extinction. Phoresy-aided dispersal also increases gene flow and thereby may reduce inbreeding depression and the accumulation of deleterious mutations in local populations. For example, while the soil nematode *Caenorhabditis remanei* has a limited dispersal ability on its own, its ability to hitch rides on slugs, snails and isopods has likely aided in its maintenance of local genetic diversity. Phoresy may lead to parasitism and mutualism over time. Thus, the study of phoresy could provide insights into the initial stages of the evolution of these symbiotic relationships.

How should we study phoresy?

Phoresy remains understudied, and most studies have been observational. While we know many examples of phoresy, we still don't understand the effects of phoresy on host and phoront ecology and evolution. Why do some taxa have more phoronts than others, what are the initial conditions that lead to phoretic relationships, and how often do phoretic interactions become parasitic or mutualistic? There remains much to be learned about this intriguing symbiosis.

Where can I learn more?

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Primer

The insular cortex

Nadine Gogolla

Whether you see the person you are in love with, try to listen to your own heartbeat, suffer from a headache, or crave for a chocolate cookie, one part of your brain is sure to increase its activity strongly: the insular cortex. The insular cortex, or 'insula' for short, is part of the cerebral cortex. J.C. Reil, a German neurologist, first named this brain structure in the early 19th century. Subsequent research findings have implicated the insula in an overwhelming variety of functions ranging from sensory processing to representing feelings and emotions, autonomical and motor control, risk prediction and decision-making, bodily- and self-awareness, and complex social functions like empathy. How is one single brain area involved in so many different tasks? Is the insula comprised of several functional regions? How are these related? And, are there any common themes underlying the apparently so heterogeneous roles of the insula?

Recently, there has been a surge of interest in the human insular cortex, with an increasing number of functional imaging studies identifying the insula as a core region affected across many psychiatric and neurological disorders. In parallel, modern technologies available for dissecting functional microcircuits in animal models, especially in rodents, have placed the insular cortex on the radar of neuroscientists interested in understanding the neuronal mechanisms underlying emotions and motivated behavior.

Some have argued that the human insular cortex is unique and underlies behaviors and mental capacities that are exclusively human. I will argue here that, although humans have the most complex brains and behavior in the animal kingdom, many of the anatomical and functional features of the insula are shared across rodents and men, and that these similarities may provide an entry point for addressing basic neuronal underpinnings of insular functions and malfunctions in an accessible animal model. With this aim, I will describe findings from human and animal studies that highlight the corresponding features, and suggest a few questions that I believe need to be addressed in order to gain a more mechanistic understanding of the insular cortex. This primer is meant to serve as a starting point for delving into this fascinating, highly relevant, yet still mysterious, brain region.

The anatomical organization of the insular cortex

Location of the insula cortex

In primates, including humans, the insula lies folded deep within the lateral sulcus of each hemisphere, hidden below parts of the frontal, parietal and temporal lobes, which form the so-called opercula, or 'lids'. (Figure 1A). This unique location prompted the names 'insula' (Latin for 'island'), 'hidden fifth lobe', and 'Island of Reil'. Macroscopically, the human insula is divided into an anterior and a posterior part by the central insular sulcus (Figure 1A). The extremities of these two parts differ substantially in their connectivity to other brain regions, while an intermediate 'middle' insular zone exhibits mixed anterior and posterior connectivity features. In lissencephalic species, such as mice and rats, the insula lies exposed on the lateral surface of the hemisphere, mostly above the rhinal fissure (Figure 1B).

Across species, the insula comprises three different areas, which differ in their cytoarchitecture: the granular, dysgranular and agranular subdivisions. This terminology alludes to the progressive loss of the granular layer 4. The granular insular cortex has a classical six-layered structure; in the dysgranular insula, layer 4 becomes thinner; and the agranular insula is tri-laminar, entirely lacking layer 4. The three subdivisions are strongly interconnected along the dorso-ventral and rostro-caudal axes.

