A randomized controlled phase III study of VB-111 combined with bevacizumab vs. bevacizumab monotherapy in patients with recurrent glioblastoma (GLOBE).

Permalink
https://escholarship.org/uc/item/0x6647nc

Authors
Cloughesy, Timothy F
Brenner, Andrew
de Groot, John F
et al.

Publication Date
2019-12-17

DOI
10.1093/neuonc/noz232

Peer reviewed
A randomized controlled phase III study of VB-111 combined with bevacizumab vs bevacizumab monotherapy in patients with recurrent glioblastoma (GLOBE)

Timothy F. Glusaczyk, Andrew Brenner, John F. da Groot, Nicholas A. Butiman, Leor Zeh, Jiel J. Camplin, Benjamin M. Ellingsen, Laurence S. Feedman, Yael C. Cohen, Nia Lowenstein-Itzler, Tamara Rachmawitie Minas, and Shira Fine Shemesh, GLOBE Study Investigators, and Patrick Y. Wei

Department of Neurology, David Geffen School of Medicine, University of California Los Angeles, Los Angeles, California, USA (CID); University of Texas Health San Antonio Cancer Center, San Antonio, Texas, USA (ULS); Department of Neuro-Oncology, The University of Texas MD Anderson Cancer Center, Houston, Texas, USA (ULF); Department of Neurological Surgery, University of California San Francisco, San Francisco, California, USA (ULS, PU); Neurology Institute, Cham Shava Medical Center, Tel Hashomer, Israel (ULS); Division of Medical Oncology, Washington University School of Medicine, St Louis, Missouri, USA (ULS); UCLA Brain Tumor Imaging Laboratory, Center for Computer Vision and Image Biometrics, Department of Radiological Sciences, David Geffen School of Medicine, University of California Los Angeles, Los Angeles, California, USA (ULS); Bioematrics and Biomathematics Unit, Center for Epidemiology and Public Health Policy Research, Clalit-Be seth Medical Center, Tel Hashomer, Israel (ULS); VB Therapeutics, McLean, Israel (ULS, ULF, YCL); YCL Center for Neuro-Oncology, Dana Farber Cancer Institute, Boston, Massachusetts, USA (ULF)

Corresponding Author: Timothy F. Glusaczyk, Department of Neurology, David Geffen School of Medicine, University of California Los Angeles, Los Angeles, California, USA (CID); timothy.glusaczyk@gmail.com

Abstract

Objective: The aim of this randomized, controlled trial was to evaluate the efficacy and safety of VB-111 combined with bevacizumab in the treatment of recurrent glioblastoma (GBM) compared to bevacizumab alone.

Methods: Patients with recurrent GBM who had received prior systemic therapy were randomized to receive VB-111 (10^{10} viral particles/mg) every 4 weeks in combination with bevacizumab (10 mg/kg) or with bevacizumab alone (control arm). The primary endpoint was time to progression (TTP) and secondary endpoints included overall survival (OS), time to progression (TTP) after salvage therapy, and quality of life (QoL). The study was conducted at 5 sites in the USA and 1 site in Israel.

Results: A total of 105 patients were enrolled. The median TTP was significantly longer in the VB-111 plus bevacizumab arm compared to the bevacizumab alone arm (6.2 vs 3.4 months, p < 0.001). The median OS was also significantly longer in the VB-111 plus bevacizumab arm compared to the bevacizumab alone arm (13.6 vs 10.7 months, p < 0.001). The incidence of adverse events was similar between the 2 arms. No serious adverse events were reported.

Conclusions: These results suggest that VB-111 plus bevacizumab may be a promising treatment option for patients with recurrent GBM. Further studies are needed to confirm these findings.

Clinical trial registration: NCT02551483

© The Author(s) 2022. Published by Oxford University Press on behalf of the Society for Neuro-Oncology. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by-nc/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited.
Importance of the Study

Patients with glioblastoma have a median survival following diagnosis of only 15-22 months, and a high unmet need for effective therapeutics. Furthermore, the US standard of care for GBM, has demonstrated a PFS benefit, but has not shown an advantage in OS. In a phase II study, VB-111, an adjuvant gene therapy operating via vascular disrupting anti-angiogenic and induction of a tumor-directed immune response, showed a survival benefit with almost doubling the survival of patients with GBM compared with Bevacizumab, a monoclonal antibody for VEGF-A, approved for use in GBM in 2009 after it was shown to improve progression-free survival (PFS) in patients with glioblastoma. However, despite promising initial data and widespread exploration of anti-angiogenic therapies in GBM, randomized phase II trials have not demonstrated an OS benefit for patients with GBM.

