

The relation of somatotypes and stress response to central serous chorioretinopathy

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Abstract

Purpose To investigate a possible relationship between central serous chorioretinopathy (CSC) and specific body types and compositions (somatotypes), and to examine the cortisol stress response among CSC patients of different somatotypes in comparison with healthy subjects.

Methods Prospective case–control study. A group of 28 patients with a previous or current diagnosis of CSC was compared with a group of 26 healthy subjects. Anthropometric measurements were used to estimate somatotype ratings in all subjects. Serum cortisol was measured at rest and following a stress-inducing computerized test in order to estimate response to stress in both groups. The main outcome measures included somatotype categorization and the change in serum cortisol following stress in both groups.

Results No significant difference in somatotype composition was found between the groups. There was no statistically significant difference between the groups in the elevation of cortisol following the stress-inducing test. The sample size was too small to exclude or find any significant difference between the different 13 subgroups of somatotype composition in the elevation of cortisol.

Conclusions Our study did not show a typical somatotype related to CSC. While previous studies showed higher cortisol values in CSC patients, we did not see a higher elevation in

blood cortisol following a stress response in this group in comparison with healthy subjects.

Keywords Central serous chorioretinopathy · CSC · Somatotypes · Cortisol · Stroop · Body composition

Introduction

Central serous chorioretinopathy (CSC) is a disease characterized by serous detachment of the neurosensory retina with or without serous detachment of the retinal pigment epithelium (RPE) caused by increased permeability of the choroidal vessels. The pathophysiology of the disease involves hyperpermeability at the level of the RPE [1, 2].

Several risk factors have been proposed for the disease. One of them is elevated corticosteroid levels, either following exogenous intake or endogenously high levels [3–9]. Type A behavior pattern (a competitive drive, a sense of urgency, an aggressive nature, and a hostile temperament) has also been shown to be a risk factor for CSC in a few studies [10–12]. Other proposed risk factors include pregnancy, alcohol consumption, untreated hypertension, antibiotic use, infection of the respiratory tract or with *Helicobacter pylori*, and bone marrow or organ transplantation [12–19].

Somatotyping is a method for description and assessment of body type and composition. It was first introduced by W. H. Sheldon in 1940 [20], and was later modified by Barbara Heath and Lindsay Carter into the Heath–Carter method of somatotyping [21], which is currently the most commonly used method for estimating physiques [22]. According to the Heath–Carter method, a person's somatotype is expressed as a three-number rating, each representing the key physique components, namely endomorphy, mesomorphy, and ectomorphy, which are always expressed in the same order. Endomorphy

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refers to relative fatness, mesomorphy to relative musculoskeletal robustness, and ectomorphy to relative linearity or slenderness of a physique (Fig. 1). An example of such a rating is 3–5–2, expressing the magnitude of each of the three components. Somatotyping has also been used in clinical research, e.g., in the study of diabetes mellitus [23] and ischemic heart disease [24, 25].

Different somatotypes share qualities with CSC patients. For example, a relationship was found between people with a strong mesomorphic component and a “type A” personality [26]. This reinforces Sheldon’s original research of temperamental traits fitting each somatotype, which stated that mesomorphs have a tendency toward assertiveness, energetic activity, love of risk and power, and physical courage [27]. In contrast, studies performed on medical students taking an oral anatomy examination showed an elevation in plasma cortisol which was higher in those with a linear body build (i.e., ectomorphic), in comparison with students with a fat or muscular physique (i.e., endomorphic and mesomorphic, respectively) [28, 29].

The aim of this study was to investigate a possible relation between CSC and specific somatotypes and to examine the cortisol stress response among CSC patients of different somatotypes.

Materials and methods

This was a prospective case–control study. It was approved by the medical center institutional review board committee. All procedures performed were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

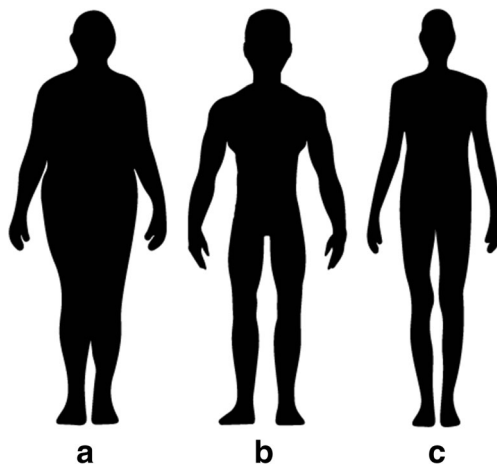


Fig. 1 The three somatotypes. *A* Endomorph — relative fatness. *B* Mesomorph — relative musculoskeletal robustness. *C* Ectomorph — relative linearity or slenderness of a physique

Study population

Patients with a previous or current diagnosis of CSC who were examined at the ophthalmology clinic between September 2013 and August 2014 were included. Diagnosis of CSC was based on evidence of subretinal fluid or active leakage as seen on fundus examination, OCT examination, and fluorescein angiography. When recruited to the study, patients presented with different disease stages — acute, chronic, or resolved CSC. The control group included age-matched (± 5 years) and gender-matched controls without CSC. Exclusion criteria included other retinal diseases, elevated blood cortisol for known medical reasons (i.e., Cushing’s syndrome, an acute illness that may raise serum cortisol), exogenous use of corticosteroids, and pregnancy.