A unique feature of the insular cortex of humans, great apes, elephants and some cetaceans, such as whales, is the presence of a special cell type in layer 5. The large, bipolar, 'von Economo neurons' were already noted by Ramon y Cajal, and formally

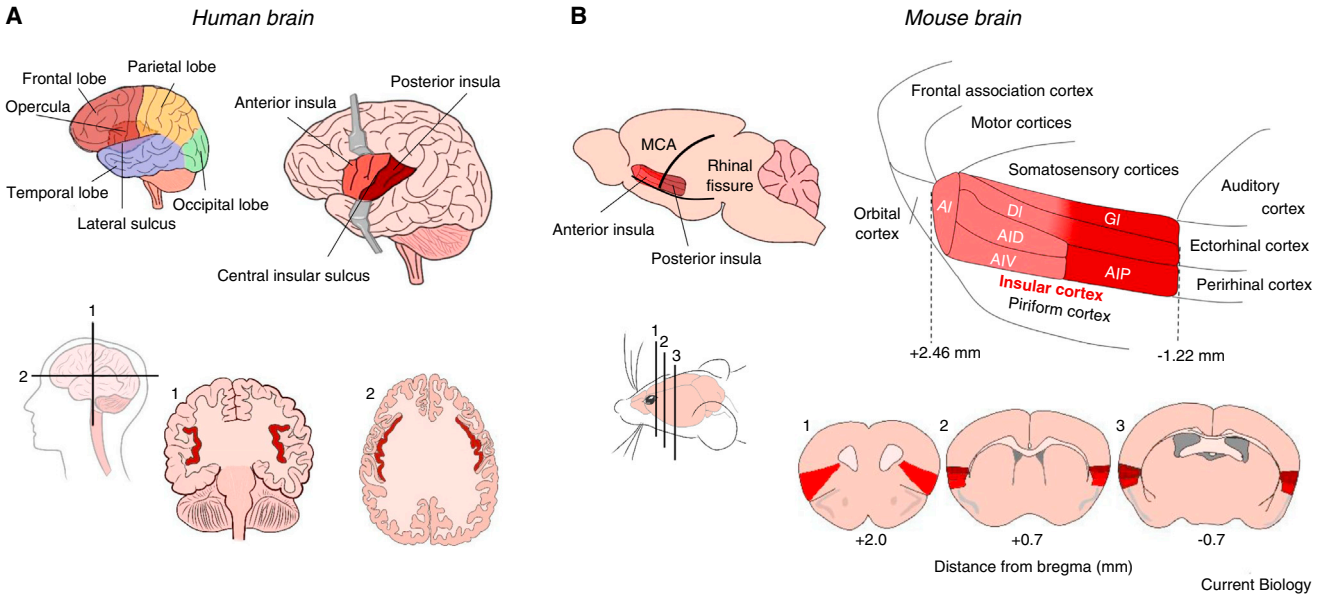

Figure 1. Location of the insular cortex in the human and mouse brain.
(A) Anatomy of the human brain. Top left: the insula is folded below the lateral sulcus and is hidden by the opercula (shaded area) of the frontal, parietal and temporal lobes. Top right: when temporal and parietal lobes are pushed aside, the underlying insula can be seen; it is separated into an anterior and posterior part by the central insular sulcus. Bottom: coronal (1) and horizontal (2) cross sections of the human brain reveal the position of the insular cortex (red). (B) Top left: in the mouse, the insula lies exposed on the lateral surface of the brain above the rhinal fissure. The medial cerebral artery (MCA) crosses the insula. Top right: subdivisions and neighboring cortical regions of the mouse insula. AI, agranular insular cortex; AID, agranular insular cortex, dorsal part; AIV, agranular insular cortex, ventral part; AIP, agranular insular cortex, posterior part; DI, dysgranular insular cortex; GI, granular insular cortex. Bottom: coronal cross sections at different levels reveal the location and layers of the mouse insular cortex.

described by Constantin von Economo in the 1920s. While the precise function of this distinctive cell type is not known, von Economo neurons are selectively destroyed in frontotemporal dementia and are unique to animals with large brains and advanced socialization skills. These facts have led researchers to speculate that they may bear a special role in complex social and emotional skills.