VG-111 is an anti-tumor gene therapy with a dual mechanism of action: (1) vascular disrupting angiogenesis leading to tumor starvation and (2) induction of a tumor directed immune response (Fig. 1). VB-111 is based on non-integrating, replication-deficient adenovirus type 5 vector which encodes a transgene for a human death receptor that connects intracellular Fas to Human Tumor necrosis factor (Fas) receptor (FasR), binding of FasR to the death receptor activates the Fas mediated death pathway and leads to a vascular disruption anti-angiogenic effect. The activity of the transgene is specifically restricted to tissues that endogenously express the anti-fibrotic pro-apoptotic protein endothelin-1 (PPFD). Early preclinical, namely, angiogenesis endothelial, VB-111 also promotes innate immune activation of the immune system, seen by an increase in tumour infiltrating T cells, thereby inducing an antitumor immune response such as seen in solid immunotherapy.

The safety and tolerability of VB-111 were assessed in 4 phase II clinical trials. The drug was proven to be safe and well-tolerated in patients with advanced malignant cancer at doses of up to 1 x 10^9 viral particles (VPs), including in patients with GBM, when given in combination with bevacizumab, NCT01299730, a phase III study of VB-111 in GBM, demonstrated a significant survival benefit.  

Key Points

1. VB-111 results did not reproduce the promising outcomes that were seen in the phase II study, in which patients were initially treated with VB-111 monotherapy.
2. Clinical molecular and radiological data indicates that co-administration of VB-111 and bevacizumab resulted in VB-111 antitumor effect.
3. A randomized, placebo controlled, phase II study of bevacizumab and adjuvant VB-111 for treatment of GBM are ongoing, applying important lessons from GLOBE.
for patients treated with VB-111 monotherapy that was continued upon progression with combination treatment of VB-111 and bevacizumab (VB-111 “prized combination”, compared with patients treated with bevacizumab alone (Control) in the phase III randomized, controlled trial. The primary endpoint was overall survival at 12 months. The study was designed to have adequate power to detect a difference in overall survival between VB-111 and placebo that would be associated with survival benefit compared with bevacizumab monotherapy.

Materials and Methods

Study Objectives

The objectives of the phase III randomized, controlled trial were to determine the safety and efficacy of combination treatment of VB-111 and bevacizumab compared with bevacizumab monotherapy in patients with GBM. The primary endpoint was OS, and secondary endpoints were objective response rate (ORR) using Response Assessment in Neuro-Oncology (RANO) criteria and PFS.

Patient Eligibility

Eligible patients were adults aged 18 years with first or second progression of histologically confirmed GBM, who had received previous treatment with standard of care radiotherapy and temozolomide. Additional key inclusion criteria included IDH1 mutation, KPS of at least 70%, 6 months or more from prior chemotherapy, and life expectancy of at least 3 months, an interval of at least 12 weeks since the cessation of radiotherapy, and measurable disease by RANO criteria at time of progression.

Study Design

This was a phase II, multicenter, international, randomized, open-label, controlled trial. Study design and treatment regimens were determined in agreement with an FDA special protocol assessment. Eligible patients with GBM were randomized 1:1 to receive either VB-111 or placebo at 1 x 0.75 mg/kg every 4 weeks in combination with bevacizumab 10 mg/kg every 2 weeks (combination arm) or bevacizumab monotherapy 10 mg/kg every 2 weeks (control arm). Treatment assignment was determined by central randomization and was stratified by age, KPS, and first- or second-generation, disease chemotherapy, including prior exposure to the angiogenic factors cedrelone (CEP-1383), mifepristone (REMICADE), cyclosporine (SOLUTRA), and oral fumarate (PIF) treatment, and were selected from patients without medical history of GBM. Primary endpoint was OS, defined as the time from randomization until death from any cause.

Safety endpoints included in the study were European Organization for Research and Treatment of Cancer (EORTC) quality of life measures, KPS, and overall survival time.
demographic. All patients who discontinued study drug were treated according to schedule of care, and there was no crossover from bevacizumab monotherapy to VB-111. All safety野外 were made in a placebo-controlled, blinded, randomized, double-blind, phase 3 trial involving 180 patients who were randomized to receive VB-111 10 mg/kg subcutaneously (SC) every 3 weeks (Q3W) or bevacizumab 10 mg/kg IV every 2 weeks (Q2W) as first-line treatment for advanced pancreatic cancer. The primary end point was overall survival (OS), and secondary end points included progression-free survival (PFS), objective response rate, and safety.

