UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K
CURRENT REPORT

Pursuant to Section 13 or 15(d) of the
Securities Exchange Act of 1934

Date of Report (Date of earliest event reported): November 14, 2011 (November 14, 2011)

REGENERON PHARMACEUTICALS, INC.
(Exact Name of Registrant as Specified in Charter)

New York	**000-19034**	**13-3444607**
(State or other jurisdiction of Incorporation)	(Commission File No.)	(IRS Employer Identification No.)

777 Old Saw Mill River Road, Tarrytown, New York 10591-6707
(Address of principal executive offices, including zip code)

(914) 347-7000
(Registrant's telephone number, including area code)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

o Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

o Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

o Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

o Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Item 7.01 Regulation FD Disclosure.

On November 14, 2011, at the American Heart Association Scientific Session 2011 in Orlando, Florida, data from a Phase 1b multidose study of REGN727/SAR236553 as mono or add-on therapy in patients with heterozygous familial and non-familial hypercholesterolemia were presented by Dr. Gary Swergold of Regeneron Pharmaceuticals, Inc. A copy of the slides that were presented is furnished as Exhibit 99.1 to this Current Report on Form 8-K.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits

99.1 Presentation entitled Inhibition of PCSK9 with Monoclonal Antibody REGN727/SAR236553 Effectively Reduces LDL-C as Mono or Add-on Therapy in Heterozygous Familial and Non-Familial Hypercholesterolemia.

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Date: November 14, 2011

REGENERON PHARMACEUTICALS, INC.

By: /s/ Joseph J. LaRosa

Name: Joseph J. LaRosa

Title: Senior Vice President, General Counsel and Secretary

Exhibit Index

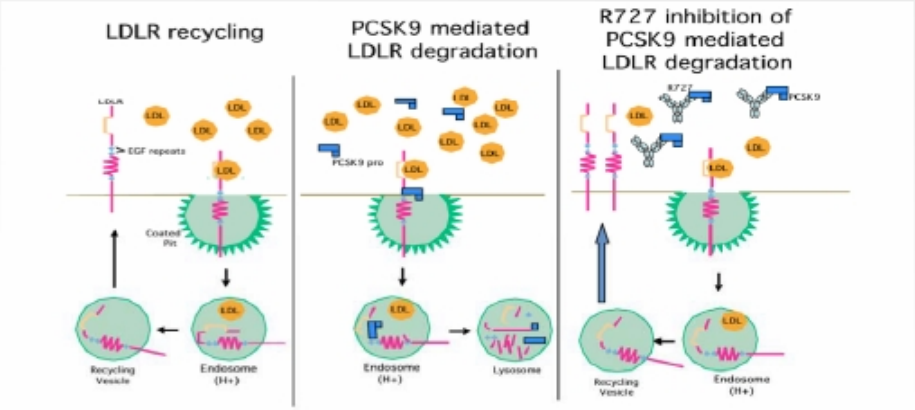
Number	Description
99.1	Presentation entitled Inhibition of PCSK9 with Monoclonal Antibody REGN727/SAR236553 Effectively Reduces LDL-C as Mono or Add-on Therapy in Heterozygous Familial and Non-Familial Hypercholesterolemia.

Inhibition of PCSK9 with Monoclonal Antibody REGN727/SAR236553 Effectively Reduces LDL-C as Mono or Add-on Therapy in Heterozygous Familial and Non-Familial Hypercholesterolemia.

Gary Swergold[1] MD, PhD, William Smith[2] MD, Scott Mellis[1], MD, PhD, Douglas Logan[3] MD, Cheryle Webb[4] MD, Richard Wu[1] PhD, Yunling Du[1] PhD, Therese Krans[4] RN, MBA, Evelyn Gasparino[1] and Evan A Stein[4] MD, PhD

[1] Regeneron Pharmaceuticals Inc.,Tarrytown, NY, USA; [2] VRG/NOCCR, University of Tennessee Medical Center Knoxville, Knoxville, TN, USA; [3] Medpace Clinical Pharmacology Unit, Cincinnati, OH, USA; [4] Metabolic & Atherosclerosis Research Center, Cincinnati, OH, USA