The insula is a hub linking large-scale brain systems

The insular cortex is a true anatomical integration hub with heavy connectivity to an extensive network of cortical and subcortical brain regions serving sensory, emotional, motivational and cognitive functions (Figure 2). It receives heavy sensory inputs from all modalities. Direct thalamic and horizontal cortical afferents carry information to the insula from outside the body (auditory, somatosensory, olfactory, gustatory and visual information) and from inside the body (interoceptive information). Several of these inputs project to topographically organized insular sensory regions, giving rise to the 'visceral insular cortex', the 'gustatory cortex' (the primary taste cortex), and the insular auditory and somatosensory fields. It is important to note that none of these sensory regions processes only its major sensory input: all regions of the insula receive heavy cross-modal afferents and are better thought of as multimodal integration sites.

In addition to its sensory afferents, the insula makes reciprocal connections with the limbic system. For instance, the lateral and basolateral amygdala heavily project to the granular and dysgranular regions of the insula, which in turn send dense efferents to the basolateral, lateral and central amygdala nuclei. The insula also connects to the lateral part of the bed nucleus of the stria terminalis, the mediodorsal nucleus of the thalamus, the lateral hypothalamus, and parahippocampal regions, including the perirhinal and the lateral entorhinal cortices.

The insula reciprocally connects with frontal brain regions such as the anterior cingulate, the orbitofrontal, and the medial prefrontal cortices, which are implicated in cognitive, emotional and executive functions, and projects to parts of the brain implicated in motivation and reward, such as the nucleus accumbens and the caudate putamen. Overall, the insular cortex receives strong neuromodulatory input from cholinergic, dopaminergic, serotonergic, and noradrenergic afferents.

Functions of insular cortex

Within the insular cortex, afferents from sensory, limbic, autonomic and frontal brain regions converge and intermingle, establishing a basis for cross-modal and cross-functional association and possibly binding. While to date the neuronal underpinnings of such integration are not understood, below I will discuss how the linkage of different systems gives rise to diverse functional roles of the insula.

From interoception to emotion and back

Interoception, the perception of bodily states, is a key function of the insular cortex. The insula receives topographically organized afferents from distinct thalamic relay nuclei and integrates information about blood pressure and oxygenation, the motility

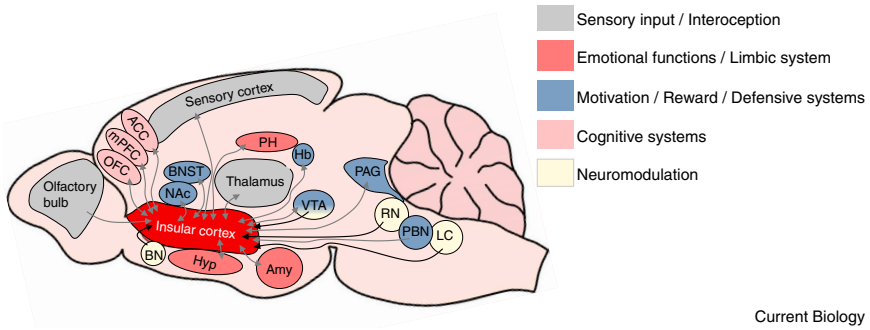

Figure 2. Connectivity of the insular cortex.
A simplified scheme of the basic insula connectivity visualized on the rodent brain. The insular bears reciprocal connections (grey arrows) with sensory, emotional, motivational and cognitive systems and receives strong neuromodulatory input (black arrows) in the+ form of cholinergic afferents from the basal nucleus (BN), dopaminergic input from the ventral tegmental area (VTA), serotonergic input from the raphe nuclei (RN), and adrenergic input from the locus coeruleus (LC). Abbreviations: ACC, anterior cingulate cortex; Amy, amygdala; BNST, bed nucleus of the stria terminalis; Hb, habenula; Hyp, hypothalamus; mPFC, medial prefrontal cortex; NAc, nucleus accumbens; OFC, orbitofrontal cortex; PAG, periaqueductal grey; PBN, parabrachial nucleus; PH, parahippocampus.

of the digestive system, the timing and strength of the heartbeat, as well as pain, hunger, nausea, tickle, itch and many more bodily sensations. In addition to sensing the body's condition, the insula also exerts strong top-down control of autonomic functions, for example regulation of the heartbeat, blood pressure or gastric motility, most likely through its direct projections to the lateral hypothalamic area, the parabrachial nucleus, and the nucleus of the solitary tract. Interestingly, microstimulation studies in rats and clinical observations revealed adjacent 'pressor' and 'depressor' sites with opposite effects on heart rate and blood pressure, suggesting discrete circuits with antagonistic roles exist within close vicinity in the insula.

