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): February 19, 2020

resTORbio, Inc.
(Exact name of Registrant as Specified in Its Charter)

Delaware	**001-38359**	**81-3305277**
(State or Other Jurisdiction of Incorporation)	**(Commission File Number)**	**(IRS Employer Identification No.)**

500 Boylston Street, 13th Floor	
Boston, MA	**02116**
(Address of principal executive offices)	**(Zip Code)**

Registrant's telephone number, including area code: (857) 315-5528

Not Applicable
(Former Name or Former Address, if Changed Since Last Report)

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:

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

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

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

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

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.0001 per share	**TORC**	**The Nasdaq Global Select Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§ 230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§ 240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD Disclosure.

resTORbio, Inc. (the "Company") from time to time presents and/or distributes to the investment community at various industry and other conferences slide presentations to provide updates and summaries of its business. A copy of its current corporate slide presentation is furnished herewith as Exhibit 99.1 and incorporated herein by reference. The Company undertakes no obligation to update, supplement or amend the materials furnished herewith as Exhibit 99.1.

Item 8.01 Other Events

On February 19, 2020, the Company announced interim results for its Phase 1b/2a trial of RTB101 in patients with Parkinson's disease and provided a corporate update. A copy of the press release is filed herewith as Exhibit 99.2 and incorporated herein by reference.

Item 9.01 Financial Statements and Exhibits

(d) Exhibits

Exhibit Number	Description
99.1	Corporate slide presentation of resTORbio, Inc., dated February 19, 2020.
99.2	Press release issued by resTORbio, Inc. on February 19, 2020.

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: February 19, 2020

resTORbio, Inc.

By: /s/ Chen Schor
Chen Schor
President and Chief Executive Officer

Exhibit 99.1

resTORbio

Targeting the biology of aging to treat aging-related diseases

Corporate Presentation

February 2020

Forward-looking statements

This presentation has been prepared by resTORbio, Inc. (“we,” ”us,” “our,” “resTORbio,” or the “Company”) and is made for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy securities. This presentation may contain “forward-looking statements” within the meaning of the Private Securities Litigation Reform Act of 1995, including, but not limited to, statements regarding the safety, efficacy and regulatory and clinical progress of our product candidates, including RTB101 alone and in combination with a rapalog, such as everolimus or sirolimus. All such forward-looking statements are based on management’s current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially and adversely from those set forth in or implied by such forward-looking statements. The use of words such as, but not limited to, “may,” “might,” “will,” “should,” “expect,” “plan,” “anticipate,” “believe,” “estimate,” “project,” “intend,” “future,” “potential,” or “continue,” and other similar words or expressions are intended to identify forward-looking statements. Forward-looking statements are neither historical facts nor assurances of future performance. Instead, they are based on our current beliefs, expectations and assumptions regarding the future of our business, future plans and strategies, our clinical results and other future conditions. All statements other than statements of historical facts contained in this presentation, including statements regarding future results of operations and financial position, business strategy, current and prospective product candidates, ongoing and planned clinical trials and preclinical activities, including the initiation, timing, enrollment, progress and results of our preclinical and clinical studies and our research and development programs, product approvals, research and development costs, current and prospective collaborations, the timing and likelihood of success of our Phase 1b/2a clinical trial of RTB101, alone or in combination with sirolimus, in Parkinson’s disease and the timing or likelihood of regulatory progress, results, filings and approvals, expectations regarding market acceptance and size, plans and objectives of management for future operations, and future results of anticipated product candidates, are forward-looking statements. New risks and uncertainties may emerge from time to time, and it is not possible to predict all risks and uncertainties. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

These statements are also subject to a number of material risks and uncertainties that are discussed in the section entitled "Risk Factors" in resTORbio’s annual report on Form 10-K for the fiscal year ended December 31, 2018, as well as discussions of potential risks, uncertainties, and other important factors in resTORbio's subsequent filings with the Securities and Exchange Commission. Any forward-looking statement speaks only as of the date on which it was made. Neither we, nor our affiliates, advisors or representatives, undertake any obligation to publicly update or revise any forward-looking statement, whether as a result of new information, future events or otherwise, except as required by law.

Certain information contained in this presentation relates to or is based on studies, publications, surveys and other data obtained from third-party sources and the Company’s own internal estimates and research. While we believe these third-party sources to be reliable as of the date of this presentation, we have not independently verified, and we make no representation as to the adequacy, fairness, accuracy or completeness of any information obtained from third-party sources. In addition, all of the market data included in this presentation involves a number of assumptions and limitations, and there can be no guarantee as to the accuracy or reliability of such assumptions. Finally, while we believe our own internal research is reliable, such research has not been verified by any independent source.

resTORbio highlights

Extensive preclinical data demonstrate that TORC1 inhibition may ameliorate multiple aging related diseases, including neurodegenerative diseases

TORC1 inhibition may be a promising approach for the treatment of Parkinson's disease (PD)

- Ameliorates levodopa-induced dyskinesia in preclinical PD models
- Induces lysosomal biogenesis and autophagy, clears alpha-synuclein aggregates and is neuroprotective in preclinical PD models
- Lead candidate, RTB101, is an oral, selective and potent TORC1 inhibitor that has been observed in preclinical models to cross the blood brain barrier and induce autophagy in neurons

Ongoing Phase 1b/2a clinical trial of RTB101 +/- sirolimus for PD

- Safety, tolerability and cerebrospinal fluid (CSF) exposure data are expected by mid-2020 in PD patients
- RTB101 has the potential to alleviate levodopa-induced dyskinesia and may offer the first opportunity to slow disease progression by inducing autophagy in the brain of PD patients
- In interim data from three cohorts in the Phase 1b/2a study we observed that RTB101 is well tolerated, crosses the blood brain barrier, and reaches concentrations in cerebrospinal fluid observed to inhibit the activity of TORC1 and induce autophagy in neuronal cells
  - Sirolimus at the dose of 2 mg, alone or in combination with RTB101, was not detected in the CSF

Cash, cash equivalents and marketable securities of $117.3 million as of September 30, 2019

Pipeline

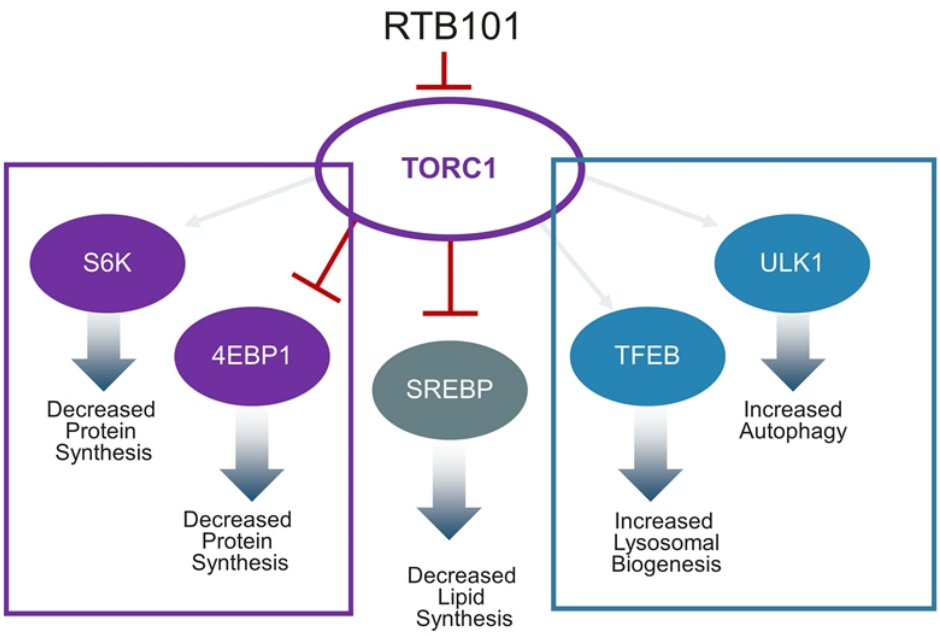
	PROGRAM	INDICATION	DISCOVERY	PRECLINICAL	PHASE 1	PHASE 2	PHASE 3
CURRENT INDICATIONS*	RTB101 or RTB101 + sirolimus	Parkinson's Disease				PHASE 1B/2A ONGOING	
POTENTIAL INDICATIONS**	RTB101 or RTB101 + rapalog	Neurodegenerative Diseases					
	RTB101 or RTB101 + rapalog	Diseases associated with TORC1 hyperactivation					

