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Visual mental imagery in congenital prosopagnosia

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ABSTRACT

Congenital prosopagnosia (cPA) is a selective impairment in the visual learning and recognition of faces without detectable brain damage or malformation. There is evidence that it can be inherited in an autosomal dominant mode of inheritance. We assessed the capacity for visual mental imagery in 53 people with cPA using an adapted Marks' VVIQ (Vividness of Visual Imaging Questionnaire). The mean score of the prosopagnosic group showed the lowest mental imagery scores ever published for a non-brain damaged group. In a subsample of 12 people with cPA, we demonstrated that the cPA is a deficit of configural face processing. We suggest that the 'VVIQ-PA' (VVIQ-Prosopagnosia) questionnaire can help to confirm the diagnosis of cPA. Poor mental imagery, a configural face processing impairment and clinical prosopagnosia should be considered as symptoms of a yet poorly understood hereditary cerebral dysfunction.

Prosopagnosia is a selective impairment in the visual learning and recognition of faces. It is associated with right or bilateral cerebral tissue damage to the temporal lobe (for an overview see [6,16]).

McConachie [41] described the first case of prosopagnosia in a person without any detectable brain damage. She called this type of prosopagnosia "developmental". By 2003, seven more single cases had been published [34]. As the term "developmental" was also used for acquired prosopagnosia in children, some authors preferred the term "congenital" for cases without detectable brain damage [1]. There is now substantial evidence for a hereditary type of prosopagnosia [15,23]. All pedigrees published so far are compatible with a simple autosomal dominant mode of inheritance, suggesting a single gene defect. A change in a single gene may indeed cause complex patterns of agnosias and/or apraxias. For example, a point mutation in the FOXP2 gene causes a complex disorder of speech production and language understanding [18,35].

Congenital prosopagnosia is not a rare disorder, although it was overlooked for a long time [24]. The prevalence of the condition in Germany was determined to be about 2.5% [30].

In an initial study we presented 38 people with congenital prosopagnosia of a familial type (hereditary prosopagnosia) identified by a typical pattern of clinical symptoms [23]. Eight of them were tested with a battery of face recognition tests revealing an objective face recognition impairment in each one of them. One finding of particular interest has been the strikingly lower vividness of visual mental imagery (VVMI) which was assessed with a modified VVIQ (Vividness of Visual Imagery Questionnaire [40]). The pattern of the impairment was somewhat inconsistent, though. While all prosopagnosic participants showed a VVIQ score of at least 1.5 SDs below controls (seven even > 2 SDs) for faces, three reported a normal VVIQ for non-face items. The effect did not seem to be familial, because one of the monozygotic twins in the study reported a normal imagery for non-face objects, the other scored 2 SDs below the controls' mean.

Visual mental imagery is a complex brain function involving several associative visual brain areas including the secondary visual cortex [32,45] and, as some have suggested, the primary visual cortex as well [31,33]. It is a distributed, modular system sharing some, but not all functional units with visual perception [27]. Brain damage can cause a total or partial loss of function [22], sometimes leading to dissociations in mental imagery abilities (cf. [28]). Levine et al. [39] reported on two patients with a dissociation of mental imagery after cerebral damage. One suffered from prosopagnosia and loss of mental imagery for faces and objects, while orientation in space, mental rotation and mental navigation was unaffected. The second showed the reverse pattern of impairments.

Barton and Cherkasova [2] studied the accuracy of mental imagery in 9 people with acquired prosopagnosia. One participant

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with an anterior temporal brain lesion was severely impaired, while others only showed a mild degradation. Mental imagery may be retained for faces learned before the onset of prosopagnosia ([4], 2nd case). On the other hand, Michelon and Biederman [44] presented a 34-year-old patient, who became prosopagnosic at the age of 5, but still had an accurate mental imagery for celebrities who became famous after the onset of his prosopagnosia. All in all, mental visual imagery impairments in acquired prosopagnosia do not seem to be consistent.

