WHAT IS CLAIMED IS:

1. Use of N-acetyl-leucine in the manufacture of a medicament for treating the prodromal phase of an alpha-synucleinopathy in a subject in need thereof, wherein a therapeutically effective amount of N-acetyl-leucine is to be administered to the subject, wherein the subject:

(i) is not diagnosed with any of the core clinical features, syndromes, or symptoms associated with the alpha-synucleinopathy; and

(ii) is diagnosed with one or more core clinical features, syndromes, or symptoms associated with the prodromal phase of the alpha-synucleinopathy,

wherein the alpha-synucleinopathy is Parkinson's disease (PD), dementia with Lewy bodies (DLB), or multiple systems atrophy (MSA).

2. The use of claim 1, wherein the subject:

(i) is not diagnosed with tremor, bradykinesia, muscle stiffness, impaired balance, and/or impaired coordination; and

(ii) is diagnosed with one or more of hyposmia, cognitive impairment, REM-sleep behavior disorder (RBD), constipation, depression, and/or anxiety.

3. The use of claim 1, wherein the subject:

(i) is not diagnosed with dementia; and

(ii) is diagnosed with one or more of RBD, constipation, hyposmia, depression, and/or orthostatic hypotension/dizziness, or

wherein the subject:

(i) is not diagnosed with (a) an autonomic dysfunction; and (b) poorly L-dopa-responsive parkinsonism and/or cerebellar syndrome; and

(ii) is diagnosed with one or more of RBD, neurogenic orthostatic hypotension within 10 minutes of standing or head-up tilt, and/or urogenital failure.

4. The use of any one of claims 1-3, wherein the subject has decreased dopaminergic function in the basal ganglia.

5. The use of any one of claims 1-4, wherein the N-acetyl-leucine increases the dopaminergic function in the subject as compared to prior to administration of the N-acetyl-leucine.

6. The use of claims 4 or 5, wherein the amount of dopaminergic function in the subject is determined by a dopamine transporter (DAT) scan.

7. The use of any one of claims 1-6, wherein the subject has decreased glucose metabolism in the brain, and/or

wherein the subject has decreased glucose metabolism in the parietal, occipital, and/or frontal regions of the brain, or

wherein the subject has decreased glucose metabolism in the occipital association cortex and primary visual area of the brain, or

wherein the subject has decreased glucose metabolism in the putamen nuclei, anterior cerebellar hemispheres, and/or the vermis.

8. The use of any one of claims 1-7, wherein the N-acetyl-leucine increases the glucose metabolism in the brain of the subject as compared to the glucose metabolism in the subject prior to administration of the N-acetyl-leucine, and/or

wherein the N-acetyl-leucine increases the glucose metabolism in the parietal, occipital, and/or frontal regions of the brain of the subject as compared to the glucose metabolism in the subject prior to administration of the N-acetyl-leucine, or

wherein the N-acetyl-leucine increases the glucose metabolism in the occipital association cortex and primary visual area of the brain of the subject as compared to the glucose metabolism in the subject prior to administration of the N-acetyl-leucine, or

wherein the N-acetyl-leucine increases the glucose metabolism in the putamen nuclei, anterior cerebellar hemispheres, and/or the vermis of the subject as compared to the glucose metabolism in the subject prior to administration of the N-acetyl-leucine.

9. The use of claims 7 or 8, wherein the glucose metabolism is determined by a positron emission tomography (PET) scan, preferably

wherein the PET scan is a fluorodeoxyglucose-positron emission tomography (FDG-PET) scan, preferably

wherein the FDG-PET scan is used to generate the subject's z-score.

10. The use of claim 9, wherein the N-acetyl-leucine reduces the subject's z-score in the parietal, occipital, and/or frontal regions of the brain, or

wherein the N-acetyl-leucine reduces the subject's z-score in the medial occipital lobes, lateral occipital lobes, occipital association cortex, and/or primary visual area of the brain, or

wherein the N-acetyl-leucine reduces the subject's z-score in the putamen nuclei, anterior cerebellar hemispheres, and/or the vermis, and/or

wherein the subject's z-score is reduced by at least about 5%, about 10%, about 20%, about 30%, about 40%, or about least 50% compared to the subject's z-score prior to administration of the N-acetyl-leucine.

11. The use of any one of claims 1-10, wherein the N-acetyl-leucine reduces the number of RBD episodes as compared to the number of RBD episodes prior to administration of the N-acetyl-leucine, and/or

wherein the subject is diagnosed with hyposmia, preferably

wherein hyposmia is diagnosed with one or more of a 12-item Brief Smell Identification Test (B-SIT) and sniffin sticks test (SST).

12. Use of N-acetyl-leucine in the manufacture of a medicament for treating REM-sleep behavior disorder (RBD) in a subject in need thereof, wherein a therapeutically effective amount of N-acetyl-leucine is to be administered to the subject.

13. The use of claim 12, wherein the subject has been diagnosed with prodromal Parkinson's disease (PD), prodromal dementia with Lewy bodies (DLB), or prodromal multiple systems atrophy (MSA), or

wherein the subject has not been diagnosed with classical Parkinson's disease (PD), classical dementia with Lewy bodies (DLB), or classical multiple systems atrophy (MSA).

14. The use of any one of claims 1-13, wherein 1 g to 15 g of the N-acetyl-leucine is to be orally administered to the subject per day, preferably

wherein 1 g to 10 g of the N-acetyl-leucine is to be administered to the subject per day, and more preferably

wherein 1 g to 5 g of the N-acetyl-leucine is to be administered to the subject per day.

15. The use of any one of claims 1-14, wherein the N-acetyl-leucine is N-acetyl-DL-leucine or N-acetyl-L-leucine.