Journal of Abnormal Psychology
2007, Vol. 116, No. 4, 796–803

0021-843X/07/$12.00 DOI: 10.1037/0021-843X.116.4.796

Movement Abnormalities Predict Conversion to Axis I Psychosis Among Prodromal Adolescents

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Evidence suggests that movement abnormalities are a precursor of psychosis. The link between movement abnormalities and psychotic disorders is presumed to reflect common neural mechanisms that influence both motor functions and vulnerability to psychosis. The authors coded movement abnormalities from videotapes of 40 adolescents at risk for psychosis (designated prodromal on the Structured Interview for Prodromal Symptoms; T. J. Miller et al., 2002). Following initial assessment, participants were evaluated for diagnostic status at 4 times annually. Ten participants converted to an Axis I psychosis (e.g., schizophrenia) over the 4-year period. Comparisons of converted and nonconverted participants at baseline indicated that the groups did not differ on demographic characteristics or levels of prodromal symptomatology, but those who converted exhibited significantly more movement abnormalities. Movement abnormalities and prodromal symptoms were strongly associated and logistic regression analyses indicated that abnormalities in the face and upper body regions were most predictive of conversion. Findings suggest that individuals with elevated movement abnormalities may represent a subgroup of prodromal adolescents who are at the highest risk for conversion. The implications for neural mechanisms and for identifying candidates for preventive intervention are discussed.

Keywords: schizophrenia, prodromal, movement abnormality, conversion, psychosis

The modal period for the onset of psychosis is early adulthood, with many patients manifesting behavioral dysfunction during adolescence (Cannon, Rosso, Bearden, Sanchez, & Hadley, 1999; Neumann & Walker, 2003). The premorbid indicators include behavioral signs, such as social withdrawal, and thought abnormalities (Walker, Baum, & Diforio, 1998), deficits in memory and executive function (Silverstein, Mavrolefteros, & Turnbull, 2003), and neuromotor abnormalities (Neumann & Walker, 2003). Because these signs often become more pronounced as the individual approaches young adulthood (Cornblatt, Lencz, & Obuchowski, 2002), it is assumed that the heightened risk associated with this developmental period results, in part, from neuromaturational processes (Walker & Diforio, 1997).

The prodromal period is typically defined as a period of functional decline that precedes the onset of psychosis (Larsen, McGlashan, Johannessen, & Vibe-Hansen, 1996). Instruments have been developed to measure the prodrome, and recent research has demonstrated that 13% to 40% of individuals who meet current criteria for the prodrome meet diagnostic criteria for schizophrenia or affective psychosis within 2 years (Haroun, Dunn, Haroun, & Cadenhead, 2006). The prodrome represents both a viable point for intervention and a developmental period with strong potential to shed light on the etiology of schizophrenia and affective disorders with psychotic features (i.e., schizoaffective disorder, bipolar disorder, and unipolar depression; Haroun et al., 2006). Within this context, premorbid movement abnormalities are of particular interest because they are more pronounced in prodromal individuals (Mittal, Dhruv, Tessner, Walder, & Walker, 2007; Mittal, Tessner, et al., 2007), independent of treatment with psychotropic medication (Boks, Liddle, Burgerhof, Knegtering, & van den Bosch, 2004; Puri, Barnes, Chapman, Hutton, & Joyce, 1999). For example, longitudinal studies have shown that children who later develop psychotic disorders manifest a higher frequency of movement abnormalities when compared to those with healthy adult outcomes (Fish, Marcus, Hans, Auerbach, & Purdue, 1992; Schiffman et al., 2004). This relationship has also been reflected in brain structure; childhood motor abnormalities are linked with greater ventricular enlargement in adult patients with psychosis (Walker, Lewine, & Neumann, 1996). Further, movement abnormalities—especially dyskinesias of the face and upper limbs—are more common in adult patients with psychosis, even patients who have never received antipsychotic medications (Gervin et al., 1998; Puri et al., 1999).