Most of these previous studies attempted to test the *accuracy* of visual mental imagery using task-based questions like "does a tractor have big wheels on the front or on the back" or "who had the bigger moustache: Hitler or Stalin?". Participants may, of course, exploit their semantic memory to help with the answers, thus limiting the specificity of the test. They may just know that Hitler's moustache was a narrow one and while Stalin's would cover the whole space between nose and upper lip. The VVMI, though, is an important additional dimension of mental imagery. You may vividly – but wrongly – imagine a tractor with two big front wheels (e.g., [36]). To our knowledge, the VVMI has never been assessed in prosopagnosics before.

53 people with congenital prosopagnosia (mean age 43.4 ys, median age 40.0 ys, range 18–94 ys) and 88 age-matched controls (mean age 42.5 ys, median age 37.5 ys, range 15–79 ys) took part in the study. 16 controls were first-degree relatives of participants in the prosopagnosic group.

If possible, we interviewed all first-degree relatives of the participants with prosopagnosia. In 17 cases no relatives were available for an interview. Therefore, the heredity of these participants' prosopagnosia could not be assessed (but not excluded either). In all other cases (36 of 53) we found one or more affected relatives.

The diagnosis for all participants (53 people with cPA, 88 controls) was made with our clinical symptom table (see for a detailed description [25,51]), which was created to identify the hereditary type of congenital prosopagnosia. The symptoms were assessed by a diagnostic interview lasting between one and two hours. The interviewer (an experienced physician, MG) asked open questions in a semi-structured interview format with three or four questions about each diagnostic item. Interviewers are held to embed the questions into conversation and make sure that questions about the same diagnostic item not asked sequentially. The interview also includes a medical history in order to exclude conditions, which may cause or mimic prosopagnosia. The diagnosis "hereditary type of congenital prosopagnosia" depends on a very specific pattern of symptoms. Affected people always report a lack of confidence with face recognition. Their feeling of familiarity (or unfamiliarity) of famous faces as well as personally familiar faces (see [8]) is always vague. Therefore, they overlook familiar people and also confuse strangers with familiar people. We found that the vague feeling of familiarity was always present and should therefore be regarded as a diagnostic hallmark. In contrast, people with acquired prosopagnosia frequently show impaired feelings of facial familiarity [20]. Therefore, the neural defect underlying the hereditary type of congenital prosopagnosia is probably different from the defect causing the acquired type.

Other symptoms include failure to recognize familiar people out of context or in crowded places, no need for eye contact, time of onset unknown (it was 'always there') and development of adaptive behaviour (other means of person recognition, ready set of excuses, avoidance of critical situations). Other face related recognition tasks are unimpaired: people with cPA report no problems with the recognition of facial emotions [17,26], facial attractiveness, gender or age. Nearly all people with cPA also reported problems with the visual recognition of objects and scenes. Only the complete symptom pattern establishes the diagnosis. A detailed discussion of the clinical diagnostic criteria can be found in [25]. Three studies with eight [23], 14 [9] and 17 [51] participants, respectively, have confirmed the validity of the clinical diagnosis so far. 16 of 22 participants of the first two studies are also in this study. As the clinical diagnosis suggests a more general processing problem, we decided to administer another test. It compares the featural and configural processing performance for faces and non-face objects (see Appendix A).

Table 1
Marks' [40] five-point scale for the assessment of visual imagery.

1. Perfectly clear and as vivid as normal vision.
2. Clear and reasonably vivid.
3. Moderately clear and vivid.
4. Vague and dim.
5. No image at all, you only know that you are thinking of the object.

Mental imagery was assessed in 53 prosopagnosics and 88 controls with the Vividness of Visual Imagery Questionnaire (VVIQ) by Marks [40], modified and extended for prosopagnosia assessment ("VVIQ-PA"). The VVIQ's reliability and construct validity has been questioned in the past [12,29], but a comprehensive meta-analysis by McKelvie [43] concluded that the test results are sufficiently reliable and reproducible.

The participants were asked to estimate the vividness of visual images with open and closed eyes in the following categories: face form, eyes, nose and mouth; emotional faces with happiness, anger, surprise and fear; sunrise—identical with Marks' original questionnaire; landscape—identical with Marks' original questionnaire.

We used the original Marks' [40] five-point scale for the assessment (see Table 1).

