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Motor and Activity Psychosis-Risk (MAP-R) Scale: An exploration of scale structure with replication and validation

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Abstract

**Background:** Motor abnormalities precede and predict the onset of psychosis. Despite the practical utility of motor abnormalities for early identification, prediction, and individualized medicine applications, there is currently no dedicated self-report instrument designed to capture these important behaviors. The current study assessed and validated a questionnaire designed for use in individuals at clinical high-risk for psychosis (CHR).

**Methods:** The current study included both exploratory (n=3,009) and validation (n=439) analytic datasets– that included individuals at CHR (n=84) who completed the novel Motor Abnormalities and Psychosis-Risk (MAP-R) Scale, clinical interviews and a finger tapping task. The structure of the scale and reliability of items were consistent across two analytic datasets. The resulting scales were assessed for discriminant validity across CHR, community sample non-psychiatric volunteer, and clinical groups.

**Results:** The scale showed a consistent structure across independent data points across two analytic datasets subscale structure. The resultant subscale structure was consistent with conceptual models of motor pathology in psychosis (coordination and dyskinesia) in both the exploratory and the validation analytic dataset. Further, these subscales showed discriminant, predictive and convergent validity. The motor abnormality scales discriminated CHR from community sample non-psychiatric controls and clinical samples. Finally, these subscales predicted to risk calculator scores and showed convergent validity with motor performance on a finger tapping task.

**Conclusion:** The MAP-R scale demonstrated good internal validity, discriminant validity, predictive validity, and convergent validity, and subscales map on to conceptually relevant motor circuits. This scale showed great promise in characterizing a novel area of detection of psychosis risk.
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Introduction

Motor abnormalities have been found to precede and predict the onset of psychosis.\textsuperscript{1–8} These motor abnormalities are tied to cognitive deficits, functional outcomes and other core features of psychotic disorders.\textsuperscript{9–14} Taken together with a growing body of supporting structural,\textsuperscript{15–18} connective, and functional imaging results,\textsuperscript{19–21} imply that motor features reflect important vulnerability and disease driving mechanism.\textsuperscript{11–13,22} Relatedly, motor symptoms may be useful in forming distinct clinical high-risk (CHR) subtypes.\textsuperscript{19} Motor behaviors may also be useful in monitoring side effects,\textsuperscript{23} promoting treatment planning,\textsuperscript{24} and tracking treatment outcome.\textsuperscript{25} In addition to motor abnormalities, sedentary behavior and level of aerobic activity are also important risk/protective indicators for individuals at CHR.\textsuperscript{26–29} Despite the clear importance of motor symptoms and activity, at the present time there is no dedicated self-report measure for assessing them in psychosis risk populations. Current motor abnormality measures often require clinical training and equipment,\textsuperscript{30,31} making current testing may be impractical for widespread clinical use. Additionally, extant self-report and clinical motor assessments tend to assess a limited number of motor abnormalities, but psychotic disorders are characterized by numerous motor abnormalities\textsuperscript{31} of clinical importance (e.g. conversion, subtyping).\textsuperscript{1,19,32} This paucity of motor abnormality items in clinical assessments limits the power to detect relevant motor abnormalities related to CHR symptoms, conversion, and subtyping.

Motor abnormalities have been found to reflected an early vulnerability to individuals at CHR for psychosis. Studies of infants that later develop psychosis in adulthood suggest that coordination deficits, delays, and dyskinesias characterize an early vulnerability.\textsuperscript{3} Similar work in people at CHR suggested that coordination deficits and delays in motor learning reflect cerebellar circuit dysfunction,\textsuperscript{17,21,33} whereas slowing as well as hyperkinetic movements reflect
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basal ganglia circuit pathology. Notably, these circuits are relevant to prominent conceptual theories of psychosis, and relatedly, both domains predicted worsening course, poor functional outcomes, and ultimate conversion to psychosis. Further, neuroimaging studies in individuals at CHR have refined our understanding of neural underpinnings, as well as the links with disease-relevant mechanisms. With respect to physical activity, individuals at CHR show decreased levels of physical activity; level of physical activity has been linked to abnormalities in the hippocampus among other regions. Indeed, exercise interventions aimed at increasing physical activity have shown evidence of increased cognition, reduced symptoms, and improved functional connectivity in the hippocampus. Due to the predictive quality, motor abnormalities and physical activity may be particularly useful to assess psychosis risk.

Despite its promise motor functioning has been largely under-utilized in the early assessment of psychosis risk; some assessments include no motor items and others only including one or two items. Indeed motor abnormalities are suggested to be a unique and independent feature of psychosis risk. Thus, adding a motor measure to the standard psychosis risk assessment may increase sensitivity and provide added benefit to prediction algorithms for conversion to psychosis. The current study developed a novel Motor Abnormalities and Psychosis-Risk (MAP-R) Scale. First, we examined structure of responses replicated across two analytic datasets; the exploratory dataset -a larger (n=3,009) screening analytic dataset who completed the MAP-R scale (as part of a larger non-motor battery) and only completed phase one of the study- and the scale validation analytic dataset- a smaller independent data points that completed additional validation measures during phase two of the study (n=439). Then we reported the psychometrics statistics (reliability) of the resulting
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subscales established by the response structure across both analytic datasets. The resultant subscales were used in subsequent analyses to probe the discriminant, predictive, and convergent validity of these scales. Discriminant validity was examined by assessing the degree to which these scales differed between relevant CHR groups compared to other disorders (depression and anxiety) and a community control sample. Convergent validity was assessed by relating scale items to motor performance during a motor neurocognitive task (i.e., finger tapping task). Finally, to examine predictive validity, these scales were related to the calculated risk for psychosis (SIPS-RC).20,62

Methods and Materials

Participants. Participants were recruited as a part of a large, multi-site community sample known as the Multisite Assessment of Psychosis-Risk study. The primary focus of that study was to evaluate markers of risk for psychosis in a large, representative community sample across multiple study sites. Study sites included the greater catchment areas of Philadelphia (Temple University), Chicago (Northwestern University), and Baltimore (University of Maryland-Baltimore County). Recruitment occurred through various outlets including ads on various internet sites (e.g., Craigslist, Facebook), student volunteer pools, refer-a-friend links, and flyers. Recruitment centered on non-clinical sources, therefore no recruitment took place at clinical locations such as outpatient psychiatric clinics and hospitals in an attempt to keep the community sample relatively unbiased.

MAP Study – Screening Phase. The study contained two phases, Figure 1. The first phase was completed by all 3,448 participants and included an online battery of measures, including established psychosis risk questionnaires- e.g., the prodromal questionnaire56 (PQ), the PRIME Screen58- and a variety of other questionnaires, including the Motor Abnormalities
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Psychosis Risk Scale. The first phase identified individuals at probable high or probable low-risk for psychosis. Notably the sites did not differ in sample demographics, such as biological sex at birth ($p=.19$) or age ($p=.38$). A random sample balanced across probable low and probable high-risk groups was invited for the second phase. Participants were labeled as questionnaire high-risk for psychosis if they endorsed eight or more distressing items on the PQ and two items with a distress rating of 5 or one item as a 6 on the PRIME scale. Subjects who did not meet either threshold were randomly selected from below these cut-offs and treated as questionnaire low-risk. The second phase also had additional inclusion criteria including participants must be proficient in English, between the ages of 16 to 30, and have normal or corrected to normal vision. Additional exclusion criteria included either an inability to provide consent for the second phase or an unwillingness to attend an in-person study visit. For the current analyses, the presence of a current psychotic diagnosis ($n=3$, as assessed during an in-person visit) was also an exclusion criterion. To preserve the representativeness of the community sample, there were no other exclusion criteria.

