



# Differences Between People Who Do and Do Not Stutter

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# Introduction

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- This study attempts to empirically test a specific hypothesis (i.e., the tendency toward psychosocial-emotional disorder pole of the bipolar stuttering threshold hypothesis) in relation to its possible role in developmental stuttering etiology.
- The primary purpose of this study is to test the tendency-toward-psychopathology pole (pole A) of the etiologic four-factor bipolar stuttering threshold hypothesis (Treon, 1995, 2002). In turn, this hypothesis is an integral aspect of the more encompassing etiologic four-factor bipolar psychopathology and neurolinguipathology based linguistic and paralinguistic processing deficit syndrome hypothesis (i.e., the PNB-LPPD syndrome hypothesis (Treon, 2002), of which stuttering behavior is but one sign and symptom.


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- **Both of these hypotheses propose that, on average, PWS have a modestly greater tendency toward psychosocial-emotional disorder (toward psychopathology) of various kinds than do PWNS. Both hypotheses assert that this average difference between PWS and PWNS is real (statistically significant), but not large in magnitude. Both hypotheses also assert that this greater tendency toward psychosocial-emotional disorder in PWS is: (1) developmentally early in origin (predominantly in the preschool years), and (2) a central etiologic factor in the emergence of the problem of developmental stuttering (Treon, 1995, 2002).**
  - **Finally, both hypotheses propose that there exists a wide range of diverse psychosocial-emotional disorder tendency types (and combinations) that can play an etiologic role in the tendency-toward-psychopathology pole of these hypotheses.**

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- **According to the four-factor bipolar PNB-LPPD syndrome hypothesis, stuttering behavior is one symptom in the phenotypic spectrum of a child with an above average degree of tendency toward psychosocial-emotional disorder and/or (most often and) an above average degree of tendency toward neurolinguistic disorder (i.e., neurolinguipathology).**
  - **These two polar etiologic factors are almost always both present to some degree and interacting with one another. In general, if the magnitude of the sum of their interaction is sufficient, it will cause manifestation of the PNB-LPPD syndrome in that child (i.e., the extent of the magnitude of this interaction will exceed the perceptual magnitude threshold of the PNB-LPPD syndrome and it will perceptibly manifest itself) (Treon, 2002).**

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**This study is concerned only with the first tendency-toward-psychopathology pole (pole A). The two factors that comprise this pole are: (1) early childhood traumatic and disruptive environmental experiences interacting with (2) innate genetically based temperament-reactivity personality tendencies which predispose the child to be vulnerable to such traumatic and disruptive experiences**

- **To widely varying degrees between PNB-LPPD syndrome individuals, pole A contributes (almost always in interaction with pole B) to the etiology of a variety of linguistic and paralinguistic processing deficits which may involve sensorimotor speech, para-language and language (including pragmatic) deficits and dysfunctions. See Figure 1 below.**



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**The tendency toward neurolinguipathology (pole B) is comprised of factors three and four. Factor three is an innate genetically based tendency toward neurolinguistic and neuro-paralinguistic processing deficits. Rarely, but sometimes, factor three interacts with factor four which is early environmentally induced-experienced neuropathology based physical-organic lesions/malformations which negatively affect the normal developmental emergence of such neurolinguistic and neuro-paralinguistic processing functions. Again, to widely varying degrees between PNB-LPPD syndrome individuals, this pole contributes (almost always in interaction with pole A) to the etiology of a variety of linguistic and paralinguistic processing deficits and dysfunctions which may include sensorimotor speech, para-language and language problems.**

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- **Almost always then, it is the interaction of these two multifactorial etiologic poles together (and more rarely almost exclusively alone) that, when elevated to a critical degree of deficit or dysfunctional severity (i.e., reach perceptible PNB-LPPD syndrome threshold), cause these varied linguistic and paralinguistic problems. These two etiologic poles, usually in interaction, may cause other non-language and non-paralanguage related phenotypic spectrum signs and symptoms as well (Treon, 2002).**

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- **However, stuttering behavior is considered a primary symptom in the phenotypic spectrum of the PNB-LPPD syndrome, but perhaps not the only one (e.g., cluttering may prove to be a primary symptom as well). By primary symptom is meant that the PNB-LPPD syndrome may manifest in a given individual through signs and symptoms other than stuttering (without stuttering), but when stuttering occurs it is always indicative of the presence of this syndrome in that individual. Which is to say that, according to the PNB-LPPD syndrome threshold hypothesis, the overt and manifest presence of this syndrome in an individual is the necessary and sufficient cause of developmental stuttering. See the diagram in Figure 2 below.**