PCSK9: Therapeutic Target for ↑ LDL-C

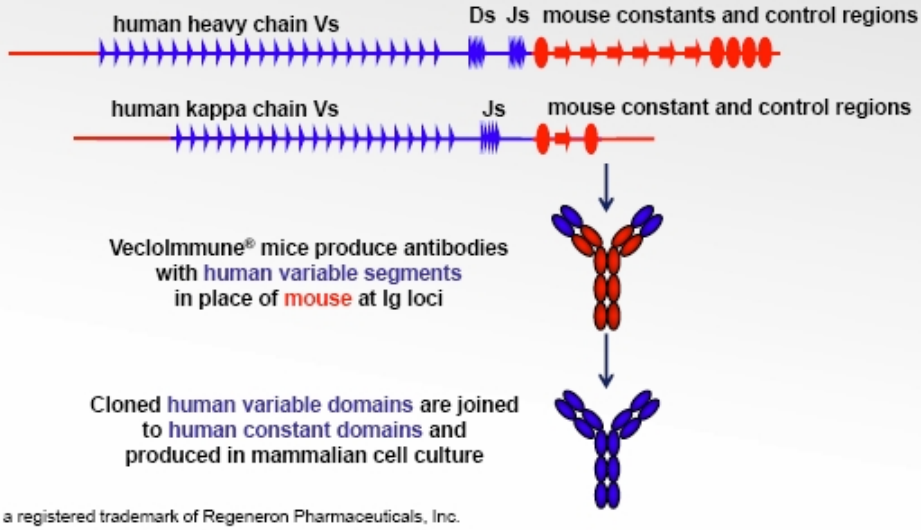


REGN727/SAR236553:

A Fully-Human mAb to PCSK9

REGN727 binds hPCSK9 with subnanomolar affinity

- Produced using Regeneron's VelocImmune technology
- Precise humanization of 6 megabases of mouse immune loci



VelocImmune® is a registered trademark of Regeneron Pharmaceuticals, Inc.

Two Initial Phase Ia Studies
in Healthy Volunteers

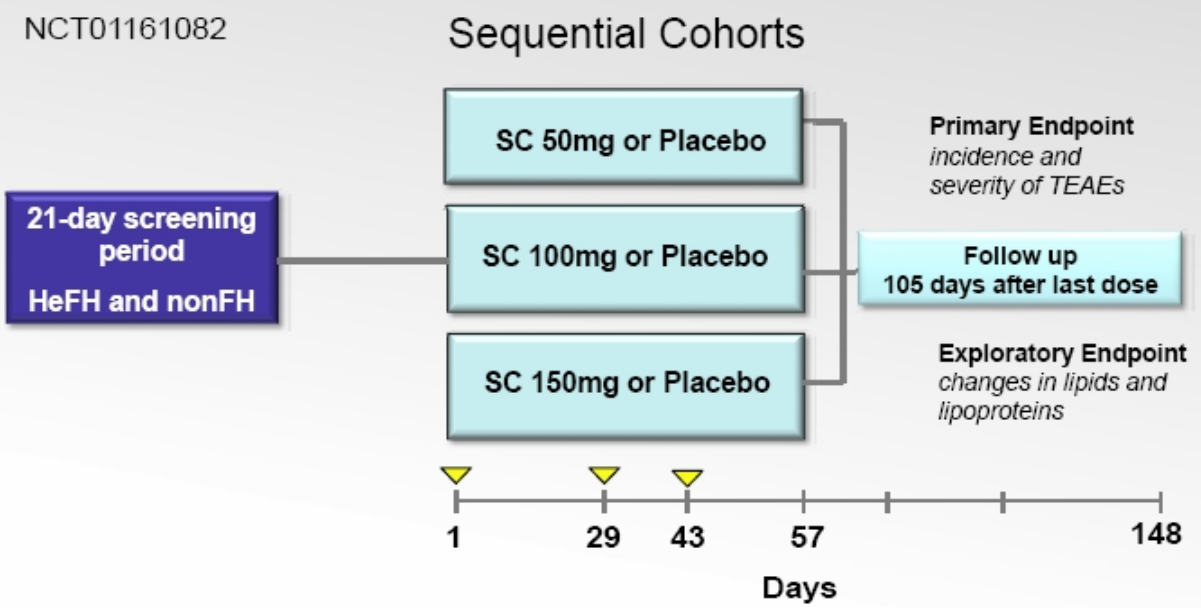
- Single dose studies
  - IV N=40*
  - SC N= 32†
- Sustained LDL-C lowering
  - exceeded 60% and lasted for at least 30 days in higher dose cohort
- Safety and tolerability supported decision to initiate studies in patients