Anthropometric measurements

Anthropometric measurements were used to estimate somatotype ratings based on the Heath–Carter method [21]. The anthropometric equipment included standardized height and weight scale with a Broca plane (Seca, USA), a sliding caliper (01407A, Neiko Tools, USA) to measure bone breadths, a steel tape measure to measure girths, and a skinfold caliper (Slim Guide skinfold caliper, Creative Health Products, USA) to measure skinfolds.

Ten anthropometric dimensions were measured in each subject in order to calculate the anthropometric somatotype. They included:

Height — the height was taken with the subject standing straight, against the edge of the scale, touching the edge with the heels, buttocks, and back, with the head oriented in the Frankfort plane (the upper border of the ear opening and the lower border of the eye socket on a horizontal line), and the heels together. Subjects were instructed to stretch upward and to take and hold a full breath. The Broca plane of the scale was lowered until it firmly touched the vertex.

Weight — subjects were measured while standing in the center of the scale platform. Weight was recorded to the nearest tenth of a Kg. A correction was later made for clothing.

Skinfolds — a fold of skin and subcutaneous tissue was raised firmly between thumb and forefinger of the left hand and pulled away from the underlying muscle. The edge of the plates on the caliper branches was applied 1 cm below the fingers of the left hand, and allowed to exert their full pressure before reading the thickness of the fold. All skinfolds were taken on the right side of the body, with the subject standing relaxed, except for the calf skinfold which was taken with the subject sitting. Skinfolds that were taken included:

- a) Triceps skinfold — taken with the subject's arm hanging loosely. A fold at the back of the arm at a level halfway along a line connecting the acromion and the olecranon processes.
- b) Subscapular skinfold — the subscapular skinfold adjacent to the inferior angle of the scapula was raised in a direction which is obliquely downwards and outwards at 45°.
- c) Supraspinale skinfold — a skinfold 5–7 cm above the anterior superior iliac spine on a line to the anterior axillary border and on a diagonal line going downwards and inwards at 45°.
- d) Medial calf skinfold — a vertical skinfold on the medial side of the leg, at the level of the maximum girth of the calf.

Bone breadths —

- a) Biepicondylar breadth of the humerus (right) — the width between the medial and lateral epicondyles of the humerus, with the shoulder and elbow flexed at 90°. The caliper was applied at an angle approximately bisecting the angle of the elbow. Firm pressure was placed on the crossbar to compress the subcutaneous tissue.
- b) Biepicondylar breadth of the femur (right) — With the subject seated with knee bent at a right angle, the greatest distance between the lateral and medial epicondyles of the femur was measured with firm pressure on the crossbars.

Girths —

- a) Upper arm girth, flexed and tensed (right) — with the subject flexing the shoulder to 90° and the elbow 45°, while clenching the hand and maximally contracting elbow flexors and extensors, measurement was taken at the greatest girth of the arm.
- b) Calf girth (right) — with the subject standing with feet slightly apart, the tape was placed around the calf, and the maximum circumference was measured.

Heights and girths were read to the nearest mm, biepicondylar diameters to the nearest 0.5 mm, and skinfolds to the nearest 1 mm.

The anthropometric measurements were entered into a software application (Somatotype version 1.2.6, M E R Goulding Software Development) in order to calculate the somatotype profile for each subject. As a result, each subject was given by the software a three-number rating representing endomorphy, mesomorphy, and ectomorphy components, respectively. Ratings of 0.5–2.5 on each component are considered low, 3–5 moderate, 5.5 to 7 high, 7.5 and above very high.

According to a subject's rating, subjects were classified by the software into one of 13 categories that reflect the dominance of each subject's somatotype components, as explained in Table 1.

According to the Heath–Carter method, we simplified the thirteen categories into four larger categories, when possible (Table 2).

The software was also used to plot each subject's somatotype on a two-dimensional graph (somatochart).

Measurement of response to stress

Cortisol at rest

A catheter (Venflon) was inserted into the cubital vein. Subjects were then required to rest while sitting for 15 min, in order to reach a resting state after the stress involved in catheter insertion. Six milliliters of venous blood were collected into an EDTA vacutainer tube for assay of cortisol. Serum total cortisol was measured by ECL method (Cobas A 411; Roche Diagnostics, Mannheim, Germany). The intra- and inter-assay coefficients of variation were 1.4% and 1.6% respectively.