* For Parkinson's disease, we may be required to file an investigational new drug application, or IND, with the U.S. Food and Drug Administration, prior to initiating Phase 2 clinical trials

** For neurodegenerative diseases and diseases associated with TORC1 hyperactivation, subject to review by the U.S. Food and Drug Administration, we believe we may have the ability to initiate Phase 2 clinical trials without the need to conduct additional Phase 1 trials.

Broad TORC1 inhibition with RTB101 has the potential to ameliorate levodopa-induced dyskinesia and to be neuroprotective in Parkinson's disease patients

Decreased phosphorylation of S6K and 4EBP1 decrease protein synthesis and are associated with amelioration of levodopa-induced dyskinesia in preclinical PD models



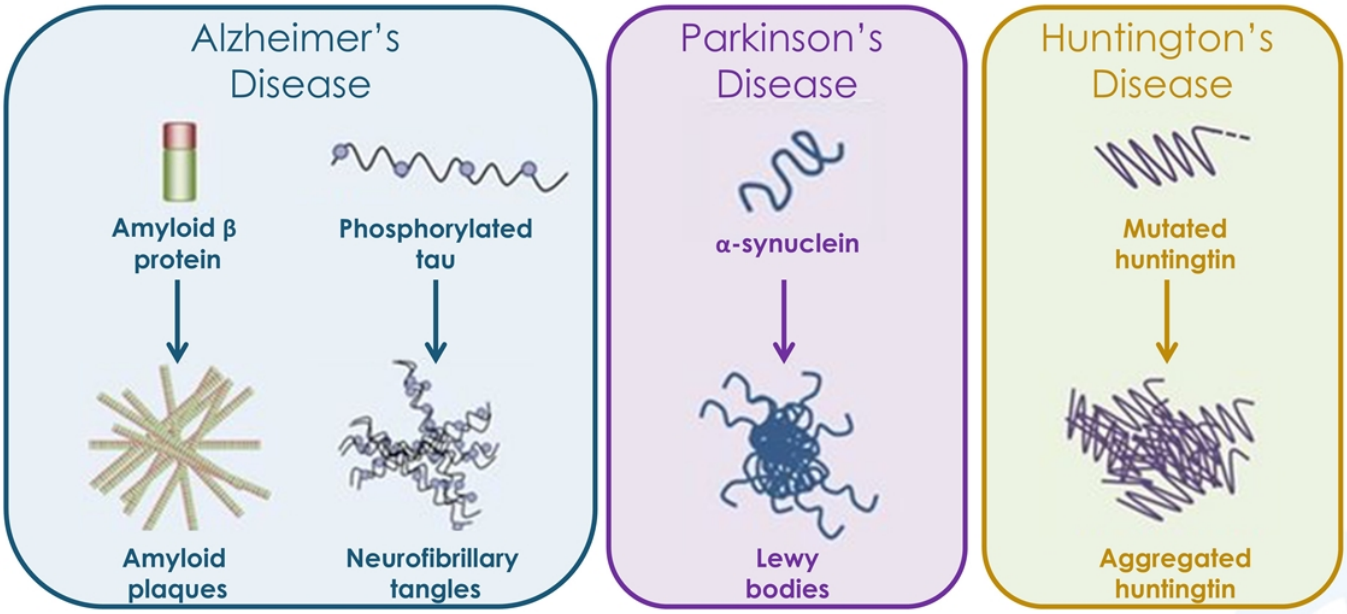
Activation of ULK1 and TFEB have been shown to promote autophagy and lysosomal biogenesis which are associated with neuroprotection in preclinical PD models

Nyfeler et al. *Molecular and Cellular Biology*, 2011; Nyfeler et al. *PLoS ONE*, 2012; Eid et al. *PNAS*, 2017; Roczniak-Ferguson et al. *Sci Signal*, 2012; Santini et al. *Sci Signaling*, 2009

Neurodegenerative Diseases

Parkinson's Disease: Disease modification

Protein aggregation is a common pathogenic mechanism in aging-related neurodegenerative diseases



Parkinson's Disease

Prevalence

- Second most common neurodegenerative disease
- Affects 1% of population over 55 years of age in the U.S.

Pathology

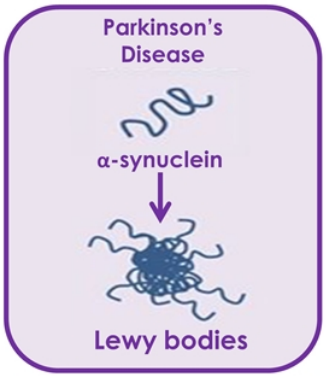
- Accumulation of Lewy body protein aggregates containing a-synuclein and death of the neurons that produce the neurotransmitter dopamine in the substantia nigra

Clinical manifestations

- Four cardinal motor symptoms:
  - Resting tremor
  - Bradykinesia (slowed movement)
  - Muscle rigidity
  - Postural instability

Current therapies treat symptoms of PD but do not alter disease progression

- Levodopa is used to treat PD; however, its effect tends to wear off over time and can lead to disabling levodopa-induced dyskinesia



