Identifying Cloninger’s Temperament Profiles as Related to the Early Development of the Metabolic Cardiovascular Syndrome in Young Men

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Abstract—Our aim in this study was to (1) identify naturally occurring temperament profiles in young adulthood by using Cloninger’s temperament dimensions and (2) examine the relationship of these profiles with the coronary heart disease (CHD) risk factors of the metabolic cardiovascular syndrome (insulin resistance syndrome, IRS) measured during adolescence and young adulthood. A randomly selected sample of 190 healthy, young adult men was divided into 4 temperament groups by cluster analysis. Physiological parameters studied were serum insulin, high density lipoprotein cholesterol, triglycerides, systolic blood pressure, body-mass index, subscapular skinfold thickness, and the IRS factor. The results showed that a temperament profile characterized by a high level of persistence and reward dependence, and a low level of novelty seeking, and a low level of harm avoidance was related to a high level of physiological CHD risk factors; in 3 study phases over a 6-year period, the subjects belonging to that cluster in adulthood were shown to have always belonged to the highest risk group in terms of the physiological risk factors in adolescence and young adulthood. The findings suggest that the temperament profile in question may predispose an individual to the development of the IRS and CHD. (Arterioscler Thromb Vasc Biol. 1999;19:1998-2006.)

Key Words: temperament □ cluster analysis □ coronary heart disease risk □ insulin resistance syndrome

Broad individual variability in sympathetic and adrenocortical responses to psychological stress both in laboratory settings and in real-life situations is a widely documented fact.1–3 Despite considerable research effort, the origin of this interindividual difference remains obscure. Recently, temperament has been seen as a promising concept for studying the sources responsible for individual differences in both psychological and physiological stress proneness.4 There are various definitions of the concept of temperament in the research literature. The most general definition is that temperament consists of biologically rooted individual differences in behavior tendencies that are present early in life and are relatively stable across time and situations; temperament highlights a person’s behavioral style, ie, the how of behavior.5,7 Temperament is also the biologically rooted core of an individual’s personality; personality develops from the interaction between innate temperament and experience.5 Temperament has been shown to be inherited, at least in part; on average, slightly <50% of the variance in temperament traits appears to be explainable by referring to the genetic factor, although the heritability estimates for different dimensions vary considerably.8–12 Although temperament is subject to changes caused by maturation and environmental factors, its dimensions have been shown to be rather stable,13,14 even from childhood to adulthood.15,16

Temperament has its origin in brain structure, and individual differences in temperament are attributable to differences in neural and physiological functions (eg, see Reference 17). In the past decade, evidence of the moderating effect, even of the cause-effect relationship, of temperament on stress-induced physiological arousal has increased markedly. This relationship has been demonstrated in both major neuroendocrine stress systems, ie, the sympathetic18–25 and adrenocortical (usually cortisol level18,21,25–30) systems. Most studies have, however, been carried out among children, even toddlers, and the stressors used have been age-appropriate, eg, separation from the mother and joining a new peer group. The findings are thus not applicable to adults for assessing daily life stress: stress in the workplace, for instance.

The role of temperament-related behavioral characteristics, like type A behavior and hostility, or temperament-rooted personality traits, like introversion-extraversion, in the pathogenesis of cardiovascular disease has been widely investigated. However, innate temperament as such, ie, with the use of temperament theories and their operationalizations, has not usually been treated as a behavioral risk factor for somatic diseases. This is surprising, because the well-known individual variation in sympathetic-adrenomedullary and hypothalamic-pituitary-adrenocortical activation by brief psychological stressors might help us to understand why daily irritations and...
stressors have greater health consequences for some individuals than for others. Inherited temperament might be especially relevant to the etiology of coronary heart disease (CHD), in which the roles of stress and sympathetic-adrenal (SA) and hypothalamic-pituitary-adrenal (HPA) axis activity are well documented. This study was undertaken with that in mind. We were interested in (1) identifying naturally occurring temperament profiles in a population-based sample of young adult men and (2) examining the relationship of those profiles with the important physiological CHD risk factors included in the metabolic cardiovascular syndrome measured during adolescence and young adulthood.