MAP Study – Validation Phase. The second, validation phase was an in-person visit at the university study site that included a variety of relevant and discriminant measures to validate the current scale. These validation measures were collected within a larger study battery that included clinical assessments using both the Structured Interview for Psychotic Risk (SIPS) and the Structured Clinical Interview for DSM-5 Research Version (SCID-5-RV). During the clinical assessment individuals were classified as clinical high-risk for psychosis (CHR) if they met one of the following criteria: attenuated positive symptoms or genetic risk and deterioration of function. Attenuated positive symptom criteria was determined by the SIPS guidelines. Finally, genetic risk and deterioration of function required that a participant have a first degree
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relative with a diagnosis with a psychotic disorder or schizotypal personality disorder and that reported that they experienced functional decline in the past year, resulting in 85 individuals being classified as CHR. In addition to assessing the presence of psychosis or CHR status, the SCID provided comorbid diagnoses across the entire community sample. The alternate diagnoses in the community sample was used to assess whether MAP-R subscales and items can discriminate between CHR status and other psychopathology detected in the community validation analytic dataset. The second phase included the completion of the Penn computerized neurocognitive battery and additional questionnaires. There were 13 individuals with lifetime antipsychotics use, which included individuals from the anxiety (n=4; 5.2%), CHR (n=8, 9.5%), and depression group (n=1, 2.9%).

Motor Abnormalities Psychosis Risk Scale. The Motor Abnormalities Psychosis Risk (MAP-R) Scale is a 14-item questionnaire that included questions about early developmental motor delays, the frequency of abnormal motor experiences, general assessments of motor function, and frequency of physical activity. Questions were developed based on a review of extant literature12,22,31,41,61 regarding motor abnormalities in individuals at CHR to include features of motor abnormalities linked to symptoms,4,10,19,21,48,48,63 conversion,1,5,32,37,38 and clinical/cognitive subtyping19 of psychosis risk. From this review of the literature a set of items were compiled to examine relevant constructs (e.g., ballistic movements, sway/balance). These items included several likert-type scale responses, such as “On a scale of 1-10, how would you rate your current motor skills?” and “Do you enjoy hobbies where you use skilled hand movements (e.g., art, crafts)? (Responses: Never Sometimes Lots)”. For an idealized version of the final measure and scoring, see Supplemental Materials.
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**SIPS Risk-Calculator.** The SIPS-RC is a Structured Interview on Prodromal Risk Symptoms (SIPS)-based risk calculator that provided practical, individualized risk assessments that can be easily implemented in a clinical setting. In this calculator, risk probability assessments are derived from four dimensions: positive and negative symptom severity, deterioration of function over the past 12 months, and low levels of dysphoric mood. In the current study, the global functioning scale was used to assess functional decline in the past year. Positive and negative symptom severity was assessed during the SIPS interview, and quantified as a composite score of endorsed positive and negative symptoms. Finally, low levels of dysphoric mood were assessed during the general symptom assessment of the SIPS. This approach has been validated against external independent risk calculators.

**Finger Tapping - Motor Validation Task.** In the current study we examined the performance on the computerized finger tapping (CTAP) performance. During the task, participants were instructed to press the spacebar as quickly as possible moving only the index finger for 10 seconds after their first button press. In this task, trial blocks alternated between the dominant and non-dominant hand for 10 blocks (5 block per hand). Median finger tapping and tapping variability behavior was used as a convergent measure of motor performance to assess the sensitivity of the MAP-R scale to motor performance.

**Analytical Strategy.** Exploratory factory analyses were used to examine structure of responses in the two analytic datasets (i.e., exploratory and validation). These two analytic datasets, with independent data-points include a larger exploratory, screening analytic dataset (n=3,109) that completed only phase one of the study and the scale validation analytic dataset (n=439) that completed both phase one and phase two. For each analysis, all participants with the data for that given analyses were included, rather than reducing the study sample size to only
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those individuals with a full and complete dataset; the intention in this approach was to both maximize power and transparency, *see Figure 1 for analyses sample sizes*. To assess the appropriate number of factors for these analyses, two approaches were used: 1) the Cattell’s scree test was used to identify the appropriate number of factors in the correlation matrix, 2) the eigen values were graphed against the increasing number of factors and components with both a threshold provided by simulated data. Factor analyses in both the exploratory analytic dataset (only completing the MAP-R scale among a battery of non-motor initial screening scales) and the validation analytic dataset (who completed both study phases) were used to determine the optimal items to composite into subscales. Then, we reported the psychometrics statistics, including internal consistency statistics (Cronbach’s alpha and Guttman’s Lambda) for the resulting subscales. Factor analyses and item reliability was assessed using the R v.4.0.0 and the Psych package.

All items that were grouped into a factor were examined as a composite subscale in the subsequent analyses. Items that were not included in the subscales (i.e. items that are not related to other items) were treated as independent items and evaluated for discriminant validity and convergent validity. Independent items that failed to show discriminant and convergent validity were excluded from the final scale. These subsequent analyses probed the discriminant and predictive validity of these composite subscales and reduced the total number of comparisons.

To examine discriminant validity, we examined the degree to which these scale factors differ between relevant CHR group (n=84) compared other disorders, i.e., depression (n=35) and anxiety (n=78), and a CSV (n=78) samples among the validation analytic dataset, using general linear models for the composite scales and chi-square for independent items. To examine predictive validity, these scale factors were related to the calculated risk for psychosis (SIPS-RC).
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and current symptom severity (SIPS total scales) within the CHR group from the validation analytic dataset. To examine convergent validity, the motor scales were compared to the motor performance (i.e., finger tapping task) in the CHR validation analytic dataset. All validation analyses were completed in SPSS version 26.

Results

Participants. There were no significant differences in key demographic variables including biological sex at birth, ethnicity, race, age, and income were compared across exploratory and validation analytic datasets, as well as discriminatory clinical samples, Table 1.