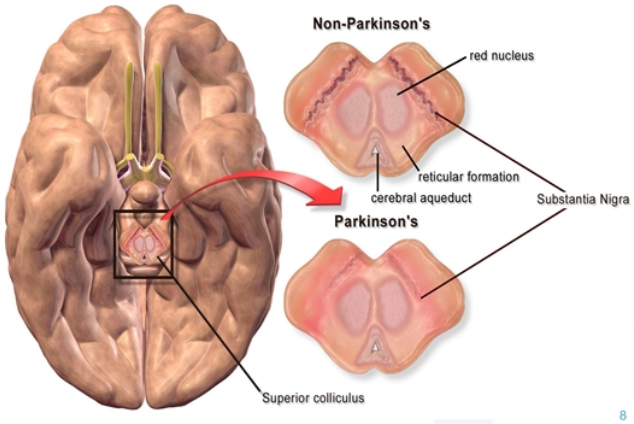
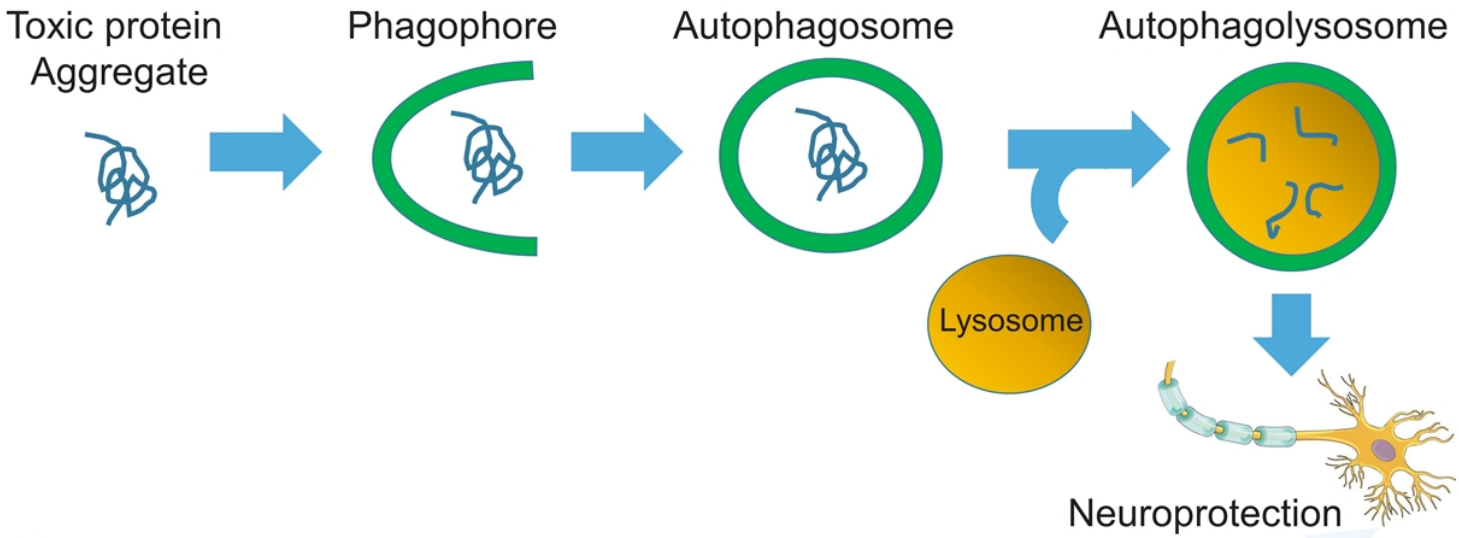


Image from Wikiwand

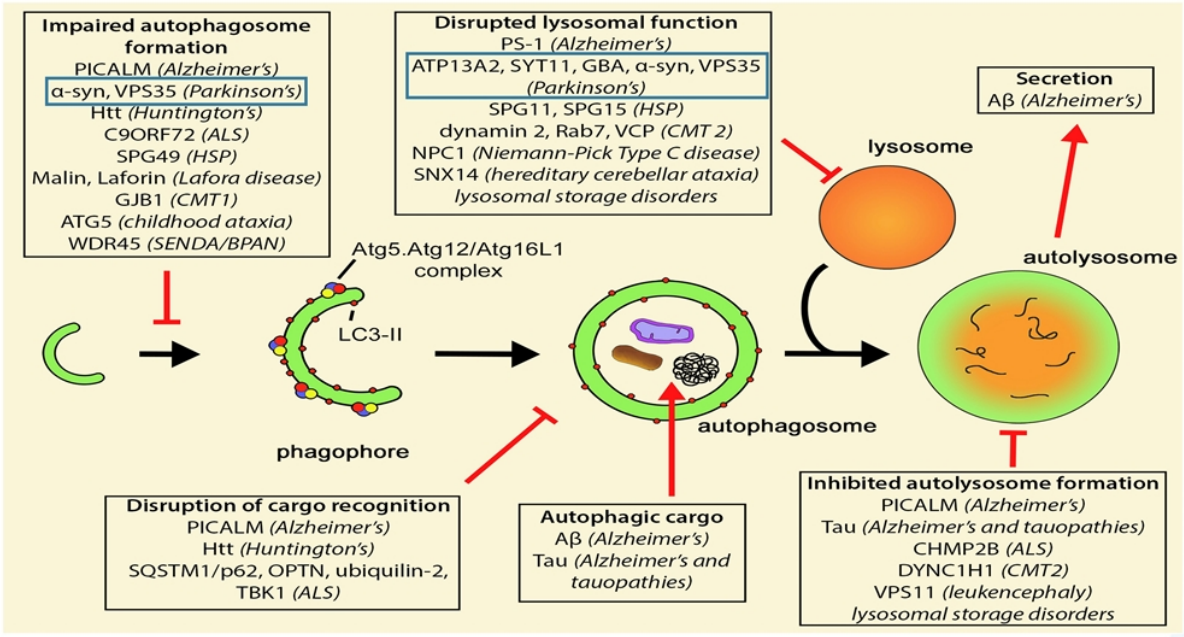
Defective autophagy may contribute to the accumulation of aggregated proteins in PD and other neurodegenerative diseases

Autophagy is a mechanism by which aggregated misfolded proteins and dysfunctional organelles are broken down and recycled in cells



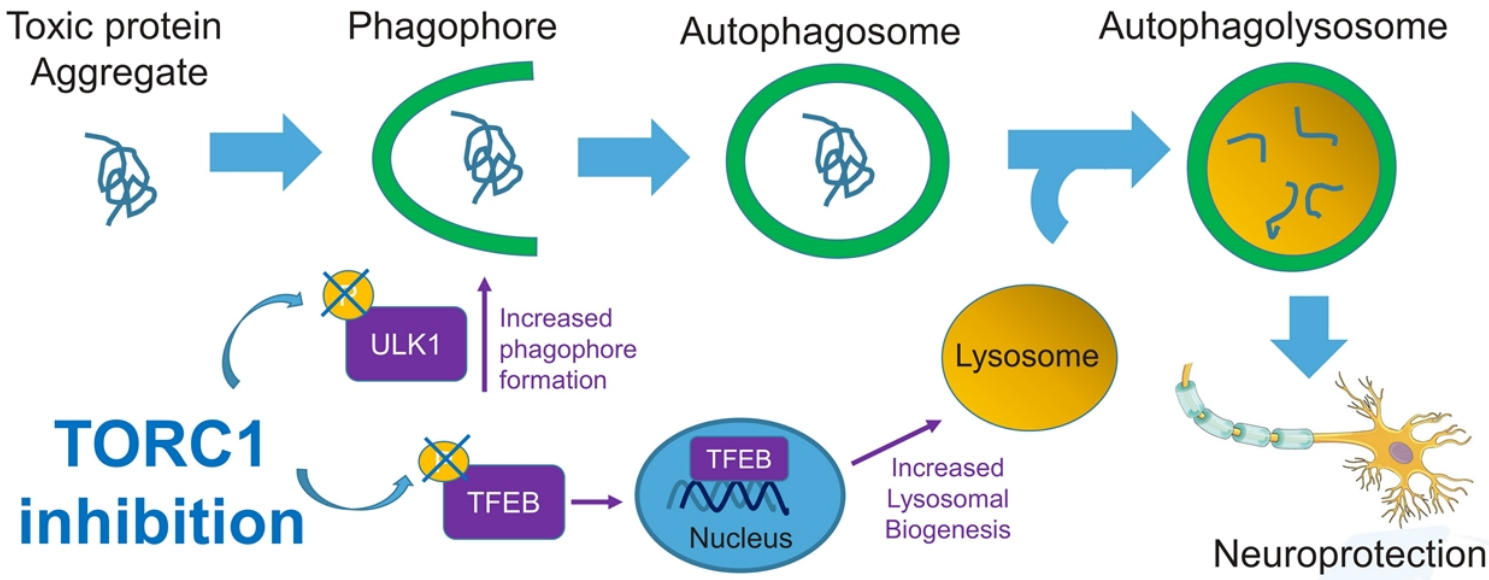
resTORbio Menzies et al. *Neuron*, 2017

Mutations in autophagy-related proteins are found in PD and other neurodegenerative diseases suggesting that deficient autophagy contributes to neurodegenerative disease pathogenesis