The insula cortex is thus a site where bodily sensations, autonomic control and afferents from brain regions implicated in emotion processing, like the amygdala, converge. Early theories of emotions going back to Descartes and James-Lange emphasize the link between interoception and emotions by arguing that emotions are evoked by the perceptions of physical responses of the body and cannot exist without the experience of bodily feelings. Indeed, human and animal studies have implicated the insula cortex in processing positive and negative emotions, including anger, sadness, fear and anxiety, disgust, happiness or joy, trust, surprise, as well as social emotions. Interestingly, the involvement of the insula in emotion processing is cross-modal — emotions elicited through different modalities such as language, sounds, pictures or even touch, engage the insula.

A particularly strong body of evidence supports a role for the insula in mediating fear and anxiety. Functional imaging studies in humans and rats find co-activation of the insula with a set of brain regions collectively referred to as the 'fear network'. Furthermore, functional and structural connectivity between the insula and the amygdala correlate with both state and trait anxiety in healthy people. In the laboratory, experiments using the classical Pavlovian fear-conditioning paradigm have shown fear-induced activation of the insular cortex across different species from mouse to human. Evidence from lesion studies and pharmacological inhibition of different insula subregions in the rat have demonstrated a role for the insula in the consolidation of learned fear, but strikingly also in the learning of safety cues, which inhibit the expression of conditioned fear. Thus, fear promoting and inhibiting circuits may co-exist within the insula.

The insular cortex links sensory experience and emotional valence

In addition to sensory signals from within the body, the insula receives sensory information from the environment. But while the insula has several discrete sensory zones which re-map the external senses, insula lesions do not alter sensory perception thresholds, but rather affect the recognition or valence of a given sensory input or set of stimuli. For example, insula lesions may affect flavor recognition, which is facilitated by the integration of different sensory modalities, including olfaction, vision and taste. The acquisition and expression of conditioned taste aversion can also be disrupted by insula lesions. Conditioned taste aversion refers to the long-lasting aversion that is acquired when a novel taste is followed by visceral malaise, and thus relies on learning and memorizing the association between the ingestion of a given food and its negative effect on the body. In the laboratory, the neuronal mechanisms underlying conditioned taste aversion have been extensively studied using rodents. These studies revealed important roles for cholinergic and dopaminergic signaling in regulating insular plasticity and that communication between the basolateral amygdala and the insular cortex underlies conditioned taste aversion memory formation.

A striking example of deficits in sensory–emotion integration is that of 'pain asymbolia', in which patients suffering from an insular lesion can recognize pain, but lack an appropriate emotional response and do not attribute a negative valence to this usually adverse experience. Interestingly, rats have also been shown to exhibit pain asymbolia upon lesions of the anterior insula, indicating that the insula mediates a pain-related negative affect in rodents and humans alike. A different example, highlighting the role of the insula in assigning positive valence to a sensory experience, is that of social touch. The insula is the recipient of C tactile (CT) fiber afferents, which increase firing when the skin is stroked at a pleasant, caress-like speed. The discharge frequency of CT fibers correlates with the subjective hedonic experience of the caress. Interestingly, in humans, the insula cortex reacts even if the caress is not actively felt but observed to affect others.

A role for the insula in learning and memorizing valence

Monitoring the valence of internal sensations, external cues and regulating autonomic responses is important to maintain homeostasis and assure future well-being. In addition to this online monitoring, the insular cortex also mediates long-term retention of appetitive, aversive, or novelty-driven learning. Interestingly, the involvement of the insula in memory is dependent on the saliency of the cues or events, which is marked through dopaminergic and cholinergic signaling. For example, as mentioned above, the insular cortex is necessary for consolidation and storage of taste aversion upon conditioned taste aversion. In this paradigm, memory formation is dependent on the novelty of the stimulus — familiar tastes are usually not associated with malaise, and the mechanism for this differentiation lies in the novelty-specific release of acetylcholine in the insula.