We also asked a number of additional questions concerning the participants' visual imagery in general:

1. Were the images there immediately without conscious effort (three answer possibilities: yes; no, only with conscious effort; no, not even with conscious effort)?
2. Did you always see a coherent image? (five answer possibilities: yes; yes, but image looked like a flat photograph; yes, but image looked like a movie scene; no, only details which could not be composed into a coherent image; no, only details which could be composed into a coherent image by intensive conscious effort).

Finally we asked people to assess the image quality:

1. Were the images sharp and crisp (two answer possibilities: sharp, blurred)?
2. Was the resolution high or low (two answer possibilities: high, low)?

One participant was removed because of irresolvable differences between her answers in the interview and the questionnaire. In all other cases, the answers were sufficiently consistent.

A sample of 12 prosopagnosic participants (9 female; mean age 37.2 ys, median age 34.5, range 24–60 ys) and 12 age-matched controls (10 female; mean age 36.0 ys, median age 38.5, range 21–58 ys) did an additional configural processing test where they had to match faces and non-face objects that varied by the degree of 2nd order relations between their cardinal features [37]. All prosopagnosic participants had also participated in an earlier configural face processing study [9] with "Thatcherized" faces [10,11,50] demonstrating clear deficits in configural face processing. All participants had a normal or corrected-to-normal vision and were identified with our clinical diagnostic procedure as prosopagnosic (target-group) or non-prosopagnosic (controls), respectively. Six of the prosopagnosics also took part in the imagery study.

The stimuli consisted of Mac-a-Mug faces and schematic drawings of houses as used in earlier face processing studies (e.g.,

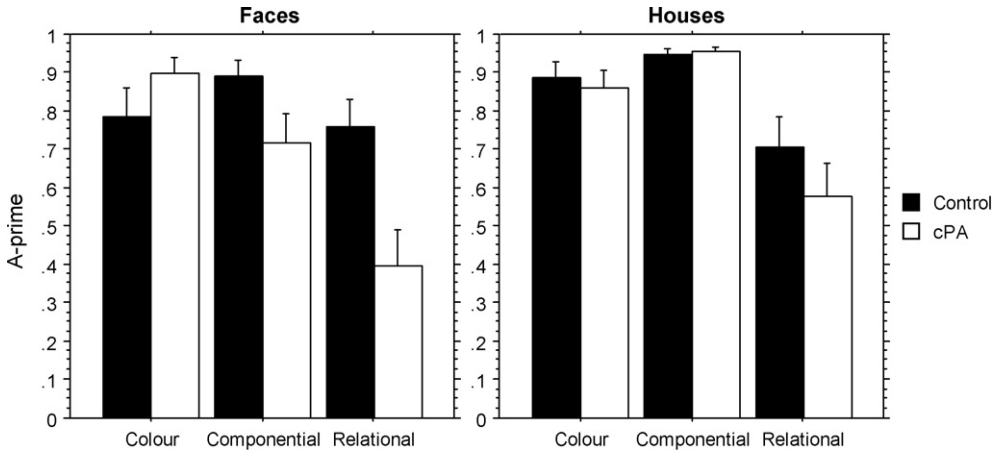

Fig. 1. A-prime data of the matching experiment for controls ($n = 12$) and congenital prosopagnosics (cPA, $n = 12$). Error bars show standard errors of the mean.

[37,49]). All faces and houses had the same facial (hairline, hair) or house context (roof, walls), respectively. For each object class, three parallel sets were constructed, in which the cardinal feature areas (eyes, nose, mouth, or windows, door, respectively) were manipulated. In the *colour* set only the shading, in the *componential* set only the shape and in the *relational* set only distance and position was manipulated. Only the relational set featured major 2nd order differences [37,38]. Each object set consisted of four unique items. Picture size was 240 × 190 pixels, presented on a 17-in. CRT monitor at a resolution of 1024 × 768 pixels with participants sitting about 65 cm away from the screen.