*Study 0902: G Swergold, et al. *Circ* 2010;122(10021): A2325; G Swergold, et al. *J Clin Lipidol* 2011; 5(3):219.
†Study 0904: G Swergold, et al. *JACC* 2011;57:2023.

Phase Ib: Multidose Study

Double-Blind, Randomized, Placebo-Controlled

NCT01161082



Study sites:
Dr. Evan Stein (Metabolic & Atherosclerosis Research Center, Cincinnati, OH)
Dr. William Smith (VRG/NOCCR, University of Tennessee Medical Center, Knoxville, TN)

▽ Dose administered

Dose Groups

a) HeFH +Atorva b) nonFH +Artorva c) nonFH Mono-Rx

REGN727 Dose	Patient Group	Total # Pts (R727:Pbo)	HeFH Status	Screening LDL-C (mg/dL)	Atorvastatin Dose
50mg	1	7 (5:2)	HeFH	>100	10-40 mg QD
	2	10 (8:2)	Non-FH	>100	10-40 mg QD
100mg	3	7 (5:2)	HeFH	>100	10-40 mg QD
	4	10 (8:2)	Non-FH	>100	10-40 mg QD
150mg	5	7 (5:2)	HeFH	>100	10-40 mg QD
	6	10 (8:2)	Non-FH	>100	10-40 mg QD
	7	10 (8:2)	Non-FH	>130	None (Diet alone)

Key Inclusion/Exclusion Criteria

- Inclusion Criteria
  - Men and women 18 – 65 yrs
  - HeFH by clinical diagnosis or non-FH
    - LDL-C > 100 mg/dL
    - Stable dose of atorvastatin (10-40 mg/day)
    - 50, 100, 150 mg dose levels
  - Non-FH with higher LDL-C
    - LDL-C > 130 mg/dL
    - Diet alone, no atorvastatin co-therapy
    - 150 mg dose level only
- Exclusion Criteria
  - Homozygous FH
  - Lipid-lowering therapies other than atorvastatin
  - Fasting TG > 300 mg/dL
  - History of MI, ACS, angina, stroke, PVD, or cardiac revascularization
  - Disorders known to cause secondary elevations of LDL-C

Demographics

	HeFH Atorva Rx		Non-FH Atorva Rx		Non-FH Diet alone	
	Placebo (N=6)	R727 (N=15)	Placebo (N=6)	R727 (N=24)	Placebo (N=2)	R727 (N=8)
Age	39	41	50	53	45	54
% Male	83%	80%	67%	54%	100%	38%
BMI (kg/m^2)	25.8	27.8	29.4	27.4	23.8	29.5
Concomitant ATORVASTATIN						
Atorva 10mg	0	20%	67%	67%	0	0
Atorva 20mg	50%	27%	33%	29%	0	0
Atorva 40mg	50%	53%	0	4%	0	0
Baseline Lipids						
Total cholesterol (mg/dL)	199	200	186	189	229	257
LDL-C (mg/dL)	135	133	115	110	152	177
HDL-C (mg/dL)	43	44	44	52	54	50
Triglycerides (mg/dL)	109	115	136	132	116	152

97 subjects screened; 62 randomized

Demographics

	HeFH Atorva Rx		Non-FH Atorva Rx		Non-FH Diet alone	
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Treatment Emergent AEs by Treatment Group