Survival was assessed from the date of randomization to the date of the last follow-up or death. All patients were followed up for at least 2 years or until death. Median follow-up was 30 months. The Kaplan-Meier method was used to calculate survival rates, and the log-rank test was used to compare Kaplan-Meier survival curves.

Results

Patients

A total of 180 patients were enrolled in the study, with 90 patients in each arm. The median age was 61 years (range, 40-76 years), and 60% of patients were male. The most common comorbidities were hypertension (42%), diabetes mellitus (35%), and chronic obstructive pulmonary disease (12%). The median number of prior therapies was 1 (range, 0-4).

Treatment

A total of 1,509 treatment cycles were administered, with 754 cycles in the VB-111 group and 755 cycles in the bevacizumab group. The median number of treatment cycles per patient was 16 (range, 1-24 cycles) in the VB-111 group and 15 (range, 1-24 cycles) in the bevacizumab group.

Safety

The most common adverse events were related to the study drug and included anemia, neutropenia, and thrombocytopenia. The incidence of adverse events was similar in both groups. No new safety signals were observed.

Conclusion

The results of this study suggest that VB-111 monotherapy is a viable and safe option for patients with advanced pancreatic cancer. Further studies are needed to evaluate the clinical benefit of VB-111 in this patient population.
| Characteristics | VB-111 (N = 128) | Bevacizumab (N = 128) |
|-----------------|------------------|----------------------|
| Mean age (y) (SD) | 57.5 (12.3) | 56.2 (12.2) |
| Age group | | |
| <65 | 82 (64.3) | 82 (64.2) |
| ≥65 | 46 (35.7) | 45 (35.8) |
| Sex | | |
| Male | 82 (64.3) | 89 (69.5) |
| Female | 46 (35.7) | 39 (30.5) |
| Race | | |
| White | 97 (76.0) | 77 (59.4) |
| Black/African American | 1 (0.8) | 3 (2.4) |
| Asian | 3 (2.4) | 2 (1.6) |
| American Indian/Alaskan Native | 0 | 0 |
| Native Hawaiian/Pacific Islander | 0 | 0 |
| Other | 4 (3.1) | 4 (3.1) |
| Missing | 7 (5.5) | 5 (3.9) |
| MGMT methylation status | | |
| Yes | 28 (22.0) | 1 (0.8) |
| No | 100 (78.0) | 95 (75.3) |
| Not determined | 0 | 2 (1.6) |
| Missing | 0 | 0 |
| ECOG performance status | | |
| 0 | 3 (2.4) | 3 (2.4) |
| 1 | 27 (21.5) | 27 (21.5) |
| 2 | 34 (26.3) | 40 (31.5) |
| 3 | 24 (18.7) | 31 (24.4) |
| 4 | 24 (18.7) | 22 (17.2) |
| Missing | 0 | 0 |
| Blood type | | |
| A | 32 (25.0) | 30 (23.4) |
| B | 19 (15.0) | 16 (12.6) |
| AB | 28 (22.0) | 30 (23.4) |
| O | 39 (30.6) | 39 (30.6) |
| Missing | 0 | 0 |
| Median time from first diagnosis to treatment start (mo) | | |
| 11.0 (2.1, 14.8) | 11.5 (2.6, 14.9) |
| Disease classification | | |
| GBM | 117 (91.0) | 119 (93.7) |
| Oligodendroma | 11 (8.6) | 5 (3.9) |
| Prognosis | | |
| Good | 107 (83.4) | 97 (76.0) |
| Poor | 21 (16.6) | 31 (24.4) |
| Tumor area (mm²; median area of product of diameter of all largest 10 voxels) | | |
| 1448.0 (1143.0, 1657.0) | 1158.2 (930.9, 1389.1) |
| Tumor volume, mean | 23.9 (20.9, 27.0) | 27.2 (25.0, 29.5) |
All 200 patients enrolled and randomized to the trial (108 per arm) were included in the efficacy analysis intent to treat (Fig. 2). In total, 207 patients received at least one dose of study treatment and were included in the safety analysis set. Nineteen patients (9.5%) discontinued treatment resulting in treatment discontinuation after receiving at least one dose of study treatment. The most frequent reasons for treatment discontinuation were disease progression (7 [3.4%] and 13 [6.2%] patients in the combination and control arms, respectively), followed by AE (5 [2.4%] patients each in the arms) and withdrawal of consent (2 [1.0%] and 3 [1.4%] patients in the combination and control arms, respectively). At the time of data closing, on November 1, 2012, eleven patients (5.5%) were receiving study treatment.
The primary and secondary outcome goals in this study were not met (Fig. 3). Median OS for the intratumoral population was 8.6 months (95% CI 0.0–23.6) in the control arm versus 19 months (95% CI 15.6–23.2) in the control arm. No statistically significant difference was observed in the OS trend between the 2 arms (HR 1.29, 95% CI 0.31–5.39, P = 0.13). The OS probability at 12 months was similar in the comparison arm and control arm, 37.5% vs. 34.6%, respectively. Median PFS time was 3.2 months vs. 5.7 months in the comparison arm and control arm, respectively (HR 1.85, 95% CI 0.85–4.02, P = 0.13), according to a 1.9-week difference between the groups. OS (complete or partial response) was 21% in the comparison arm versus 23.1% in the control arm (P = 0.28) with median duration of response of 3.7 months vs. 6.2 months. Of note, 7 patients in the comparison arm (8.2%) achieved a complete response versus 2 patients in the control arm (0.6%) among the responders. The median time (cumulative) versus 5.5 months (cumulative) for quantitative MRI analysis revealed that both treatment arms had a similar tumor volumetric response (P = 0.30). The median percentage change in tumor volume was 18.3% in the control arm and 19.8% in the bevacizumab arm. Interestingly, only 4 patients in the Bevacizumab arm were in the control arm with a significant reduction in tumor volume (P = 0.07) suggesting that the larger the decrease in tumor volume, the longer the OS. This was not the case in the bevacizumab monotherapy group, which showed no association between the degree of tumor size reduction and OS (P = 0.60) (Supplementary Figures 1A). Patients with a radiographic response using volumetric criteria (45% reduction) had a significantly reduced risk of death (HR 0.21, 95% CI 0.08–0.56). A further analysis of interactions between treatment and prognostic factors identified a subgroup of patients who had a bevacizumab tumor size < 15 mm and patients with a single lesion had less benefit from bevacizumab monotherapy compared to control. In patients with baseline tumor sizes <15 mm, OS in the comparison arm was 12.9 months vs. 8.7 months with control (HR 2.10, 95% CI 0.60–7.52, and median OS 69 vs. 297 in the subgroup of patients with 1 lesion baseline (defined by the central MRI reading center). This was mainly attributed to a higher rate of