The mechanisms underlying the relation between movement abnormalities and psychosis have been of interest to investigators because the neurocircuitry hypothesized to be implicated in psychotic symptoms is partially shared by the circuits that are known to give rise to dyskinetic movements (Gray, Kumari, Lawrence, & Young, 1999; Graybiel, 1997; Walker, 1994). Specifically, overactivation of dopaminergic pathways in striatal regions (proximal to the region regulating hyperkinesias) has been hypothesized to contribute to psychotic symptoms (Graybiel, 1997; Walker, 1994). Thus similar circuitry malfunctions may result in two manifestations: movement abnormality and psychotic symptoms (Walker,

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This research was supported by National Institute of Mental Health Grant RO1-MH4062066 to Elaine F. Walker.

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Lewine, & Neumann, 1996). Thus it is possible that for some individuals, movement abnormalities are the first signs of compromised neurocircuitry that may later give rise to psychosis (Mittal, Tessner, et al., 2007).

As it is becoming increasingly important to identify youth who are most likely to benefit from preventive intervention (Haroun et al., 2006), research examining potential predictive markers is needed. There have been no previous reports on the potential of movement abnormalities to predict conversion to Axis I disorders in prodromal individuals. But the existing literature and theory on the relation between movement abnormalities and psychoses provides a basis for hypothesizing an association. The present investigation tests the hypothesis that, among prodromal adolescents, movement abnormalities will predict conversion to psychosis. To test this hypothesis, prodromal adolescents were assessed for movement abnormalities and evaluated for psychiatric status over a period of 4 years.

Diagnostic Specificity of Outcome for Prodromal Individuals

Research has indicated that individuals identified as prodromal using current criteria are at increased risk for developing schizophrenia as well as affective disorders with psychotic features (Haroun et al., 2006; Miller et al., 2002; Yung et al., 1998). These findings are consistent with genetic evidence of shared etiological factors among psychotic disorders defined by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV*; American Psychiatric Association, 1994) (Cardno, Rijsdijk, Sham, Murray, & McGuffin, 2002; Riley & Kendler, 2006). As a result, prodromal researchers are focusing on psychosis as the outcome variable. In an effort to provide consistency with the growing prodromal research literature, the present study examines *DSM–IV* Axis I psychosis, including both schizophrenia and affective disorder with psychotic features. However, because it is also of interest to determine whether the movement abnormalities under investigation are more predictive of schizophrenia in particular, compared with affective psychosis, the study includes comparative analyses of both diagnostic outcomes.

Method

Participants

Participants were recruited for a prospective study of at-risk adolescents conducted at Emory University. Adolescents from the Atlanta area were recruited through announcements, describing prodromal symptoms in lay terminology, directed at parents. This study presents data on 40 adolescents, ranging in age from 12 to 18 years ($M = 14.38$, $SD = 1.73$), who underwent an initial assessment and three annual follow-up assessments.

Assent and written consent was obtained from all participants and a parent, in accordance with the guidelines of the Emory University Human Subjects Review Committee. Demographic characteristics of the sample are presented in Table 1. Exclusion criteria were neurological disorder, mental retardation, substance addiction (*DSM–IV* criteria for a substance disorder), and current Axis I disorder. In the present study, it was not necessary to exclude any potential prodromal participants due to substance abuse.

Although priority was given to the recruitment of participants who had never received a psychotropic drug, a subgroup of participants was on one or more psychotropics. This reflects national trends, in that there has been a significant increase in the number of children—especially adolescents with adjustment problems—who are taking psychotropic medications (Zito et al., 2003). The psychotropic drugs for which increased prescriptions to children have been most clearly documented are stimulants, antidepressants, and to a lesser extent, antipsychotics. This trend was apparent in the present sample; the most common psychotropic was

Table 1
Demographic and Clinical Characteristics of Sample

Characteristic	Nonconverted prodromal ($n = 30$)	Converted to psychotic disorder ($n = 10$)	Total
Gender			
Male	23	5	28
Female	7	5	12
Age (years)			
M (*SD*)	14.43 (1.65)	14.60 (1.71)	14.48 (1.65)
Ethnicity			
African American	4	3	7
Caucasian	23	7	30
Asian American	2	0	2
Other	1	0	1
Medication			
Stimulants	11	3	14
Antidepressants	11	2	13
Antipsychotics	7	1	8
Total prodromal symptoms[a]			
M (*SD*)	37.70 (11.45)	41.60 (22.76)	38.67 (14.83)

