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Mechanisms of synesthesia: cognitive and physiological constraints

Peter G. Grossenbacher and ChristopherT. Lovelace

Synesthesia is a conscious experience of systematically induced sensory attributes that are not experienced by most people under comparable conditions. Recent findings from cognitive psychology, functional brain imaging and psychophysiology have shed considerable light on the nature of synesthesia and its neurocognitive underpinnings. These cognitive and physiological findings are discussed with respect to a neuroanatomical framework consisting of hierarchically organized cortical sensory pathways. We advance a neurobiological theory of synesthesia that fits within this neuroanatomical framework.

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Synesthesia is the conscious experience of sensory attributes induced by particular conscious mental events, appearing in addition to any sensations that are normally experienced by most people during such events[1,2]. For example, in one form of synesthesia, perception of a letter induces the phenomenal experience of a color, with each letter inducing its own distinct color. Synesthesia is neither strategy nor attitude; it is an involuntary concrete sensory experience that can be quite vivid. Only a small proportion of people routinely experience synesthesia. Such people ('synesthetes') typically experience synesthesia on a daily basis, beginning in early childhood. Many synesthetes exhibit surprise upon first learning that others do not share the synesthetic part of their perceptual experience.

Although experienced as a unitary whole, synesthesia is composed of two interrelated components. We use the terms 'inducer' and 'concurrent' to refer to the inducing event and the synesthetically induced sensory attribute(s), respectively. For example, one synesthete describes the sound of her crying baby as having an unpleasant yellow color. In her synesthesia, sounds (the inducers) were experienced as having colors (the concurrents) in addition to the customary auditory attributes. For most individuals, synesthesia is unidirectional: if sounds induce the experience of colors, colors typically do not induce the experience of sounds[3]. The relationship between inducers and concurrents is systematic in that each specific concurrent is typically induced by only one inducer[4].

Forms and types of synesthesia

Each form of synesthesia, such as sound inducing color, involves a set of triggering inducers (e.g. sounds) mapped to a corresponding set of synesthetic concurrents (e.g. colors). To avoid ambiguities of phrases like 'colored hearing' we use notation of the general form (I)→(C) where 'I' and 'C' designate inducer and concurrent sets, respectively. Thus '(sound)→(color)' efficiently refers to the form of synesthesia in which sound induces color.

In many forms of synesthesia, the inducer set and concurrent set belong to separate sense modalities. However, not all forms of synesthesia span two sense modalities. Figure 1a shows one relatively common form having visual inducers and visual concurrents[3,5]. In (visual letter)→(color) synesthesia, (visual) colors are induced by (visual) letters of the alphabet that are read by the synesthsete[6]. In addition, some forms of synesthesia involve inducers that are conceptual rather than sensory[1,7-10].

We distinguish between two types of synesthesia, according to whether inducers are sensory or conceptual. In *synesthetic perception*, concurrents are induced by perceiving particular sensory stimuli. In *synesthetic conception*, concurrents are induced by thinking about particular concepts. In (number)→(location) synesthesia, each counting number has a location in space relative to its neighbors[11,12]. Similarly, in the (time period)→(location) form of synesthetic conception, periods of time are conceptualized in a spatial layout (Fig. 1b,c)[10,13].

After several decades of relative neglect, synesthesia is again the subject of increasing interest[14]. In recent years, the field has progressed from seeking objective

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evidence that synesthesia exists to uncovering its component processes and neural substrates. Despite this advance, the recent literature shows little agreement among definitions of synesthesia. Our definition includes both crossmodal and intramodal sorts of synesthetic perception as well as synesthetic conception, and builds on previous proposals to differentiate among varieties of synesthesia[15–17].

Etiology of synesthesia

The experience of synesthesia can arise in one of three ways. A small proportion of the population have developmental synesthesia. These people routinely experience one or more forms of synesthetic perception and/or synesthetic conception, beginning in early childhood[1,2,7–9]. Although the cause of developmental synesthesia has yet to be determined, it appears to have a genetic basis, perhaps with autosomal dominant or X-linked dominant mode of transmission[18] (see Box 1).