Menzies et al. *Neuron*, 2017

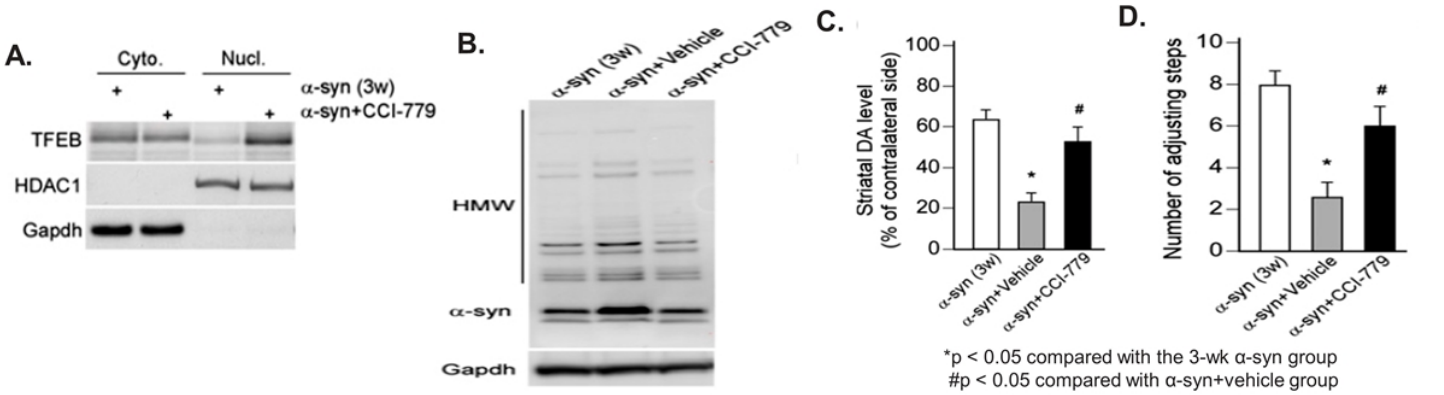
TORC1 inhibitors have the potential to be disease modifying in neurodegenerative diseases by upregulating autophagy and lysosomal biogenesis



 Menzies et al. *Neuron*, 2017; Roczniak-Ferguson et al. *Sci Signal*, 2012; Nyfeler et al. *Molecular and Cellular Biology*, 2011

TORC1 inhibition is neuroprotective in PD pre-clinical models

Increased TFEB nuclear translocation → **Clearance of toxic a-syn aggregates** → **Improved Neuronal Survival** → **Improved Motor Performance**



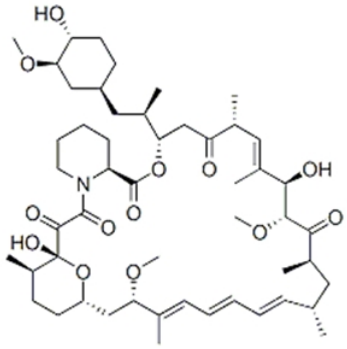
*p < 0.05 compared with the 3-wk α-syn group
#p < 0.05 compared with α-syn+vehicle group

In a rat PD model that overexpresses a-syn in the substantia nigra, the TORC1 inhibitor CCI-779 started 3 weeks after adenoviral delivery of a-syn (3w) and given every other day for 5 weeks was shown **(A)** to correct impaired TFEB function (as reflected by increased TFEB nuclear translocation), **(B)** decreased striatal a-syn levels (both monomeric and high molecular weight (HMW) aggregates), **(C)** increased dopaminergic neuron survival and **(D)** improved motor function.

resTORbio Decressac et al. *PNAS*, 2013

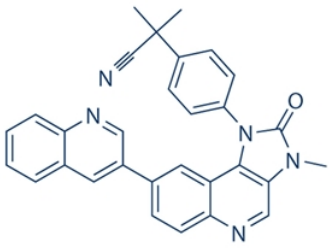
TORC1 inhibitors under evaluation in a Phase 1b/2a trial in Parkinson's disease

sirolimus (rapamycin):
- Allosteric inhibitor of TORC1
- Partial TORC1 inhibitor: only consistently inhibits S6K downstream of TORC1
- Approved for use in humans



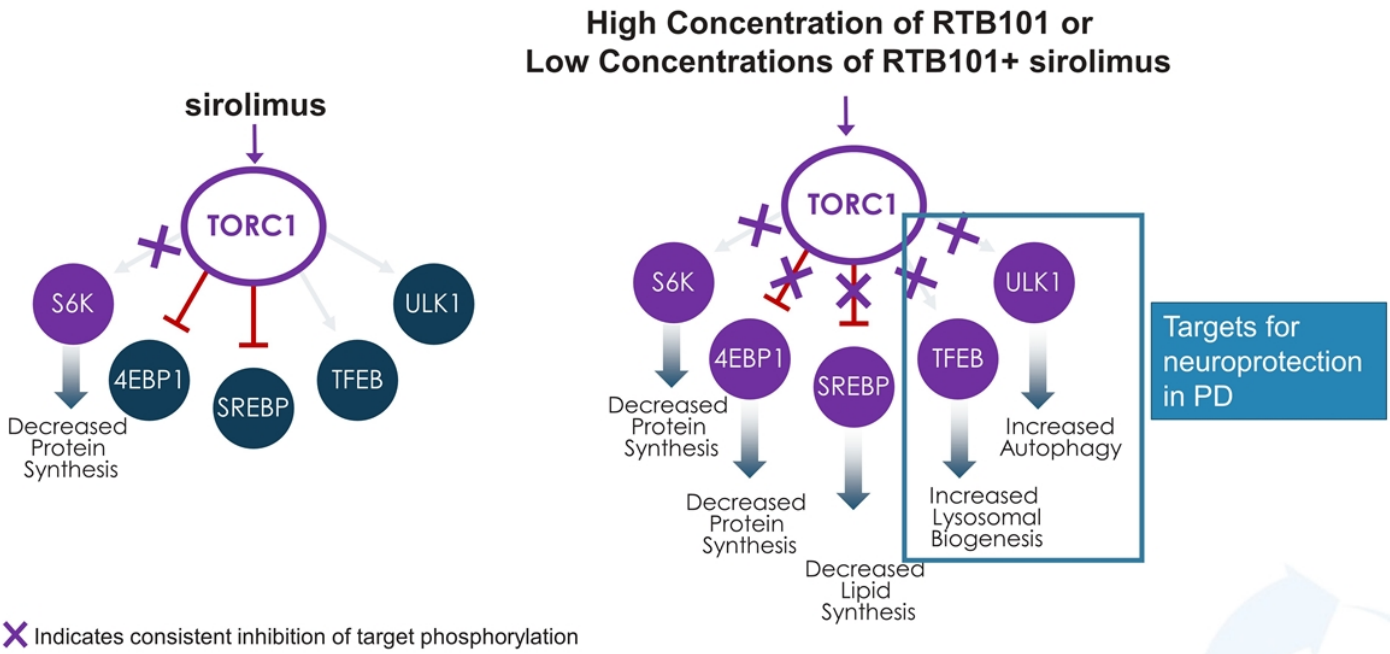
sirolimus (rapamycin)