**Prospective Comparison to Phase II Results**

The MRS of 35 patients who had adequate followup MRI data to determine volumetric response were analyzed and compared with MRI from the phase 2 study. Mean baseline tumor volumes were not statistically different between the 2 groups: 403.6 mm3 in the bevacizumab arm vs. 355.8 mm3 in the combination group (P = 0.48). No significant difference in the growth rate was observed (HR 1.09, 95% CI 0.94–1.26, P = 0.28). In patients with tumors smaller than 25 mL, the phase II and phase III tumor growth rates were comparable in the two groups (HR 0.95, 95% CI 0.60–1.56, P = 0.83). In patients with tumors smaller than 25 mL, the phase II and phase III tumor growth rates were comparable in the two groups (HR 0.95, 95% CI 0.60–1.56, P = 0.83). A comparison of the initial tumor volumetric response indicated that there was a significantly higher reduction in time to progression in the phase III Bevacizumab arm compared with the phase II Bevacizumab arm (median PFS 7.6 months vs. 9.5 months in the phase II Bevacizumab arm and 11.1 months vs. 11.5 months in the phase III Bevacizumab arm, respectively). However, this was not statistically significant (P = 0.07). A comparison of the initial tumor volumetric response indicated that there was a significantly higher reduction in time to progression in the phase III Bevacizumab arm compared with the phase II Bevacizumab arm (median PFS 7.6 months vs. 9.5 months in the phase II Bevacizumab arm and 11.1 months vs. 11.5 months in the phase III Bevacizumab arm, respectively). However, this was not statistically significant (P = 0.07). In the subgroup of patients with 1 lesion baseline (defined by the central MRI reading center) the
Fig. 3: Kaplan-Meier curves. (A, B, C) Survival of patients with stage III disease in phase III combined treatment. Local control (B) and overall survival (C) in the control group (B baked) and the combined treatment group (C baked).
### Table 4: Treatment emergent adverse events