Alternatively, synesthesia may begin later in life as a result of brain injury[19] or sensory deafferentation[20,21]. This rare condition, acquired synesthesia, might involve only synesthetic perception (we know of no report of acquired synesthetic conception). Finally, a person who ingests hallucinogenic drugs (e.g. LSD or mescaline) may experience pharmacological synesthesia during the drugged state[22]. Pharmacological synesthesia can produce forms of synesthetic perception not reported in developmental synesthesia[23]. We know of no reported case of pharmacological synesthetic conception.

We now review recent research on developmental synesthesia. Psychological and physiological investigation of synesthetic inducers and concurrents guides our thinking about underlying mechanisms and contributes to a theoretical framework that may be used for constructing theories of synesthesia.

Inducers – the events that trigger synesthesia

Although a given inducer repeatedly produces the same highly specified concurrent for an individual person, in some forms there is greater flexibility in the sensory parameters of the inducer. For example, if seeing the letter 'B' induces a particular shade of red, the red color might remain constant despite different handwriting. This flexibility raises questions about the nature and scope of synesthetic inducers.

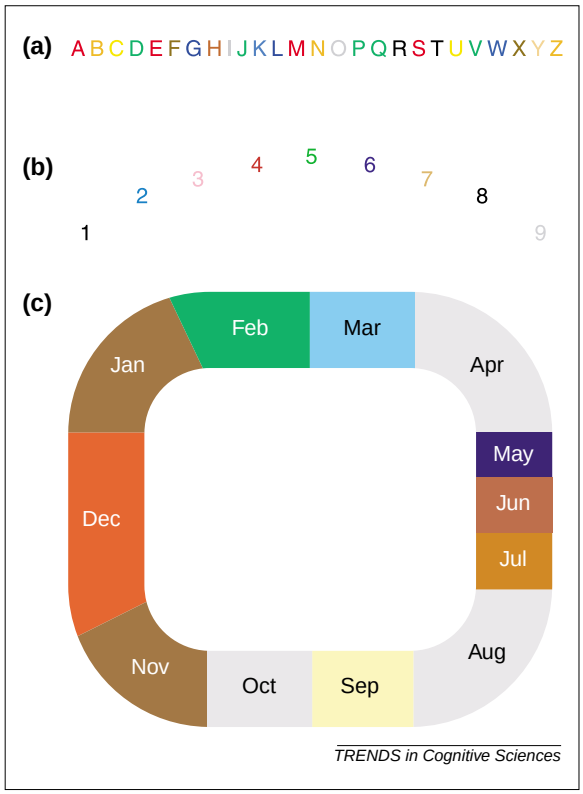

Fig. 1. Some forms of synesthesia. (a)The color experienced for each letter by a synesthete who experiences (letter)→(color) synesthesia. She also experiences (numeric digit)→(color and shape) synesthesia, with digits 1 through 9 positioned from left to right in an arc at chest level in front her (b). (c) Another synesthete conceptualizes the twelve months arrayed in a flat horizontal loop surrounding her, canonically oriented with February in front. This can be described as (time period)→(color and shape) synesthesia.

Representational level of inducers

Many synesthetic inducers are stimuli that were created to convey meaning; the majority of synesthetes have forms of synesthesia with linguistic or musical inducers[24,25]. This tendency for meaningful or symbolic stimuli to induce synesthesia has implications for understanding the cognitive processes involved. In (letter)→(color) synesthesia, it is the letter's identity that determines its color. For spoken letters, the sound of the voice (e.g. male versus female) typically has no effect on a letter's color. For written letters, most synesthetes report that the way the letter is written (e.g. font or case) has no effect on a letter's color. Thus the neural networks that represent visual shape or auditory timbre without letter identity do not represent the information required to mediate this form of synesthesia.

