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 17, 2019

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

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

	300 Boyiston Street, 13th	11001					
	Boston, MA (Address of principal executive	02116 (Zip Code)					
	(Address of principal executive	offices)	(Zip Code)				
	Registrant's telepho	one number, including area code:	(857) 315-5528				
	(Former Name	Not Applicable or Former Address, if Changed Since Las	t Report)				
	ck the appropriate box below if the Form 8-K filing is into wing provisions:	ended to simultaneously satisfy the	filing obligation of the registrant under any of the				
	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))						
eci	urities registered pursuant to Section 12(b) of the Act:						
	Title of each class	Trading Symbol(s)	Name of each exchange on which registered				
C	ommon Stock, par value \$0.0001 per share	TORC	The Nasdaq Global Select Market				
	cate by check mark whether the registrant is an emerging oter) or Rule 12b-2 of the Securities Exchange Act of 193		: 405 of the Securities Act of 1933 (§ 230.405 of this				
me	erging growth company 🗵						
	n emerging growth company, indicate by check mark if the						

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 (the "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 9.01 Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number

Description

99.1 <u>Corporate slide presentation of resTORbio, Inc., dated November 17, 2019.</u>

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 18, 2019

resTORbio, Inc.

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



Forward-looking statements

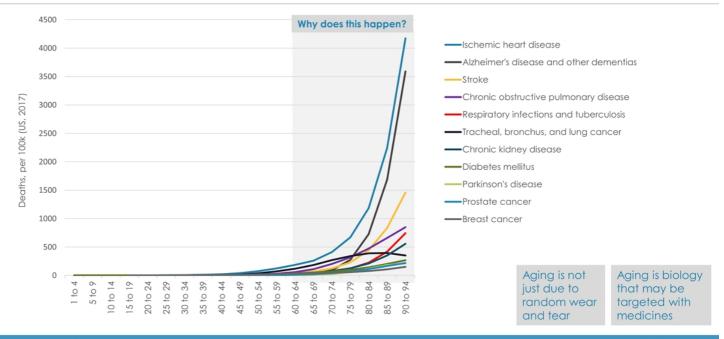
This presentation has been prepared by resTORbio, Inc. ("we," "us," "our," "resTORbio," or the "Company") and is made for informational purposes only and does not constitute an offer to sell or a solicitation of an offer to buy securities. This presentation may contain "forward-looking statements" within 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 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, fiming, 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 clinic

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 fillings 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.

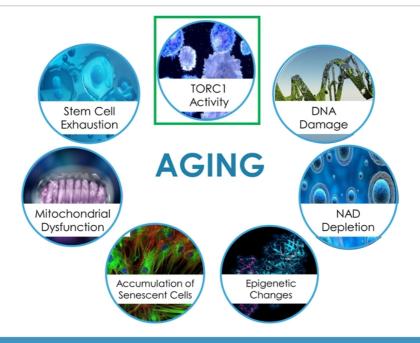


Aging is the biggest risk factor for most chronic diseases



Global Burden of Disease Collaborative Network. Global Burden of Disease Study 2017 (GBD 2017) Results

Well-characterized pathways associated with aging and aging-related diseases



TORC1 Pathway:

TORC1 is an evolutionarily conserved pathway that regulates aging

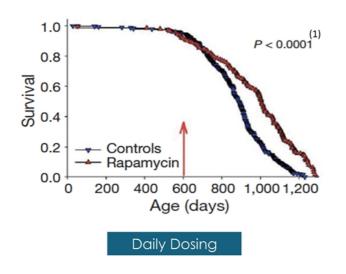


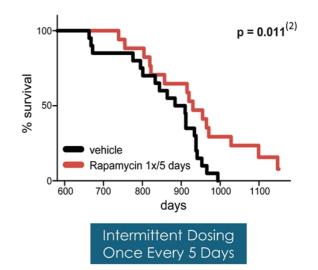


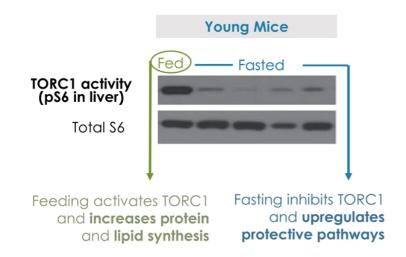
TORC1 inhibition extended lifespan and healthspan in multiple preclinical species

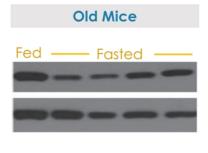
Lamming et al. Journal of Clinical Investigation, 2013

TORC1 inhibitors extend lifespan in mice even when started late in life and given intermittently









TORC1 activity remained aberrantly elevated during fasting, which may **prevent upregulation** of protective pathways

Inhibition of TORC1 has the potential to improve the function of multiple aging organ systems