RTB101:
- ATP competitive catalytic site inhibitor of mTOR protein kinase
- Consistently inhibits phosphorylation of all targets downstream of TORC1
- Crosses the blood brain barrier in animal models
- Tested in >1,000 humans
- Human maximum tolerated dose: 1,200 mg/day



RTB101

Potential spectrum of TORC1 inhibition with RTB101 and sirolimus



✕ Indicates consistent inhibition of target phosphorylation

Nyfeler et al. *Molecular and Cellular Biology*, 2011; Nyfeler et al. *PLoS ONE,* 2012; Eid et al. *PNAS*, 2017; Roczniak-Ferguson et al. *Sci Signal*, 2012

The combination of RTB101 and sirolimus synergize to inhibit TORC1 and may lower the RTB101 brain exposure required to induce autophagy

SK-N-SH neuroblastoma cells were exposed to increasing concentrations of RTB101 (y axis) and/or increasing concentrations of sirolimus (x axis). Orange shaded areas indicate concentrations of RTB101 and/or sirolimus that induce >50% maximal autophagy activation. Autophagocytic flux was measured using an mCherry-GFP-LC3 cell-based assay

% maximal activation of autophagy

RTB101 (nM)

Total	Free (5%)									
87.50	4.38	130.58	114.37	156.80	170.61	173.28	181.56	196.15	174.61	158.67	216.07
43.75	2.19	91.48	71.47	123.06	118.25	166.88	154.73	189.63	194.12	190.70	214.89
21.88	1.09	31.89	25.16	81.50	100.98	125.12	137.82	212.58	197.37	166.87	218.33
10.94	0.55	0.02	4.25	29.25	41.45	88.97	138.95	155.32	184.65	146.93	179.15
5.47	0.27	-12.14	-14.12	-1.11	8.36	44.22	81.09	103.23	143.72	120.56	123.57
2.73	0.14	-12.10	-6.71	-0.19	-1.19	25.53	43.99	75.14	96.76	73.48	100.10
1.37	0.07	-7.40	-17.37	0.03	0.09	13.03	29.69	41.98	54.65	60.23	68.35
0.68	0.03	-23.25	-25.36	3.41	-2.42	5.87	16.31	26.84	52.55	33.51	31.80
0.34	0.02	-16.81	-28.70	-7.67	-5.83	5.18	14.95	9.42	33.10	21.35	43.68
0	0	-11.63	-20.72	-6.80	-6.46	-1.54	9.74	2.82	13.34	10.25	-4.02

Free (2.5%)	0	0.000053	0.000214	0.000854	0.003418	0.013672	0.054688	0.218750	0.875000	3.5
Total	0	0.002136	0.008545	0.034180	0.136719	0.546875	2.187500	8.75	35.00	140.00

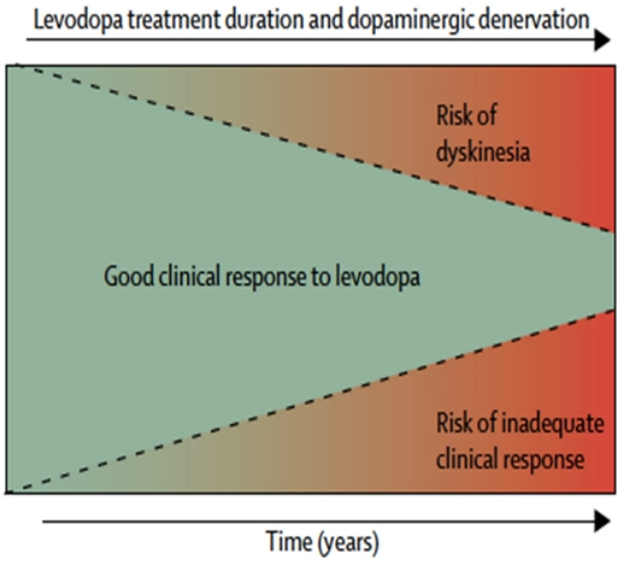
sirolimus (nM)

RTB101 300 mg monotherapy cerebrospinal fluid concentration observed in PD patients

Neurodegenerative Diseases

Parkinson's Disease: Levodopa-induced dyskinesia

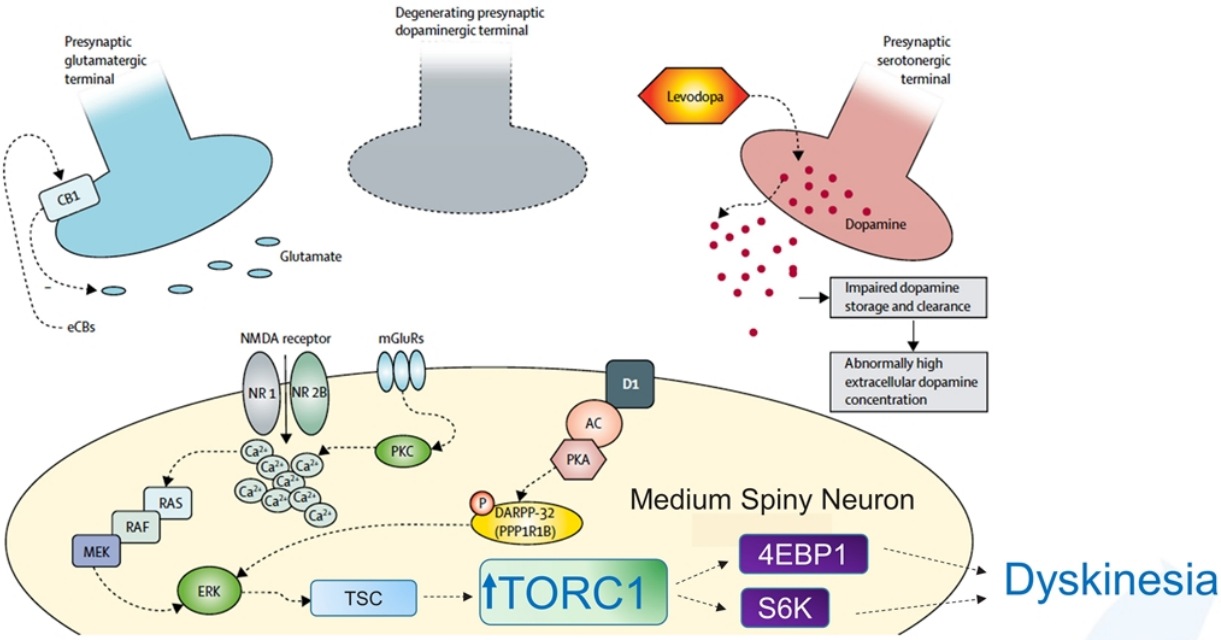
Advantage of PD as a clinical indication for TORC1 inhibitors: levodopa-induced dyskinesia (LID) is a potential clinical endpoint that can be assessed in a shorter time frame than disease progression



- LID is a disabling side effect of chronic levodopa treatment associated with significantly impaired quality of life and increased health care costs[1]
- Most common dyskinesias:
  - chorea (jerky involuntary movements, especially around the face, shoulders, and hips)
  - dystonia (involuntary muscle contraction leading to twisting, repetitive movements, or abnormal posture in affected muscle or muscle group)
- Average time to onset of dyskinesia estimated at 6.5 years[1]
- > 90% of PD patients will have LID after 15 years of levodopa therapy[3]