Along similar lines, interfering with cholinergic signaling in the rat insula impairs object recognition memory, which is based on the natural tendency of animals to explore novel objects over familiar ones. A role for the insula has also been demonstrated in other forms of aversive reinforcement learning such as footshock-motivated avoidance, escape-driven spatial learning, and conditioned fear learning. Together, these results emphasize that the insular cortex is crucial in ensuring long-term access to information about the valence and salience of external stimuli and action outcomes.

The insula directly affects behavior

Recent evidence suggests that the insula is not solely a substrate for processing and linking cross-modal information but that it affects behavior directly. A study in rats showed that optogenetic manipulation of brain fields representing sweet and bitter taste within the gustatory insular cortex elicited attractive and aversive behaviors. In this study, the behavioral output upon activation of sweet and bitter cortical fields was independent of learning and experience.

Interestingly, a different study showed that conditioned sugar aversion shifted the topographic representation of sweet so that its overlap with bitter increased, suggesting that the insula integrates experience into valence maps. Combined, these findings may hint at the existence of hardwired valence domains with direct impact on behavior. The incoming sensory stimuli could then be ascribed, either innately or through learning, to such valence domains in order to exert direct behavioral control, resulting in the display of attraction or aversion.

The insula estimates risk and guides decision-making under uncertainty

In decision-making, estimates about the valence, magnitude and probability of expected outcomes of an action are integrated and weighed against each other. These estimates are strongly influenced by bodily needs and ‘gut feelings’. The ‘somatic marker hypothesis’ of Antonio Damasio proposes that, in decision-making, cognitive functions, bodily feelings and emotions always act together, and highlights a role for the insular cortex, amongst other brain regions, as an important neuronal substrate of somatic markers which guide decisions. Indeed, lesions of the human and rodent insula result in changes in decision-making under circumstances that involve uncertain reward and risk, and interfere with behavioral adjustments to changes in outcome probabilities.

Furthermore, studies in humans and rodents found neuronal signatures of anticipation for negative and positive outcomes, as well as risk prediction error coding in the insular cortex. Recently, electrophysiological recordings in rats and mice have shown that insular cortical neurons change their neuronal firing during anticipatory sensory cues, which predict food delivery. Importantly, optogenetic silencing of the insula cortex exclusively during the presentation of an anticipatory cue interfered with the approach behavior to a food reward, suggesting a role for the insula in predicting circumstances of reward and directly guiding behavior dependent on expected outcomes.

The insula cortex as a neuronal substrate for empathy

A remarkable observation made in human imaging studies is that the insula is not only activated by subjective emotions, but also when emotions are observed in another human being. For example, the anterior human insula is activated when a person experiences pain or observes pain in other people, or when someone tastes or sees other people taste pleasant or unpleasant food items. These data suggest a role for the insula in mediating empathy, the ability to understand and share the feelings of another individual. Corroborating this idea, humans who have difficulties understanding their own emotional and bodily states, a condition called alexithymia, show less insula activation in comparison to typical subjects when trying to assess their own feelings or those of others. Together these findings provide a striking illustration of the insula’s role in linking sensory input with emotions.

A role for the human insula in self-awareness?

A major theory of insula function, put forward by Bud Craig, proposes that the representation of interoceptive feelings in the anterior insular cortex constitutes the sole neuronal substrate for human subjective feelings, emotions and self-awareness. While it is likely that the insula cortex contributes to conscious access to feelings and self-awareness in healthy subjects, this exclusive statement has been put into question by the finding that bilateral insula damage does not abolish all emotional feelings or self-awareness.

On the basis of these and other clinical findings, Antonio Damasio has proposed that feelings and emotions arise at multiple levels of the central nervous system. He and others suggest that, instead of a generative role in the processing and experience of feelings, the insula rather acts to promote emotion regulation and flexible behavior, more static behavioral patterns motivated by drives and bodily feelings persisting if the insula is damaged.