Imagery of inducers can produce synesthesia

For many people who experience synesthetic perception, concurrent phenomena can arise through voluntary imagery of an inducer without exposure to the inducer stimulus (Ref. 26, p. 41). Imagery involves many parts of the brain that are also active during perception[27,28]. As no adequate stimulus is present

Box 1. Developmental synesthesia

- Origin: the majority of synesthetes have developmental synesthesia.
- Automaticity: synesthetic concurrents are involuntarily induced.
- Specificity: synesthetic concurrents entail highly specific sensory attributes.
- Prevalence: synesthesia occurs in at least 1 in 2000 persons[a].
- Age: synesthesia is more common among children than among adults[b].
- Sex: synesthesia is more common in women than men[c].
- Familiality: synesthesia runs in families[a].

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and no afferent signals need enter the brain during imagery, this means that synesthesia can occur with incomplete activation of the entire cascade of sensory signaling normally propagated during perception.

Concurrents – the phenomena of synesthesia

Although different individuals can experience the same form of synesthesia, and can even share the same inducer set (e.g. all the letters of an alphabet), synesthetic concurrents vary greatly across people. For example, it would be highly unlikely to find two people for whom every alphabetical letter induces the identical color.

Although there is vast inter-individual variation in concurrents, a given individual's concurrents are highly specific and consistent. Synesthetes often describe their concurrents with meticulous care and attempt to convey specific hues by combining color terms in phrases like 'bluish gray' and 'orangish black'[2,29]. One study compared the consistency with which synesthetes and non-synesthetes assigned color names to 117 letters and words[30]. After one week, non-synesthetes named the same color for only 38% of the items. With impressively greater consistency, and after a longer time interval, synesthetes named the same color for 92% of the items one year later.

Representational level of concurrents

Synesthetic concurrents do not occur at all levels of perceptual representation. Concurrents comprise simple features such as color or spatial location, or their combination, rather than highly integrated percepts like an image of a face[26]. This restricted range of concurrent phenomena suggests the activation of particular stages of cortical processing near, but not necessarily involving, primary sensory cortices. A PET study compared blood flow in the brains of six (spoken word)→(color) synesthetes with that of six non-synesthetes[25]. The two groups differed in their cortical activity when listening to words, with synesthetes showing more activation in some extrastriate visual cortical areas (bilateral superior occipital gyrus/superior parietal lobe, bilateral posterior inferior temporal cortex) and less activation in other cortical areas (left lingual gyrus). This differential activity pattern suggests that color concurrents result from partial activation of higher-order visual cortical networks, rather than arising at the earliest levels of cortical visual processing.

Another aspect of synesthetic phenomena may further reveal their neural basis: individuals vary in where concurrents appear spatially. Some (visual letter)→(color) synesthetes say that color fills the printed letter. Others say that the color appears on an invisible screen located within arm's reach in front of their eyes, not in the letter itself. For yet others, concurrents appear in 'the mind's eye,' rather than outside the body. This variation in subjective concurrent location could reflect variation in the neural representation of the concurrent, and may depend on the recruitment of spatiotopic networks.

Rapid concurrent onset

Synesthetic concurrents are reported to appear at the same time as non-synesthetic components of synesthetic perception[25,31]. A recent study used scalp-recorded electrical brain activity to explore the physiological time course of synesthesia. This study measured the brain's response to visually presented letters by recording ERPs in 17 people with (visual letter)→(color) synesthesia compared with 17 non-synesthetes[6]. The two groups differed in their average evoked brain electrical activity beginning 200 ms after a visual letter had been presented, with synesthetes showing enhanced positivity at frontal scalp locations lasting several hundred ms. The timing of this electrophysiological effect is consistent with the previously described PET findings of cortical involvement in synesthesia.