Exploratory factor analyses. In the exploratory analytic dataset, the traditional Cattell’s scree analyses suggested that the intercorrelation matrix showed 4 components; scree analyses compared to simulated data suggested the presence of 3 components or 2 factors, Supplemental Figure 1. Both analytic datasets showed similar scale structures with 2 components dividing the scale into Motor Abnormality items and Physical Activity items; when reorganized into three factors, the Motor Abnormality items divided into sub-facets coordination abnormalities items and dyskinesia items. In the validation analyses, the traditional Cattell’s scree analyses suggested that the intercorrelation matrix showed 3 components; scree analyses compared to simulated data suggested the presence of 3 components or 2 factors, Figure 2. There were two items that did not load onto these factors including an item on motor delays (i.e., “Are you aware of any delays in walking, talking, or toilet training when you were an infant or toddler?”) and an item on fine motor hobbies (i.e., “Do you enjoy hobbies where you use skilled hand movements (art, crafts)?”). These items were treated as independent items in the following validation analyses. Both exploratory and validation analytic datasets showed similar internal consistency (Table 2).
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**Discriminant validity among clinical samples.** Composite subscales totals were compared across diagnostic groups in independent analyses of variance model (ANOVA), Figure 3. For each of the independent items a limited set of Likert-responses were compared across groups using a chi-square analysis.

**Motor Abnormalities Subscale by Diagnostic Group.** The *Motor Abnormality* subscale significantly differed among groups, $F(270,3)=9.50, p<.001$, *partial $\eta^2*=.10$, such that CHR ($M=6.72, StD=3.25, SEM=.36$) showed increased *Motor Abnormality* score than individuals with depression ($M=5.32, StD=2.78, SEM=.37, p=.02$), anxiety ($M=5.18, StD=3.20, SEM=.37, p=.006$), and the CSV ($M=4.29, StD=2.21, SEM=.25, p<.001$).

**Abnormalities Sub-facets by Diagnostic Group.** The *Coordination Abnormalities* sub-facet significantly differed among groups, $F(271,3)=6.193, p<.001$, *partial $\eta^2*=.07$, such that CHR ($M=5.33, StD=2.55, SEM=.28$) showed increased *Coordination Abnormalities* scores than individuals with depression ($M=4.40, StD=2.44, SEM=.412, p=.05$), anxiety ($M=4.51, StD=2.74, SEM=.317, p=.043$), and the CSV ($M=3.70, StD=1.83, SEM=.21, p<.001$). The *Dyskinesia* sub-facet significantly differed among groups, $F(270,3)=8.54, p<.001$, *partial $\eta^2*=.09$, such that CHR ($M=1.41, StD=1.26, SEM=0.14$) showed an increased *Dyskinesia* score than individuals with depression ($M=0.88, StD=1.15, SEM=0.20, p=.02$), anxiety ($M=0.67, StD=0.96, SEM=0.11, p<.001$), and the CSV ($M=0.636, StD=1.01, SEM=0.12, p<.001$).

**Physical Activity Subscale by Diagnostic Group.** The *Physical Activity* subscale significantly differed among groups, $F(250,3)=8.536, p<.001$, *partial $\eta^2*=.09$, such that CHR ($M=7.47, StD=2.67, SEM=0.31$) showed less physical activity than the CSHV ($M=9.19, StD=2.74, SEM=0.32, p<.001$). Although the CHR group showed less physical activity than individuals...
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with depression ($M=8.59$, $StD=3.18$, $SEM=0.48$, $p=.076$) and anxiety ($M=8.19$, $StD=3.53$, $SEM=0.45$, $p=.16$); this difference was not significant.

*Independent Items by Diagnostic Group.* The motor delay response (no delays, some delays, a lot of delays) were compared across the diagnostic groups (CHR, CSV, depression, anxiety); groups differed in terms of motor delays $\chi^2(223)=9.45$, $p=.009$. The CHR group showed the highest percentage of motor delays with 16.5% of the CHR group reporting some or severe motor delays, and 100% of individuals reporting severe motor delays were also members of the CHR group. The frequency of engagement in motor hobbies (never, sometimes, often) were compared across the diagnostic groups (CHR, CSV, depression, anxiety); groups did not differ in terms of their engagement in fine motor hobbies, $\chi^2(275)=8.94$, $p=.27$.

*Convergent validity to motor performance.* Composite scales were compared to performance on a finger tapping task performance. Within the CHR group, a repeated-measure general linear model compared motor speed across hands (dominant, non-dominant) by median and variance as the within-subject factor and the MAP-R subscales, respectively, as between-subject factors. Finger tapping performance related to the *Motor Abnormalities* subscale, $F(71)=5.83$, $p=.016$; higher self-reported *Motor Abnormalities* subscale related to lowered median taps on both the dominant ($r_{partial}=-.23$) and non-dominant hands ($r_{partial}=-.18$). Finger tapping performance also related to the *Coordination Abnormalities* sub-facet, $F(71)=5.95$, $p=.017$, such that increased *Coordination Abnormalities* sub-facet scores were related to lowered median taps on both the dominant ($r_{partial}=-.26$) and non-dominant hands ($r_{partial}=-.22$), *Figure 4*. Finger tapping did not relate to any other subscales or items, $p's>.13$.

*Predictive and convergent validity to clinical features.* Composite subscales totals related to the psychosis risk score (SIPS-RC) in a correlation. *Motor Abnormalities* subscale
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related to SIPS-RC scores, \( r(312)=.15, p=.008 \). Coordination Abnormalities sub-facet related to SIPS-RC scores, \( r(310)=.16, p=.006 \). Dyskinesia sub-facet related to SIPS-RC scores, \( r(308)=0.118, p=.04 \). Physical Activity subscale related to SIPS-RC scores, \( r(284)=-.150, p=.01 \). For each of the independent items a limited set of Likert-responses were compared across groups using a general linear model. SIPS-RC did not relate to infant delays \( (p=.23) \) or frequency of fine motor hobbies \( (p=.63) \).

Discussion

The MAP-R scale showed a replicable structure that grouped items into Motor Abnormalities and Physical Activity subscales. Motor Abnormalities further divided into Coordination Abnormalities and Dyskinesia subscales. Each of these factors was of significant interest to understanding, identifying, predicting, and treating early psychosis, and so a motor scale that differentiates distinct motor categories has significant potential. Further, these subscales discriminated individuals at CHR from clinical groups (anxiety and depression) and a community sample of CSV. This discriminant validity suggested that this scale has sensitivity to psychosis-risk over issues related to general psychopathology, which would also appear in clinical samples in general. There was support for convergent validity; the general motor abnormality scale and the coordination scale were related to finger tapping performance. Additionally, these scales related to a validated measure of risk for psychosis indicating potential predictive validity of this scale to be relevant to psychosis course and risk for conversion to psychosis. Collectively this scale may provide unique insight into early risk for psychosis by examining motor abnormalities, which is largely under-utilized in current approaches to psychosis risk and may reflect a novel target for risk treatment.
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The exploratory and validation analytic dataset factor analyses suggested that items of the scale could be grouped into two subscales (Motor Abnormalities and Physical Activity subscales) with 2 sub-facets (Coordination Abnormalities, Dyskinesia) in the exploratory analytic dataset. Scale structure was consistent across independent data points in the analytic datasets providing converging evidence grouping the same items into the same subscale structure, providing increased confidence in the stability of these subscale structures. Additionally, these scales showed similar reliability across the exploratory and validation analytic datasets further suggesting the stability of these subscales. It is also notable that both the exploratory and the validation analytic datasets reflected a diverse community sample, which should both increase our sensitivity to individual differences and increase the generalizability of the current scale structures. The current paper identified three scales that may reflect distinct vulnerabilities/mechanisms/circuits that are central to the etiology of psychosis, and reflected by motor abnormalities. For example, motor behaviors ranging from hippocampal vulnerability, BDNF, and neurogenesis in the case of sedation/physical activity, to cerebellar-thalamic dysfunction for poor coordination, balance and ataxia, to aberrant dopamine. Finally, the validation analyses provided initial evidence of the utility of these scales.