	Placebo HeFH & Non-FH ± Atorva (N=14), %	R727 HeFH Atorva (N=15), %	R727 Non-FH Atorva (N=24), %	R727 Non-FH Diet Only (N=8), %
Serious TEAE	0	0	0	0
D/C due to TEAE	0	0	0	0
Any TEAE	4 (28.6)	7 (46.7)	17 (70.8)	6 (75.0)
Any treatment-related TEAE	1 (7.1)	1 (6.7)	3 (12.5)	4 (50.0)
TEAE by preferred term that occurred in >1 patient/group				
Headache	0	0	6	2
Nasopharyngitis	0	3	2	0
Oropharyngeal pain	0	0	2	0
URTI	0	1	2	0
Viral gastroenteritis	0	2	0	0
Sensation of blood flow	0	0	0	2
Clinically- relevant abnormalities in clinical laboratory values				
CK >5 ULN, < 10 ULN	2	0	2	0
ALT or AST >3 ULN	1	0	0	0

No clinically meaningful injection site reactions.
No clinically meaningful anti-drug antibodies observed.

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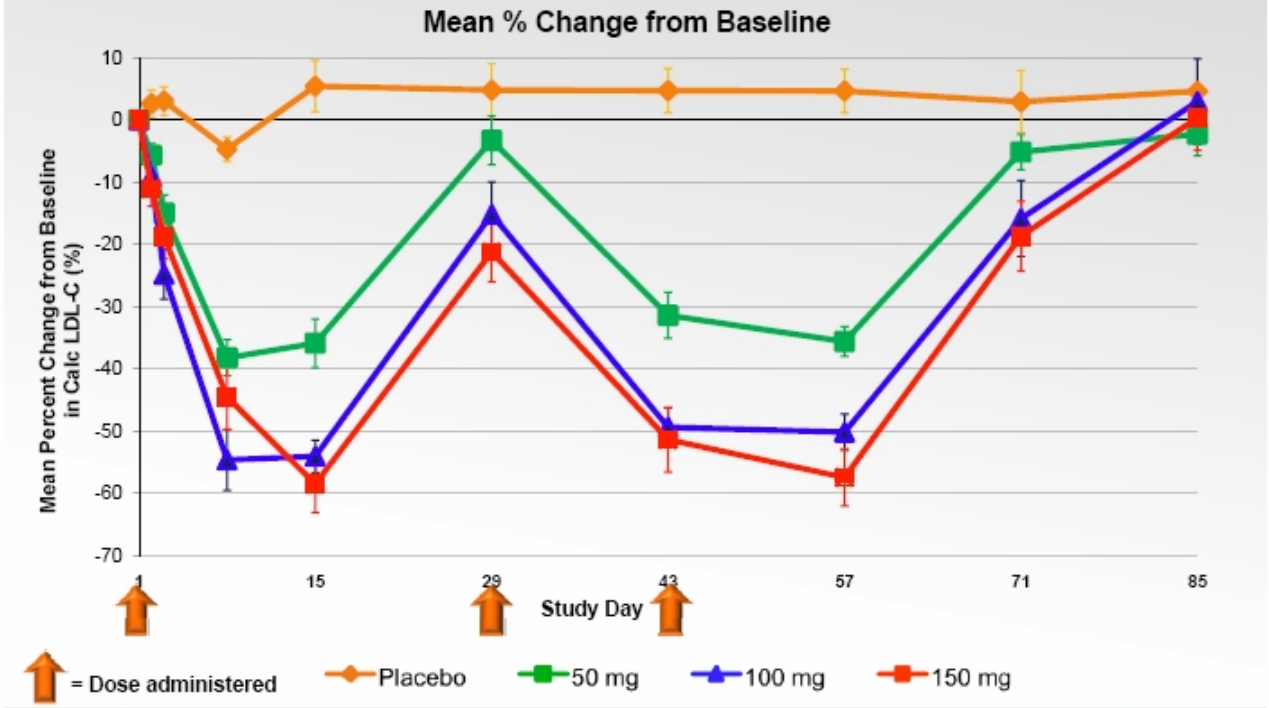
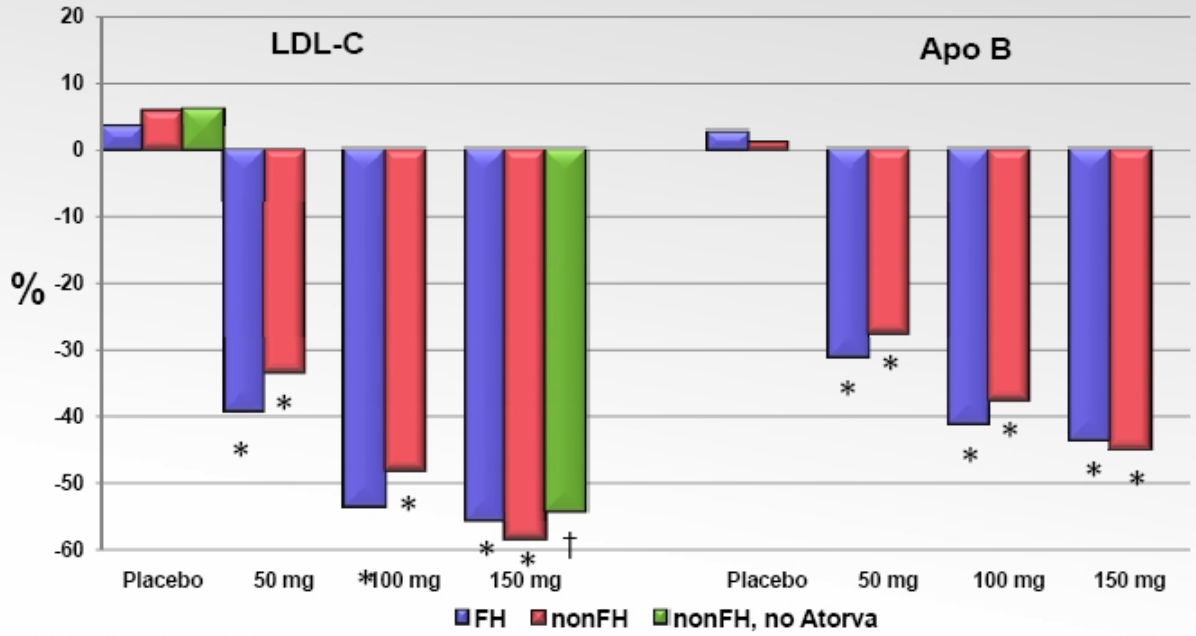
LDL-C Dose Response