For technical reasons, cortisol at rest was measured at different times of the day for different patients. The daytime of sampling was not recorded. Since endogenous levels of cortisol vary during the day (with a peak in the morning and a trough at night); only the difference between cortisol at rest and cortisol after stress was used to compare between the groups, and cortisol at rest was not used as an independent parameter.

Stress test

To generate stress, we used the Stroop test, which was shown to induce stress and elevate cortisol levels. [30, 31]. The 20-min test, described previously [30, 32], was recreated into a computerized program (Fig. 2). It consists of a brief presentation of a word (either “red”, “blue”, “yellow”, or “green”) on the screen. The word is presented in one of those four colors (red, blue, yellow, or green), at random intervals of 0.8–1.7 s. Simultaneously with each projected word, a voice announces the name of one of the four colors, not necessarily matching the word or the color that appears on the screen. The subject is asked to respond to each word by clicking a button that matches only the color of the word presented on the screen (which may be different from the word read or heard). The duration of each stimulus lasts between 0.4–1.0 s. If the subject does not click the correct button or fails to do so promptly, a red x appears on the screen, while a right answer shows a green √. Subjects took a practice session prior to the test in order to become familiar with test requirements.

Table 1 Classification of subjects into categories according to dominance of somatotype components

Somatotype category	Definition of category
Central	No component differs by more than 1 unit from the other two
Balanced endomorph	Endomorphy is dominant, and mesomorphy and ectomorphy are equal (or do not differ by more than 0.5 unit)
Mesomorphic endomorph	Endomorphy is dominant, and mesomorphy is greater than ectomorphy
Mesomorph–endomorph	Endomorphy and mesomorphy are equal (or do not differ by more than 0.5 unit), and ectomorphy is smaller
Endomorphic mesomorph	Mesomorphy is dominant, and endomorphy is greater than ectomorphy
Balanced mesomorph	Mesomorphy is dominant, and endomorphy and ectomorphy are equal (or do not differ by more than 0.5 unit)
Ectomorphic mesomorph	Mesomorphy is dominant, and ectomorphy is greater than endomorphy
Mesomorph–ectomorph	Mesomorphy and ectomorphy are equal (or do not differ by more than 0.5 unit)
Mesomorphic ectomorph	Ectomorphy is dominant, and mesomorphy is greater than endomorphy
Balanced ectomorph	Ectomorphy is dominant, and endomorphy and mesomorphy are equal (or do not differ by more than 0.5 unit)
Endomorphic ectomorph	Ectomorphy is dominant, and endomorphy is greater than mesomorphy
Endomorph–ectomorph	Endomorphy and ectomorphy are equal (or do not differ by more than 0.5 unit), and mesomorphy is lower
Ectomorphic endomorph	Endomorphy is dominant, and ectomorphy is greater than mesomorphy

Cortisol after stress

Immediately following the stress test, blood was drawn from the catheter according to the methods described above for a second assay of plasma cortisol.

Statistical methods

Differences of nominal variables between two groups (CSR vs controls) were carried out by Chi-square or Fisher's exact test, each when appropriate. Continuous data were checked for normality (Shapiro–Wilk test). *T*-test or Mann–Whitney non-parametric rank test were used between two groups, each as appropriate. $P < 0.05$ was considered statistically significant.

The power calculation was based on an estimated elevation of 0.5 in cortisol in the CSC group, and no change in the control group. To have an 80% power of detecting such a change (at a statistically significant level of $\alpha = 5\%$), 30 patients are required in each group.

Statistical analyses of somatotype data were carried out with Somatotype version 1.2.6, M E R Goulding Software Development. All other statistical analyses were carried out with SPSS version 23 (IBM, Armonk, NY, USA).

Table 2 Simplified classification of somatotype components

Somatotype category	Definition of category
Central	No component differs by more than 1 unit from the other two
Endomorph	Endomorphy is dominant, mesomorphy and ectomorphy are more than 0.5 unit lower
Mesomorph	Mesomorphy is dominant, endomorphy and ectomorphy are more than 0.5 unit lower
Ectomorph	Ectomorphy is dominant, endomorphy and mesomorphy are more than 0.5 unit lower

Results

Demographic data

Fifty-four subjects were included in the study, of whom 28 were in the CSC group and 26 in the control group. The mean age was 46.8 ± 11.75 in the CSC group and 46.1 ± 12.5 in the control group ($p = 0.83$). Most of the subjects in both groups were males (82.14% in the CSC group, 84.62% in the control group, $p = 0.8$).