The validation analytic dataset was a diverse community sample that included CSV as well as individuals with psychiatric diagnoses including major depression disorder and anxiety disorders (which are associated with some degree of motor abnormalities, such as psychomotor slowing or psychomotor agitation). In these analyses, the CHR group showed significantly more motor abnormalities, coordination abnormalities, and dyskinesia indicating that these scales are particularly sensitive to the presence of psychosis risk, beyond general psychopathology or general psychomotor agitation/slowing, which may have been present in depression and anxiety.
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In contrast, the *Physical Activity* subscale did not discriminate between the CSV and the CHR group. However, the *Physical Activity* subscale was sensitive to the presence of general psychopathology as the psychiatric groups did not differ from each other, but differed from the CSV sample.61,72 It is notable that both the *Motor Abnormalities* subscale and the *Physical Activity* subscale distinguished other anxiety and depression from CSV. This sensitivity to other psychopathology suggests that this scale may also have some utility in examining motor abnormalities in depression and anxiety. In summary, the scales showed both a specific relationship to psychosis risk and a general sensitivity to discriminate individuals with psychopathology from a CSV group. However, this self-report measure relied on the patient’s insight and, as a result, may not reliably reflect actual motor behavior.73–75 To this end we have further validated these scales against a measured motor behavior.

The finger tapping task has been known to reflect underlying disturbances in the function of underlying neurocircuitry of motor systems.30,76,77 This task is particularly probative of cortico-cerebellar interactions that govern the execution of sub-second responses, but also motor slowing generally. Among the subscales examined, the coordination subscale was the most relevant scale related to cortico-cerebellar function,76 and was related to performance on the finger tapping task. Critically, this result suggests that individuals at CHR were able to faithfully report on their coordination in a way that reflected independent measures of motor behavior. Although the *Motor Abnormalities* subscale related to the performance on this task, this was driven by the coordination abnormalities items.

All of the MAP-R subscales related to the calculated risk for developing psychosis- a composite score that weights the predictive features of clinical high-risk for psychosis (i.e., SIPS-RC score).64 Calculated risk scale serves as an estimation of the likelihood of an individual
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to convert to psychosis, but it also leverages critical features of early clinical risk, e.g., symptom severity and decreased global function.62,64 Taken together, the relationship of these subscales for calculated risk may reflect the relevance of *Motor Abnormalities* subscale to risk for conversion to psychosis, as estimated by calculated risk for psychosis, consistent with previous research.1,37

Despite the promise of the MAP-R subscales to reflect theoretical factors, there were two items that did not map onto any particular subscale- motor developmental delays and engaging in fine motor skill hobby items. The motor developmental delays item distinguished between clinical groups with a larger proportion of individuals in the CHR group who endorsed minor motor developmental delays,3,78 and endorsements of major motor developmental delays were made up entirely of individuals at CHR. The motor delay item was included in future versions of the MAP-R scale, but the fine motor hobby item will not be included in the final scale as it did not reflect group membership, clinical risk features, or motor performance.

This study included many strengths, but there were limitations that should be explored in future studies. Although the current paper identified three scales that may reflect distinct vulnerabilities/mechanisms/circuits, we did not directly assess mechanistic ties and specificity in the current study. Further, we omitted additional motor behaviors such as gestures and catatonia that are likely to tap into other distinct vulnerabilities.16,71,79–85 Finally, in the absence of longitudinal data, it is unknown whether motor abnormalities relate to clinical course or ultimate conversion to psychosis.

The MAP-R scale fills a gap in current self-report and clinical interviews that assess clinical risk as these measures rely on a limited set of items on motor abnormalities, while the current scale includes diverse items that include abnormalities. This scale allows researchers to assess motor phenotypes without expensive equipment or expertise, and can be used in many
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different psychosis assessment settings as a low-cost assessment of mechanisms underlying emerging symptoms of psychosis. As a result the MAP-R Scale may be a beneficial tool in CHR screening in clinical practice and has the potential to be an important target for clinical trials.31
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References

1. Mittal VA, Walker EF. Movement abnormalities predict conversion to Axis I psychosis among prodromal adolescents. *Journal of Abnormal Psychology*. 2007;116(4):796-803. doi:10.1037/0021-843X.116.4.796

2. Mittal VA, Tessner KD, Trotman HD, et al. Movement Abnormalities and the Progression of Prodromal Symptomatology in Adolescents at Risk for Psychotic Disorders. *Journal of Abnormal Psychology*. 2007;116(2):260-267.

3. Walker EF. Developmentally Moderated Expressions of the Neuropathology Underlying Schizophrenia. *Schizophr Bull*. 1994;20(3):453-480. doi:10.1093/schbul/20.3.453

4. Mittal VA, Neumann C, Saczawa M, Walker EF. Longitudinal Progression of Movement Abnormalities in Relation to Psychotic Symptoms in Adolescents at High Risk of Schizophrenia. *Arch Gen Psychiatry*. 2008;65(2):165-171. doi:10.1001/archgenpsychiatry.2007.23

5. Schiffman J, Mittal V, Kline E, et al. Childhood dyspraxia predicts adult-onset nonaffective–psychosis-spectrum disorder. *Development and Psychopathology*. 2015;27(4pt1):1323-1330. doi:10.1017/S0954579414001436

6. Schiffman J, Sorensen HJ, Maeda J, et al. Childhood motor coordination and adult schizophrenia-spectrum disorder. *Am J Psychiatry*. 2009;166(9):1041-1047. doi:10.1176/appi.ajp.2009.08091400

7. Schiffman J, Walker E, Ekstrom M, Schulsinger F, Sorensen H, Mednick S. Childhood videotaped social and neuromotor precursors of schizophrenia: a prospective investigation. *Am J Psychiatry*. 2004;161(11):2021-2027. doi:10.1176/appi.ajp.161.11.2021

8. Schiffman J. Motor Issues in the Clinical High Risk Phase of Psychosis. *Schizophr Bull*. 2017;43(5):937-938. doi:10.1093/schbul/sbx086

9. Dean DJ, Mittal VA. Spontaneous parkinsonisms and striatal impairment in neuroleptic free youth at ultrahigh risk for psychosis. *NPJ Schizophr*. 2015;1:14006. doi:10.1038/npjjschz.2014.6

10. Dean DJ, Kent JS, Bernard JA, et al. Increased postural sway predicts negative symptom progression in youth at ultrahigh risk for psychosis. *Schizophrenia Research*. 2015;162(1):86-89. doi:10.1016/j.schres.2014.12.039

11. Hirjak D, Thomann PA, Kubera KM, Wolf ND, Sambataro F, Wolf RC. Motor dysfunction within the schizophrenia-spectrum: A dimensional step towards an underappreciated domain. *Schizophrenia Research*. 2015;169(1):217-233. doi:10.1016/j.schres.2015.10.022