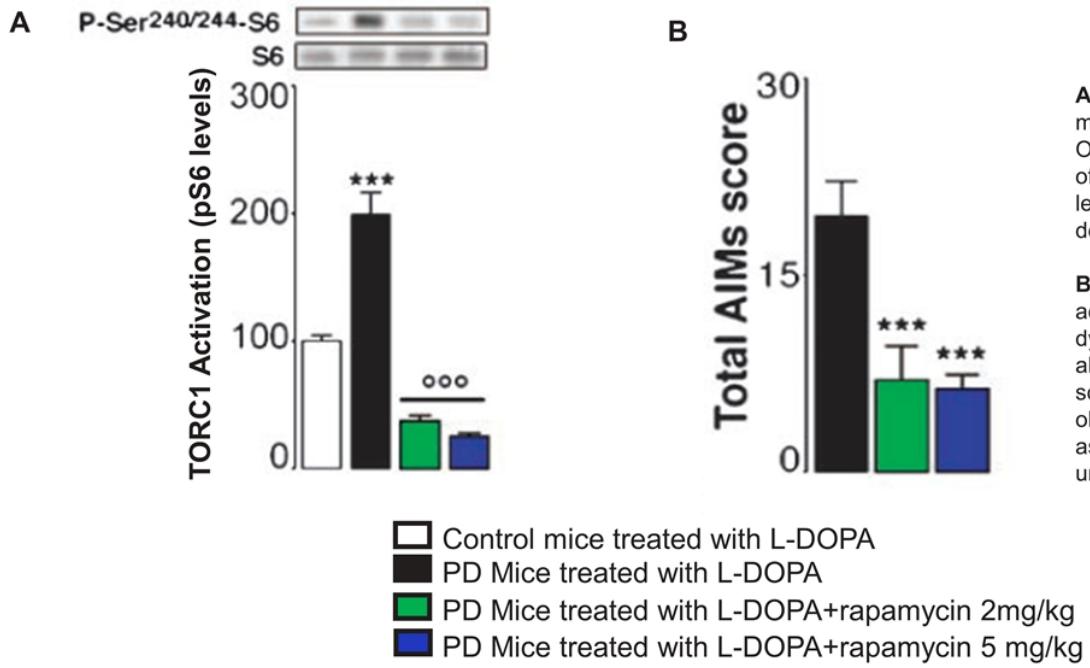
 [1] and figure Calabresi *et al*., 2010; [2] Tran *et al*., 2018; [3] Ahlskog and Muenter 2001 and Hely *et al* 2005

Multiple pathways implicated in levodopa-induced dyskinesia (LID) pathogenesis converge on ERK and downstream TORC1 hyperactivation



Adapted from Calabrisi P et al., Lancet Neurol, 2010 Santini et al. *Sci Signaling*, 2009

TORC1 is hyperactivated in medium spiny neurons in preclinical PD models of LID and TORC1 inhibition alleviates LID



A: Administration of levodopa in a mouse model of PD (unilateral 6-OHDA lesion) led to hyperactivation of TORC1(as assessed by pS6 levels) in medium spiny neurons and development of dyskinesia.

B: Rapamycin inhibited TORC1 activation and ameliorates dyskinesia, as assessed by an abnormal involuntary movement score (AIMs), determined by an observer blind to treatment assignment. ^{ooo},*** P<0.001 versus untreated control.

Control mice treated with L-DOPA
PD Mice treated with L-DOPA
PD Mice treated with L-DOPA+rapamycin 2mg/kg
PD Mice treated with L-DOPA+rapamycin 5 mg/kg

Santini et al. *Sci Signaling*, 2009

resTORbio Phase 1b/2a Parkinson's disease trial

Design	**Randomized, Placebo-Controlled Phase 1b/2a Study (4-week dosing)** • Mild-moderate PD patients (mH&Y I-III) • On standard of care PD drugs • Once weekly dosing
Study Size	N=45 (2:1 randomization)
Key Endpoints	**Primary endpoint:** • Safety and tolerability **Secondary endpoint:** • Exposure in blood, plasma and CSF **Exploratory endpoints:** • Biomarkers in plasma and CSF • Clinical assessments, wearables

Cohort	RTB 101 dose (mg)	sirolimus dose (mg)
1	300	0
2	0	2
3	300	2
4	300	4
5	300	6

or matching placebo

- **Study initiated in 1Q19**
- **Data expected by mid-2020**

RTB101 CSF Concentrations in 6 PD patients dosed with RTB101 300 mg once weekly

The combination of RTB101 and sirolimus synergize to inhibit TORC1 and may lower the RTB101 brain exposure predicted to be required for clinical benefit in PD

SK-N-SH neuroblastoma cells were exposed to increasing concentrations of RTB101 (y axis) and/or increasing concentrations of sirolimus (x axis). Orange shaded areas indicate concentrations of RTB101 and/or sirolimus that induce >50% maximal S6K or 4EBP1 phosphorylation inhibition or >50% maximal autophagy activation

% maximal Inhibition of **pS6K**

Total	Free (5%)										
87.50	4.38	100	99	104	99	101	104	102	101	105	99
43.75	2.19	97	101	100	102	95	102	104	104	102	103
21.88	1.09	84	86	99	98	100	101	104	102	99	104
10.94	0.55	51	62	79	99	102	104	103	104	105	104
5.47	0.27	26	38	54	91	98	104	103	103	105	104
2.73	0.14	12	30	39	74	97	102	103	104	104	104
1.37	0.07	8	18	27	56	89	101	103	103	104	102
0.68	0.03	14	12	30	49	85	101	103	105	103	104
0.34	0.02	6	7	21	49	79	100	104	104	105	103
0	0	6	12	16	45	73	98	103	103	104	103

% maximal Inhibition of **p4EBP1**

Total	Free (5%)										
87.50	4.38	55	57	65	73	79	87	93	97	99	96
43.75	2.19	25	33	39	54	65	72	83	71	77	89
21.88	1.09	5	16	13	28	38	62	62	68	62	76
10.94	0.55	-6	-7	-7	0	13	28	36	40	45	51
5.47	0.27	-7	-12	-21	-44	-8	6	13	12	25	30
2.73	0.14	-12	-10	-8	-10	-12	-2	-6	1	9	18
1.37	0.07	-9	-10	-10	-22	-25	-9	-4	-3	6	5
0.68	0.03	-7	-6	-4	-16	-33	-10	-6	-3	5	4
0.34	0.02	-17	-15	-11	-21	-31	-17	-9	-10	9	0
0	0	-16	-15	-8	-13	-25	-11	-9	-10	1	-3

% maximal activation of **autophagy**

Total	Free (5%)										
87.50	4.38	130.58	114.37	156.80	170.61	173.28	181.56	196.15	174.61	158.67	216.07
43.75	2.19	91.48	71.47	123.06	118.25	166.88	154.73	189.63	194.12	190.70	214.89
21.88	1.09	31.89	25.16	81.50	100.98	125.12	137.82	212.58	197.37	166.87	218.33
10.94	0.55	0.02	4.25	29.25	41.45	88.97	138.95	155.32	184.65	146.93	179.15
5.47	0.27	-12.14	-14.12	-1.11	8.36	44.22	81.09	103.23	143.72	120.56	123.57
2.73	0.14	-12.10	-6.71	-0.19	-1.19	25.53	43.99	75.14	96.76	73.48	100.10
1.37	0.07	-7.40	-17.37	0.03	0.09	13.03	29.69	41.98	54.65	60.23	68.35
0.68	0.03	-23.25	-25.36	3.41	-2.42	5.87	16.31	26.84	52.55	33.51	31.80
0.34	0.02	-16.81	-28.70	-7.67	-5.83	5.18	14.95	9.42	33.10	21.35	43.68
0	0	-11.63	-20.72	-6.80	-6.46	-1.54	9.74	2.82	13.34	10.25	-4.02