Somatotypes

In the CSC group, the mean values for endomorphy, mesomorphy, and ectomorphy were 4.16 ± 1.47 , 4.20 ± 1.17 , and 2.08 ± 1.27 respectively. The most common somatotype categories were mesomorphic endomorph and endomorphic mesomorph, each with seven subjects (25%), followed by mesomorph–endomorph (four subjects, 14.3%).

In the control group, the mean values for endomorphy, mesomorphy, and ectomorphy were 3.88 ± 1.60 , 4.84 ± 1.63 , and 1.91 ± 1.43 respectively. The most common somatotype categories were endomorphic mesomorph (eight subjects, 30.8%), followed by mesomorph–endomorph (four subjects, 15.4%), and mesomorphic endomorph (three subjects, 11.5%).



Fig. 2 Computerized Stroop test. In this example, the word yellow appears in blue, while a voice in the background announces the word “red”. The examinee clicked the “blue” button, corresponding to the color of the word shown, meaning the answer was right, and therefore a ✓ appeared on the screen

When comparing the mean values for the combined somatotype component (endomorph, mesomorph, and ectomorph combined) between groups, no significant difference was found ($p = 0.29$). Additionally, when comparing each somatotypic component between groups, no significant difference was found ($p = 0.48$ for endomorph, $p = 0.09$ for mesomorph, and $p = 0.354$ for ectomorph).

The distribution of somatotype categories in both groups is outlined in Table 3. No statistically significant difference was found between groups when each category was compared. Additionally, no difference was found when the simplified categorization to only four categories was used.

Figure 3 illustrates the distribution of different subjects from both groups in a somatochart.

Table 3 Distribution of somatotype categories in both groups

Category name	CSC ($n = 28$)	Controls ($n = 26$)
Central	2 (7.1%)	4 (15.4%)
Balanced endomorph	1 (3.6%)	1 (3.9%)
Mesomorphic endomorph	7 (25%)	3 (11.5%)
Mesomorph–endomorph	4 (14.3%)	4 (15.4%)
Endomorphic mesomorph	7 (25%)	8 (30.8%)
Balanced mesomorph	2 (7.1%)	2 (7.7%)
Ectomorphic mesomorph	0 (0%)	1 (3.8%)
Mesomorph–ectomorph	1 (3.6%)	1 (3.8%)
Mesomorphic ectomorph	2 (7.1%)	2 (7.7%)
Balanced ectomorph	2 (7.1%)	0 (0%)
Endomorphic ectomorph	0 (0%)	0 (0%)
Endomorph–ectomorph	0 (0%)	0 (0%)
Ectomorphic endomorph	0 (0%)	0 (0%)

Cortisol

In the CSC group, mean serum cortisol at rest was 9.0 ± 4.5 . Following the Stroop test, it was 9.9 ± 6.6 . In the control group, cortisol at rest was 11.46 ± 4.9 , and after the Stroop test it rose to 11.6 ± 3.9 . There was a significant difference between the groups for cortisol at rest ($p = 0.04$) and for cortisol levels following the Stroop test ($p = 0.048$), but not for the difference between the value after the test and the value at rest [$+ 0.75 (\pm 3.8)$ for the CSC group, $- 0.14 (\pm 2.8)$ for the control group] ($p = 0.516$).

Discussion

The aim of the study was to find a correlation between specific somatotypes and CSC, and to examine the change in serum cortisol levels in response to stress among CSC patients in comparison with healthy controls. We did not find a specific somatotype related to CSC. No difference was found in the elevation of cortisol in response to stress between CSC patients and healthy controls.

This is the first study, to our knowledge, that addresses the body composition of CSC patients, specifically their somatotype. The reason for choosing this anthropometric method over others was the possible relationship between the disease and different somatotypes. In 1986, Yannuzzi reported an association between CSC and a “type A” personality pattern, characterized by a competitive drive, a sense of urgency, an aggressive nature, and a hostile temperament [10]. This finding was later found in a small number of other studies [11, 12]. In a study by Quinn and Wilson [26], 82 men and 112 women were assessed for “type A” behavior using a survey, and their somatotype was then determined. “Type A” behavior as tested in the survey was more prevalent in mesomorphic individuals compared with endomorphs and ectomorphs ($p < 0.05$).

A different characteristic of CSC was demonstrated in relation to ectomorphs. In a study by Bridges and Jones [28], 32 medical students underwent an assessment of their physique. This evaluation was not based on the Heath–Carter method, but rather the Parnell method, which gives a seven-point scale for each of three physical components — fat, muscularity, and linearity. Cortisol at rest was measured 2 months prior to an oral anatomy exam, and then again 30 min after the examination began. The group with primary linearity was found to have a significantly higher stress response, manifested by increased cortisol levels following the exam, in comparison with other types of physique. Another study by the same group examined an additional 40 students [29], and similarly showed a statistically significant difference between different physique groups, where the linear subjects experienced higher elevations in serum cortisol levels in comparison with others.