The metabolic cardiovascular syndrome—also called the insulin resistance syndrome (IRS)—is a plurimetabolic disorder consisting of insulin resistance, compensatory hyperinsulinemia, glucose intolerance, an increased concentration of serum triglycerides (TGs), a decreased concentration of serum HDL cholesterol (HDLC), and hypertension. Cellular resistance to insulin-stimulated glucose uptake has been postulated as the primary metabolic defect responsible for the other manifestations of the syndrome. Even though CHD usually manifests itself in middle age or later, the atherosclerotic process underlying it originates in childhood and adolescence, and coronary risk factors are already present then. Thus, it is important to understand the pathogenic processes associated with the development of high levels of metabolic CHD risk factors, even in the young. The metabolic cardiovascular syndrome was considered to be of special relevance here, because the role of stress and stress-induced arousal of the SA and HPA axes in the development of this syndrome is widely accepted.

Cloninger’s biosocial temperament theory was adopted for assessing temperament. According to this theory, genetically homogeneous and independent temperament dimensions exist, each of which is 50% to 65% inheritable. These 3 dimensions are defined in terms of individual differences in the sensitivity to specific environmental stimuli and the behavioral responses to those stimuli, and they reflect variation in 3 brain systems; ie, each dimension is associated with a specific neurotransmitter. The temperament dimensions are novelty seeking (NS), harm avoidance (HA), and reward dependence (RD). NS reflects behavioral activation; it is a heritable tendency toward frequent exploratory activity and intense excitement in response to novel stimuli, as well as active avoidance of monotony. It is correlated with low basal dopaminergic activity. HA is connected with behavioral inhibition and high serotonergic activity. It reflects a heritable tendency to respond intensely to aversive stimuli and to learn to avoid punishment, novelty, and frustrating nonreward. RD is connected with behavioral maintenance. It reflects a heritable tendency to respond intensely to signals of reward (particularly verbal signals of social approval and succor) and to maintain or resist extinction of behavior that has previously been associated with rewards or relief from punishment. It is correlated with low basal noradrenergic activity. Persistence (P) later emerged as a distinct fourth dimension, although it was originally thought to be a component of RD and was measured in terms of perseverance despite frustration and fatigue.

Previous literature does not give a basis for specific hypotheses. There is, however, evidence for an association of NS with some behavioral risk factors of CHD, such as smoking and alcohol abuse. Also, HA is close to the concept of social inhibition, which in turn has been associated with social aloneness and high reactivity of the SA and HPA axes during social challenge, whereas RD includes aspects totally opposite to the traditional psychological CHD risk factors. That being so, HA and NS were expected to be related to high levels of metabolic CHD risk factors whereas RD was suggested to be a protective factor.

Methods

Sample

The participants were 190 randomly selected healthy boys and men who participated in the prospective epidemiological Cardiovascular Risk in Young Finns (CRYF) study, in which the development of risk factors for CHD has been monitored at intervals of 3 years beginning in 1980; in addition, in 1997, a temperament survey was performed. The subjects of the CRYF study, a total of 3596 healthy Finnish children, adolescents, and young adults, were a randomly selected sample of 360 rural and 360 urban girls and boys in the age cohorts of 3, 6, 9, 12, 15, and 18 years in 1980. The design of the CRYF study and the selection of the sample have been described previously.

The participants in the current study were the 3 oldest male age cohorts of the CRYF study who, at the baseline of the present study (year 0, n = 866) were 12 to 18 years of age and consequently were 15 to 21, 18 to 24, and 29 to 35 years of age during the first (year-3 follow-up, n = 645), second (year-6 follow-up, n = 565), and third (year-17 follow-up, n = 400) follow-up examinations in 1983, 1986, and 1997, respectively. At years 0, 3, 6, and 17, complete data were available from 190 participants. Despite the high dropout rate, there was no systematic bias in attrition.