12. Hirjak D, Meyer-Lindenberg A, Kubera KM, Thomann PA, Wolf RC. Motor dysfunction as research domain in the period preceding manifest schizophrenia: A systematic review.
CHR MOTOR SCALE

Neuroscience & Biobehavioral Reviews. 2018;87:87-105. doi:10.1016/j.neubiorev.2018.01.011

13. Walther S, Mittal VA. Motor System Pathology in Psychosis. Curr Psychiatry Rep. 2017;19(12):97. doi:10.1007/s11920-017-0856-9

14. Ellman LM, Vinogradov S, Kremen WS, et al. Low maternal hemoglobin during pregnancy and diminished neuromotor and neurocognitive performance in offspring with schizophrenia. Schizophrenia Research. 2012;138(1):81-87. doi:10.1016/j.schres.2012.04.008

15. Mittal VA, Daley M, Shiode MF, Bearden CE, O’Neill J, Cannon TD. Striatal volumes and dyskinetic movements in youth at high-risk for psychosis. Schizophrenia Research. 2010;123(1):68-70. doi:10.1016/j.schres.2010.08.002

16. Stegmayer K, Bohlhalter S, Vanbellingen T, et al. Structural brain correlates of defective gesture performance in schizophrenia. Cortex. 2016;78:125-137. doi:10.1016/j.cortex.2016.02.014

17. Dean DJ, Bernard JA, Orr JM, et al. Cerebellar Morphology and Procedural Learning Impairment in Neuroleptic-Naive Youth at Ultrahigh Risk of Psychosis. Clinical Psychological Science. 2014;2(2):152-164. doi:10.1177/2167702613500039

18. Bernard JA, Orr JM, Mittal VA. Cerebello-thalamo-cortical networks predict positive symptom progression in individuals at ultra-high risk for psychosis. NeuroImage: Clinical. 2017;14:622-628. doi:10.1016/j.nicl.2017.03.001

19. Dean DJ, Walther S, Bernard JA, Mittal VA. Motor Clusters Reveal Differences in Risk for Psychosis, Cognitive Functioning, and Thalamocortical Connectivity: Evidence for Vulnerability Subtypes. Clinical Psychological Science. 2018;6(5):721-734. doi:10.1177/2167702618773759

20. Osborne KJ, Damme KSF, Gupta T, Dean DJ, Bernard JA, Mittal VA. Timing dysfunction and cerebellar resting state functional connectivity abnormalities in youth at clinical high-risk for psychosis. Psychological Medicine. Published online 2020:1-10. doi:10.1017/S0033291719004161

21. Bernard JA, Dean DJ, Kent JS, et al. Cerebellar networks in individuals at ultra high-risk of psychosis: Impact on postural sway and symptom severity. Human Brain Mapping. 2014;35(8):4064-4078. doi:10.1002/hbm.22458

22. Mittal VA, Bernard JA, Northoff G. What Can Different Motor Circuits Tell Us About Psychosis? An RDoC Perspective. Schizophr Bull. 2017;43(5):949-955. doi:10.1093/schbul/sbx087

23. Caligiuri MP, Teulings H-L, Dean CE, Niculescu AB, Lohr JB. Handwriting Movement Kinematics for Quantifying EPS in Patients Treated with Atypical Antipsychotics. Psychiatry Res. 2010;177(1-2):77-83. doi:10.1016/j.psychres.2009.07.005
24. Murck H, Laughren T, Lamers F, et al. Taking Personalized Medicine Seriously: Biomarker Approaches in Phase IIb/III Studies in Major Depression and Schizophrenia. *Innov Clin Neurosci.* 2015;12(3 SupplA):26S-40S.

25. Mittal VA, Hasenkamp W, Sanfilipo M, et al. Relation of neurological soft signs to psychiatric symptoms in schizophrenia. *Schizophrenia Research.* 2007;94(1):37-44. doi:10.1016/j.schres.2007.04.017

26. Mittal VA, Gupta T, Orr JM, et al. Physical activity level and medial temporal health in youth at ultra high-risk for psychosis. *Journal of Abnormal Psychology.* 2013;122(4):1101-1110. doi:10.1037/a0034085

27. Dean DJ, Bryan AD, Newberry R, Gupta T, Carol E, Mittal VA. A Supervised Exercise Intervention for Youth at Risk for Psychosis: An Open-Label Pilot Study. *J Clin Psychiatry.* 2017;78(9):e1167-e1173. doi:10.4088/JCP.16m11365

28. Newberry RE, Dean DJ, Sayyah MD, Mittal VA. What prevents youth at clinical high risk for psychosis from engaging in physical activity? An examination of the barriers to physical activity. *Schizophrenia Research.* 2018;201:400-405. doi:10.1016/j.schres.2018.06.011

29. Deighton S, Addington J. Exercise practices in individuals at clinical high risk of developing psychosis. *Early Intervention in Psychiatry.* 2015;9(4):284-291. doi:10.1111/eip.12107

30. Damme KSF, Osborne KJ, Gold JM, Mittal VA. Detecting motor slowing in clinical high risk for psychosis in a computerized finger tapping model. *Eur Arch Psychiatry Clin Neurosci.* 2020;270(3):393-397. doi:10.1007/s00406-019-01059-0

31. Mittal VA, Walther S. As Motor System Pathophysiology Returns to the Forefront of Psychosis Research, Clinical Implications Should Hold Center Stage. *Schizophr Bull.* 2019;45(3):495-497. doi:10.1093/schbul/sby176

32. Masucci MD, Lister A, Corcoran CM, Brucato G, Girgis RR. Motor dysfunction as a risk factor for conversion to psychosis independent of medication use in a psychosis-risk cohort. *J Nerv Ment Dis.* 2018;206(5):356-361. doi:10.1097/NMD.0000000000000806

33. Gupta T, Dean DJ, Kelley NJ, Bernard JA, Ristanovic I, Mittal VA. Cerebellar Transcranial Direct Current Stimulation Improves Procedural Learning in Nonclinical Psychosis: A Double-Blind Crossover Study. *Schizophr Bull.* 2018;44(6):1373-1380. doi:10.1093/schbul/sbx179

34. Howes OD, Kapur S. The Dopamine Hypothesis of Schizophrenia: Version III—The Final Common Pathway. *Schizophr Bull.* 2009;35(3):549-562. doi:10.1093/schbul/sbp006

35. Osborne KJ, Vargas T, Mittal VA. Early childhood social communication deficits in youth at clinical high-risk for psychosis: Associations with functioning and risk. *Development and Psychopathology.* 2020;32(2):559-572. doi:10.1017/S0954579419000385
CHR MOTOR SCALE

36. Mittal VA, Jalbrzikowski M, Daley M, Roman C, Bearden CE, Cannon TD. Abnormal movements are associated with poor psychosocial functioning in adolescents at high risk for psychosis. *Schizophrenia Research*. 2011;130(1):164-169. doi:10.1016/j.schres.2011.05.007