Free (2.5%)	0	0.000053	0.000214	0.000854	0.003418	0.013672	0.054688	0.218750	0.875000	3.5
Total	0	0.002136	0.008545	0.034180	0.136719	0.546875	2.187500	8.75	35.00	140.00

RTB101 (nM)

sirolimus (nM)

RTB101 300 mg monotherapy cerebrospinal fluid concentration observed in PD patients

Target inhibition for levodopa-induced dyskinesia

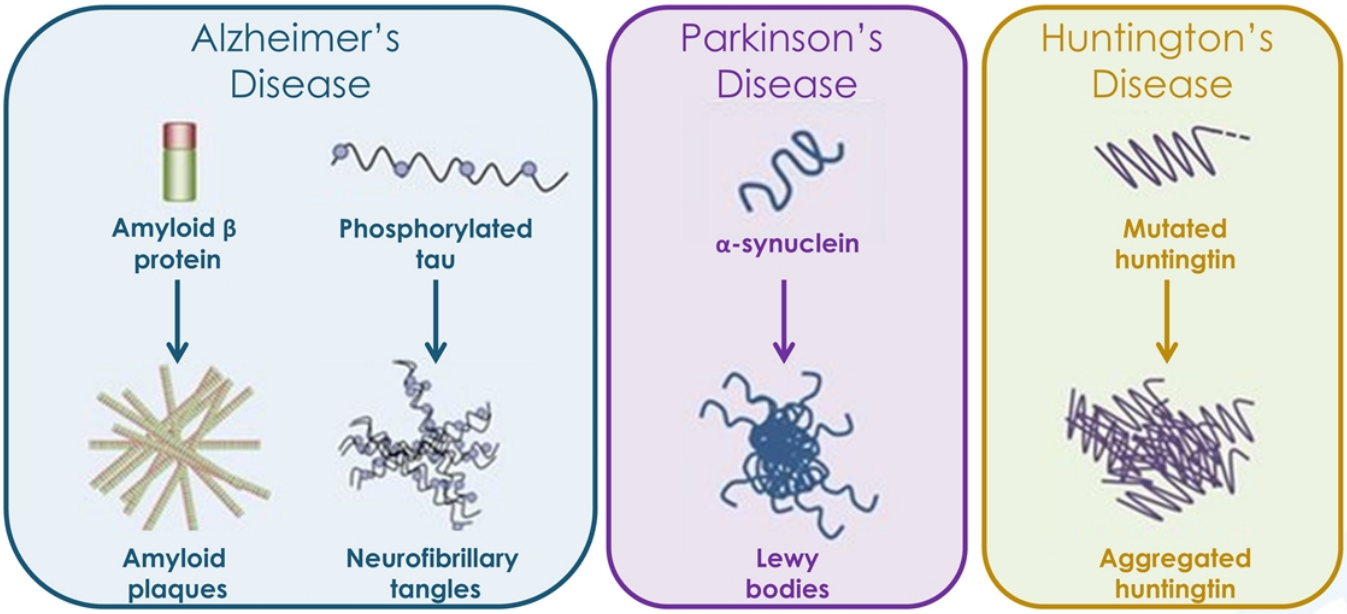
Target inhibition for neuroprotection and disease modification

resTORbio

Neurodegenerative Diseases

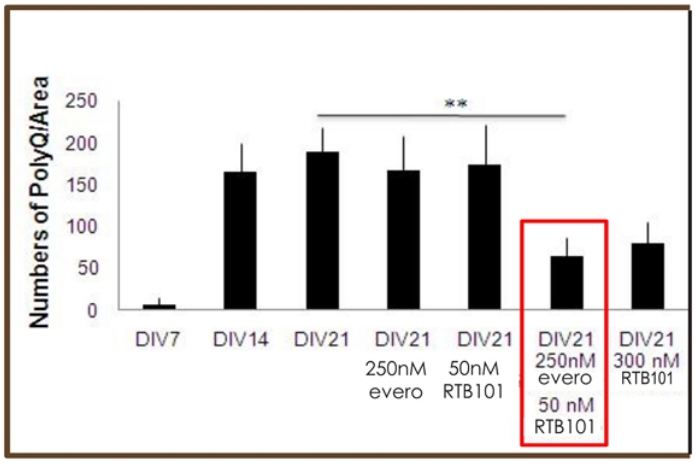
Huntington's disease

Induction of autophagy with TORC1 inhibitors may have potential benefit in multiple neurodegenerative diseases in which protein aggregation contributes to disease pathogenesis

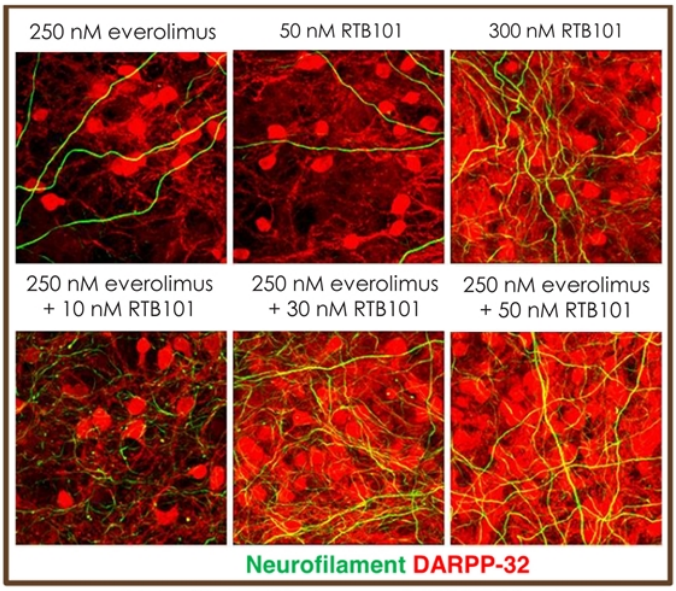


In a preclinical model of Huntington's disease, the combination of RTB101 and everolimus (an analog of sirolimus) cleared protein aggregates and was neuroprotective

Aggregated mHtt protein levels in cultured cortico-striatal slices from R6/2 Huntington's disease mouse.



