The Bayer Hemophilia Awards Program (BHAP) was initiated in 2002, with the first awards being presented in 2003. Since then, the program has supported many important projects from around the world.

The graph below shows the impact of the program in terms of the numbers of abstracts, full papers and other outputs published per annum since its inception. It clearly shows how the impact of the program is increasing over time (the figures are annual, not cumulative).

BHAP awardees published 19 papers in 2014 in a variety of peer-reviewed journals (selected papers shown below; awardee names in bold)

- Sack BK, … Markusic D. Immune responses to human factor IX in haemophilia B mice of different genetic backgrounds are distinct and modified by TLR4. Haemophilia 2015; 21(1): 133–139.
The program’s awards are available in five categories

There are five different awards currently available. Awards categories may change for the 2015 / 2016 cycle, so please check the website (www.bayer-hemophilia-awards.com) for updates before applying. They offer treaters, researchers and allied healthcare professionals working in hemostasis/hemophilia various options tailored to their aspirations and career backgrounds:

**Outcomes Research Award**

The award is intended to facilitate the development of outcomes research in hemophilia. Examples of the type of projects that might be considered for this award include, but are not limited to, those related to:

- use of different clinimetric instruments to assess bleeding disorders and the outcome of therapeutic interventions
- patient-reported outcomes and patient preferences
- quality of life.

- Duration of award: 1–2 years
- Number of new awards per year: 1
- Award: up to US$25,000 per year
- Any individual involved in hemostasis/hemophilia research, treatment or care may apply

**Fellowship Project Award**

This is a mentored award that is intended to facilitate the development of clinical and research expertise in the field of hemophilia for applicants who have completed medical training and have an interest in pursuing a career as a hemophilia clinician.

- Duration of award: 2 years
- Number of new awards per year: up to 4
- Award: up to US$80,000 per year
- Applicants must have received their medical degree less than 8 years ago

**Special Project Award**

This award supports a wide variety of clinical and/or basic research projects. These may be undertaken by any individual affiliated with a facility providing care to people with hemophilia.

- Duration of award: 1 or 2 years
- Number of new awards per year: up to 4
- Award: up to US$100,000 per year
- Any individual involved in hemostasis/hemophilia research, treatment or care may apply

**Early Career Investigator Award**

This award provides salary support and research funding for a junior faculty member to undertake a clinical or basic research project in the field of hemophilia or related bleeding disorders, under the guidance of a mentor.

- Duration of award: 2 years
- Number of new awards per year: up to 4
- Award: up to US$100,000 per year
- Applicants must have received their last academic degree less than 10 years ago

**Caregiver Award**

This award is designed to promote the essential role of caregivers and allied healthcare professionals involved in the care of patients with hemophilia.

- Duration of award: 1 year
- Number of new awards per year: up to 6
- Award: up to US$25,000 per year
- Medically-qualified hematologists are not eligible

For full details of the award categories and the full eligibility criteria, please consult the BHAP website:
www.bayer-hemophilia-awards.com
Bayer’s commitment to hemophilia and hemostasis

The BHAP began in 2002 and since its inception has pledged grants totaling over US$31 million to researchers, treaters and other healthcare professionals working in hemophilia.

Bayer believes the awards program will enable healthcare professionals currently working in hemostasis to push forward the scientific understanding of diseases such as hemophilia.

Furthermore, the program will provide an opportunity for the treatment of patients to be improved, and provide attractive educational opportunities to allied healthcare professionals such as nurses and physiotherapists.

One of the key aims of the program is to encourage younger physicians to pursue a career as a hemophilia treater, to ensure that the next generation of patients has access to high-quality care.

A unique aspect of the awards program is its global nature. Bayer encourages applications from all around the world. To date, applicants from 32 different countries have received an award.

In 2015, the program made its first awards to Mauritius and Zambia.

Green indicates countries where awards have been presented.
Research focus

The program’s research priorities for the 2015/2016 cycle are as follows:

**Patient-related**
e.g. comorbidities, inhibitors, joint disease

**Treatment-related**
e.g. new products, regimens, gene therapy

**Mechanistic**
e.g. molecular mechanisms, experimental models

**Outcomes research**
e.g. patient-reported outcomes, quality of life

Research priorities may change for the 2015 / 2016 cycle, so please check the website (www.bayer-hemophilia-awards.com) for updates before applying.