Note. Except where indicated, data presented are *n*s for each characteristic.
[a] Total prodromal symptoms measure is derived from the Scale of Prodromal Symptoms; in this study the movement abnormality item was excluded from analyses to prevent overlap with the dependent variable.

stimulants (27%), followed by antidepressants (25%) and antipsychotics (14%). Most psychotropic medications had been prescribed by pediatricians, off-label, with antipsychotics primarily directed at controlling conduct problems rather than treating psychotic symptoms. Because these medications can affect movement abnormalities, medication status was treated as a covariate in all statistical analyses.

Procedure

Assessing symptomatology. Participants in the present study met criteria for attenuated positive symptom syndrome (Miller et al., 2002), defined by the presence of moderate to severe positive symptoms. The Structured Interview for Prodromal Symptoms (Miller et al., 2002) was administered to obtain data on prodromal signs during the initial assessment. The Structured Interview for Prodromal Symptoms contains an instrument, the Scale of Prodromal Symptoms, which rates the severity of relevant symptoms along dimensions ranging from *healthy* to *pathological*. The Scale of Prodromal Symptoms is comprised of five symptom domains that are classified as *positive* (unusual thoughts or ideas, suspiciousness, grandiosity, perceptual abnormalities, conceptual disorganization); *negativ*e (social isolation, avolition, decreased expression of emotion, decreased experience of emotion, decreased ideational richness, deteriorated role function); *disorganized* (odd behavior, bizarre thinking, trouble with focus and attention, impairment in personal hygiene or social attention); and *general* (sleep disturbance, dysphoric mood, impaired stress tolerance, and motor disturbance—in the present study, to avoid overlap with the movement ratings, the motor disturbance item was not included in the ratings of prodromal status). Although all subjects met the symptom severity criteria for attenuated positive symptom syndrome, the duration criteria could not be established for all participants and were not considered a central focus in the present study. Each symptom was rated on a 6-point scale that ranged from *absent* to *severe*. The mean of the combined category scores was used as an indicator of global symptomatology.

To assess for the presence of Axis I disorders, the Structured Clinical Interview for Axis I *DSM–IV* Disorders (First, Spitzer, Gibbon, & Williams, 1995) was administered during the initial evaluation and subsequent yearly follow-up assessments (for a 4-year period). The Structured Clinical Interview for Axis I *DSM-IV* Disorders has been demonstrated to have excellent interrater reliability in adolescent populations (Martin, Pollock, Bukstein, & Lynch, 2000) and has been used in several studies that focused on adolescent populations with schizophrenia spectrum disorders (Mittal et al., 2006; Walker, Lewis, Loewy, & Palyo, 1999; Weinstein, Diforio, Schiffman, Walker, & Bonsall, 1999). The Axis I psychotic disorders identified in the study sample are listed in Table 2.

Interviews were conducted by either Elaine F. Walker (a clinical psychologist) or an advanced (4th year or beyond) psychology doctoral student. Training of interviewers was conducted over a 2-month period, and interrater reliabilities exceeded the minimum study criterion of .80 (Pearson correlation). All interviews were videotaped throughout the course of the study so that interrater reliability could be monitored. Videotapes were reviewed by Elaine F. Walker and/or a psychiatrist to confirm diagnostic reliability.