37. Mittal VA, Walker EF, Bearden CE, et al. Markers of Basal Ganglia Dysfunction and Conversion to Psychosis: Neurocognitive Deficits and Dyskinestias in the Prodromal Period. *Biological Psychiatry*. 2010;68(1):93-99. doi:10.1016/j.biopsych.2010.01.021

38. Studerus E, Papmeyer M, Riecher-Rössler A. Neurocognition and Motor Functioning in the Prediction of Psychosis. *Early Detection and Intervention in Psychosis*. 2016;181:116-132. doi:10.1159/000440919

39. Callaway DA, Perkins DO, Woods SW, Liu L, Addington J. Movement Abnormalities Predict Transitioning to Psychosis in Individuals at Clinical High Risk for Psychosis. *Schizophr Res*. 2014;159(0):263-266. doi:10.1016/j.schres.2014.09.031

40. Ebel H, Gross G, Klosterkötter J, Huber G. Basic Symptoms in Schizophrenic and Affective Psychoses. *Psychopathology*. 1989;22(4):224-232. doi:10.1159/000284602

41. Dazzan P, Murray RM. Neurological soft signs in first-episode psychosis: a systematic review. *British Journal of Psychiatry*. 2002;181:50-57.

42. Andreasen NC, Paradiso S, O’Leary DS. “Cognitive Dysmetria” as an Integrative Theory of Schizophrenia: A Dysfunction in Cortical-Subcortical-Cerebellar Circuitry? *Schizophr Bull*. 1998;24(2):203-218. doi:10.1093/oxfordjournals.scbull.a033321

43. Kent JS, Hong SL, Bolbecker AR, et al. Motor Deficits in Schizophrenia Quantified by Nonlinear Analysis of Postural Sway. *PLoS One*. 2012;7(8). doi:10.1371/journal.pone.0041808

44. Petruzzelli MG, Margari L, Craig F, et al. Markers of neurodevelopmental impairments in early-onset psychosis. *Neuropsychiatr Dis Treat*. 2015;11:1793-1798. doi:10.2147/NDT.S83904

45. Wilquin H, Delevoye-Turrell Y. Motor Agency: A New and Highly Sensitive Measure to Reveal Agency Disturbances in Early Psychosis. *PLoS One*. 2012;7(2). doi:10.1371/journal.pone.0030449

46. Dandash O, Pantelis C, Fornito A. Dopamine, fronto-striato-thalamic circuits and risk for psychosis. *Schizophrenia Research*. 2017;180:48-57. doi:10.1016/j.schres.2016.08.020

47. Dandash O, Fornito A, Lee J, et al. Altered Striatal Functional Connectivity in Subjects With an At-Risk Mental State for Psychosis. *Schizophr Bull*. 2014;40(4):904-913. doi:10.1093/schbul/sbt093

48. Cortese L, Caligiuri MP, Malla AK, Manchanda R, Takhar J, Haricharan R. Relationship of neuromotor disturbances to psychosis symptoms in first-episode neuroleptic-naïve
CHR MOTOR SCALE

schizophrenia patients. Schizophrenia Research. 2005;75(1):65-75. doi:10.1016/j.schres.2004.08.003

49. Mittal VA, Dean D, Pelletier A, Caligiuri M. Associations between spontaneous movement abnormalities and psychotic-like experiences in the general population. Schizophr Res. 2011;132(2-3):194-196. doi:10.1016/j.schres.2011.06.028

50. Bernard JA, Seidler RD. Cerebellar contributions to visuomotor adaptation and motor sequence learning: an ALE meta-analysis. Front Hum Neurosci. 2013;7. doi:10.3389/fnhum.2013.00027

51. Bernard JA, Mittal VA. Dysfunctional Activation of the Cerebellum in Schizophrenia: A Functional Neuroimaging Meta-Analysis. Clin Psychol Sci. 2015;3(4):545-566. doi:10.1177/2167702614542463

52. Koivukangas J, Tammelin T, Kaakinen M, et al. Physical activity and fitness in adolescents at risk for psychosis within the Northern Finland 1986 Birth Cohort. Schizophrenia Research. 2010;116(2):152-158. doi:10.1016/j.schres.2009.10.022

53. Harrison PJ. The hippocampus in schizophrenia: a review of the neuropathological evidence and its pathophysiological implications. Psychopharmacology. 2004;174(1):151-162. doi:10.1007/s00213-003-1761-y

54. Stranahan AM, Khalil D, Gould E. Running induces widespread structural alterations in the hippocampus and entorhinal cortex. Hippocampus. 2007;17(11):1017-1022. doi:10.1002/hipo.20348

55. Pajonk F-G, Wobrock T, Gruber O, et al. Hippocampal plasticity in response to exercise in schizophrenia. Arch Gen Psychiatry. 2010;67(2):133-143. doi:10.1001/archgenpsychiatry.2009.193

56. Loewy RL, Pearson R, Vinogradov S, Bearden CE, Cannon TD. Psychosis Risk Screening with the Prodromal Questionnaire – Brief version (PQ-B). Schizophr Res. 2011;129(1):42-46. doi:10.1016/j.schres.2011.03.029

57. Karcher NR, Barch DM, Avenevoli S, et al. Assessment of the Prodromal Questionnaire-Brief Child Version for Measurement of Self-reported Psychoticlike Experiences in Childhood. JAMA Psychiatry. 2018;75(8):853-861. doi:10.1001/jamapsychiatry.2018.1334

58. Miller TJ, McGlashan TH, Rosen JL, et al. Prodromal Assessment With the Structured Interview for Prodromal Syndromes and the Scale of Prodromal Symptoms: Predictive Validity, Interrater Reliability, and Training to Reliability. Schizophr Bull. 2003;29(4):703-715. doi:10.1093/oxfordjournals.schbul.a007040

59. Nakamura BJ, Ebesutani C, Bernstein A, Chorpita BF. A Psychometric Analysis of the Child Behavior Checklist DSM-Oriented Scales. J Psychopathol Behav Assess. 2009;31(3):178-189. doi:10.1007/s10862-008-9119-8
CHR MOTOR SCALE

60. First MB, Williams JB. *Structured Clinical Interview for DSM-5: Research Version*. American Psychiatric Association; 2015.