The Grants Review and Awards Committee – bringing external scrutiny to the program

In order to ensure that the awards program is completely independent, Bayer has appointed a group of eminent healthcare professionals working within hemostasis to oversee the appraisal process. The role of the Grants Review and Awards Committee (GRAC) is to fully review each Letter of Intent and Full Proposal and to determine the suitability of each application for funding. The GRAC is an independent body and Bayer personnel have no input into the judging process.

Finally, a consensus score for each application is agreed upon and the applications that score highly enough attract funding. Funding occurs on a regional basis.

Current worldwide membership of the GRAC

<table>
<thead>
<tr>
<th>Name</th>
<th>Location</th>
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<tbody>
<tr>
<td>Alok Srivastava, MD (Chairman)</td>
<td>Vellore, India</td>
</tr>
<tr>
<td>Valder Arruda, MD, PhD</td>
<td>Philadelphia, PA, USA</td>
</tr>
<tr>
<td>Craig Kessler, MD</td>
<td>Washington, DC, USA</td>
</tr>
<tr>
<td>David Lillicrap, MD</td>
<td>Kingston, ON, Canada</td>
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<tr>
<td>Pier Mannucci, MD</td>
<td>Milan, Italy</td>
</tr>
<tr>
<td>Michael Nichol PhD</td>
<td>Los Angeles, CA, USA</td>
</tr>
<tr>
<td>Johannes Oldenburg, MD, PhD</td>
<td>Bonn, Germany</td>
</tr>
<tr>
<td>Andrea Pritchard, PhD, RN, MN</td>
<td>Calgary, AB, Canada</td>
</tr>
<tr>
<td>Brenda Riske, MS, MBA, MPA</td>
<td>Denver, CO, USA</td>
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<tr>
<td>Midori Shima, MD, PhD</td>
<td>Nara, Japan</td>
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<tr>
<td>Marijke van den Berg, MD, PhD</td>
<td>Utrecht, The Netherlands</td>
</tr>
<tr>
<td>Gilbert White II, MD</td>
<td>Milwaukee, WI, USA</td>
</tr>
<tr>
<td>Riitta Lassila</td>
<td>Helsinki, Finland</td>
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</table>

Please see the BHAP website for more details: www.bayer-hemophilia-awards.com
Guide to the application process

The BHAP uses the Internet and email as its application platform. The purpose of this is to give the largest possible number of applicants an opportunity to submit an application. If you have any queries about the program, please feel free to email the Program Administrator at programadministrator@bayer-hemophilia-awards.com. Applications should also be sent to this email address.

Letter of Intent

There are two key steps in the application process. The first is the completion and submission of a Letter of Intent (LOI). The LOI is a short form that asks for key information, including biographical details of the applicant and, in the case of some awards, their mentor. The proposed project, program or activity should also be described in 500 words or fewer.

You can download LOIs for each award category from the awards website all year round (see www.bayer-hemophilia-awards.com). Furthermore, LOIs can be submitted all year round, although they will only be reviewed for the 2015/2016 cycle after November 2015.

The GRAC (see page 4) reviews each LOI and assigns each a numeric score. LOIs are then ranked by score and region, and for those exceeding the threshold score, the applicants will be asked to submit a Full Proposal.

Full Proposal

Full Proposals are reviewed by at least two members of the GRAC, selected for their familiarity with the subject matter of the application. As well as providing a numeric score for each Full Proposal, GRAC members will also provide written comments, which are available to applicants if they wish to see them.

The GRAC looks favorably on applications that are accurately budgeted. Proposed equipment purchases and time allocations should be commensurate with the scope of the project. A detailed budget should be provided with an explanation of all associated costs. We strongly advise applicants to include letters of support from their mentors and letters of agreement from their co-investigators.

Funding decisions are taken at a yearly meeting of the GRAC and the results of the review process and funding decisions are communicated to applicants after the meeting. The decision of the GRAC is final and is not subject to appeal or discussion.

The timelines for the 2015/2016 Bayer Hemophilia Awards are shown below.