Coding of movement abnormalities. The Dyskinesia Identification System: Condensed User Scale (DISCUS; Kalachnik, Young, & Offerman, 1984) was used to code involuntary movements. The empirically developed DISCUS contains 15 items rated on a 0–4 (*absent* to *severe*) scale and employs a methodology that uses six different quality of item indices (Sprague, White, Ullman, & Kalachnik, 1984). The DISCUS was chosen because it yields high interrater reliability ($\geq$ .90) for mentally ill and nonclinical samples (Kalachnik & Sprague, 1993). The measure also yields separate indexes for different body regions: *facial* (consisting of tics, grimaces, blinking, chewing/lip smacking, puckering/sucking/thrusting lower lip, tongue thrusts, tonic tongue, tongue tremor, athetoid/myokymic/lateral tongue), *upper body* (retrocollis/torticollis, shoulder/hip torsion, athetoid/myokymic finger–wrist–

Table 2
Characteristics of Participants Who Converted From Prodromal Status to Axis I Psychotic Disorder

Participant	Age at index	Gender	Baseline medication	Age at conversion	Axis I disorder
1	12	F		14	Schizophrenia
2	15	M		16	Bi-Polar with psychotic features
3	15	F	Adderall[a] (50mg)	16	Bi-polar with psychotic features
4	13	M		15	Schizophrenia
5	15	F	Abilify[b]	16	Schizoaffective
6	13	M	Concerta[c] (72mg)	15	Schizophrenia
7	18	M		21	Schizoaffective
8	14	F	Zoloft[d] (50mg)	16	Depression with psychotic features
9	16	F		18	Schizoaffective
10	15	M	Lexapro[e] (50mg) Depakote[f] (750mg) Adderall XR (20mg) Lorazepam (.5mg PRN)	16	Bipolar with psychotic features

Note. PRN = as needed.
[a] Adderall is supplied by Shire Pharmaceuticals (Wayne, PA). [b] Abilify is supplied by Bristol-Myers Squibb, Otsuka America Pharmaceutical (New York, NY). [c] Concerta is supplied by McNeill-PPC (Ft. Washington, PA). [d] Zoloft is supplied by Pfizer (New York, NY). [e] Lexapro is supplied by Forest Pharmaceuticals (New York, NY). [f] Depakote is supplied by Abbott Laboratories (Chicago, IL).

arm, pill rolling, writhing, and alternating extensions and flexions of the fingers or wrist), and *lower body* (ankle flexion/foot tapping, toe movement). The sum of movement abnormalities in each body region was used for the present analyses.

Following the procedures used in previous research (Walker et al., 1999), movement abnormalities were coded from videotapes made during the baseline clinical interview. Interviews were conducted in private rooms and the participant was videotaped while seated in a chair facing a wall-mounted camera behind the interviewer. The chair was positioned so that the entire body was visible on tape. A total of 45 min of each videotape was coded. Raters were blind to the participants' clinical status, and coding was conducted without audio. Research assistants were trained in the application of the coding procedures over a 1-month period using tapes of nonparticipants. Coding of the participant tapes began after all pairs of raters had achieved a minimum interrater reliability of .80 for coding (Pearson correlation), independently, each body region and movement type, in a 6-min segment. The mean reliability at the end of the training period was .86, ranging from .72 to .95 across body regions.

Results

Of the 40 putatively prodromal participants, 10 (25%) converted to an Axis I psychotic disorder during the period of the study (initial assessment followed by three annual assessments). (See Table 2 for a description of the characteristics of the conversion group.) None of the prodromal individuals converted to an Axis I disorder that did not contain psychotic features (this was likely a function of the sampling strategy that focused recruitment on individuals with schizotypal and other symptoms linked with risk for developing psychosis). Analyses were conducted to test for demographic differences between the converted and nonconverted groups. Chi-square tests revealed no significant differences between converters and nonconverters in gender ratio or ethnic distribution, and *t* tests indicated no group differences in age or level of prodromal symptomatology at baseline. Screening the data using Kolmogorov–Smirnov tests revealed that distributions of movement abnormality variables were normal and met the assumptions for parametric statistics.

Associations Between Movement Abnormalities and Psychotropic Medication

Point-biserial correlations between the movement abnormality region (face, upper body, lower body) and each dummy coded medication class (yes vs. no: stimulant, antidepressant, antipsychotic) were examined to provide a framework with which to interpret the movement findings. There were no significant associations between any of the psychotropic medications and movement scores. It is important to note that prescription medication was not a controlled component of the present study; individuals undergoing medication treatment were observed under a naturalistic paradigm. As such, any interpretations, particularly given the small number of participants being treated (see Table 1), should be made with caution.