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- **The PNB-LPPD syndrome hypothesis asserts that stuttering is only one expression of a broader linguistic and paralinguistic processing deficit. The resultant singly manifesting or variously configured symptom clusters of linguistic and paralinguistic processing dysfunction and disorder are the central and most prominent feature of the PNB-LPPD syndrome. These linguistic and paralinguistic processing deficits, expressed through speech (including stuttering), language, and/or paralanguage disorders, include possible deficits and dysfunctions of sensorimotor speech, prosodic, and language (semantic, syntactic, phonologic, morphologic and/or pragmatic) processing. These speech-language and paralanguage processing deficits may be of capacity, efficiency, complexity, organization and/or synchrony. See Figures 3 and 4 below.**

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- **Both the bipolar stuttering threshold hypothesis and its more inclusive bipolar PNB-LPPD syndrome hypothesis propose that over all individuals with the PNB-LPPD syndrome (i.e., thus over all PWS) the average etiologic contribution of each of the two poles to this syndrome (and thus to stuttering) is approximately equal. However, to account for the proposed wide variance in relative degree of etiologic contribution of these two poles between PWS (between people with PNB-LPPD syndrome), there are hypothesized to be seven sub-syndromes categories of this syndrome for individuals who perceptibly stutter (Treon, 2002).**

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- **The following sub-syndromes designate the relative (proportionate) degree of etiologic contribution of each of these two poles within each sub-syndrome: (1) predominantly psychosocial-emotional disorder based and very secondarily neurolinguipathology based PNB-LPPD sub-syndrome, (2) primarily psychosocial-emotional disorder based and strongly secondarily neurolinguipathology based PNB-LPPD sub-syndrome, (3) predominantly neurolinguipathology based and very secondarily psychosocial-emotional disorder based PNB-LPPD sub-syndrome, (4) primarily neurolinguipathology based and strongly secondarily psychosocial-emotional disorder based PNB-LPPD sub-syndrome, (5) approximately equally balanced psychosocial-emotional disorder and neuolinguipathology based PNB-LPPD sub-syndrome, (6) almost exclusively psychosocial-emotional disorder based PNB-LPPD sub-syndrome, and (7) almost exclusively neurolinguipathology based PNB-LPPD sub-syndrome.**


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- **Conceptually, three functional levels of the etiologic four-factor bipolar PNB-LPPD syndrome hypothesis are proposed. The first two are etiologic variables and the third is a symptomatic variable.**
    - **First is the genetically based deep etiologic functional level which is comprised of either or both of the two genetically based factors (factors two and three). This is the foundational etiologic basis of the PNB-LPPD syndrome, and plays a critical originating role in its etiology.**
    - **Second is the environmentally based precipitating etiologic functional level which is composed of either or both of the two environmentally based etiologic factors (especially factor one and rarely factor four alone). This is the mediating etiologic basis of the PNB-LPPD syndrome, and plays a central precipitating role in its etiology.**


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- **The third level is expressed when perceptible linguistic and paralinguistic processing deficits (including stuttering) occur, as they almost always do, in the phenotypic spectrum of this syndrome. This level is the psychosocially learned conceptual, attitudinal, affective, and behavioral reactionary coping and adaptive surface functional level. From very early in its onset, such learning factors may function to maintain, and possibly even elaborate upon and increase, the severity of any speech-language-paralanguage disorder symptoms that may initially emerge.**

# Methods

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- **The revised MMPI -A and MMPI -2 were used to compare personality characteristics of people who stutter (PWS) and people who do not stutter (PWNS).**
- **Subjects consisted of 60 PWS (people who stutter) and 60 PWNS people who do not stutter).**
- **Subjects were matched for: gender, education, socio-economic background, racial/ethnic background, social outgoingness, emotional expressiveness, & primary language.**

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- **All PWS were administered the Stuttering Severity Index.**
  - **All PWS completed a Perception of Stuttering Inventory.**
  - **All subjects (PWS & PWNS) were administered the MMPI-2 or MMPI-A.**
  - **All MMPI-2/A outcomes were computer scored.**