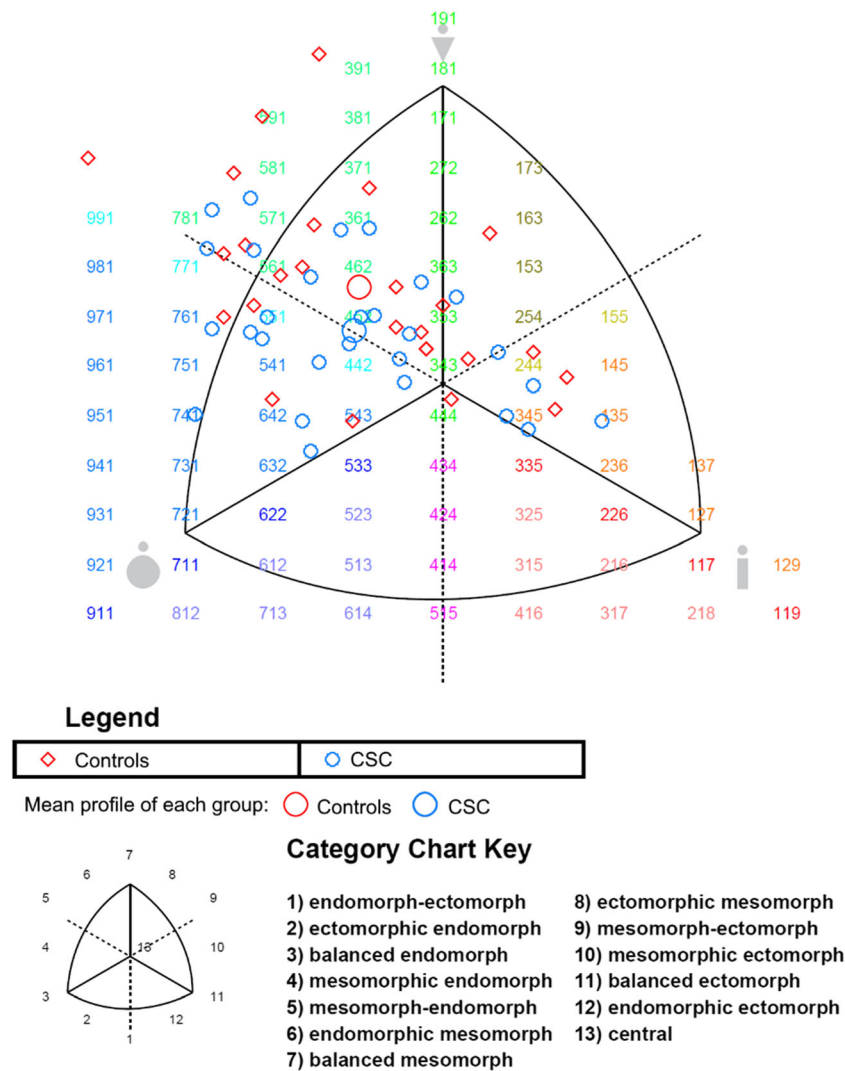


Fig. 3 Somatochart demonstrating the distribution of somatotypes among groups. The figure shows the distribution of subjects in the two groups, as well as the mean profile of all subjects in each group. The edges of the graph, marked by gray symbols, represent balanced mesomorph (top), balanced endomorph (bottom left), and balanced ectomorph (bottom right). The center of the graph marks a central

distribution of somatotypes. The areas between these spots stand for different combinations of somatotypes, as presented in the chart key. The numbers represent a somatotype composition, with the first number representing endomorphy, the second mesomorphy, and the third ectomorphy. It can be seen that the means of both groups are close to each other

In our study, no difference was found between CSC patients and the control group when the combined somatotype profile (endomorph, mesomorphy, and ectomorphy) was compared. In addition, we did not find a difference when each of the components was compared between the two groups. Furthermore, no difference was found when we compared the elaborate 13 somatotype categories between CSC patients and healthy controls. However, it is of note that most CSC patients in our study did not suffer from an active disease. An individual’s somatotype tends to change throughout life. Men tend toward increases in mesomorphy and endomorphy, while women become more endomorphic [21]. While it is not known if our subjects have undergone a change in somatotype since the time of first disease activity and to what extent, it is possible that

determination of their somatotype at the time of active disease would have resulted in different values. Studies on patients with active CSC are required to assess this.

In humans, cortisol is the principal glucocorticoid. The association between elevated endogenous or exogenous levels of glucocorticoids and CSC has been well established. As early as 1977, Gass reported that glucocorticoids, which were used at the time for the treatment of CSC, seemed to worsen the condition [33]. Later, CSC was found in patients with endogenous Cushing’s syndrome [7, 34–36], and in patients with disturbing psychological events that preceded the visual symptoms of CSC [37, 38].