<table>
<thead>
<tr>
<th>LOI submissions</th>
<th>Permitted throughout the year</th>
</tr>
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<tbody>
<tr>
<td>Deadline for consideration</td>
<td>November 30, 2015</td>
</tr>
<tr>
<td>GRAC reviews LOIs</td>
<td>December 2014 / January 2016</td>
</tr>
<tr>
<td>GRAC informs applicants</td>
<td>January 31, 2016</td>
</tr>
<tr>
<td>Applicants develop Full Proposals</td>
<td>February/March 2016</td>
</tr>
<tr>
<td>Full Proposals submitted</td>
<td>March 2016</td>
</tr>
<tr>
<td>GRAC reviews Full Proposals</td>
<td>April/May 2016</td>
</tr>
<tr>
<td>Applicants notified of GRAC decisions</td>
<td>May/June 2016</td>
</tr>
<tr>
<td>Funding available</td>
<td>July/August 2016</td>
</tr>
</tbody>
</table>
**Guide to the application process**

The BHAP website, www.bayer-hemophilia-awards.com, is dedicated to providing applicants with all the information they need for successful completion of a grant application. There are four main sections:

- **About the Program** contains an overview of the application process, details of current GRAC members and an insight into the review process. Prospective applicants are particularly advised to view the research priorities listed in this section of the website, where they can find areas of particular interest to the program as well as topics that are not covered by the awards.

- **Awards** explains the various grants available and—of great importance—describes the eligibility requirements for each.

- **Essential Info** contains important notes on a variety of issues, including those relating to additional funding, expenses, clinical trials, publications and termination of support.

- **Submit a Proposal** contains the Letter of Intent document to download, and advice on how to complete a successful application.

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**STEP 1. Choose your category**

- Fellowship Project Award
- Early Career Investigator Award
- Special Project Award
- Caregiver Award
- Outcomes Research Award

“Make sure you are eligible!”

**STEP 2. Complete a Letter of Intent**

- Forms can be downloaded from the website at any time of the year.
  Go to: www.bayer-hemophilia-awards.com.

**STEP 3. Submit Letter of Intent**

- Email LOI to: programadministrator@bayer-hemophilia-awards.com
  (deadline for the 2015/2016 cycle is November 30, 2015).

**STEP 4. Complete and submit Full Proposal (March)**

- Selected applicants will be emailed a Full Proposal form. See the section “Hints on Writing a Successful Grant” on the Awards website for advice.
- Email completed Full Proposal to: programadministrator@bayer-hemophilia-awards.com.

**STEP 5. Learn about the funding decision**

- Awardees will be notified and asked to send a letter of acceptance.
**Fellowship Project Award:**

**Etiology of the increased activity of FIX variants with substitutions at position 338**

Dr. Ben Samelson-Jones  
The Children’s Hospital of Philadelphia, PA, USA

**PROJECT OVERVIEW**

In this proposal, I seek to systematically decipher the etiology of the increased activity of FIX variants with substitutions at position 338. A twenty amino acid screen at this position revealed that FIX Arg338Leu is the most active variant, while wild-type FIX is one of the least active enzymes that is still functional, and FIX Arg338Ala is intermediate between these two. The activity differences between Factor IX wild-type, Arg338Ala, and Arg338Leu creates an attractive experimental system where the relevant molecular interactions can be evaluated in a dose-dependent manner.

I hypothesize that the hyperactivity is most likely due to two possible mechanisms:

1. Enhanced cofactor interaction with factor VIII (FVIII).
2. Resistance to regulatory inhibition by endogenous anticoagulants. The identification of either mechanism would provide new insight into the role and regulation of FIX in coagulation. These two hypotheses will be methodically evaluated in Specific Aim #1 and #2.

**Specific Aim #1: Determine whether hyperactive substitutions at position 338 of FIX modify the interaction with FVIII.** I will use several approaches to definitively determine whether these substitutions increase binding affinity and/or complex lifetime. The Michaelis–Menten kinetics of each FIX variant will be determined using commercial and novel chromogenic substrates for activated FIX and FX. This approach will determine the apparent dissociation constant (KD) between activated FIX and FVIII, as well as the strength of FX binding to the FIxa/FVIIa complex and the rate of FX cleavage. I will also assay the binding of FIxa and FVIIa directly using fluorescein active site labeled FIxa or surface plasmon resonance.