Associations Between Movement Abnormalities and Symptoms at Baseline

Relationships between movements and symptoms at baseline were assessed by conducting partial correlation analyses for each of the respective movement regions and global, positive, and negative symptoms rated with the Scale of Prodromal Symptoms. Correlations controlled for the classes of psychotropic medications using the dummy coding procedure. Abnormalities in the face region were significantly and positively associated with the three symptom scores. In the upper body region, negative symptoms were positively associated with movement abnormalities, and global symptomatology showed a strong trend in the same direction. There were no significant associations for lower body scores (see Table 3).

Movement Abnormalities and Conversion Group Differences

Univariate analysis of covariance (ANCOVA) with medication status (dummy coded: stimulant, antidepressant, and antipsychotic) treated as covariates was used to test for group differences in movement abnormalities. To clarify the relationship of psychotropic medications to movement abnormalities and outcome, the same series of analyses were also conducted without the dummy coded medication covariates in an analysis of variance (ANOVA).

Face region. As predicted, ANCOVA indicated significant differences in movement abnormalities in the facial region between the nonconverted ($M = 0.65$, $SD = 1.20$) and the converted groups ($M = 2.10$, $SD = 1.52$), $F(1, 38) = 2.08$, $p \leq .05$, $\eta^2 = .24$. Among the classes of psychotropics, no covariates approached significance. Analyses for the facial region without covariates indicated the same pattern of results for group differences, $F(1, 38) = 9.34$, $p \leq .01$, $\eta^2 = .20$.

Upper body region. For the upper body region, the ANCOVA indicated that there were significant differences between the nonconverted ($M = 2.10$, $SD = 2.17$) and the converted groups ($M = 5.10$, $SD = 3.10$), $F(1, 38) = 2.84$, $p \leq .05$, $\eta^2 = .25$. As with the previous analyses, none of the dummy coded psychotropic medication covariates approached significance. When ANOVAs were conducted, the same pattern of group differences remained significantly different, $F(1, 38) = 11.20$, $p \leq .01$, $\eta^2 = .23$.

Lower limb region. For the lower limb region, both ANCOVA and ANOVA indicated that there were no significant differences between the nonconverted ($M = 1.46$, $SD = 1.31$) and the con-

Table 3
Associations Between Prodromal Symptomatology and Movement Abnormality at Baseline

Region	Positive	Negative	Total
Face	.31*	.39**	.36**
Upper body	.08	.30*	.24
Lower body	.00	.24	.18

Note. Data presented are partial correlations controlling for psychotropic medication.
* $p \leq .05$. ** $p < .0$.

verted groups ($M = 1.60$, $SD = 1.26$). See Figure 1 for an illustration of the mean group differences.

Subtypes of Psychotic Outcomes

In the present study, 3 of the prodromal individuals converted to a schizophrenia outcome and 7 converted to affective illness (bipolar, schizoaffective, or unipolar) with psychotic features (see Table 1). To determine whether the group differences in movement abnormalities were related to one of these outcomes in particular, subgroup comparisons were conducted. ANCOVAs and ANOVAs were conducted for each movement region using three groups: the nonconverted group, a group converted to a schizophrenia outcome ($n = 3$), and a group converted to affective disorder with psychotic features ($n = 7$). It is important to note that these are exploratory analyses, and that the low number in the conversion subgroups limits statistical power. As such, results should be interpreted with caution.

Face region. For the face region, omnibus analyses were significant, ANCOVA, $F(1, 38) = 3.53$, $p \leq .01$, $\eta^2 = .34$ (no covariates were significant); ANOVA, $F(1, 38) = 7.22$, $p \leq .01$, $\eta^2 = .28$. Post hoc analyses indicated that both converted groups were significantly elevated when compared to the nonconverted prodromal adolescents ($p \leq .05$) and the schizophrenia group showed a trend toward exhibiting more movement abnormalities than the group with affective disorder with psychotic features ($p = .06$).