Measures

Temperament

At year 17, temperament of the subjects was measured by Cloninger’s Temperament and Character Inventory (TCI), a 240-item, self-administered questionnaire. The TCI was designed to assess the 4 temperament dimensions and 3 character dimensions as defined by Cloninger’s theory. In the present study, however, only the 4 temperament scales comprising 107 items were used. The temperament dimensions addressed by the TCI are as follows: (1) NS (4 subscales: exploratory excitability versus stoic rigidity, impulsiveness versus reflection, extravagance versus reserve, and disorderliness versus regimentation), (2) HA (4 subscales: anticipatory worry and pessimism versus uninhibited optimism, fear of uncertainty, shyness with strangers, and fatigability and asthenia), (3) RD (3 subscales: sentimentality, attachment, and dependence), and (4) P.

Examples of NS items from the TCI include “I’m slow to get excited about new ideas” (reverse scored), “I think in detail before deciding” (reverse scored), “I’m better at saving money than most” (reverse scored), and “I do things spontaneously.” Examples of HA items include “I’m confident that things will go well” (reverse scored), “I get tense and worried in unfamiliar situations,” “I avoid meeting strangers,” and “I have less energy than most.” The RD scale consists of items such as “I’m strongly moved by sentimental appeals,” “I don’t open up much even with friends” (reverse scored), and “Others think I am too independent” (reverse scored). The P scale consists of items such as “I often push myself to exhaustion,” “I work long after others give up,” and “I am satisfied with my accomplishments, and have little desire to do better” (reverse scored). Items of each temperament scale were added up to yield total scores of NS, HA, RD, and P.

The content, construct, and predictive validity as well as reliability of the TCI scales in a Finnish sample have previously been shown to be acceptably high.

Metabolic Coronary Risk Factors

The following measurements relevant for the IRS were obtained for all participants: serum insulin, serum HDL-C, serum TGs,
systolic blood pressure (SBP), weight (in kilograms), height (in meters), and subcapular skinfold thickness (SSF). These physiological parameters were measured at years 0, 3, and 6.

BP was measured with a standard mercury gravity sphygmomanometer on the right arm, after a rest of at least 3 minutes, to the nearest even figure. SSF was measured by Harpenden calipers (Holtain and Bull-British Indicators instruments) to the nearest 0.2-mm reading. SSF was used as an index of upper-body subcutaneous fat distribution. Height was measured by Seca anthropometer and weight by Seca scales. Body-mass index (BMI) was calculated as weight divided by height squared. The procedures for anthropometric measurements have been described in detail elsewhere.

After an overnight fast, venous blood samples were taken from the right antecubital vein of recumbent subjects. Serum insulin was measured by a modification of the immunossay method of Herbert et al in the Research Laboratory of the Department of Pediatrics, University of Oulu, Oulu, Finland. The intra-assay CV was 1.6% and the interassay CV 8.4%, the sensitivity of the assay being 0.5 mU/L. All lipid determinations were done in duplicate, with standard enzymatic methods used for serum cholesterol (Boehringer CHOD-PAP) and TGs (Boehringer) in the laboratory of the Rehabilitation Research Center of the Social Insurance Institution, Turku, Finland. This laboratory continuously cross-checks the lipid determinations with the World Health Organization laboratory in Prague, Czech Republic. Serum HDL-C concentrations were measured from the serum supernatant after precipitation of VLDL and LDL lipoproteins with dextran sulfate 500 000. All analyses were performed as simultaneously as possible. The averaged within-run and between-run CVs were 1.7% and 3.8% for HDL-C and 2.6% and 4.4% for serum TGs. A more detailed description of our assessment protocol has been reported elsewhere.

**Design of the Study**

In principle, given the hypothesis that temperament may influence the IRS, temperament should have been measured before, or at the same time as, the metabolic CHD risk factors. In the present study, however, temperament (independent variable) was measured after the metabolic risk factors (dependent variables); that being so, the design of the study might be regarded as inappropriate. Several facts support the view that the present study design is warranted: (1) As mentioned above, the atherosclerotic process underlying CHD originates in childhood and adolescence, and coronary risk factors are already present then. In addition, both individual risk factors making up the IRS and the clustering of these risk factors show considerable tracking* from childhood to adulthood. (2) The Cloninger temperament model is likely to be particularly relevant in the present connection, given that it focuses on the underlying neurobiological basis of temperament; such a focus enables more specific suggestions as to the mechanisms through which temperament is likely to influence metabolic CHD risk factors. In addition, no childhood version of the TCI is available; although temperament scales for use in childhood are available, they are based on other temperament models. (3) Importantly, in regard to Cloninger’s model, the number and structure of temperament dimensions based on teacher ratings throughout childhood have been shown to be similar to those observed in self-reports by adults; ie, these temperament dimensions exhibit continuity from childhood to adulthood. Of course, although the structure of temperament (conceptually similar temperament factors) and the neurophysiological basis of the different dimensions remain the same from age to age, the behavioral manifestations of these dimensions may change, at least slightly, across ages (ie, heterotypic continuity). (4) If an association is found between adulthood temperament dimensions (the focus of the present study) and adolescence metabolic CHD risk factors, this result is in effect a strong argument for the stability of the temperament dimensions measured and the robustness of the association studied.