In our study, although we found a significant difference in plasma cortisol at rest (with a higher value for the control group), this measurement was not considered reliable,

since resting levels of cortisol were measured at different times of the day in different patients. We did not find a significant difference in the change in cortisol levels between rest and following the Stroop test.

In a study by Garg et al., endogenous plasma and urine cortisol were measured in 30 patients with acute CSC and compared with 30 controls with new-onset retinal detachment. The levels of cortisol both in the urine and in plasma were significantly higher compared with the control group ($p < 0.001$) [6]. Kapetanios et al. compared the measurement of endogenous urinary free cortisol in 16 patients with CSC between 1 and 7 days after the onset of the disease with the same measurements in a control group. A statistically significant difference was reported here as well, with higher levels in the CSC group [39].

To our knowledge, no other study examined the change in cortisol following stress among CSC patients. We chose to examine this parameter since previous studies showed that in ectomorphs, the elevation in plasma cortisol was higher than in endomorphs and mesomorphs [28, 29]. While we did not find an abundance in ectomorphism among CSC patients, our results shed more light on the relationship between glucocorticoids and the development of CSC. Adrenocorticotrophic hormone (ACTH), secreted by the pituitary gland, is the primary regulator of adrenal cortisol secretion. Corticotropin-releasing hormone (CRH), secreted by the hypothalamus, is the main stimulator of ACTH production. The activity of the hypothalamic–pituitary–adrenal axis (HPA axis) is regulated by negative feedback from cortisol, while factors such as metabolic, physical, and emotional stress increase glucocorticoid secretion by increasing the hypothalamic secretion of CRH [40, 41]. Our results suggest that it may be the basic level of cortisol, rather than a more reactive HPA axis, that leads to the development of CSC.

It is possible also with regard to plasma cortisol that measurement of cortisol while our subjects suffered from active disease would have yielded different results. Studies measuring the change in cortisol in response to stress in active CSC patients are needed.

Our study had several limitations. First, plasma cortisol at rest was measured at different times of the day in different subjects. Since there is a diurnal variation in plasma cortisol, we could not use these measurements to compare baseline cortisol values. However, the aim of the study was to assess the change in cortisol between rest and a stressful event, which should not depend on the time of day [42–44]. Second, most of our patients did not suffer from acute CSC at the time of the study. As discussed above, it is possible that the somatotype and change in cortisol may have been different at the time of active disease, rather than inherent to the individual. Third, our sample size was relatively small. The power calculation done prior to the study showed a need for 30 subjects in each group for an elevation of 0.5 in cortisol in response to stress in

the CSC group and no change in the control group. The actual difference found was elevation of 0.75 in the CSC group and drop of -0.14 in the control group, a larger difference than expected. A repeat power calculation for these values with our current sample size in each group showed the actual power to be 78%. Larger studies are needed to test the hypotheses in this study. However, the relative scarcity of CSC, combined with the rigorous examinations conducted as part of this study, make recruitment difficult. Another limitation of the study is in the selection of the control group, in light of the small sample size. Parameters such as socioeconomic status or physical activity were not taken into account in the selection process. Such factors may affect the somatotype composition of the control group. However, since the somatotype composition in this group was taken at a specific point in time, and since the study aim was to find a correlation between CSC and specific somatotypes rather than investigate the origin for the measured somatotype, we believe that the selection process was sufficient for the goals of this study. Last, we chose the Stroop method to induce stress among our subjects. It is possible that different methods of inducing stress (e.g., an exam or public speaking) would have resulted in different measurements. We chose this method thanks to its high reproducibility.

In summary, we found no difference in the somatotypes among CSC patients in comparison with healthy age- and sex-matched controls. There was no difference in cortisol elevation in response to stress among the two groups. Further, larger, studies are needed to examine these characteristics, possibly including other parameters that may be compared with somatotypes, such as weight, weight development, physical activity per year, or BMI.

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Compliance with ethical standards

Conflict of interest All authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest (such as honoraria; educational grants; participation in speakers' bureaus; membership, employment, consultancies, stock ownership, or other equity interest; and expert testimony or patent-licensing arrangements), or non-financial interest (such as personal or professional relationships, affiliations, knowledge, or beliefs) in the subject matter or materials discussed in this manuscript.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