Upper body region. For the upper body region, omnibus analyses were significant, ANCOVA, $F(1, 38) = 4.77$, $p \leq .01$, $\eta^2 = .42$, and no covariates were significant; ANOVA was also significant, $F(1, 38) = 11.49$, $p \leq .01$, $\eta^2 = .39$. Post hoc analyses indicated that the schizophrenia converted group was significantly elevated in comparison to the affective disorder with psychotic features group and the prodromal nonconverted group ($p \leq .05$). There was a trend ($p = .11$) indicating the affective disorder group exhibited elevated movement disorders in comparison to the nonconverted prodromal group.

Lower body region. For the lower body region, omnibus analyses were not significant for ANCOVA ($\eta^2 = .21$) or ANOVA ($\eta^2 = .01$). See Table 4 for standard deviations and mean group differences.

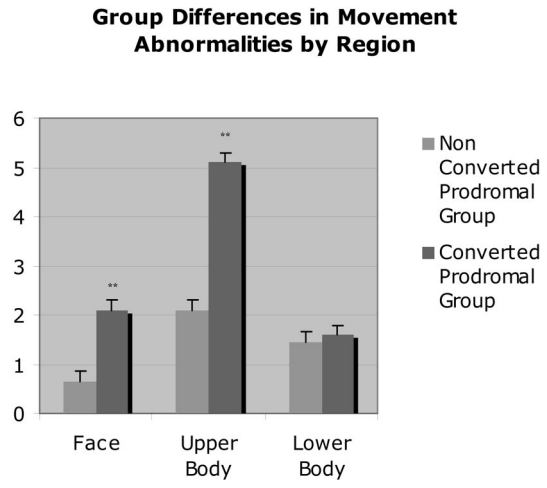

Figure 1. Group differences in movement abnormalities by body region. Movement abnormalities in the face and upper body regions were significantly more frequent in the converted prodromal group ($^{**}p \leq .01$). Error bars represent standard error of the mean.

Logistic Regression of Movement Abnormality Predicting Conversion

To provide an index of the power of movement abnormalities in predicting conversion, we conducted logistic regression analyses with movement abnormality in the respective region as a continuous predictor variable and conversion status (yes/no) as a dichotomous outcome. For these regression equations, psychotropic medications (dummy coded stimulant, antidepressant, and antipsychotic classes) were entered as control variables. Odds ratios were highly significant for the facial region and the upper body region, suggesting that abnormalities in these regions are strong predictors of conversion (see Table 5 for estimated odds ratios). Results for the lower body region were not significant. The psychotropic medication covariates did not approach significance.

Discussion

Our finding that 25% of participants converted to Axis I psychosis within a 4-year period is highly similar to the rate reported in other prospective longitudinal studies of prodromal youth (Yung & McGorry, 1996; Yung et al., 1998). Consistent with our hypothesis, the converted group exhibited significantly more movement abnormalities in the face and upper body regions than did the nonconverted group. Thus it appears that the kinds of upper body and face movement abnormalities rated on the DISCUS are most common in prodromal adolescents, who convert to an Axis I disorder within 4 years.

Congruent with our previous research comparing at-risk to healthy control youngsters (Walker et al., 1999), there were no significant group differences in the lower extremities. It is possible that the current methodology, in which individuals were seated during the assessment of movement abnormalities, may confound assessment by limiting movements in the lower extremities. Indeed, the earlier study (Walker et al., 1999) also used a coding procedure with seated individuals. However, consistent with the present findings, other reports examining movement abnormalities have noted a trend for more pronounced movement abnormalities in facial and upper body regions. For example, in a report examining movement abnormalities in adults with psychosis, researchers, using a coding method in which participants were not seated, observed abnormalities in the oral/facial region roughly three times as often—and in the upper body region roughly twice as often—as abnormalities in the lower extremities (Puri, Barnes, Chapman, Hutton, & Joyce, 1999). One potential explanation for the region differences observed in the present investigation lies in the neurological basis of movement abnormalities; because there are topographically organized motor subcircuits for different body regions, it is possible for movement abnormalities to be limited to one area (Walker, 1994).