Atorvastatin Combo-Rx, heFH & Non-FH Combined



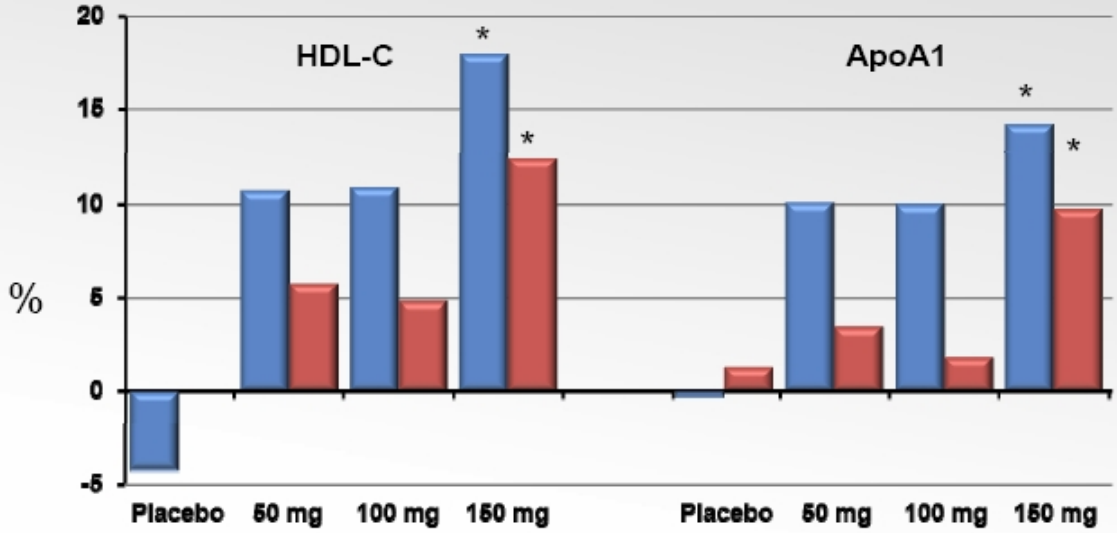
ApoB & LDL-C Response
Mean % Change from Baseline, Day 57