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- **There are 99 possible personality scales or subscales on the MMPI-2, depending on the number that are commonly shared between matched pairs.**
  - **93 of these personality scales/subscales had a full complement of subject pairs.**







# Results


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- **Only the 93 personality scales/subscales that had a full complement of subject pairs were used in the analysis of T-score differences between stuttering and nonstuttering groups.**

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- The following results were found for the 93 scales/subscales:
    - **The mean T-score for stuttering subjects was higher than for nonstuttering subjects in 80 (86%) of these 93 scales/subscales.**
    - **The mean T-score for nonstuttering subjects was higher in 13 (14%) of the 93 scales/subscales.**

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- **Analysis of variance indicated that the mean T-scores of the 60 stuttering subjects were statistically significantly higher than their 60 nonstuttering controls at or below the .05 level on 24 (25.8%) of the 93 scales/subscales.**

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- **Of the 93 scales/subscales, 83 (89.3%) had higher mean standard deviation values for stuttering than for nonstuttering subjects.**
  - **The mean standard deviation value for all stuttering subjects across all 93 scales/subscales was 11.79.**
  - **The mean standard deviation value for all nonstuttering subjects across all 93 scales/subscales was 9.51.**
  - **Using an ANOVA, this difference yields an F-value of 115.419 (df = 1) which is statistically significant below the  $p = .001$  level.**

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- **The following scales/subscales were found to be statistically significantly higher for stuttering subjects than for nonstuttering subjects at or below the .05 level.**
    - **1 of the 2 Fear Subscales falling under the Content Component Scales**
    - **Self-depreciation, one of the Depression Subscales**
    - **General Health Concerns, 1 of the 3 Health Concerns Subscales**
    - **5 Schizophrenia subscales - Bizarre Sensory Experiences, Social Alienation, Lack of Ego Mastery-Conative, Emotional Alienation.**

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- **Average T-scores for each matched pair of stuttering versus nonstuttering subjects were also calculated using each of their respective T-scores in every scale in which they had paired scores.**


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- **The mean T-score for all 60 stuttering subjects across all MMPI -2/A scales was 52.19 (SD = 6.462).**
  - **The mean T-score for all 60 nonstuttering subjects across MMPI -2/A scales was 49.75 (SD = 4.341).**


# Interpretation & Discussion

- **One of the central findings of this study is that, averaged across all MMPI-2/A scales/subscales, subjects who stutter (SWS) score statistically significantly higher than matched subjects who do not stutter (SWNS) ( $p = .017$ ) in overall tendency toward psychosocial-emotional disorders (i.e., overall tendency toward psychopathology). This finding agrees with the explicit projection of the tendency-toward psychopathology pole (Treon, 2002) that: (1) there exists a greater tendency toward such disorder in PWS versus PWNS, and (2) that this tendency is relatively small but statistically significant (i.e. appears to be a real average difference).**



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- **Another central finding that may help to clarify the nature of the distribution (i.e. range and variability) of this apparent greater average tendency toward psychosocial-emotional disorder in PWS concerns the T-score standard deviation differences found between SWS versus SWNS in this study. The mean standard deviation value for all stuttering subjects was greater than the mean standard deviation value for all nonstuttering subjects in eighty-three (89.25%) of the ninety-three MMPI-2/A scales/subscales sampled. The mean standard deviation value across all ninety-three scales/subscales was 11.79 for all stuttering subjects versus 9.51 for all nonstuttering subjects. This difference was statistically significant below the  $p = .001$  level.**


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- **This outcome indicates that stuttering subjects, on average, displayed greater within scale T-score variance than do nonstuttering subjects across these ninety-three MMPI-2/A scales/subscales. This statistically significant MMPI-2/A finding of greater within scale variance in tendency toward psychosocial-emotional disorders among stuttering versus nonstuttering subjects can be interpreted as supportive of the tendency toward psychopathology pole hypothesis of the bipolar stuttering threshold hypothesis.**


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- **In agreement with the great majority of previous personality inventory studies comparing SWS to SWNS (see Bloodstein, 1995 for a review of such studies), the results of this study, according to MMPI-2/A T-score standardization values, suggest that, in general and on average, PWS are not neurotic, borderline, or psychotic (i.e., are not psychopathologic), but rather fall within the normal range of psychosocial-emotional functioning and adjustments. This finding also agrees with specific projections to this effect put forth in relation to the tendency-toward- psychopathology pole of the bipolar stuttering threshold hypothesis (Treon, 2002).**