The participants were asked whether two simultaneously presented pictures showing houses or faces were the same or different. In 50% of all cases both pictures were identical, in 50% they were different but belonged to the same object set. Houses and faces were tested in different blocks; the order was counterbalanced. Each trial started with a blank screen (100 ms), followed by a fixation cross in the centre of the screen (500 ms), another blank screen (200 ms), and finally the pair of objects (4000 ms), either in upright or inverted orientation. All experimental factors were counterbalanced over all trials by the experimental software PsyScope [13]. The assignment of keys was counterbalanced across the subjects. The first two trials of both test blocks were randomly selected practice trials and were omitted from further analyses. The whole experiment consisted of 2 [response type: same, different] × 2 [orientations: upright vs. inverted] × 2 [object classes: houses vs. faces] × 3 [object sets: colour, componential, relational] × 4 [exemplars] = 96 test trials.

We will first present general results on the house vs. face test (see detailed analysis in Appendix A) to demonstrate face-specific problems in people with cPA, then we will analyze imagery data for both experimental groups in detail.

We analyzed A′ data (A′ is a discriminability index that integrates hits and false alarms into one parameter, see [48]) for both experimental groups. People with cPA had a distinctive impairment of matching performance in faces, but not for houses

This general pattern of results was confirmed by Analysis of Variance (ANOVA), described in detail in Appendix A. People with cPA performed worse than controls only for the *relational face* set, but not for the *relational house* set (see Fig. 1).

Most participants with cPA reported a markedly reduced ability to call up mental images which was not confined to mental images for faces, but also extended to objects and scenes. Five prosopagnosics added written notes to the effect that they did not have any visual mental images at all. Another prosopagnosic insisted that she had perfect mental imagery, but still did not recognize faces, because “they do not always look like I imagine them”.

Vividness scores were submitted to a three-way mixed design ANOVA with *group* (cPA vs. control) as between-subjects factor, and *eyes-condition* (eyes close vs. eyes open) and *material* (face shapes, facial emotions, non-face objects and scenes) as within-subjects factors (see average vividness scores in Table 2). The main effects of *group*, $F_{1,138} = 162.3$, $p < .0001$, $\eta_P^2 = .494$, and *object class*, $F_{2,276} = 38.1$, $p < .0001$, $\eta_P^2 = .216$, were qualified by an interaction between *group* and *material*, $F_{2,276} = 16.4$, $p < .0001$, $\eta_P^2 = 106$. No other effect was found significant. Thus, vividness of imagery was *not* modulated by the *eyes-condition*.

Although people with cPA showed lower vividness scores than controls in general, they had specifically problems to imagine face-specific content, e.g. face shapes and facial emotions.

To directly test for face-specific imagery deficits, additional simple-main analyses of *material* were performed for both experimental groups. Vividness differed significantly between *material* for the prosopagnosic group, $F_{2,137} = 27.8$, $p < .0001$, $\eta_P^2 = .289$, but not for the controls, $F_{2,137} = 1.9$, $p = .1500$, *n.s.* Post hoc tests indicated significant differences for prosopagnosics only between any face-specific material and non-face material, p's < .0001, but not between both face-specific materials, $p = .4557$, *n.s.*

In order to exclude a social or more general familial factor we calculated the mean score for the 16 first-degree relatives among the controls separately. Their mean vividness score was 1.90 (SD = 0.56), which is not significantly different from that of the other controls.

Fig. 2 shows the percentage of the respective answers 1 (most vivid) to 5 (least vivid) for faces (Fig. 2, left), sunrise and landscape (Fig. 2, right) in the prosopagnosic group and in the control group. While the answers 1 and 2 (most vivid) dominate in the control group, very few prosopagnosics claim to have vivid imagery.

Fig. 3 shows the distribution of average scores for faces. Only 6 of 53 (11.3%) persons in the prosopagnosic group claim a vividness score of 2.99 or better, as compared to 79 of 88 (89.8%) in the control

Table 2
Means and standard deviations for the prosopagnosics and the controls on the Marks' [40] five-point rating scale. A score of 1 stands for most vivid, a score of 5 for no mental image at all. Standard errors are denoted in parentheses, p-values and corresponding effect sizes (η_P^2) of the simple main effect of group are shown in the last two columns.