**Specific Aim #2: Determine how hyperactive substitutions at position 338 of FIX modify the inhibition of FIX by endogenous anticoagulants.** Previous studies of the Arg338Ala variant and structural analysis suggest that substitutions at position 338 may make FIX more resistant to inhibition by endogenous anticoagulants, including antithrombin, protein S, and α2-macroglobulin, as well as exogenous therapeutics, including unfractionated heparin and low-molecular-weight heparin. These reports specifically examined antithrombin and heparins; however, recent work suggests a potential role for protein S in FIxa inhibition. I will take several approaches to testing this hypothesis, using both plasma and *in vitro* assays.

As an initial approach, I will determine how the activity of each variant is affected in plasma-based clotting assays by different concentrations of inhibitors, using purified inhibitors and inhibitor-deficient plasma. I will also determine the inhibition constant (Ki) of each inhibitor, using the *in vitro* chromogenic substrate assays similarly described in Aim #1. Finally, I will determine the effect of each inhibitor on thrombin generation of each variant using the thrombin generation assays, which provides additional information regarding the kinetics of thrombin production, even after a clot has formed in clotting assays.

**EXPECTED OUTCOMES**

This research project will answer critical questions regarding the mechanism, and thus safety, of hyperactive FIX variants that are already being used in Phase I/II clinical trials. I anticipate that results from this investigation would unlock novel avenues of study regarding FIX activity or regulation, with therapeutic implications for treating bleeding and clotting disorders.
Immune tolerance induction (ITI) is the mainstay of treatment for hemophilia A (HA) patients with inhibitors to factor VIII (FVIII). However, this treatment is protracted, not effective in all patients, very expensive, and highly demanding. It is not clear why some patients develop FVIII inhibitors while others do not. It is accepted that B lymphocytes play a key role in the development and maintenance of neutralizing FVIII antibody response in HA. Interestingly, IgG1 and IgG4 are the most abundant IgG subclasses in HA patients with inhibitors, suggesting that a distinctive immune regulatory pathway is involved in the production of neutralizing FVIII antibodies.¹

PROJECT OVERVIEW
To better understand the characteristics of these FVIII inhibitors, we propose a simple, high-throughput method to isolate and expand single cell clones of FVIII-specific memory B cells from patients with HA receiving FVIII replacement therapy. The antibody reactivity of these clones will be compared with the Ig gene sequence information assessed to determine specific signatures. This information will be very important in identifying patients at high risk of inhibitor formation.

Objectives:
1. To determine if there is a difference in Ig gene rearrangement signature between HA patients who developed neutralizing FVIII antibodies when compared with those who did not develop inhibitor
2. To determine the kinetic of change in Ig rearrangement and establish if this can be used as an early marker to identify those that are likely to be refractory to ITI

Blood will be collected from HA patients (including previously untreated patients) referred to Great Ormond Street Hospital (Drs. Liesner and Mathias) and Evelina London Children’s Hospital (Dr. Alamelu). We have established that the anti-FVIII-specific memory B cells are capable of generating an antibody-secreting cell. A flow cytometric assay has been established for the isolation and single cell cloning of individual anti-FVIII-specific memory B cells. These clones will be further characterized by sequencing the variable heavy and light Ig chain genes. The affinity and specificity of the isolated antibodies for FVIII will be determined.

EXPECTED OUTCOMES
Our hypotheses are:
1. A specific Ig gene rearrangement pattern will determine if a patient is more likely to develop an inhibitor or not
2. The Ig gene rearrangement pattern will provide a tractable and highly sensitive marker to study the kinetics of the loss of FVIII-specific memory B cells following commencement of ITI, and will likely provide an early biomarker for those that are likely to be refractory to ITI.
Hemophilia A is a common blood disorder that is caused by mutations in the coagulation factor VIII (FVIII). To date, most hemophilia patients are treated with intravenous recombinant or plasma concentrate FVIII, which restores blood coagulation in the majority of cases. However, a therapeutic conundrum is the development of FVIII antibodies due to frequent infusions. Therefore, there is a pressing need to develop novel strategies to treat hemophilia A. About half of the patients with severe FVIII deficiency carry inversions within the \textit{F8} gene that result in elimination of protein production. Two common inversions have been described. One originates from an approximately 1 kb sequence in intron 1 (int1h-1) with a homologous sequence annotated int1h-2, about 15 kb downstream of exon 1, and the other between intron 22 and 9.5 kb homologous regions, denoted int22h-2 and int22h-3, located approximately 300 and 400 kb distal to \textit{F8}.