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*Tracking of risk factors refers to the maintenance of relative rank of risk status over time compared with other individuals in the same birth cohort.

**Statistical Analyses**

**Temperament Patterns**

In the person-oriented pattern approach, each subject’s complete profile of available data are considered, and individuals are grouped together on the basis of similarities in these profiles. This approach deals with data and presents them in terms directly applicable to individuals, in contrast to the traditional regression models, which yield results referring to “the average person,” who simply does not exist. Cluster analytic techniques are 1 useful method of pattern description.

Cluster analysis served to identify subgroups of individuals with similar patterns or profiles on the 4 temperament variables. The cluster analysis was performed with SLEIPNER, a statistical package for pattern-oriented analyses. Before the cluster analysis, we identified a residue of multivariate outliers with the RESIDUE module of SLEIPNER and removed those subjects who belonged to the residue. For the cluster analysis, raw scores for each temperament variable were standardized as z scores. To perform a preliminary classification, the standardized scores for NS, HA, RD, and P were entered into SLEIPNER’S CLUSTER program with use of the Ward clustering algorithm. This is an agglomerative hierarchical clustering technique that uses the squared euclidean distance to determine the similarity between subjects’ profiles on the variables. The Ward algorithm iteratively joins those clusters, the fusion of which minimizes an increase in the within-cluster or error sum of squares (error SS) while maximizing the between-cluster sum of squares. (The error SS is defined as the sum of the distances from each individual’s profile to the centroid of its parent cluster.) Subjects whose profiles have similar elevations and patterns will consequently be grouped together.

A major issue with cluster analysis is how to select the number of clusters. Unfortunately, no standard, objective selection procedure exists. However, the optimal solution for the number of clusters to retain in a cluster analysis can be based on the substantial increase in the error SS associated with a particular number of clusters compared with the error SS associated with the previous iteration. Small step-by-step increases in the error SS indicate additional clustering of cases and/or clusters of relatively homogeneous characteristics. The merging of cases and/or clusters of dissimilar characteristics is indicated by a large increase in the error SS. Thus, the optimum number of clusters is reached at the stage of the clustering process preceding the substantial increase in the error SS. It has been suggested, however, that it is probably best to compute a number of different cluster solutions (eg, 3, 4, and 5) and then decide among the alternative solutions by using a priori criteria, practical judgment, common sense, or theoretical foundations.

After the preliminary classification, the subjects were relocated to the clusters by using SLEIPNER’S RELOCATE module. RELOCATE starts from an initial classification and moves cases from 1 cluster to another if that reassignment leads to a reduction in the total error SS of the cluster solution. This produces more homogeneous clusters.

**IRS Factor**

Physiological parameters, ie, serum insulin, HDL-C, TGs, SBP, BMI, and SSF, were subjected to principal-components analysis. The resulting IRS factor is described under Results.

**Relationship of Temperament Patterns to Metabolic Coronary Risk Factors**

Differences in metabolic risk factors, ie, serum insulin, HDL-C, TGs, SBP, BMI, SSF, and IRS factor, between the groups (temperament clusters) were analyzed by ANCOVA with age as the covariate (see also the footnote).