- Gass JD (1967) Pathogenesis of disciform detachment of the neuroepithelium. *Am J Ophthalmol* 63(Suppl):1–139. [https://doi.org/10.1016/0002-9394\(67\)90027-X](https://doi.org/10.1016/0002-9394(67)90027-X)
- Meyerle CB, Spaide R (2008) Central serous chorioretinopathy. In: Albert DM, Mill JW, Azar DT, Blodi BA (eds) *Albert Jakobiec's principles and practice of ophthalmology*, volume 3. Saunders, Philadelphia, pp 1871–1880
- Abu El-Asrar AM (1997) Central serous chorioretinopathy complicating systemic corticosteroid therapy. *Eur J Ophthalmol* 7:297–300. <https://doi.org/10.1136/bjo.68.5.329>
- Polak BC, Baarsma GS, Snyers B (1995) Diffuse retinal pigment epitheliopathy complicating systemic corticosteroid treatment. *Br J Ophthalmol* 79:922–925. <https://doi.org/10.1136/bjo.79.10.922>
- Haimovici R, Gragoudas ES, Duker JS et al (1997) Central serous chorioretinopathy associated with inhaled or intranasal corticosteroids. *Ophthalmology* 104:1653–1660. [https://doi.org/10.1016/S0161-6420\(97\)30082-7](https://doi.org/10.1016/S0161-6420(97)30082-7)
- Garg SP, Dada T, Talwar D, Biswas NR (1997) Endogenous cortisol profile in patients with central serous chorioretinopathy. *Br J Ophthalmol* 81:962–964
- Bouzas EA, Scott MH, Mastorakos G et al (1993) Central serous chorioretinopathy in endogenous hypercortisolism. *Arch Ophthalmol* 111:1229–1233. <https://doi.org/10.1001/archoph.1993.01090090081024>
- Haimovici R, Rumelt S, Melby J (2003) Endocrine abnormalities in patients with central serous chorioretinopathy. *Ophthalmology* 110:698–703. [https://doi.org/10.1016/S0161-6420\(02\)01975-9](https://doi.org/10.1016/S0161-6420(02)01975-9)
- Zakir SM, Shukla M, Simi Z-U-R et al (2009) Serum cortisol and testosterone levels in idiopathic central serous chorioretinopathy. *Indian J Ophthalmol* 57:419–422. <https://doi.org/10.4103/0301-4738.57143>
- Yannuzzi LA (1987) Type-a behavior and central serous chorioretinopathy. *Retina* 7:111–131. <https://doi.org/10.1097/00006982-198707020-00009>
- Xu SH, Zhang AZ, Wang YF, Fu B (1994) Association between central serous chorioretinopathy and type of personality. *Chin Behav Sci Med* 3:29–30
- Haimovici R, Koh S, Gagnon DR et al (2004) Risk factors for central serous chorioretinopathy: a case-control study. *Ophthalmology* 111:244–249. <https://doi.org/10.1016/j.ophtha.2003.09.024>
- Tittl MK, Spaide RF, Wong D et al (1999) Systemic findings associated with central serous chorioretinopathy. *Am J Ophthalmol* 128:63–68
- Errera MH, Kohly RP, da Cruz L (2013) Pregnancy-associated retinal diseases and their management. *Surv Ophthalmol* 58:127–142. <https://doi.org/10.1016/j.survophthal.2012.08.001>
- Casella A, Berbel R, Bressanim G et al (2012) *Helicobacter pylori* as a potential target for the treatment of central serous chorioretinopathy. *Clinics* 67:1047–1052. [https://doi.org/10.6061/clinics/2012\(09\)11](https://doi.org/10.6061/clinics/2012(09)11)
- Eom Y, Oh J, Kim S-W, Huh K (2012) Systemic factors associated with central serous chorioretinopathy in Koreans. *Korean J Ophthalmol* 26:260. <https://doi.org/10.3341/kjo.2012.26.4.260>
- Weenink AC, Borsje RA, Oosterhuis JA (2001) Familial chronic central serous chorioretinopathy. *Ophthalmologica* 215:183–187. <https://doi.org/10.1159/000050855>
- Spahn C, Wiek J, Burger T, Hansen L (2003) Psychosomatic aspects in patients with central serous chorioretinopathy. *Br J Ophthalmol* 87:704–708. <https://doi.org/10.1136/bjo.87.6.704>
- Fawzi AA, Holland GN, Kreiger AE et al (2006) Central serous chorioretinopathy after solid organ transplantation. *Ophthalmology*. <https://doi.org/10.1016/j.ophtha.2006.01.031>
- Sheldon WH (1940) *The varieties of human physique*. Harper and Brothers, New York. [with the collaboration of Tucker WB and Stevens SS]
- Carter JEL, Heath B (1990) *Somatotyping — development and applications*. Cambridge University Press, Cambridge
- Yang L-T, Wang N, Li Z-X et al (2015) Study on the adult physique with the Heath-Carter anthropometric somatotype in the Han of Xi'an, China. *Anat Sci Int* 91(2):180–187. <https://doi.org/10.1007/s12565-015-0283-0>
- Buffa R, Floris G, Putzu PF et al (2007) Somatotype in elderly type 2 diabetes patients. *Coll Antropol* 31:733–737
- Williams SRP, Goodfellow J, Davies B et al (2000) Somatotype and angiographically determined atherosclerotic coronary artery disease in men. *Am J Hum Biol* 12:128–138. [https://doi.org/10.1002/\(SICI\)1520-6300\(200001/02\)12:1<128::AID-AJHB14>3.0.CO;2-X](https://doi.org/10.1002/(SICI)1520-6300(200001/02)12:1<128::AID-AJHB14>3.0.CO;2-X)
- Valkov J, Matev T, Hristov I (1996) Relationship between somatotype and some risk factors for ischemic heart disease. *Folia Med* 38:17–21
- Quinn TJ, Wilson B (1989) Somatotype and type a behavior in college-age adults: psychological reports. *Psychol Rep* 65:15–18
- Sheldon WH, Stevens S (1942) *The varieties of temperament*. Harper and Brothers, New York
- Bridges PK, Jones MT (1967) Personality, physique and the adrenocortical response to a psychological stress. *Br J Psychiatry* 113:601–605. <https://doi.org/10.1192/bjp.113.499.601>
- Bridges PK, Jones MT (1968) Relationship of personality and physique to plasma cortisol levels in response to anxiety. *J Neurol Neurosurg Psychiatry* 31:57–60
- Dugué B, Leppänen EA, Teppo AM et al (1993) Effects of psychological stress on plasma interleukins-1 beta and 6, C-reactive protein, tumour necrosis factor alpha, anti-diuretic hormone and serum cortisol. *Scand J Clin Lab Invest* 53:555–561. <https://doi.org/10.3109/00365519309092553>
- Hamer M, Endrighi R, Venuraju SM et al (2012) Cortisol responses to mental stress and the progression of coronary artery calcification in healthy men and women. *PLoS ONE* 7(2):e31356. <https://doi.org/10.1371/journal.pone.0031356>
- Tulen JH, Moleman P, van Steenis HG, Boomsma F (1989) Characterization of stress reactions to the Stroop color word test. *Pharmacol Biochem Behav* 32:9–15. [https://doi.org/10.1016/0091-3057\(89\)90204-9](https://doi.org/10.1016/0091-3057(89)90204-9)
- Gass JDM (1977) *Stereoscopic atlas of macular diseases: diagnosis and treatment*, 2nd edn. CV Mosby Co, St Louis
- Daniele S, Schepens CL, Daniele C, Angeletti G (1995) Fundus abnormalities in Cushing's disease: a preliminary report. *Ophthalmologica* 209:88–91
- Harada T, Harada K (1985) Six cases of central serous chorioidopathy induced by systemic corticosteroid therapy. *Doc Ophthalmol* 60:37–44
- Thoelen AM, Bernasconi PP, Schmid C, Messmer EP (2000) Central serous chorioretinopathy associated with a carcinoma of the adrenal cortex. *Retina* 20:98–99
- Gelber GS, Schatz H (1987) Loss of vision due to central serous chorioretinopathy following psychological stress. *Am J Psychiatry* 144:46–50
- Rouvas AA, Chatziralli IP, Ladas ID et al (2014) The impact of financial crisis on central serous chorioretinopathy in Greece: is there any correlation? *Eur J Ophthalmol* 24:559–565. <https://doi.org/10.5301/ejo.5000403>