	Prosopagnosics (cPA) ($n = 53$)	Controls ($n = 88$)	p-value	η_P^2
Face, form and details	3.96 (0.94)	2.05 (0.95)	<.0001	.498
Face, emotions	4.02 (0.84)	2.04 (0.85)	<.0001	.571
Non-facial (Sunrise, landscapes)	3.01 (1.30)	1.84 (0.79)	<.0001	.268

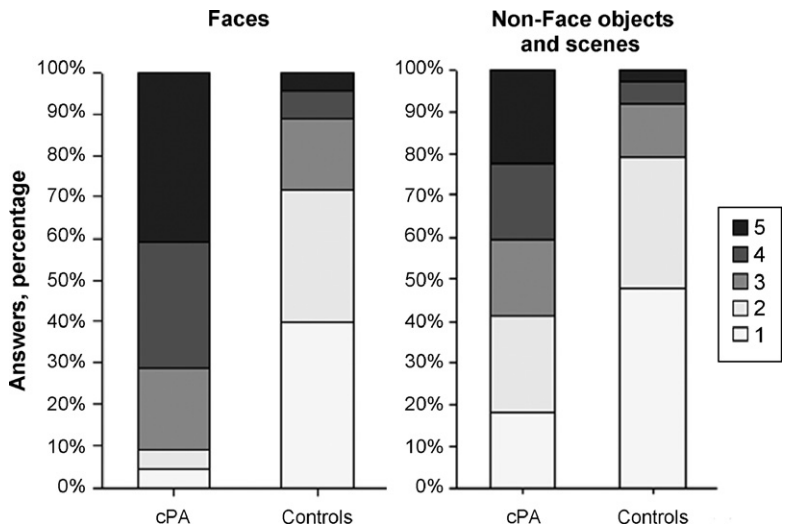

Fig. 2. Left: Percentage of answers 1 (best image) to 5 (no image) in prosopagnosics (cPA, $n = 53$) and controls ($n = 88$) for faces in the imagery experiment. Right: Percentage of answers for non-face objects and scenes (landscapes, sunrise) in the imagery experiment.

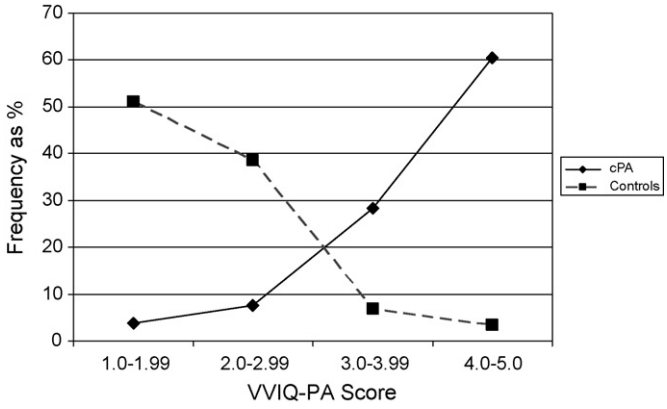

Fig. 3. Distribution of mean scores for the prosopagnosic group (cPA; diamonds) and the control group (squares), $n = 53$ and $n = 88$, respectively.

group. A score of 3 or more is generally considered as an indication of poor imagery [43].

Most prosopagnosics reported that they needed a conscious effort to conjure a mental image (Table 3). This is more pronounced for faces (96.2% as compared to 17.0% in controls) than for other objects (66.1% as compared to 17.0% in controls). The prosopagnosics' imagery is composed of scattered details (Table 4). Most participants in the prosopagnosia group describe the overall quality of their visual imagery as blurred (82.7%) and the resolution as low (81.1%). Respective figures for the control group are 14.8% and 13.6%. All differences are highly significant concerning individual Z-tests (p's < 0.001).

Our results indicate that mental visual imagery as measured by the VVIQ-PA scale is significantly reduced in people with the hereditary type of congenital prosopagnosia. The controls' mental imagery scores fit well with the results of a comprehensive meta-analysis conducted by McKelvie [43]. He reported a mean VVIQ score of 2.30 (SD = 0.69) for 38 studies including more than 2600 participants, while we found a score of 1.93 (SD = 0.71).