**Results**

**Temperament Patterns**

Residue analysis resulted in the identification of 8 individuals who were multivariate outliers (4% of the original sample). These 8 individuals were removed from the data set before performance of the cluster analysis. Analysis was thus performed on the data from 182 individuals. Based on the criteria...
mentioned above, cluster data were best represented by a 4-cluster solution; ie, 4 clusters of individuals appeared with relatively similar patterns or profiles on the temperamental variables. After the preliminary cluster analysis, the relocation cluster analysis was performed next. Figure 1 displays the results of this final clustering by both mean data and a pictorial representation. Inspection of the mean standardized scores for the temperament variables indicated that cluster 1 was characterized by a notable elevation of P and RD, low values for HA, and an average level of NS. Cluster 2 was distinguished by rather high P and low RD, NS, and HA. Cluster 3 was characterized by high NS, rather high RD, and rather low P and HA. Cluster 4 was marked by high HA, rather low P and NS, and an average level of RD. All temperament variables significantly differentiated the temperament clusters, all F values (3178) were >3.9, and all P values were <0.001.†

**IRS Factor**

Principal-components analysis of the physiological parameters resulted in 1 factor that accounted for 44%, 42%, and 43% of the total variance of these parameters at years 0, 3, and 6, respectively. This factor was named the IRS factor and is presented in Table 1.

**Relationship of Temperament Patterns to Metabolic Coronary Risk Factors**

Table 2 shows the mean values for the physiological parameters in the study group at years 0, 3, and 6 separately for the age cohorts. Figure 2 is a graphical representation of the relationship between year-17 temperament clusters and metabolic coronary risk factors measured at years 0, 3, and 6. The IRS factor score significantly differentiated the temperament clusters at years 0, 3, and 6: F(3177) = 2.94 and 3.27, P = 0.035 and 0.022, respectively, and marginally significantly at year 0: F(3177) = 2.61, P = 0.053. The ANCOVA also showed that the temperament clusters were significantly differentiated by insulin at year 3: F(3177) = 6.09, P = 0.001. In addition, the temperament clusters were marginally significantly differentiated by year-3 BMI, year-6 TGs, and year-6 HDL-C: F = 2.23, 2.25, and 2.31, and P = 0.087, 0.085, and 0.078, respectively. In addition, the IRS factor score significantly differentiated the temperament clusters at years 0, 3, and 6: F(3177) = 2.84, 5.69, and 3.17, and P = 0.040, 0.001, and 0.026, respectively.‡

### Discussion

Four naturally occurring temperament clusters (groups of individuals) were identified in the present sample. In regard to the association of temperament with metabolic CHD risk factors, the results were totally opposite to what was hypothesized. Individuals characterized in adulthood by high P, high RD, average NS, and low HA always (ie, in every study phase during the 6-year follow-up period) belonged to the highest risk group for each physiological parameter. Persons in the opposite-pattern cluster systematically expressed the lowest risk, whereas persons belonging to the other 2 clusters were usually intermediate in physiological risk level.

Individuals with high P express perseverance despite frustration and fatigue. Individuals with high RD, in turn, are described as being sympathetic persons eager to help and please others and highly dependent on emotional support and intimacy with others, even vulnerable because of their openness. They are oversocialized, with an excessive tendency to conform to peer pressures, and highly sensitive to social cues of approval/praise and rejection from others, leading to excessive reward-seeking behaviors such as overeating or overworking in response to social rejection or frustration. As workers, like those manifesting high P, they are confident, hard-working, and please others and highly dependent on emotional support and intimacy with others, even vulnerable because of their openness. They are oversocialized, with an excessive tendency to conform to peer pressures, and highly sensitive to social cues of approval/praise and rejection from others, leading to excessive reward-seeking behaviors such as overeating or overworking in response to social rejection or frustration.

### Table 1. IRS Factors

<table>
<thead>
<tr>
<th>Variable</th>
<th>Year and Factor Loading</th>
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<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>TGs</td>
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<tr>
<td>HDL-C</td>
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<td>Insulin</td>
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<td>SBP</td>
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<td>BMI</td>
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<tr>
<td>SSF</td>
<td>0.81</td>
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<tr>
<td>Variance accounted for, %</td>
<td>43.8</td>
</tr>
</tbody>
</table>

†The reproducibility of these empirically derived clusters is critical. We have obtained rather similar clusters when the clustering was carried out in other samples of men and women who participated in the CRYF Study (N.R. et al, unpublished data, 1998). Thus, it is likely that the present solution has some generality.