Concurrents cannot be ignored

Most synesthetes say that concurrents arise unbidden in their mind and that each inducer to which they are sensitive always produces synesthesia. Two recent single-subject studies of (numeric digit)→(color) synesthesia assessed the automaticity of synesthesia by examining interference in the naming of physically presented colors as a result of synesthetically induced colors[3,32]. Both found significant levels of interference, reflecting the involuntary nature of synesthesia. One of these studies also demonstrated the unidirectionality of synesthesia: speed of naming the video color of digits was affected by synesthetically induced colors, but speed of naming the digits was not influenced by their video color[3]. That is, the induced concurrent colors interfered with color naming, but there was no digit synesthetically induced by the color to interfere with digit naming. The other study found that the inhibition of an actively ignored concurrent color lasted after the inducer stimulus has disappeared, persisting at least through the next trial – an impressive demonstration of negative priming via synesthesia[32]. These two studies confirm the long-held observation that synesthesia arises involuntarily and cannot be entirely ignored[5].

Hierarchical framework for neurocognitive analysis of synesthesia

Some aspects of synesthesia are idiosyncratic, such as the specific set of concurrents experienced by each synesthete. Other aspects are more widely shared, such as the general forms of synesthesia, their constituent inducer sets, the sensory quality of their concurrents, the consistency and specificity of concurrents, and the rapid and automatic induction of these concurrents. These commonalties suggest that all forms of developmental synesthesia could be explained by a single neurocognitive theory. First though, discussion of the neural mechanisms of synesthesia requires an understanding of the neuroanatomy of sensory processing.

Much of the primate cortex operates on the principle of hierarchically organized parallel systems[33]. A sensory hierarchy consists of simple representations feeding

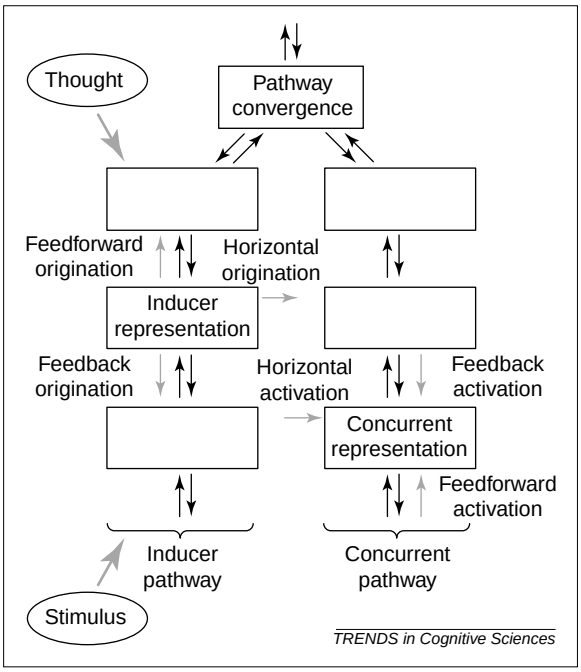

Fig. 2. Schematic depiction of neural mechanisms in synesthesia. Synesthesia could be mediated via neural signals between an inducer pathway (left) and a concurrent pathway (right). Each box depicts a representation within a pathway (a single representation may be anatomically distributed over multiple brain areas). Afferent flow of information is conveyed by bottom-up signals via feedforward neuronal projections (upward black arrows), and top-down signals are carried by feedback connections (downward black arrows). Synesthesia stems from activity in the inducer pathway during either synesthetic perception of a stimulus or synesthetic conception of a thought, and the concurrent representation could become activated either via horizontal connections between the pathways or as a result of pathway convergence.

forward to higher levels that support increasingly complex representations. Feedback connections are found at all levels of the brain's hierarchies of sensory pathways[34]. That is, ascending neuronal projections that convey afferent (bottom-up) signals within a sensory pathway are reciprocated by top-down signals carried by feedback connections. Hierarchies of converging cortical pathways provide an appropriate framework for analyzing the functional anatomy of synesthesia.

Where does synesthetic induction originate?

We define synesthetic induction as the process of neural communication that presumably underlies synesthesia. Given that any experience of synesthesia involves two components, the inducer and concurrent, we now explore how synesthetic induction might relate their respective neural representations.