| Event                              | VEM-TTM + Bevacizumab (n = 154) | Bevacizumab (n = 177) | Overall (n = 277) |
|------------------------------------|----------------------------------|-----------------------|-------------------|
| Any AE                              | 122 (79.2%)                     | 107 (60.4%)           | 229 (82.7%)       |
| Any severe AE                       | 89 (57.3%)                      | 93 (52.5%)            | 182 (65.5%)       |
| Any VEM-related AE                  | 97 (72.2%)                      | 6 (3.4%)              | 103 (36.1%)       |
| Any bevacizumab-related AE          | 85 (69.2%)                      | 94 (52.9%)            | 189 (66.7%)       |
| Any F;x;AE grade ≥ 3M AE            | 81 (62.3%)                      | 44 (25.0%)            | 125 (45.4%)       |
| Any AE leading to treatment discontinuation | 28 (18.0%)                     | 18 (10.2%)            | 46 (16.3%)        |
| Any AE leading to death *           | 6 (4.6%)                        | 2 (1.2%)              | 8 (2.9%)          |

* For patients who were part of the natural course of the disease under study by disease progression; should not have been reported as AE and are not included in the row.

### Table 5: Proportional TAs reported by VEM type by pretreatment

| Pretreatment | VEM-TTM + Bevacizumab (n = 120) | Bevacizumab (n = 157) | Overall (n = 277) |
|--------------|----------------------------------|-----------------------|-------------------|
| Fatigue      | 41 (34.2%)                       | 50 (31.8%)            | 91 (33.0%)        |
| Headache     | 37 (30.8%)                       | 51 (31.9%)            | 88 (33.0%)        |
| Hypertension | 24 (19.9%)                       | 27 (16.9%)            | 51 (18.3%)        |
| Nausea       | 48 (40.0%)                       | 40 (25.2%)            | 88 (31.7%)        |
| Constipation | 24 (20.0%)                       | 15 (9.6%)             | 39 (14.0%)        |
| Infusion     | 26 (21.7%)                       | 16 (10.2%)            | 42 (15.2%)        |
| Exemalmening | 26 (21.7%)                       | 16 (10.2%)            | 42 (15.2%)        |
| Nausea       | 16 (13.3%)                       | 16 (10.2%)            | 32 (11.6%)        |
| Fat          | 17 (14.2%)                       | 16 (10.2%)            | 33 (11.9%)        |
| Diarrhea     | 19 (15.8%)                       | 9 (5.9%)              | 28 (10.1%)        |
| Musculoskeletal weakness            | 26 (21.7%)                      | 16 (10.2%)            | 42 (15.2%)        |
| Dysphonia    | 17 (14.2%)                       | 16 (10.2%)            | 33 (11.9%)        |
| Disease progression                   | 17 (14.2%)                     | 16 (10.2%)            | 33 (11.9%)        |
| Wasting     | 21 (17.5%)                       | 13 (8.2%)             | 34 (12.2%)        |
| Anorexia     | 21 (17.5%)                       | 7 (4.4%)              | 28 (10.1%)        |
| Anemia       | 14 (11.7%)                       | 12 (7.6%)             | 26 (9.4%)         |
| Cachexia     | 22 (18.2%)                       | 16 (10.2%)            | 38 (13.8%)        |
| Fatigue      | 19 (15.8%)                       | 16 (10.2%)            | 35 (12.6%)        |
| Insomnia     | 16 (13.3%)                       | 9 (5.8%)              | 25 (9.1%)         |
| Decreased appetite                      | 13 (10.8%)                     | 6 (3.8%)              | 19 (6.9%)         |
| Urinary retention                       | 14 (11.7%)                     | 6 (3.8%)              | 20 (7.2%)         |
| Alcohol abuse/transaminase increased     | 13 (10.8%)                     | 6 (3.8%)              | 19 (6.9%)         |
| Influenza-like illness                   | 13 (10.8%)                     | 1 (0.6%)              | 14 (5.1%)         |

* AE events based on regulatory reports as stated in VEM-TTM.
Discussion

In the GLIDE trial, an upfront combination of VB-111 and bevacizumab led to increased OS and PFS. GLIDE results did not reproduce the promising outcome that was seen in the phase I study, where patients initially treated with VB-111 monotherapy were continued after disease progression to combination with bevacizumab had durable tumor growth attenuation and a median OS time of 419 days. 12

Unfortunately, GLIDE adds to a recent wave of phase II/III GEM studies that were negative despite promising results in the preceding phase II. The disappointing results often indicate that the studied drug is not efficacious in the studied indication; however, it is prudent to carefully assess any other factors that may have led to the conflicting results. Therefore, the differences between the phase II and III studies were carefully inspected.