‡Inspection of the means of the physiological risk factors suggested that in some instances, there might be a puberty-related quadratic relationship between age and the physiological parameters. That being so, we repeated all ANCOVAs after adding age squared as another covariate to control for these potential nonlinear associations. However, this did not change the results. We also verified that the assumptions regarding homogeneity of the regression slopes was not violated.
persistent, ambitious, heroic, and forceful overachievers who frequently push themselves to exhaustion.39

For a naturally occurring cluster, being a combination of different traits with balancing effects, a low level of HA is also significant. Low HA is characterized by a lack of inhibition and of appropriate caution, even when the situation requires them; by activities with a risk of physical injury; and by high energy levels with quick recovery from exertion, stress, and minor illness and rapid adaptation to changes in familiar routines. If low-HA persons are at the same time low in NS, as here, they will usually be cheerful, boastful, and overconfident.39

The “at-risk person” portrayed in the current study appears to challenge the traditional coronary-prone personality, at least to some extent. Actually, most of the widely documented coronary-prone characteristics were not only lacking in the present high-risk person, but also many of them, such as cynicism, pessimism, anticipatory worrying, tenseness, and slow recovery from stress, were determinants of a low-risk profile. Of course, the risk factor status of hostility, for example, is so well established that we are not trying to question previous findings; it is noteworthy, however, that the most recent meta-analytic review suggested that the relationship between the cognitive-experiential measures of hostility and CHD is rather modest (weighted mean $r=0.08^{60}$). Although this discrepancy may be difficult to explain, it should be recognized that the present temperament dimensions reflecting individual differences in associative learning in response to novelty, danger or punishment, and reward (ie, preconceptual biases in learning$^{40}$) are not identical to any of the personality traits traditionally examined in relation to CHD (eg, hostility), the development of which involves insight learning or reorganization of self-concepts.

The present findings are, however, in line with those studies suggesting that ambitious overachieving and the resulting exhaustion are risk factors for CHD: the present high-risk person is described as an effort-oriented one who easily pushes himself to exhaustion. Van Doornen and Van Blokland$^{61}$ have, for instance, suggested that a combination of ambitious striving for achievement and exhaustion might be the core concept of coronary-prone behavior. The pathogenic role of exhaustion that people attribute to overwork is

<table>
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<th>Measure and Year</th>
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<tbody>
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<td>n</td>
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</tr>
<tr>
<td>n</td>
<td>70</td>
<td>67</td>
<td>45</td>
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<td>BMI, kg/m²</td>
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<td>8</td>
</tr>
<tr>
<td>Year 3</td>
<td>8</td>
<td>2</td>
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</tr>
<tr>
<td>Year 6</td>
<td>9</td>
<td>3</td>
<td>12</td>
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<tr>
<td>TG, mmol/L</td>
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<td></td>
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<tr>
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<td>0.80</td>
</tr>
<tr>
<td>Year 3</td>
<td>0.79</td>
<td>0.30</td>
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<td>Year 6</td>
<td>0.88</td>
<td>0.34</td>
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<td>HDL-C, mmol/L</td>
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<td>1.59</td>
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<td>1.37</td>
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<tr>
<td>Year 3</td>
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<td>0.27</td>
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<td>1.31</td>
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<td>11.5</td>
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<tr>
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<tr>
<td>Year 6</td>
<td>10.3</td>
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<td>SBP, mm Hg</td>
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<tr>
<td>Year 3</td>
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<td>0.68</td>
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<td>Year 3</td>
<td>0.21</td>
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<td>0.88</td>
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<tr>
<td>Year 6</td>
<td>0.34</td>
<td>0.65</td>
<td>0.74</td>
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</table>
Figure 2. Age-adjusted mean values of physiological risk factors among boys and men in different temperamental clusters at years 0, 3, and 6.
in turn well documented in several CHD studies by Appels and coworkers.62–64

Temperament, though apparent at the behavioral level, also refers to the neurobiological and genetic determinants of behavior; so far as Cloninger’s theory is concerned, it refers to the neurobiology of motivation and learning.79 As mentioned before, Cloninger’s 3 temperament dimensions (with P added later) reflect variation in the 3 brain systems and the biogenetic predispositions to stimulus-response characteristics. RD, 1 of the characteristics of the risk profile, is responsible for the maintenance of behavior that has previously been socially appreciated or rewarded (overworking or overresponsibility, for instance) despite frustration of one’s efforts; relevant stimuli are conditioned signals of rewards, especially social cues and social approval. In other words, signals of social approval (secondary reinforcements) are of such a strong influence that behavior (overworking) is likely to be maintained despite lack of actual benefits and rewards (primary reinforcements).