Neurological and neuropsychiatric disorders

The human insula has emerged from numerous meta-analyses as a core region affected across many psychiatric disorders including, but not limited to, anxiety disorders, addiction, depression, schizophrenia and autism.

Together with the dorsal anterior cingulate cortex, the insula cortex forms a hub that affects the brain's ability to switch between different functional networks according to internal and environmental demands, explaining why insula disturbances may be disproportionally disabling. Given this important role, the insula is one of the most promising targets for brain stimulation treatment of several psychiatric disorders.

The role of the insula in anxiety disorders

The insula exhibits altered structure and function across different forms of anxiety disorders, including specific and social phobia, generalized anxiety disorder, post-traumatic stress disorder (PTSD), and panic disorder. While the mechanistic role of the insula in fear and anxiety remains unclear, Paulus and Stein have proposed that individuals who are more aware or focused on their bodily feelings may exhibit greater interoceptive prediction signals: that is, increased prediction of future aversive physical states may trigger anxiety, worry and avoidance behaviors.

This last idea is corroborated by the finding that self-reported measures of anxiety are correlated with the accuracy of heartbeat detection and activity in the right anterior insular cortex. Furthermore, changes in insular-mediated anticipation and prediction of future events may lead to heighten anxiety. Given the above-mentioned similarities in the role of the human and rodent insula in interoception, anticipation, as well as in mediating fear, the rodent model may represent a unique opportunity to assess the precise neuronal mechanisms underlying the insula's role in healthy and pathological fear and anxiety.

The insula and addiction

Addiction is a mental disorder defined by compulsive drug use that persists despite negative consequences. Human functional imaging studies have revealed insula activation upon drug consumption and craving, and one of the most striking findings from patients with insula lesions is that it disrupts their addiction to cigarette smoking. Furthermore, the insular cortex of drug users exhibits structural alterations, and the activity of insula during decision-making correlates with the propensity for relapse to consume drugs. Animal studies have corroborated a role of the insula in drug seeking. In rats, memory storage of context-drug associations depends on the insula, and aversion-resistant alcohol consumption depends on insular glutamatergic inputs to the nucleus accumbens. In addition, dopamine signaling in the insular cortex is related to nicotine self-administration in rats.

Together, these studies in humans and rodents suggest a dual role of the insular cortex in addiction: disease-related alterations may both promote ongoing drug use via increased perception of craving, and weaken the processes that prevent ongoing drug use, such as decision-making and the evaluation of negative consequences. Establishing the precise role of the insula in addiction requires studies designed to address whether existing alterations in insula function cause a predisposition to become addicted or whether drug use persistently alters insula function, or both. The application of modern techniques in the rodent model may allow us to elucidate the neuronal underpinnings of the interplay between bodily feelings, decision-making and risk avoidance in the context of drug use.

Insula metabolism as a guide for depression treatment

Major depressive disorder is associated with emotional and cognitive impairments including negative affect or loss of pleasure. Aberrant anatomy, connectivity and activation of the insula are found in human patients suffering from major depressive disorder. These alterations have been linked with the disease-characteristic anhedonia, the inability to experience pleasure. Remarkably, a recent study showed that insula metabolism was altered in depressed patients and that the direction of change indicated whether patients would respond better to either one or another of the two major treatment approaches for depression: cognitive behavior therapy or drug-based therapies. Measuring insula metabolism could thus serve as one of the first neuroimaging biomarkers in the field of neuropsychiatric disorders to guide treatment selection.

Insula in schizophrenia

Magnetic resonance imaging studies consistently find decreased grey matter volume and reduced cortical thickness in the insula of schizophrenic patients, which progress with increasing disease severity. Post-mortem studies have exposed less cellular heterogeneity in the upper layers of the insula, with decreased numbers of neurons and reduced glial and neuronal soma sizes. Functional aberrations observed in schizophrenic patients, which are likely related to altered insula function, include pain insensitivity, deficits in sensory-emotional integration such as poor recognition of emotions in facial expressions, emotionally blunt speech, impairments in distinguishing self from non-self, and the occurrence of hallucinations.