In his meta-study, McKelvie [43] defines a score of 2.93 (mean of 33 studies, SD = 0.38, average sample size 17.3) and above as "poor imaging". Using this definition, the vast majority of persons with cPA have poor or very poor mental imagery. It should be noted that the prosopagnosia group showed the lowest VVIQ score ($M = 3.51$, SD = 0.87) ever reported for any group of otherwise healthy people. Additional experimental testing suggested that the face processing impairment is at least in part due to reduced configural face processing; the subsample tested with this additional test did *not* differ from the rest of the prosopagnosic sample.

This raises the question, whether cPA is a symptom or a consequence of poor mental imagery. The latter alternative is unlikely, however, as there are a number of control participants with poor mental imagery, but no symptoms of prosopagnosia (4 of 88 with VVIQ-score > 3.5). Also, an earlier study about the VVIQ as a predictor of facial recognition memory performance failed to show any relation between facial memory performance and VVIQ score [42].

Therefore, we argue that a reduced vividness of mental imagery is a symptom, in fact a common symptom, of the hereditary type of congenital prosopagnosia. However, three prosopagnosics in our study reported normal or even vivid mental imagery. At this stage it is difficult to say whether they may suffer from a type of congenital prosopagnosia without degradation of mental imagery vividness or whether their symptoms are not fully captured by our assessment methods.

To our knowledge, the association between prosopagnosia and degraded vividness of mental imagery has not been systematically studied before, although a study on eyewitness testimony by Riske

Table 3
First VVIQ-PA-specific question "Were the images there without conscious effort?". One prosopagnosic participant did not complete the face-specific question.

Response	Prosopagnosics (cPA)		Controls	
	Face	Non-face	Face	Non-face
Yes	2 (3.8%)	18 (33.9%)	73 (83.0%)	73 (83.0%)
No, only with conscious effort	27 (50.1%)	25 (47.2%)	13 (14.8%)	14 (15.9%)
No, no coherent image even with conscious effort	24 (46.1%)	10 (18.9%)	2 (2.3%)	1 (1.1%)
Total Yes/No	2/51 (3.8%/96.2%)	18/35 (33.9%/66.1%)	73/15 (83%/17%)	73/15 (83%/17%)

Table 4
Second VVIQ-PA-specific question "Did you always see a coherent image?" for face and non-face objects. One prosopagnosic participant and one control participant did not complete the face-specific question. The table shows total numbers (percentages in parentheses).

Response	Prosopagnosics (cPA)		Controls	
	Face	Non-face	Face	Non-face
Yes (unconditional)	1 (1.9%)	11 (20.8%)	65 (74.7%)	64 (72.7%)
Yes, but image looked like a flat photograph	4 (7.7%)	6 (11.3%)	5 (5.8%)	9 (10.2%)
Yes, but image looked like a movie scene	5 (9.6%)	6 (11.3%)	7 (8.1%)	7 (8.0%)
No, I saw only details, which would not fit together	17 (32.7%)	9 (17.0%)	5 (5.8%)	1 (1.1%)
No, but I could fit the details with a conscious effort	25 (48.1%)	21 (39.6%)	5 (5.8%)	7 (8.0%)
Total Yes/No	10/42 (19.2%/80.8%)	23/30 (43.4%/56.6%)	77/10 (88.5%/11.5%)	80/8 (90.9%/9.1%)

et al. [46] has suggested a link between face recognition and mental imagery.

Congenital prosopagnosia may be caused by a so-called point mutation, a single gene defect, which may indeed cause a complex pattern of changes and impairments. The normal score of the first-degree relatives supports this hypothesis. If the condition was caused by a complex pattern of inheritance, we would expect at least some degree of degradation of mental visual imagery in this group.

In addition to the reduced mental imagery, congenital prosopagnosics showed a distinct failure to recognize second order (configural) differences in Mac-a-Mug faces. This effect was confined to faces and did not show up for houses. Indeed, many prosopagnosics report problems in composing mental imagery parts into a complete picture.