Improved Neurologic Function

Tain et al., Nature Neuroscience, 2009 Malagelada et al., J Neurosci, 2010 Spilman et al., PLoS ONE, 2010 Halloran et al., Neuroscience, 2012 Majumder et al., Aging Cell, 2012 Neff et al., JCI, 2013

Reversal of aging-related cardiac dysfunction

Flynn et al., Aging Cell, 2013 Dai et al., Aging Cell, 2014 Chiao et al., Aging, 2016



Reversal of agingrelated immune dysregulation

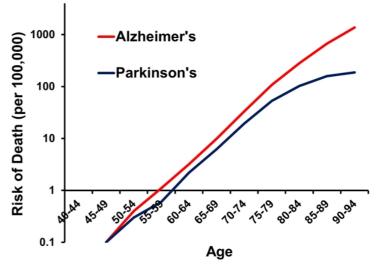
Chen et al., Science Sig, 2009 Selman et al., Science, 2011 Neff et al., JCI, 2013 Hurez et al., Aging Cell, 2015

Improvement in physical activity

Selman et al., Science, 2011 Harrison et al., Nature, 2009 Wilkinson et al., Aging Cell, 2014 Flynn et al., Aging Cell, 2013

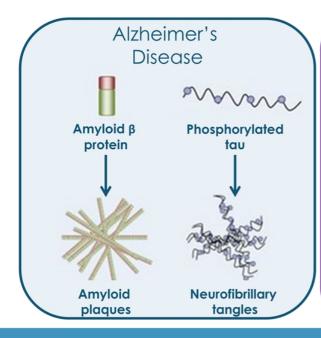
Age is the greatest risk factor for neurodegenerative disease

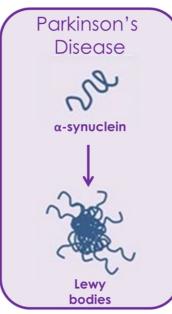


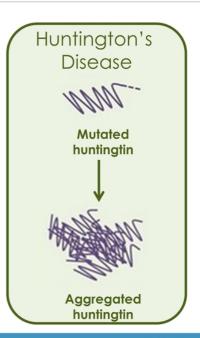


http://www.spring.org.uk

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

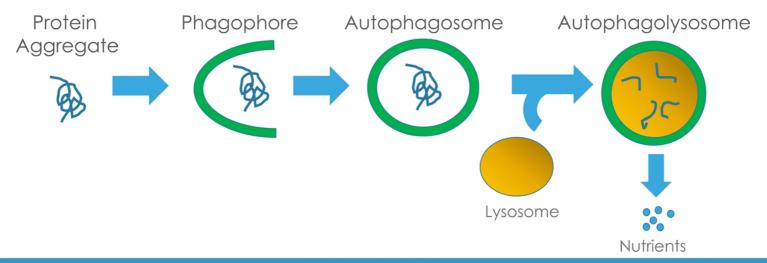






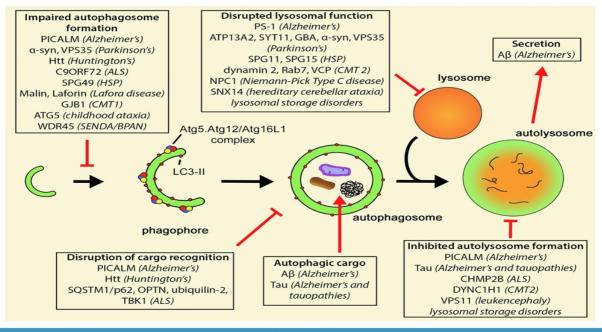
Defective autophagy may contribute to the accumulation of aggregated proteins in neurodegenerative diseases

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



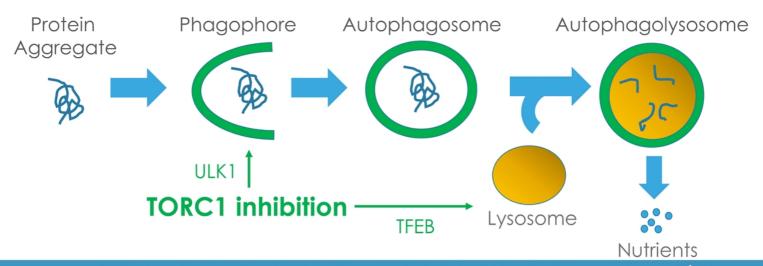
Menzies et al. Neuron, 2017

Mutations in autophagy-related proteins are found in multiple neurodegenerative diseases



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

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



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

Parkinson's Disease

Prevalence:

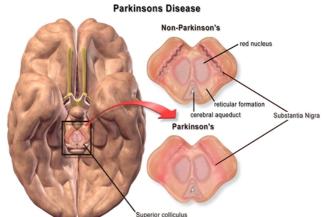
 Parkinson's disease (PD) is the second most common neurodegenerative disease and affects 1% of population over 55 years of age