In considering the possible origins of synesthetic induction, we assume that the neural network responsible for representing the inducer lies at a particular level within a neural pathway and that neural activity in this inducer representation fully represents the inducer (see Fig. 2). Could synesthetic induction originate at a level of the inducer pathway either below or above the inducer representation? We assume that the inducer percept is not fully represented at levels of the inducer pathway below the inducer representation, as these areas do not have complete access to the information content of the inducer representation. So origination of synesthetic induction at levels below the inducer representation conflicts with the fact that different inducers each induce their own distinct concurrent.

At levels of the inducer pathway above the inducer representation any representation that preserves all relevant aspects of the inducer could contain sufficient information for initiating synesthetic induction. However, any network that distinguishes among the identities of inducers (e.g. the identity of a letter regardless of its font or case) is so redundant with the inducer representation that it may be best understood as an anatomically distributed part of the inducer representation itself. Hence, having ruled out origination of synesthetic induction below or above the inducer representation, we propose that synesthetic induction stems from activation of the inducer representation itself.

Three possible routes for the origination of synesthetic induction suggest themselves. Feedforward origination from the inducer representation upwards within the inducer pathway provides one intra-pathway route (Fig. 2). Feedback origination downwards from the inducer representation provides another intra-pathway route. Horizontal origination via connections from the inducer representation going outside this pathway constitutes a third, inter-pathway route. Whatever its route, this origination stemming from the inducer representation constitutes only part of the entire process of synesthetic induction.

How is the concurrent representation activated?

We now consider the possible neural basis of synesthetic concurrents appearing in the contents of consciousness. Conscious sensory experience is associated with activity in cortical sensory pathways during perception[35,36], imagery[28], and hallucination[37,38]. Although alternative views have been suggested[39], most investigators agree that synesthesia probably obeys the same rule as other conscious experience: conscious experience of concurrent phenomena depends on neural activity in appropriate sensory cortical areas[17,40,41]. Indeed, the PET blood flow results described earlier support this view[25].

We assume that, for each form of synesthesia, a cortical neural network responsible for representing concurrent phenomena lies at some level of a concurrent pathway (see Fig. 2). Synesthesia results when induced neural signals provoke neural activity in this concurrent representation. If the process of synesthetic induction is serial, then it could begin when neural signals propagate out from the inducer representation and before ultimately activating the concurrent representation. The ERP study described above provides some support for this, because significant differences between synesthetes and non-synesthetes did not appear in the ERP waveform until 200 ms following stimulus onset[6].

In the previous section, we delineated three possible ways that synesthetic induction could originate. We now delineate three possible routes leading to activation of the concurrent representation (Fig. 2). Feedback activation of the concurrent representation could be mediated by feedback connections within the concurrent pathway. Horizontal activation of the concurrent would require receiving signals from outside the concurrent pathway, stemming from the inducer pathway. In a strictly hierarchical organization, feedforward activation of the concurrent representation also requires

horizontal connections, but at a level lower than the concurrent representation. Horizontal connectivity might be peculiar to the brains of synesthetes, if no horizontal connections between inducer and concurrent pathways exist in the normal human brain.

Disinhibited feedback: a neurobiological theory of synesthesia

As mentioned previously, connections feeding forward into brain areas that receive signals from multiple pathways ('Pathway convergence' in Fig. 2) are always reciprocated by feedback connections. In most people, top-down signaling via these feedback connections may be sufficiently inhibited to avoid synesthetic induction. In synesthetes, information entering such a convergence area through the inducer pathway could propagate down the concurrent pathway through disinhibition of these feedback signals. We have previously suggested that feedforward signaling in the inducer pathway activates neurons in a brain area where the inducer and concurrent pathways converge and that feedback signaling from this area propagates down the concurrent pathway to activate the concurrent representation[17]. We may now add that with respect to induction mechanisms, feedforward origination of synesthetic induction leads to feedback activation of the concurrent representation.