The present findings support the theory that that hyperkinesias and psychotic symptoms involve shared neural mechanisms (Gray-

Table 4
Means and Standard Deviations of Movement Abnormalities by Group

	Nonconverted ($n = 30$)		Schizophrenia converted ($n = 3$)		Affective disorder with psychotic features ($n = 7$)		
Region	*M*	*SD*	*M*	*SD*	*M*	*SD*	Group difference
Face	0.65	1.20	3.33	1.52	1.57	1.27	$2 > 1, 3^{*}; 3 > 1^{a}$
Upper body	2.10	0.97	8.33	2.17	3.71	3.05	$2 > 1, 3^{*}; 3 > 1^{a}$
Lower body	1.46	1.31	2.00	1.00	1.42	1.39	$1 = 2 = 3$

Note. Analyses are controlled for psychotropic medications.
[a] Statistical trend, $p \leq .15$.
[*] $p \leq .05$.

biel, 1997; Mittal, Dhruv, et al., 2007; Mittal, Tessner, et al., 2007; Walker, 1994;). Hyperkinetic movements are assumed to be a reflection of overactivation of ascending dopamine pathways, specifically the striatal pathway mediated by the D2 receptor subtype (Alexander, Crutcher, & Delong, 1990; Smith, 1982). Striatal D2 receptor irregularity has been strongly implicated in Axis I psychosis (Kestler, Walker, & Vega, 2001; Seeman & Kapur, 2005). Given this overlap, some researchers have suggested that cortico–striato–pallido–thalamic circuit malfunction, moderated by dopaminergic function, is responsible for symptomatology that includes deficits in motivation and cognitive functioning (Gray et al., 1999) as well as both positive and negative psychotic symptomatology (Graybiel, 1997; Walker, 1994). If this theory is correct then it may be possible to draw tentative hypotheses with regard to which dysfunctional neural regions are responsible for movement abnormalities and risk for conversion to psychosis.

In the present study, facial and upper body region movement abnormalities were uniquely associated with symptoms and predicted conversion. Because the orofacial and upper limb regions are predominantly represented in the ventromedial and adjacent areas of the putamen (Walker, 1994), our results point to these regions as prime candidates for neural dysfunction. In contrast, because movement abnormality findings were uniformly nonsignificant for the lower body region, our results suggest that dorsolateral areas of the putamen are not associated with the relationship between movement and psychosis (Walker et al., 1999).

Graybiel (1997) is among those who have suggested that the abnormalities in the basal ganglia may be involved in the generation of dyskinesia and psychotic symptoms. The basal ganglia are linked with forebrain structures that play a role in planning, goal-directed behavior, and monitoring intentions. It has been suggested that irregular neuroactivation, and/or compromised structural or neural circuitry in the basal ganglia, could contribute to psychotic symptomatology. For example, if the striatum plays a role as a goal selector for potential adaptive processes (Schulz et al., 1986), then a disturbance in this subcortical region might result in a "disconnect" between the goals represented in the prefrontal cortex and the basal ganglia's selection of a particular response. In other words, a psychotic patient might experience this disconnection as a feeling that they are not in control of their own behavior or motivations (Neumann & Walker, 2003). Consistent with this hypothesis, Moller and Husby (2000) characterized the initial prodrome of psychosis in first-episode schizophrenia as self-disturbances that were related to losing control of cognitive and affective experiences. Another possibility is that because cortico–striato–pallido–thalamic pathways play a role in linking motor and emotional pathways (Lichter & Cummings, 2001), movement functioning is potentially linked with symptomatology relating to emotion.

The findings reported in the prodromal literature suggest that the diagnostic outcome of at-risk individuals is often either schizophrenia or affective disorders with psychotic components (Cardno, Rijsdijk, Sham, Murray, & McGuffin, 2002; Miller et al., 2002; Riley & Kendler, 2006; Yung et al., 1998). Given this finding, and to remain consistent with the convention in prodromal research, the outcome variable in the present study was conversion to Axis

Table 5
Logistic Regression Results (Odds Ratio) for Prodromal Individuals Who Converted to Axis I Schizophrenia Spectrum Disorders and Mood Disorders With Psychotic Features Compared With Nonconverted Individuals

						95% CI	
Region	Estimate	*SE*	Wald	*p*	OR	Lower	Upper
Facial	1.09	0.45	5.90	.01	2.98	1.23	7.19
Upper body	0.48	0.19	6.10	.01	1.60	1.10	2.37
Lower body	−0.04	0.32	0.02	.88	0.23	0.02	2.37

Note. The regressions that controlled for psychotropic medications included stimulants, antidepressants, and antipsychotics. OR = odds ratio; CI = confidence interval.

