which lithium ions bind to oxygen sucked in from the atmosphere. There, the ions are hard to recover for recharging and tend to be poisoned by other atmospheric gases.

In that field, says Nazar, scientists are still finding out about the problems. ■

HOW BATTERIES STACK UP

No battery can beat the energy density and power of petrol (gasoline) — but lithium–sulphur batteries would outperform lithium-ion ones.



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INFECTIOUS DISEASE

Bid to cure HIV ramps up

Clinical trial will aim to replicate virus-expunging therapy that worked in US infant.

BY ERIKA CHECK HAYDEN

HIV-positive mothers who take antiretroviral therapies while pregnant can be prevented from transmitting the virus to their babies 99% of the time — a resounding success story in the decades-long fight against the virus. But what about infants whose mothers do not receive the drugs? Energized by the case of the 'Mississippi baby' — who seemed to be cured of HIV after aggressive treatment was begun within hours of birth — researchers are hoping to show that these infants, too, can get off to a healthy start.

At a symposium on HIV cure research on 29 June at the International AIDS Society's biennial meeting in Kuala Lumpur, Malaysia, investigators will describe how they are racing to design a clinical trial to test whether the early treatment works, and why. They hope to treat the first babies by the end of this year.

The trial, sponsored by the International Maternal Pediatric Adolescent AIDS Clinical Trials (IMPAACT) Group, marks a change for the field: so far, most research worldwide has focused on adults. In 2012, the US National Institute of Allergy and Infectious Diseases in Bethesda, Maryland, spent US$18 million on HIV cure research in adults and adolescents, and just $45,000 on children. Yet 3.3 million children worldwide have HIV.

"Children have been an afterthought," says Jeffrey Safrit, director of clinical and basic research for the Elizabeth Glaser Pediatric AIDS Foundation, who is based in Los Angeles, California. "But the immune system of the child might be more easily manipulated to allow a cure."

This was highlighted in March, when virologist Deborah Persaud of the Johns Hopkins Children's Center in Baltimore, Maryland, announced that a baby in Mississippi who received treatment for HIV within 31 hours of birth stopped medication at 18 months without the virus rebounding (see *Nature* http://doi.org/m2d; 2013). Researchers knew that early treatment could help infants to control HIV, but were surprised that they could essentially wipe it out from an infant's body using existing drugs.

Early HIV treatment is helpful for patients of any age. It stops the virus from replicating before it can infect central memory T cells, the main immune-cell reservoir where HIV 'hides' from drugs. But researchers think that babies are better targets for HIV cures than adults because of their immature immune systems, which respond more mildly when provoked. Because the cells involved in this 'inflammatory response' are the same ones that are ▶

GIDEON MENDEL/CORBIS FOR UNICEF

A baby in Lesotho is given anti-HIV drugs at birth.

most susceptible to HIV, this could mean that infants are less prone to the infection spreading. Moreover, babies are born without central memory T cells, so they are likely to have a smaller reservoir of infected cells, says Mike McCune, an immunologist at the University of California, San Francisco.

The IMPAACT study, to be conducted across some of the group's 71 sites worldwide, will screen and treat hundreds of babies to find 20–30 infants who have acquired HIV from untreated mothers or from those whose HIV was not well controlled during pregnancy. Because diagnosing HIV takes up to 7 days, all screened babies will automatically receive a similar treatment to the Mississippi baby: a cocktail of three drugs within 48 hours of birth. Physicians will add a fourth drug if babies then test positive for HIV. Around the age of three, the 20–30 children will be tested to see whether their immune systems make antibodies to HIV or if it can be detected in their blood. Those testing negative on both counts would then be taken off the drugs to see whether they can remain HIV-free.

The practical and ethical challenges of the trial are significant. Babies of untreated HIV-positive women have only a 15–30% chance of infection at birth, so the trial will need to recruit many babies to try to cure the few who are infected. Those who do not contract HIV will be treated anyway, perhaps exposing them to drug side effects. These are usually mild, but can deplete certain blood cells.

But children born to untreated HIV-positive women are already given up to three drugs after birth as a precaution. The potential for finding a cure far outweighs the risks of adding another drug, or of stopping treatment to test whether the cure has worked, says Yvonne Bryson, a physician at the Mattel Children's Hospital at the University of California, Los Angeles, and co-chair of the trial. "There's much more benefit to be gained than risk," she says.

Physicians are already considering changing the way they treat children infected by the virus. Bryson says that families of HIV-positive teenagers who were treated soon after birth and kept on medication are now asking that the teens be taken off the drugs.

Ultimately, the 34 million people worldwide who live with HIV could also benefit, researchers say. If it turns out that infants are more amenable to cures because they have a less active inflammatory response, that might encourage physicians to prescribe treatments that are less likely to trigger inflammation in adults, McCune says.

Bryson, who has worked on HIV since the first case was detected, thinks that the end could be in sight. "I saw patient zero," Bryson says. "I've always been so excited to think that I would see the day that we would arrive at a cure, and I think we're here." ■

LEAH FASTEN

Hugh Rienhoff prepared his daughter's DNA for sequencing at home using second-hand equipment.

PERSONAL GENOMICS

Father's genetic quest pays off

Mutation provides clue to daughter's undefined syndrome.

BY BRENDAN MAHER

Hugh Rienhoff says that his nine-year-old daughter, Bea, is "a fire cracker", "a tomboy" and "a very sassy, impudent girl". But in a forthcoming research paper, he uses rather different terms, describing her hypertelorism (wide spacing between the eyes) and bifid uvula (a cleft in the tissue that hangs from the back of the palate). Both are probably features of a genetic syndrome that Rienhoff has obsessed over since soon after Bea's birth in 2003. Unable to put on much muscle mass, Bea wears braces on her skinny legs to steady her on her curled feet. She is otherwise healthy, but Rienhoff has long worried that his daughter's condition might come with serious heart problems.

Rienhoff, a biotech entrepreneur in San Carlos, California, who had trained as a clinical geneticist in the 1980s, went from doctor to doctor looking for a diagnosis. He bought lab equipment so that he could study his daughter's DNA himself — and in the process, he became a symbol for the do-it-yourself biology movement, and a trailblazer in using DNA technologies to diagnose a rare disease (see *Nature* **449,** 773–776; 2007).

"Talk about personal genomics," says Gary Schroth, a research and development director at the genome-sequencing company Illumina in San Diego, California, who has helped Rienhoff in his search for clues. "It doesn't get any more personal than trying to figure out what's wrong with your own kid."

Now nearly a decade into his quest, Rienhoff has arrived at an answer. Through the partial-genome sequencing of his entire family, he and a group of collaborators have found a mutation in the gene that encodes transforming growth factor-β3 (TGF-β3). Genes in the TGF-β pathway control embryogenesis, cell differentiation and cell death, and mutations in several related genes have been associated with Marfan syndrome and Loeys–Dietz syndrome, both of which have symptomatic overlap with Bea's condition. The mutation, which has not been connected to any disease before, seems to be responsible for Bea's clinical features, according to a paper to be published in the *American Journal of Medical Genetics*.

Hal Dietz, a clinician at Johns Hopkins University School of Medicine in Baltimore, Maryland, where Rienhoff trained as a geneticist, isn't surprised that the genetic culprit is in this pathway. "The overwhelming early hypothesis was that this was related," says Dietz, who co-discovered Loeys–Dietz syndrome in 2005.

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For more on Hugh Rienhoff's genetic search, see:
go.nature.com/qvvtks