The disinhibited-feedback theory differs from other cortically based theories of synesthesia[30,42]. For example, Maurer has suggested that human infants are born with dense interconnections between cortical sensory systems and that synesthesia results from a partial failure of the normal pruning process that eliminates these connections[42]. In terms of synesthetic induction, this would involve horizontal connections between pathways as exemplified by horizontal origination and horizontal activation (Fig. 2). The disinhibited-feedback theory, on the other hand, posits no abnormal (e.g. horizontal) neural connections, but proposes instead that synesthesia is entirely mediated by neural connections that exist in normal adult human brains. This feature is shared by another theory, one that emphasizes limbic mediation and cortical depression instead of cortical mediation[39]. In support of theories of synesthesia that posit normal connectivity, the ability of hallucinogenic drugs to induce synesthetic experiences in non-synesthetes[17] suggests that such experiences rely on normally existing adult networks, rather than on the formation of new connections between pathways.

Concluding remarks

We have proposed that synesthesia depends on synesthetic induction, a process of neural communication that originates from activity in the inducer representation and culminates with activation of the concurrent representation. Considered within a hierarchy of neural pathways, this view provides a framework for relating theories of synesthesia to empirical findings. We have suggested that synesthetic induction must either (a) rely on inter-pathway connections or (b) originate in feedforward signals from the inducer representation, and must culminate in activation of the concurrent representation via feedback signals (by virtue of convergence between inducer and concurrent pathways).

An understanding of multisensory convergence in the brain helps to delineate the issue of pathway convergence. Any polysensory brain area that might mediate synesthesia must have access to the sensory representations underlying concurrent phenomena. Cortical areas in the primate superior temporal sulcus (STS) send feedback connections to unisensory cortical areas involved in processing visual features[43]. STS also contains neurons responsive to feature-level aspects of stimuli in multiple sense modalities[44–46]. This agreement between the anatomical and physiological findings makes STS a strong candidate for mediating pathway convergence in synesthetic induction.

Only some synesthetes report synesthetic experience consequent to mental imagery of an inducer. Whether imagery induces synesthetic concurrents could be a theoretically important distinction. It might be that imagery only produces synesthesia in forms in which the inducer representation is situated relatively high in the inducer pathway, or it might simply reflect variation in imagery ability across individuals. The known neural substrates of perception and imagery of letters and color can suggest candidate neural representations of inducers and concurrents, at least for those forms of synesthesia that involve letter inducers or color concurrents (but this will not be discussed further in the present review).

The recent literature focuses on frequently occurring and easily studied forms of synesthesia. Most of the experimental findings concern forms of synesthetic perception in which linguistic symbols induce colors. This narrow focus makes for straightforward comparison among studies, but may not reveal whether other forms of synesthesia involve different mechanisms. In order to determine whether all forms involve identical mechanisms, the field must broaden its focus by studying a greater variety of forms.

Knowing the neural substrates of synesthesia could illuminate several related multisensory issues. On the one hand, if synesthesia involves abnormal neural connections, then synesthetes constitute an important population to investigate with regard to the cause (and effects) of these abnormal connections. On the other hand, if synesthesia involves only normal neural connections, then uncovering the mechanisms of synesthesia might improve our grasp of multisensory issues in normal, non-synesthetic perception. As knowledge of the mechanisms underlying developmental synesthesia accumulates, comparison with synesthesia of other etiologies, such as pharmacological and acquired synesthesia, may shed light on phenomena such as pathological hallucination. The study of synesthesia thus has wide implications for the understanding of both normal and abnormal perception.

Outstanding questions

- Does synesthesia depend on abnormal neural connectivity (found only in the brains of synesthetes) or is it mediated by neural connections that exist in normal brains?
- To what extent is synesthesia more common in children than adults?
- In cases of developmental synesthesia, in what proportion do various forms of synesthetic perception occur?
- What is the genetic basis for developmental synesthesia, and do synesthetes differ from non-synesthetes in other ways?
- How can acquired synesthesia be reliably differentiated from developmental synesthesia?
- Can synesthesia be learned via explicit training, or lost via conditioning?

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