I psychosis (i.e., encompassing outcomes of schizophrenia and affective disorders with psychotic features). Whereas our study found that the conversion group was heterogeneous with both psychotic and affective outcomes, the results of more fine-grained analyses (partitioning the converted group into both schizophrenia and affective disorder with psychotic features outcomes) suggest that movement abnormalities, like many other impairments, are more pronounced in those with schizophrenia outcomes. Although movement abnormalities were elevated in both converted groups in comparison to the nonconverted prodromal adolescents, the schizophrenia group exhibited considerably elevated movement abnormalities in the facial and upper body regions relative to the group with affective disorder with psychotic features. However, because of the small samples that resulted when the groups were divided, any interpretation is tentative. Future studies with larger samples are necessary to determine whether this effect is an artifact of the small sample or reflects a real phenomenon.

Results of the logistic regression analyses provide support for the potential of combining behavioral and physical markers to enhance prediction of psychosis (Cannon et al., 1999). More specifically, movement abnormalities in the limbs and facial area may hold promise for predicting Axis I disorders in youth with prodromal signs. Although research is beginning to support the notion that pharmacological intervention can ameliorate the course of illness—or even prevent the onset of psychosis (Haroun et al., 2006)—the side effects of available medications (e.g., weight gain, diabetes) make providing blanket drug treatment to all prodromal individuals an inappropriate strategy. Movement abnormalities are relatively easy to measure, and thus have potential to serve as risk indicators that, in combination with other behavioral and biological measures, may enhance prediction of psychiatric outcome.

The subgroup of adolescents who were being treated with psychotropic medications presented a methodological challenge as well as a tentatively interesting pattern of preliminary results. Although medications were statistically controlled in each of the present analyses (e.g., group comparisons were conducted with and without covariates), this procedure did not eliminate the potential confound of medication effects. Prescription of psychotropics is expected to target individuals with more severe behavioral dysfunction and, perhaps, concomitant movement abnormalities. Thus, controlling for medication can affect the variance in ratings of symptoms and movements, thereby attenuating the covariance between these two factors. In observing the demographic differences between converted and nonconverted groups, it was noteworthy that a relatively small number of participants treated with antipsychotic medications were in the converted group. Although this observation may indicate a protective effect of medication, such a conclusion is premature. First, there may have been premedication clinical differences between adolescents prescribed and not prescribed this medication (i.e., a selection bias). Second, the biserial correlations conducted in the present study between the different classes of medication and regions of movement were not significant. Finally, the present study was not intended to address this question, and the number of participants prescribed antipsychotic medication in the present sample ($n = 8$) was too small to draw conclusions about medication effects. As such, future research aimed at examining this question directly is warranted.

Another limitation of the present study concerns the coding procedure which was applied to videos of seated participants and may have masked movement abnormalities in the lower limbs. Future research should examine movement abnormalities using other methodologies; one good candidate methodology is illustrated in a recent study conducted by Schiffman and colleagues (2004) in which movement abnormalities were coded by observing videotapes of playground and social interactions. Finally, future studies should aim to examine demographic differences in outcome groups. In the present study, there were sizable (but nonsignificant) group differences in the ratios of converted individuals who were female and who were African American.

Despite these limitations, the present findings add to the accumulating body of literature indicating that research on prodromal adolescents and young adults holds great promise for identifying the most at-risk individuals prior to the onset of clinical episodes. In addition, this work has the potential to elucidate neural mechanisms that may be linked with the conversion from prodromal status to Axis I psychotic disorders. Both objectives are critical to the long-term goal of preventive intervention.

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Received December 11, 2006
Revision received March 12, 2007
Accepted March 12, 2007 ■