39. Kapetanios AD, Donati G, Bouzas E et al (1998) Serous central chorioretinopathy and endogenous hypercortisolemia. *Klin Monatsbl Augenheilkd* 212:343–344. <https://doi.org/10.1055/s-2008-1034901>
40. Garavanis A, Margioris AN (2001) Pharmacology of glucocorticoids: an overview. In: Margioris AN, Chrousos GP (eds) *Adrenal disorders*. Humana Press, Totowa NJ, pp 59–70
41. Hirsch Pescovitz O, Cutler G, Loriaux D (1990) Synthesis and secretion of corticosteroids. In: Becker KL (ed) *Principles and practice of endocrinology and metabolism*. Lippincott Williams and Wilkins, Philadelphia, pp 579–591
42. Simons SSH, Cillessen AHN, de Weerth C (2017) Associations between circadian and stress response cortisol in children. *Stress* 20:52–58. <https://doi.org/10.1080/10253890.2016.1276165>
43. Kidd T, Carvalho LA, Steptoe A (2014) The relationship between cortisol responses to laboratory stress and cortisol profiles in daily life. *Biol Psychol* 99:34–40. <https://doi.org/10.1016/j.biopsycho.2014.02.010>
44. van Eck MM, Nicolson NA, Berkhof H, Sulon J (1996) Individual differences in cortisol responses to a laboratory speech task and their relationship to responses to stressful daily events. *Biol Psychol* 43:69–84