61. Bernard JA, Mittal VA. Updating the research domain criteria: the utility of a motor dimension. *Psychological Medicine*. 2015;45(13):2685-2689. doi:10.1017/S0033291715000872

62. Zhang T, Li H, Tang Y, et al. Validating the Predictive Accuracy of the NAPLS-2 Psychosis Risk Calculator in a Clinical High-Risk Sample From the SHARP (Shanghai At Risk for Psychosis) Program. *AJP*. 2018;175(9):906-908. doi:10.1176/appi.ajp.2018.18010036

63. Bermanzohn PC, Siris SG. Akinesia: A syndrome common to parkinsonism, retarded depression, and negative symptoms of schizophrenia. *Comprehensive Psychiatry*. 1992;33(4):221-232. doi:10.1016/0010-440X(92)90045-R

64. Osborne KJ, Mittal VA. External validation and extension of the NAPLS-2 and SIPS-RC personalized risk calculators in an independent clinical high-risk sample. *Psychiatry Res*. 2019;279:9-14. doi:10.1016/j.psychres.2019.06.034

65. Hall RCW. Global Assessment of Functioning: A Modified Scale. *Psychosomatics*. 1995;36(3):267-275. doi:10.1016/S0033-3182(95)71666-8

66. Gur RC, Richard J, Hughett P, et al. A cognitive neuroscience-based computerized battery for efficient measurement of individual differences: Standardization and initial construct validation. *Journal of Neuroscience Methods*. 2010;187(2):254-262. doi:10.1016/j.jneumeth.2009.11.017

67. Popper K. *The Logic of Scientific Discovery*. Routledge; 2005.

68. R: The R Project for Statistical Computing. Accessed June 8, 2020. https://www.r-project.org/

69. psych citation info. Accessed June 8, 2020. https://cran.r-project.org/web/packages/psych/citation.html

70. Schiffman J, Ellman LM, Mittal VA. Individual Differences and Psychosis-Risk Screening: Practical Suggestions to Improve the Scope and Quality of Early Identification. *Front Psychiatry*. 2019;10. doi:10.3389/fpsyt.2019.00006

71. Osborne KJ, Bernard JA, Gupta T, et al. Beat gestures and postural control in youth at ultrahigh risk for psychosis. *Schizophrenia Research*. 2017;185:197-199. doi:10.1016/j.schres.2016.11.028

72. An Examination of Psychomotor Disturbance in Current and Remitted MDD: An RDoC Study. Accessed June 8, 2020. https://www-ncbi-nlm-nih-gov.turing.library.northwestern.edu/pmc/articles/PMC7255437/
CHR MOTOR SCALE

73. Baranowski T. Methodologic Issues in Self-Report of Health Behavior. *Journal of School Health*. 1985;55(5):179-182. doi:10.1111/j.1746-1561.1985.tb04115.x

74. Carol EE, Mittal VA. Self-Reported Cannabis Use is Inconsistent with the Results From Drug-Screening in Youth at Ultra High-Risk for Psychosis in Colorado. *Schizophr Res*. 2014;157(0):317-318. doi:10.1016/j.schres.2014.05.032

75. Lindwall M, Ljung T, Hadžibajramović E, Jonsdottir IH. Self-reported physical activity and aerobic fitness are differently related to mental health. *Mental Health and Physical Activity*. 2012;5(1):28-34. doi:10.1016/j.mhpa.2011.12.003

76. Buijink AWG, Broersma M, van der Stouwe AMM, et al. Rhythmic finger tapping reveals cerebellar dysfunction in essential tremor. *Parkinsonism Relat Disord*. 2015;21(4):383-388. doi:10.1016/j.parkreldis.2015.02.003

77. Carroll CA, O’Donnell BF, Shekhar A, Hetrick WP. Timing dysfunctions in schizophrenia as measured by a repetitive finger tapping task. *Brain and Cognition*. 2009;71(3):345-353. doi:10.1016/j.bandc.2009.06.009

78. Keskinen E, Marttila A, Marttila R, et al. Interaction between parental psychosis and early motor development and the risk of schizophrenia in a general population birth cohort. *European Psychiatry*. 2015;30(6):719-727. doi:10.1016/j.eurpsy.2015.04.006

79. Mittal VA, Tessner KD, McMillan AL, Delawalla Z, Trotman HD, Walker EF. Gesture behavior in unmedicated schizotypal adolescents. *J Abnorm Psychol*. 2006;115(2):351-358. doi:10.1037/0021-843X.115.2.351

80. Millman ZB, Goss J, Schiffman J, Mejias J, Gupta T, Mittal VA. Mismatch and Lexical Retrieval Gestures are Associated with Visual Information Processing, Verbal Production, and Symptomatology in Youth at High Risk for Psychosis. *Schizophr Res*. 2014;158(0):64-68. doi:10.1016/j.schres.2014.06.007

81. Beat and metaphoric gestures are differentially associated with regional cerebellar and cortical volumes - Bernard - 2015 - Human Brain Mapping - Wiley Online Library. Accessed June 9, 2020. https://onlinelibrary.wiley.com/doi/full/10.1002/hbm.22894

82. Walther S, Mittal VA. Why We Should Take a Closer Look at Gestures. *Schizophr Bull*. 2016;42(2):259-261. doi:10.1093/schbul/sbv229

83. Walther S, Eisenhardt S, Bohlhalter S, et al. Gesture Performance in Schizophrenia Predicts Functional Outcome After 6 Months. *Schizophr Bull*. 2016;42(6):1326-1333. doi:10.1093/schbul/sbw124

84. Walther S, Vanbellingen T, Müri R, Strik W, Bohlhalter S. Impaired gesture performance in schizophrenia: Particular vulnerability of meaningless pantomimes. *Neuropsychologia*. 2013;51(13):2674-2678. doi:10.1016/j.neuropsychologia.2013.08.017
CHR MOTOR SCALE

85. Nonverbal Social Communication and Gesture Control in Schizophrenia | Schizophrenia Bulletin | Oxford Academic. Accessed June 9, 2020.
   https://academic.oup.com/schizophreniabulletin/article/41/2/338/2526113
Table 1. Demographic Metrics: A comparison of diagnostic groups within the validation sample for discriminatory analyses