Norepinephrine is the principal monoamine neuromodulator for the system. Norepinephrine plays a critical role in the learning and memory of new paired associations. Individuals with reduced basal activity in the dorsal noradrenergic bundle, and hence greater sensitivity to norepinephrine, are expected to respond to signals of social sentiment and to persist in reward-seeking behavior, even when frustrated, whereas those with lower sensitivity to norepinephrine quickly cease activity that is no longer gratifying.39

Regarding the “risk temperament” discovered herein and the role of achievement and effort orientation in it, catecholamine secretion has repeatedly been shown to be related to performance or striving for achievement.65,66 Superior performance has also been associated with an increase in norepinephrine, reflecting a challenge to perform well.66,67 Release of catecholamines also occurs in stress situations, especially during acute stress.68 As to the potential role of the SA system in the genesis of the IRS, it has been shown, for example, that catecholamines exert marked insulin resistance in humans.69,70 Also, Lembo and coworkers71 have recently demonstrated that an acute increase in skeletal muscle noradrenergic activity, as measured by norepinephrine outflow, is able to antagonize insulin-stimulated muscle glucose disposal. In addition to the metabolic hormonal effects, chronic sympathetic overactivity may also induce insulin resistance by inducing hemodynamic changes.70,72 Of course, given that a combination of high RD and P is likely to expose an individual to chronic work stress, for example, an increased release of cortisol may also be important; cortisol is also known to exert marked insulin resistance.35,36

Although this study’s associations between temperament and physiological CHD risk factors did not always reach statistical significance, they did show a high consistency across all physiological parameters. A relationship to the whole IRS was most evident, and single associations with insulin and SSF were also significant. Compensatory hyperinsulinemia is the final common denominator ultimately responsible for the other changes of the IRS, and insulin itself is also a major risk factor for the development of CHD.32,73

The present empirical associations between physiological risk factors and temperament based on Cloninger’s concept support the view that there is a neurobiological basis for that temperament, and the well-known connections between adrenergic responsivity and the pathogenesis of coronary atherosclerosis allow construction of a model in which all pieces fit. The relevance of the current findings is in part dependent on the continuity and stability of the temperament dimensions measured. As mentioned before, the temperament dimensions included in the Cloninger temperament model have previously been shown to exhibit continuity from childhood to adulthood88; of course, it is possible that the manifestations of these dimensions change slightly from young adulthood to later ages, at which time myocardial infarction may occur.

Some methodological limitations should also be recognized. First, there are problems when one attempts to measure temperament by means of a questionnaire. For example, although Cloninger’s temperament dimensions by definition reflect heritable biases in information processing by the perceptual memory system40 and the heritability estimates for the TCI temperament dimensions are higher than is often the case,10 substantial proportions of the variance of these dimensions are determined by other than genetic factors. Second, if a different method of cluster analysis had been used, the behavioral clusters would have been slightly different. Of course, the results must also be cross-validated in other samples, although there is some evidence that the present cluster solution has some generality (see the †footnote). Thus, the present findings should not be overinterpreted.

To summarize, the present study demonstrated that a temperament profile characterized by a high level of P and RD, an average level of NS, and a low level of HA is related to a high level of physiological CHD risk factors included in the IRS. This association may be predicated on several factors: these temperament factors may (1) increase exposure to stressors; (2) affect appraisal of stressors, thereby modulating physiological responses to stress; and (3) be markers of the same biological processes that underlie the development of high levels of metabolic CHD risk factors. The findings suggest that the temperament profile in question may predispose an individual to the development of the IRS and CHD.

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References
Temperament: Early Developing Personality Traits


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