* P < 0.0001 vs. Placebo
† P < 0.01 vs. Placebo

HDL-C and ApoA1 Response

Mean % Change from Baseline, Day 57

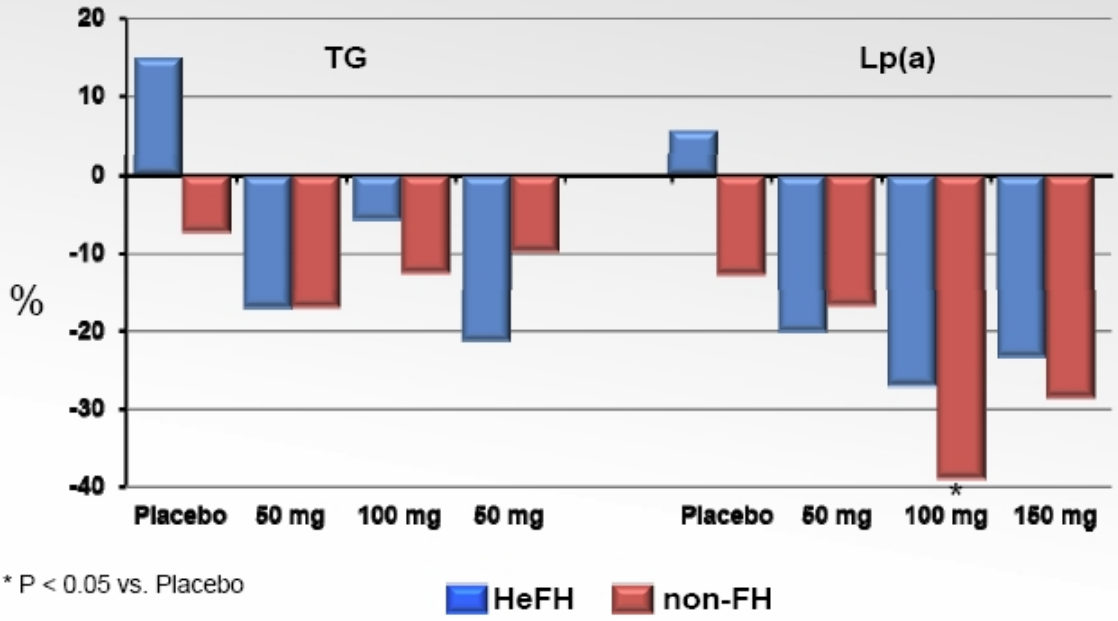


HeFH non-FH

* P < 0.05 vs. Placebo

Triglycerides & Lp(a) Response

Median % Change from Baseline, Day 57



* P < 0.05 vs. Placebo

HeFH non-FH

Conclusions

REGN727/SAR236553 was generally safe and well-tolerated

- No SAE
- No discontinuations for TEAE or any reason
- No apparent hepatotoxicity
- Numerically more TEAEs in REGN 727 treated vs placebo treated patients

Lipid Changes

- LDL-C:
  - 50-60% mean reduction from baseline on top of atorvastatin or as monotherapy
  - Similar lipid and lipoprotein effects in HeFH and nonFH
  - 2-week effect with doses of 100mg and 150 mg REGN727
- Favorable trends (especially in patients receiving atorvastatin):
  - HDL-C/ApoA1
  - Lp(a)
  - TG

Inhibition of PCSK9 is a promising approach for the treatment of hypercholesterolemia

Ongoing Phase II Studies

- Phase II dose ranging trial in HeFH
- Phase II trial in primary hypercholesterolemia with high dose atorvastatin
- Phase II dose ranging trial in primary hypercholesterolmia
- Full details to be presented at future medical conference

Thank You

Pre-Clinical
George D. Yancopoulos
Neil Stahl
Mark Sleeman

Clin Ops
Evelyn Gasparino
Stephanie Biedermann
Rumiana Renard

Translational Med
Scott Mellis

Statistics
Yunling Du
Richard Wu

Medpace Pharmacology Unit
Evan Stein

VRG/NOCCR
William Smith

Our Patient Volunteers