Drug concentrations in the figures are total concentrations



Neurofilament is a marker of axons

DARPP-32 is a marker of cell soma

resTORbio highlights

Extensive preclinical data demonstrate that TORC1 inhibition may ameliorate multiple aging related diseases, including neurodegenerative diseases

TORC1 inhibition may be a promising approach for the treatment of Parkinson's disease (PD)

- Ameliorates levodopa-induced dyskinesia in preclinical PD models
- Induces lysosomal biogenesis and autophagy, clears alpha-synuclein aggregates and is neuroprotective in preclinical PD models
- Lead candidate, RTB101, is an oral, selective and potent TORC1 inhibitor that has been observed in preclinical models to cross the blood brain barrier and induce autophagy in neurons

Ongoing Phase 1b/2a clinical trial of RTB101 +/- sirolimus for PD

- Safety, tolerability and cerebrospinal fluid (CSF) exposure data are expected by mid-2020 in PD patients
- RTB101 has the potential to alleviate levodopa-induced dyskinesia and may offer the first opportunity to slow disease progression by inducing autophagy in the brain of PD patients
- In interim data from three cohorts in the Phase 1b/2a study we observed that RTB101 is well tolerated, crosses the blood brain barrier, and reaches concentrations in cerebrospinal fluid observed to inhibit the activity of TORC1 and induce autophagy in neuronal cells
  - Sirolimus at the dose of 2 mg, alone or in combination with RTB101, was not detected in the CSF

Cash, cash equivalents and marketable securities of $117.3 million as of September 30, 2019


resTORbio
Targeting the biology of aging
to treat aging-related diseases
Corporate Presentation
February 2020

resTORbio Announces Interim Results for Phase 1b/2a Trial of RTB101 in Patients with Parkinson's Disease and Provides Corporate Update

- Interim data from three cohorts in the Phase 1b/2a study demonstrate that RTB101 is well tolerated, crosses the blood brain barrier, and reaches concentrations in cerebrospinal fluid observed in preclinical models to inhibit the activity of TORC1 and induce autophagy in neuronal cells —
- TORC1 Inhibition in the brain with RTB101 may offer a new treatment paradigm for multiple neurodegenerative diseases associated with the accumulation of protein aggregates, such as Parkinson's, Huntington's and Alzheimer's disease —

Boston, MA, Feb 19, 2020 – resTORbio (Nasdaq: TORC) today announced interim results from the ongoing Phase 1b/2a trial of RTB101, an orally-administered small molecule potent target of rapamycin complex 1 (TORC1) inhibitor product candidate, alone or in combination with sirolimus, in Parkinson's disease (PD).

"We are pleased to have observed that RTB101 is well tolerated and crosses the blood brain barrier in Parkinson's disease patients at concentrations that have the potential to induce autophagy, the process by which cells break down toxic misfolded protein aggregates. Preclinical data suggest that induction of autophagy has the potential to slow the progression not only of Parkinson's disease but also of multiple other neurodegenerative diseases that are characterized by the accumulation of toxic protein aggregates in cells such as Huntington's and Alzheimer's disease," said Dr. Joan Mannick, Co-Founder and Chief Medical Officer of resTORbio.

Phase 1b/2a Trial of RTB101 alone and in combination with sirolimus in Parkinson's disease interim results

The multicenter, 2:1 randomized, double-blind, placebo-controlled Phase 1b/2a trial is evaluating the safety and tolerability of RTB101 alone or in combination with escalating doses of sirolimus (2 mg, 4 mg and 6 mg) once weekly for 4 weeks in patients with Parkinson's disease. To date, patients have been enrolled in three cohorts and dosed once weekly with 300 mg of RTB101 alone, 2 mg of sirolimus alone, or a combination of 300 mg RTB101 and 2 mg of sirolimus. Results of the interim study analysis indicated that all 3 dosing regimens were well tolerated and RTB101 300 mg once weekly was observed to cross the blood brain barrier. The concentrations of RTB101 in cerebrospinal fluid (CSF) in subjects dosed with RTB101 300 mg once weekly monotherapy were higher than expected and based on preclinical models, have the potential to induce autophagy in the brain. Sirolimus at the dose of 2 mg, alone or in combination with RTB101, was not detected in the CSF. Data from the first three cohorts in the study suggest that the concentrations of RTB101 observed in the CSF four hours after dosing were highest when RTB101 was given as a monotherapy. Enrollment of the RTB101 300 mg in combination with sirolimus 4 mg once weekly cohort is ongoing.

Corporate Update

The company has initiated a process to evaluate external opportunities, such as partnerships, acquisitions, mergers and other financial and strategic alternatives to maximize shareholder value. The company has engaged JMP Securities LLC to act as a strategic advisor for this process. There can be no assurance that this strategic review process will result in the company pursuing any transaction or that any transaction, if pursued, will be completed. The company has not set a timetable for completion of this strategic review process, and the company does not intend to comment further unless or until its Board of Directors has approved a definitive course of action, the review process is concluded, or it is determined other disclosure is appropriate.

About Parkinson's Disease

Parkinson's disease (PD) is a progressive neurodegenerative disease that affects approximately 7.5 million people worldwide. The incidence of PD increases rapidly in people 60 years of age and older, with a mean age at diagnosis of 70.5 years. Inhibition of TORC1 has been shown in multiple preclinical models to ameliorate neurodegenerative diseases including Parkinson's disease. TORC1 inhibition with RTB101 alone or in combination with sirolimus, may provide a therapeutic benefit to PD patients by ameliorating levodopa-induced dyskinesia and/or by inducing autophagy which leads to the breakdown of protein aggregates and improved neuronal survival.

About RTB101

RTB101 is an oral, selective, and potent TORC1 inhibitor product candidate that inhibits the phosphorylation of multiple targets downstream of TORC1. Inhibition of TORC1 has been observed to extend lifespan and healthspan in aging preclinical species and to improve the function of aging organ systems, including neurologic function, suggesting potential benefits in several aging-related diseases.

About resTORbio

resTORbio, Inc. is a clinical-stage biopharmaceutical company developing innovative medicines that target the biology of aging to treat aging-related diseases. resTORbio's lead program selectively inhibits TORC1, an evolutionarily conserved pathway that contributes to the decline in function of aging organ systems, including neurologic function. Learn more about resTORbio, Inc. at www.resTORbio.com.

Forward Looking Statements

This press release contains forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995, as amended. Investors are cautioned that statements in this press release which are not strictly historical statements, including, without limitation, our proposed timing and anticipated results of our Phase 1b/2a clinical trial of RTB101 alone or in combination with sirolimus in patients with mild to severe Parkinson's disease, including the announcement of interim results; our future plans to develop RTB101 alone or in combination with rapalogs, such as everolimus or sirolimus, including the therapeutic potential and clinical benefits thereof; our expectations on the potential patient populations that may be addressed by our product candidates; our ability to replicate results achieved in our clinical trials in any future trials; and our engagement of JMP Securities LLC and our plans to explore and evaluate strategic alternatives and external opportunities, constitute forward-looking statements identified by words such as, but not limited to, "believe," "expect," "may," "will," "should," "seek," "anticipate," or "could" and similar words or expressions.

Any forward-looking statements in this statement are based on management's current expectations of future events and are subject to a number of risks and uncertainties that could cause actual results to differ materially from those anticipated, including, without limitation, risks associated with: our ongoing Phase 1b/2a clinical trial of RTB101 alone or in combination with sirolimus in Parkinson's disease, including the announcement of interim results; the timing and anticipated results of our clinical trials; the risk that the results of our clinical trials will be predictive of future results in connection with future clinical trials; our ability to explore and evaluate strategic alternatives and external opportunities, the timing and outcome of our planned interactions with regulatory authorities; and obtaining, maintaining and protecting our intellectual property as well as those risks more fully discussed in the section entitled "Risk Factors" in the Annual Report on Form 10-K filed by resTORbio, Inc. with the Securities and Exchange Commission, as well as discussions of potential risks, uncertainties, and other important factors in our subsequent filings with the Securities and Exchange Commission. In addition, any forward-looking statements are neither historical facts nor assurances of future performance. Instead, they represent our beliefs, expectations, assumptions and views only as of today and should not be relied upon as representing our beliefs, expectations, assumptions and views as of any subsequent date. resTORbio explicitly disclaims any obligation to update any forward-looking statements. No representations or warranties (expressed or implied) are made about the accuracy of any such forward-looking statements.

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