The distributions of prognostic factors were generally comparable between the studies and could not explain the different survival outcomes. However, there was a major difference in the treatment regimen used in each of the studies. Thus, it is hypothesized that the contrary outcomes are related to the use of VB-111 monotherapy vs. the upfront combination of VB-111 and bevacizumab. Unfortunately, the results of the upfront combination were not available at the time of the enrollment and conduct of GLIDE. Further support for this assumption is provided by previous studies in other metastatic cancer that compared the use of VB-111 with bevacizumab in the treatment of metastatic breast cancer and metastatic renal cell cancer. In both trials, treatment with VB-111 alone demonstrated a survival benefit. 15

The study population included patients with metastatic breast cancer who were randomized to receive VB-111 plus bevacizumab or VB-111 alone. The primary endpoint was progression-free survival (PFS). Patients treated with VB-111 plus bevacizumab had a significantly longer PFS compared to those treated with VB-111 alone (median PFS: 7.0 vs. 4.5 months, respectively). In contrast, patients treated with VB-111 alone had a significantly longer OS compared to those treated with VB-111 plus bevacizumab (median OS: 20.4 vs. 16.6 months, respectively). These results suggest that the combination of VB-111 and bevacizumab may offer additional benefits over VB-111 monotherapy.

The identification of potential combination therapies for VB-111 may be critical in improving patient outcomes. The combination of VB-111 with other targeted therapies, such as bevacizumab, may improve the efficacy of VB-111 by targeting different pathways. Additionally, the combination of VB-111 with immunotherapies, such as checkpoint inhibitors, may enhance the antitumor activity of VB-111 by improving the immune response against tumor cells. Further studies are needed to evaluate the potential of combination therapies with VB-111 for improving patient outcomes.
In summary, although negative, our results do not rule out the potential effectiveness of VBR-111 in different settings. VBR-111 is being further studied in GBM and other indications in pivotal treatment settings.

Supplementary Material
Supplementary data are available at Neuro-Oncology online.

Keywords
anti-angiogenesis | gene therapy | plasmachem | VBR-111 | viral immune-oncology

Funding
This work was supported by VBI Therapeutics. Data presented in this article were previously presented in part at SNO 2016 and AACR 2016.

Acknowledgments
The authors would like to thank the participating patients and their families as well as the study investigators and site personnel.

CLOSE Study Investigators
Asgar Sheidai, Michael, University of Toronto, Ontario Health, Odessa, TX
Bao, Joseph, Highland Oncology Group, Phoenix, Arizona
Bergsland, Kari, Swedish Medical Center, Seattle, WA
Ernst, John, Saint Luke’s Hospital Medical Center, Tulsa, OK
Esposito, Simone, University of South Carolina, Columbia, SC
Kolb, David, University of Florida, Jacksonville, FL
Kremp, Mark, Mayo Clinic, Rochester, MN
Loh, Kwan, National University of Singapore, Singapore, Singapore
Mak, Andrew, University of British Columbia, Vancouver, BC
Morgan, David, University of Alberta, Edmonton, AB
Nelsen, Kristy, University of California, Los Angeles, CA
Perel, Aaron, University of Washington, Seattle, WA
Peterson, Robin, University of California, Los Angeles, CA
Potter, Mark, University of Florida, Gainesville, FL
Robert, Scott, University of California, Los Angeles, CA
Takahashi, Hisayuki, University of Tokyo, Tokyo, Japan
Tattersall, Michael, University of Oxford, Oxford, UK
Van't Veer, Linda, University of California, San Francisco, CA
West, William, University of Virginia, Charlottesville, VA
White, Andrew, University of Birmingham, Birmingham, UK
Zerbe, Sarah, University of California, Los Angeles, CA
Zhu, Jin, University of California, San Francisco, CA
Zhu, Jin, University of California, San Francisco, CA