A deprivation of patterned visual input in the first few weeks of life by a bilateral congenital cataract leads to a significantly reduced facial identity recognition, while facial expression recognition is left unimpaired. Sensitivity to low spatial frequencies and, consequently, configural face processing is permanently damaged, while object processing is not affected [21]. Facial identity recognition matures very early, but probably relies on adequate visual stimuli, while facial expression evaluation may be "hardwired". It has been shown that face recognition is functional at birth [7] and that a damage on the first day after birth can never be fully compensated for [19]. The deficits in the hereditary type of congenital prosopagnosia resemble those found by Geldart et al. [21]. While facial expression recognition is normal, the identity processing is impaired as demonstrated by the selective deficit in the configural processing of faces. We propose two alternative explanations: (a) a lack of preferential gaze towards faces, or (b) a relative developmental delay or hypoplasia of the face recognition system or the visual system in general at birth or in the first year of life.

Most people with cPA reported that they do not feel the need to look at their counterpart's face during conversation. Also, people with cPA show abnormal face-focused gaze behaviour [47]. This may favour the idea of a defect in the face attentiveness module, although, this deficit may also be a symptom of a general delay of visual facial processing development. In the first few months of brain development, the rate of synaptogenesis greatly increases and a relative developmental delay of some weeks could markedly disturb the organisation of synaptic pathways. The development of the visual areas is believed to be hierarchical [5], and therefore we would expect to see a grey matter deficit somewhere down the ventral visual stream in people with cPA, which has indeed been observed [3]. A possible early disruption in synaptic connectivity may lead to a less detailed visual memory especially for faces, but to a lesser extent for other objects as well and subsequently to a blurred and less vivid mental imagery. Cui et al. [14] have reported that the VVIQ score correlates well with the activation of the early visual cortex during a mental imagery task. Therefore we assume that the lack of imagery vividness in cPA should have a neurophysiologic equivalent. Besides, it would be interesting to assess the vividness of visual imagery in the participants of Geldart et al.'s [21] study with our VVIQ-PA. Currently, not very much is known about the relative and absolute maturation of higher visual areas. Therefore, it is impossible to know whether a deficit in the face attentiveness module or a relative hypoplasia due to a gene expression defect underlies the condition.

In summary, we argue that the VVIQ-PA is a useful means to support the diagnosis of congenital prosopagnosia. We suggest that the hereditary type of congenital prosopagnosia and the degradation of mental imagery should be considered as associated symptoms of a multifaceted hereditary cerebral dysfunction.

Appendix A. Analysis of the house vs. face test

A′ data was analyzed by two independent three-way mixed design Analysis of Variance (ANOVA) for faces and houses. Both ANOVAs used *group* (cPA vs. control) as between-subjects factor, and *manipulation* (colour, componential, relational) and *orientation* (upright vs. inverted) as within-subjects factors.

For faces, the main effects of *manipulation*, $F_{2,44} = 5.8$, $p = .0058$, $\eta_P^2 = .209$, and *orientation* ("inversion effect"; A-prime data for upright faces > A-prime data for inverted faces), $F_{1,22} = 13.4$, $p = .0014$, $\eta_P^2 = .378$, were qualified by interactions between *group* and *manipulation*, $F_{2,44} = 4.0$, $p = .0246$, $\eta_P^2 = .155$, and between *manipulation* and *orientation*, $F_{2,44} = 4.4$, $p = .0181$, $\eta_P^2 = .167$. For houses, *manipulation* was the only significant effect, $F_{2,40} = 10.4$, $p < .0001$, $\eta_P^2 = .343$.

People with cPA were performing worse than controls only for the *relational* face set. There was only an effect of *group* for the *relational* manipulation, $F_{1,22} = 6.9$, $p = .0153$, $\eta_P^2 = .239$, but not for the *colour* or *componential* manipulation, F's$_{1,22} < 2.3$, p's > .15, *n.s.* In fact, people with cPA performed the matching task with relational faces on chance level, one-group t_{11} (against 0.5) < 1, *n.s.* For faces differing in colour, people with cPA were numerically, but not statistically, *better* than controls. A significant main effect of *orientation* for faces but not houses replicated earlier findings by Leder and Carbon [37] indicating specific configural processing of faces but not houses.

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