|          | Exploratory Sample | Validation Sample | Sample Comparison | CHR | CHV | MDD | Anxiet y | Discriminan t Sample | Discriminant Group |
|----------|--------------------|-------------------|-------------------|-----|-----|-----|-----------|----------------------|-------------------|
|          | n=3,009            | n=439             | Statistics        | n=84| n=78| n=35| n=78      | n=275                |                    |
| Age -M(StD) | 20.31 (2.36)       | 20.13 (1.84)      | t(3436)=1.523 p=.13 |     |     |     |           |                      |                   |
| Sex (% Female) | 67.75%            | 77.47%            | χ² (1)=.285 p=.59 |   | 70.50% | 80% | 84.60% | 74.69% | χ² (3)=4.70, p=.20 |
| Hispanic Ethnicity | 10.38%        | 10.25%            | χ² (1)=.007 p=.93 | 7.31% | 8.97% | 14.29% | 5.26% | 8.98% | χ² (3)=2.76, p=.43 |
| Race                                               |                    |                   |                   |     |     |     |          |                      |                   |
| American Indian                                    | 0.36%             | 0.46%             | 0.35%             | 0.00% | 0.00% | 0.00% | 0%       | χ² (3)=2.31, p=.51 |
| Asian                                              | 24.06%            | 25.06%            | 6.36%             | 8.13% | 2.47% | 8.13% | 28.98% | χ² (3)=2.45, p=.49 |
| Native Hawaiian                                   | 0.03%             | 0.00%             | 0.35%             | 0.35% | 0.00% | 0.00% | 0.82%   | χ² (3)=1.417, p=.70 |
| Black/African American                              | 13.51%            | 14.81%            | 4.95%             | 4.59% | 1.41% | 4.24% | 17.55%   | χ² (3)=.629, p=.089 |
| White                                              | 51.34%            | 50.57%            |                   | %    | 13.43% | 7.42% | 13.07% | 58.37% | χ² (3)=2.59, p=.46 |
| Multiracial                                        | 6.16%             | 6.61%             | 1.06%             | 2.12% | 1.06% | 1.41% | 6.53%    | χ² (3)=1.78, p=.62 |
| Unknown                                            | 7.45%             | 2.51%             | 0.71%             | 1.06% | 0.71% | 0.00% | 2.86%    | χ² (3)=3.978, p=.26 |
| Income                                             |                    |                   |                   |     |     |     |          |                      |                   |
| <$2,000                                            | 44.76%            | 45.04%            | %                 | 10.26% | 4.27% | 13.25% | 44%     | χ² (18)=26.481, p=.089 |
| 2,000-2,999                                        | 9.69%             | 8.65%             | 1.71%             | 2.56% | 1.28% | 2.14% | 7.70%    | χ² (18)=57.8, p=.24 |
| 3,000-3,999                                        | 5.26%             | 6.11%             | 2.14%             | 2.56% | 0.85% | 2.56% | 8.10%    |                     |
| 4,000-4,999                                        | 3.78%             | 4.33%             | 0.85%             | 1.28% | 0.85% | 2.14% | 5.10%    |                     |
| 5,000-5,999                                        | 3.63%             | 5.34%             | 0.85%             | 2.14% | 1.28% | 0.43% | 4.70%    |                     |
| 6,000-6,999                                        | 2.53%             | 4.07%             | 1.28%             | 2.14% | 0.43% | 1.28% | 5.10%    |                     |
| 7,000-7,999                                        | 1.89%             | 1.78%             | 0.43%             | 1.28% | 0.43% | 0.00% | 2.10%    |                     |
| 8,000-8,999                                        | 1.89%             | 2.54%             | 0.85%             | 0.43% | 0.43% | 1.28% | 3%       |                     |
| 9,000-9,999                                        | 1.55%             | 1.78%             | 0.43%             | 0.00% | 0.43% | 0.00% | 0.90%    |                     |
| 10,000-12,499                                      | 5.07%             | 6.36%             | 2.56%             | 2.99% | 0.43% | 0.85% | 1.70%    |                     |
| 12,500-14,999                                      | 1.89%             | 1.27%             | 0.00%             | 0.00% | 0.85% | 0.00% | 0.90%    |                     |
| 15,000-16,999                                      | 1.36%             | 2.04%             | 0.43%             | 0.43% | 0.43% | 0.43% | 1.70%    |                     |
|                      | 0.98% | 1.53% | 0.00% | 0.00% | 0.00% | 0.85% | 0.90% |
|----------------------|-------|-------|-------|-------|-------|-------|-------|
| 17,000-19,999        |       |       |       |       |       |       |       |
| 20,000-24,999        | 2.50% | 1.27% | 0.00% | 0.43% | 0.00% | 0.43% | 0.90% |
| 25,000-34,999        | 3.93% | 3.31% | 2.14% | 0.00% | 0.43% | 0.85% | 3.40% |
| 35,000-49,999        | 4.05% | 3.31% | 0.43% | 0.43% | 0.43% | 1.71% | 3%    |
| 50,000-69,999        | 2.65% | 0.51% | 0.00% | 0.43% | 0.00% | 0.00% | 0.40% |
| 75,000-99,999        | 1.55% | 0.76% | 0.43% | 0.00% | 0.00% | 0.85% | 1.30% |
| 100,000+             | 1.02% | 0.00% | 0.00% | 0.00% | 0.00% | 0.00% | 0%    |
Table 2. Reliability of scales replicated across samples

| Factor Scales          | Exploratory Sample |                      | Validation Sample |                      |
|------------------------|---------------------|----------------------|-------------------|----------------------|
|                        | Cronbach’s Alpha   | Guttman’s Lambda    | Cronbach’s Alpha  | Guttman’s Lambda    |
| Motor Abnormalities    | 0.69                | 0.71                 | 0.71              | 0.73                 |
| Coordination Scale     | 0.71                | 0.68                 | 0.75              | 0.71                 |
| Dyskinesia Scale       | 0.67                | 0.51                 | 0.68              | 0.51                 |
| Activity Scale         | 0.68                | 0.64                 | 0.69              | 0.64                 |
Figure 1. Study Sample: Guide on data collection phases, sample recruitment overlap, and measure sample sizes.

**Phase One: Online Screening Battery (n=3,448)**

**Overview.** Participants completed an online battery of measure that included established psychosis risk questionnaires and the included the MAP-R Scale.

**Exploratory Factor Analyses.** Most participants only completed this phase were treated as an exploratory analytic dataset (n = 3,009) which, these data points were included in the exploratory factor analyses to examine scale structure.

**Phase Two: In-Person Visit Clinical Interviews and Behavioral Battery (n=439)**

**Overview.** Participants completed an in-person visit which included clinical interviews (e.g., SIPS, SCID, GAF) and behavioral measures (e.g., Finger Tapping).

**Exploratory Factor Analyses (n=439).** Participants who completed both phases were treated as a validation analytic data set which, these data points were included in the exploratory factor analyses to examine scale structure separately from the larger sample to ensure that the scale structure was consistent between these independent analytic datasets.

**Discriminant Validity Analyses (n=275).** Diagnoses from SCID and SIPS clinical interviews were used to group individuals into Clinical High-Risk for Psychosis (CHR; n=84), Major Depression Disorder (MDD; n=25), Anxiety (n=78), and Community Sample Non-Psychiatric Volunteer (CSV; n=78) groups – all comorbid and alternative diagnoses were excluded from this analysis. These groups were compares on the subscales and independent scale items.

**Convergent Validity Analyses (n=73).** Performance on behavioral finger tapping was compared to the self-reported MAP-RS subscales among individuals at clinical high-risk for psychosis (CHR) for whom finger tapping data was available.

**Predictive validity Analyses (n=312).** Calculated psychosis risk score (SIPS-RC) was compared to MAP-RS subscales and independent items.
Figure 2. Factor analyses and data structure for the validation analytic dataset: A. Depicts the intercorrelation structure of the items, B. Depicts the Cattell scree plot of the intercorrelation matrix, C. Depicts the factor analyses compared to simulated data in a scree plot, D. Depicts the factor structure for both a 2 factor (Motor Abnormalities and Physical Activity Scales) and three factor solution (Dyskinesia Abnormalities sub-facets, Coordination abnormalities sub-facets, and Physical Activity Scale).
Supplemental Figure 1. Factor analyses and data structure for the exploratory analytic dataset: A. Depicts the intercorrelation structure of the items, B. Depicts the Cattell scree plot of the intercorrelation matrix, C. Depicts the factor analyses compared to simulated data in a scree plot, D. Depicts the factor structure for both a 2 factor (Motor Abnormalities and Physical Activity Scales) and three factor solution (Dyskinesia Abnormalities sub-facets, Coordination abnormalities sub-facets, and Physical Activity Scale)
Figure 3. Discriminant Validation Analyses: Composite Scales by Group