Pathobiology:

 PD is characterized by loss of >50% of the neurons that produce the neurotransmitter dopamine in a specific area of the brain (substantia nigra)

Clinical manifestations:

- Four cardinal motor symptoms:
 - Resting tremor
 - Bradykinesia (slowed movements)
 - Muscle rigidity
 - Postural instability
- All current therapies treat symptoms of PD but do not alter disease progression



TORC1 inhibitors may be of therapeutic benefit in Parkinson's disease

Induction of autophagy with a TORC1 inhibitor leads to the clearance of α -synuclein aggregates in a preclinical PD model

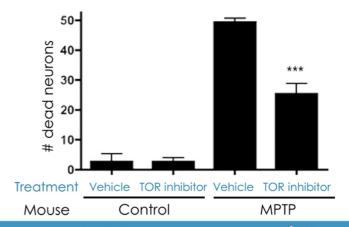
Mouse: Control Vehicle a-syn Transgenic Torcal inhibitor

A B C C

a-synuclein staining (brown)

LC3 autophagosome staining (marker of autophagy activation)

TORC1 inhibition prevents neuronal loss and improves motor function in multiple PD preclinical models



Malagelada et al. J Neurosci, 2010; Crews et al. PLoS one, 2010

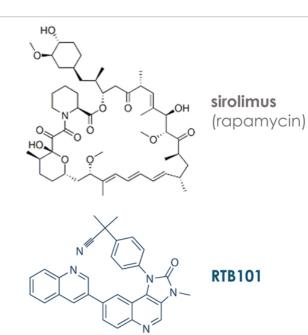
TORC1 Inhibitors that will be evaluated in a Phase 1b/2a trial in Parkinson's Disease

SIrolimus:

- Allosteric inhibitor of TORC1
- Consistently inhibits phosphorylation of only some targets downstream of TORC1
- Approved for use in humans

RTB101:

- ATP competitive catalytic site inhibitor of mTOR protein kinase
- Inhibits phosphorylation of all targets downstream of TORC1
- · May have advantages over rapalogs for PD
- Crosses the blood brain barrier in preclinical models

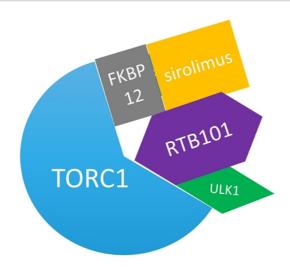


RTB101 and sirolimus synergize to induce autophagy in neuronal cells

	Total	Free (5%)	>80% Maximal Induction of Autophagy									
	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
(mM)	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
RTB101	2.73	0.14	-12.10	-6.71	-0.19	-1.19	25.53	43.99	75.14	96.76	73.48	100.10
R	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
	Fre	e (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)									

SK-N-SH neuroblastoma cell line assay

Potential Mechanism Underlying Synergistic Inhibition and Autophagy Activation by sirolimus + RTB101



 Sirolimus may induce a conformation change in TORC1 that allows lower concentrations of RTB101 to inhibit TORC1

Adapted from Nyfeler et al. PloS one, 2012

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

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

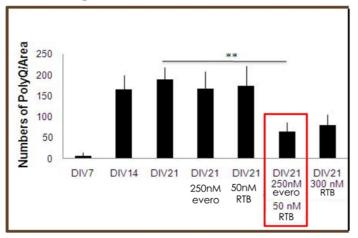
Cohort	RTB 101 dose (mg)	Sirolimus dose (mg)
1	300	0
2	0	2
3	300	2
4	300	4
5	300	6

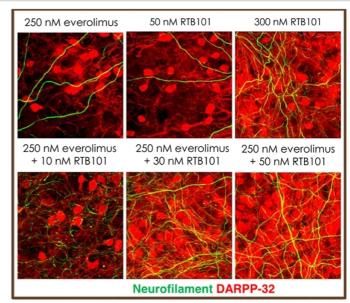


- Study initiated in 1Q19
- Data expected in mid-2020

The combination of RTB101+ a rapalog may have potential benefit in other neurodegenerative diseases including Huntington's disease

Aggregated protein levels in cultured brain slices from a Huntington's Disease mouse model





Neurofilament is a marker of axons

DARPP-32 is a marker of cell soma

Source: Novartis Data on tile

Summary

- Protein aggregation is a common pathogenic mechanism underlying multiple neurodegenerative diseases
- Induction of autophagy may have therapeutic benefit in neurodegenerative diseases by clearing toxic protein aggregates
- In preclinical models, TORC1 inhibition with RTB101 alone or in combination with a rapalog induces autophagy
- Phase 1b/2a study or RTB101 alone and in combination with sirolimus is underway