**PROJECT OVERVIEW**

Site-specific recombinases, such as the Cre/loxP systems, are able to effectively and specifically remove or invert DNA segments in the genome of living systems. A limitation of their utility has been the restricted number of sequences that they can recombine. To overcome this limitation, we have developed the directed molecular evolution approach ‘SLiPE’ for the streamlined generation of recombinases with altered target site specificities. The algorithm SeLOX allows for an efficient identification of suitable target sites to start the \textit{in vitro} evolution procedure. We have demonstrated the power of this approach for the generation of an HIV-1–specific recombinase acting on sequences present in the long terminal repeat of the virus. This recombinase has demonstrated efficacy in an HIV-1 mouse model and is currently being evaluated for use in a clinical trial.

**EXPECTED OUTCOMES**

With this application, we propose to develop a site-specific recombinase that recognizes target sequences present in inverted sequences of \textit{F8}. We have already analyzed the inverted sequences and have identified suitable sites that should be amenable for SLiPE. Through the iterative process of mutagenesis, DNA shuffling, and selection, we propose to evolve \textit{F8} recombinases that recombine these sequences. Initial tests in \text{E. coli} will be followed by transient delivery of the evolved \textit{F8} recombinase into human cells.

The expression of the \textit{F8}-specific recombinase should result in the inversion of the sequence between the two target sites, which will be tested by polymerase chain reaction and Southern hybridization. To ensure recombination specificity without adverse side effects, next-generation sequencing will be performed on cells that express the recombinase. Finally, we will investigate whether expression of the \textit{F8} recombinase in affected patient cells reverts the rearranged sequence of \textit{F8} and leads to expression of the gene as tested by RT-PCR. This approach should repair the endogenous \textit{F8} gene back to wild-type function, offering a cure for hemophilia A without the potential adverse side effects of other gene therapy approaches.
Outcomes Research Award:
The role of biomarkers to predict long-term joint outcome in patients with hemophilia

Dr. Michiel Coppens
Academic Medical Center, Amsterdam, Netherlands

Hemarthrosis is the hallmark of hemophilia, accounting for 70–80% of all bleeding episodes. Intra-articular bleeding results in active synovitis, characterized by neoangiogenesis and inflammation. Progression to late stages of joint damage, such as cartilage erosion and arthropathy, is frequently observed. Unexpectedly, previous studies have shown joint abnormalities on magnetic resonance imaging (MRI) in boys with hemophilia without overt joint bleeding. It is hypothesized that this is attributable to subclinical bleeding. It is uncertain, however, to what extent these MRI findings reflect reversible changes and/or normal developmental changes in a growing child.

PROJECT OVERVIEW

Tools that predict long-term adverse joint outcome in patients with early joint damage are potentially relevant, as more intense prophylaxis in these patients may reduce long-term complications. Biomarkers may prove valuable for this. The pathogenesis of hemophilia arthropathy is characterized by both degenerative damage, resembling osteoarthritis (OA), and inflammatory processes, as found in rheumatoid arthritis (RA) and juvenile idiopathic arthritis (JIA). In this setting, the predictive value of biomarkers has been repeatedly demonstrated. The best candidates for biomarkers of early joint damage in hemophilia are discussed below.

a. **Proangiogenic mediators** (e.g., high vascular endothelial growth factor levels in peripheral blood) may indicate active synovitis. Similar to findings in JIA patients, these levels are increased in hemophilic patients with joint disease compared with healthy controls.

b. **Cartilage degradation markers** (e.g., urinary type II collagen C-telopeptide, serum cartilage oligomeric matrix protein) correlate with radiological findings and predict long-term radiological progression of early joint damage in RA and OA patients. Similarly, these markers have been associated with plain X-ray–based joint scores in hemophilia patients.

c. **MRP8/14 and elastase** are released by neutrophils and macrophages, infiltrating inflamed tissues such as the affected synovium. Serum myeloid-related MRP8/14 predicts radiological progression in RA patients. Moreover, MRP8/14 seems to be a strong biomarker that detects subclinical disease activity in JIA. We have evidence that neutrophil-derived elastase is an equally strong, but more stable, serum marker (not yet published). Hemophilic synovial tissue contains increased numbers of CD68+ cells, suggesting myeloid-cell involvement. Therefore, MRP8/14 and elastase may also predict joint outcome in hemophilic patients.