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- **In this study, the tendency for SWS to have higher mean T-scores than SWNS across the ninety-three MMPI-2/A scales/subscales examined was very evident. SWS scored higher on mean T-score than SWNS on eighty (86%) of these ninety-three scales/subscales. Of the twenty-four scales/subscales (25.8%) that had statistically significantly higher mean T-scores for SWS versus SWNS, the following tendency toward psychosocial-emotional disorder trends appeared.**

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- **The six most prominent findings in this regard (i.e., prominent by virtue of the number of related scales/subscales involved and/or the levels of statistical significance of those scales/subscales) are in scales/subscales related to schizophrenia, depression, health concerns-somatic complaints, psychasthenia (tendency toward phobia, obsession and compulsion), anxiety-fearfulness, and self-doubt-self-depreciation. Stuttering subjects in this study had statistically significantly higher mean T-scores than nonstuttering subjects at or below the .05 level in various dimensions of all of these psychological problem areas. This was especially apparent in the areas of schizophrenia and depression.**


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- **The Depression Scale in the Basic Scales Profile: Clinical section had the highest level of significance at  $p = .002$  of any of the ninety-three scales/subscales studied. In general, these depression findings agree with those of Walnut (1954) in a study using the original MMPI, and of Richardson (1944).**
  - **The greater tendency toward health concerns in the Content Profile Scale ( $p = .019$ ), general health concerns in the Health Concern Subscales ( $p = .008$ ) and somatic complaints in the Hysteria Subscales ( $p = .026$ ) in stuttering versus nonstuttering subjects was quite evident in this data. Also evident was the increased tendency toward phobia, obsession and compulsion (i.e., the Psychasthenia scale) ( $p = .013$ ) in stuttering versus nonstuttering subjects.**

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- **The findings of this study indicate that the tendency toward Content Scale Profile anxiety ( $p = .018$ ) and Supplementary Scale Profile anxiety ( $p = .056$ ) as well as generalized fearfulness in the Fear Subscales ( $p = .032$ ) were notably higher in stuttering than in nonstuttering subjects.**
  - **In general, these anxiety findings agree with those of Ezrati-Vinacour & Levin (2004), Craig et al. (2003), Guitar (2003), Gabel et al. (2002), Mahr & Torosian (1999), Stein et al. (1996), Craig (1990), Kraaimaat et al. (1991), Fitzgerald et al. (1992), Santostefano, (1960). Also, self-doubt ( $p = .039$ ) and self-depreciation ( $p = .015$ ) mean T-scores were higher in stuttering than in nonstuttering subjects. In general, these self-doubt and self-esteem related findings agree with those of Perkins (1947).**

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- **Whether or not any given factor regularly associated with the problem of stuttering is a part of its cause, its effect, or both has been frequently debated since the beginning of the scientific study of stuttering (Bloodstein, 1995).**
  - **It seems reasonable to suppose that the psychosocial- communicative stigma that a person who stutters (or has any other type of pronounced speech or language disorder for that matter) must daily encounter, confront and respond to, has a psychosocial-emotional influence (i.e., has its own psychosocial-emotional “rebound” effect) on that individual).**



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- **Such an effect could be expected to account for at least some of the statistically significant scale/subscale differences in this study (e.g., perhaps the social anxiety dimension of generalized anxiety)**
  - **In summary, the overall range, direction and magnitude of the statistically significant findings of this study (and these findings tend to be congruent with the multiple experimental findings cited earlier of greater temperamental sensitivity and emotional reactivity of CWS versus CWNS), together with the likelihood that many, if not most, of these tendencies toward psychosocial-emotional disorder have their origins in early childhood, can reasonably be interpreted as generally supportive of the tendency toward psychosocial-emotional disorder pole hypothesis of the etiologic bipolar stuttering threshold hypothesis.**

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- **Interpreted in this way, the data of this study tends to support the hypothesis that, in general, a child's early traumatic-disruptive experiences interacting with his or her genetically based temperament-reactivity predisposition to be psychologically vulnerable to such traumatic-disruptive experiences (i.e., a child's above average tendency toward psychosocial-emotional disorder) plays a significant role in the etiology of developmental stuttering.**

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