This project is a cross-sectional pilot study to identify biomarkers that correlate with MRI-based joint scores and with the functional Hemophilia Joint Health Score in patients 8–40 years of age with moderate or severe hemophilia A on primary prophylaxis (31 eligible patients identified).

EXPECTED OUTCOMES

We hypothesize that biomarkers correlate with functional and MRI joint scores and help to identify patients at high or low risk of developing arthropathy. This risk assessment can facilitate personalized medicine, in which prophylactic treatment is modified according to the risk of arthropathy. We aim to enroll the patients in this pilot study into a prospective longitudinal study with periodic reassessment of biomarkers and joint status (HJHS and MRI).
The BHAP would like to thank all the applicants who submitted a proposal to the 2014/2015 cycle. Those fortunate enough to receive an award in 2015 came from 17 separate institutions and 9 countries. We congratulate them on their achievement.

### FELLOWSHIP PROJECT AWARD

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<tr>
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<tr>
<td>Ben Samelson-Jones</td>
<td>USA</td>
<td>Etiology of the increased activity of FIX variants with substitutions at position 338</td>
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<tr>
<td>Marta Milan</td>
<td>Italy</td>
<td>Bleeding risk profile of patients with FVII deficiency</td>
</tr>
<tr>
<td>Julie Tarrant</td>
<td>Canada</td>
<td>Influence of VWF on the innate immune response to FVIII</td>
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<tr>
<td>Beth Warren</td>
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<td>Joint Outcomes Study: predictors of prophylaxis outcome</td>
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<td>Yizhou Dong</td>
<td>USA</td>
<td>mRNA nanomedicines for treating hemophilia A</td>
</tr>
<tr>
<td>Behnaz Pezeshkpoor</td>
<td>Germany</td>
<td>Detection of domain specific inhibitors of FVIII in a large cohort of samples using a new immunoassay</td>
</tr>
<tr>
<td>Ammon Fager</td>
<td>USA</td>
<td>Rational Design of New Therapeutic Agents for the Treatment of Hemophilia Patients with Inhibitors</td>
</tr>
<tr>
<td>Vânia Coelho</td>
<td>UK</td>
<td>Identification of a B cell signature associated with inhibitors in Haemophilia A</td>
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### SPECIAL PROJECT AWARD

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<tr>
<td>Qizhen Shi</td>
<td>USA</td>
<td>Platelet Gene Therapy of Murine Hemophilia B with Inhibitors</td>
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<tr>
<td>Frank Buchholz</td>
<td>Germany</td>
<td>Development of designer recombinases that correct factor VIII gene inversions</td>
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<tr>
<td>Christopher Doering</td>
<td>USA</td>
<td>Bioengineering Factor VIII through Ancestral Reconstruction</td>
</tr>
<tr>
<td>Kazuo Ohashi</td>
<td>Japan</td>
<td>Creation of Micro-organoids-based Cellular Therapies Toward Hemophilia</td>
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### CAREGIVER AWARD

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<tbody>
<tr>
<td>Silvia Riva</td>
<td>Italy</td>
<td>RE.CO.VERY: REsponsibility for treatment COMpliance is VERY important. A study in elderly population with hemophilia</td>
</tr>
<tr>
<td>Mala Kuneeram / Urvashi Rughoo</td>
<td>Mauritius</td>
<td>Improving hemophilia care in Mauritius through improved laboratory diagnostic services and physiotherapy support</td>
</tr>
<tr>
<td>Nicki Mackett</td>
<td>UK</td>
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<tr>
<td>Nyondwa Zulu</td>
<td>Zambia</td>
<td>The impact of stigma on Hemophiliacs living in the rural parts of Zambia</td>
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### OUTCOMES RESEARCH AWARD

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<th>Name</th>
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<tr>
<td>Michiel Coppens</td>
<td>The Netherlands</td>
<td>The role of biomarkers to predict long-term joint outcome in patients with hemophilia</td>
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