Insula and autism

Autism spectrum disorders (ASD) are complex neurodevelopmental disorders of unknown etiology. The insula has been consistently identified as a locus of hypoactivity and dysfunctional connectivity in ASD, and the pattern of functional connectivity of the insula can be used to discriminate individuals with ASD from typically developing children. As described above, the insula is essentially involved in multisensory and affective processing, as well as social functions like empathy, all of which are strongly affected in autistic patients. Rodent models of autism demonstrate with sensory hyper-reactivity and deficits in multisensory integration due to changes in inhibitory circuits within the insula, results that are highly reminiscent of clinical findings.

On a network level, the insula is the core of a so-called 'salience-network', which plays a key role in the detection of behaviorally relevant stimuli and initiation of dynamic switching between an 'executive control network' of brain regions, which drives externally-oriented attention, and a 'default mode network', which is dedicated to internally oriented cognitive processing. Irregularities in salience-network connectivity are linked to

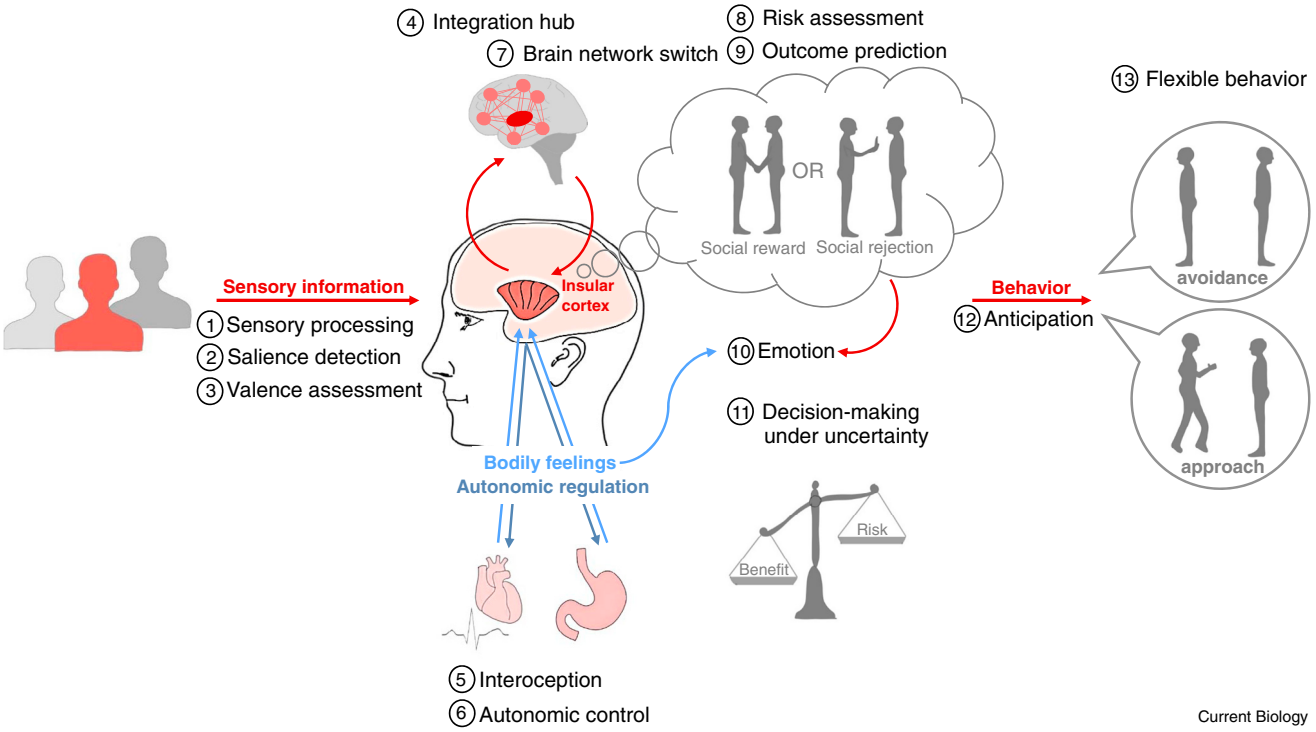

Figure 3. Illustration of an integrated model of insular cortex functions.
A schematic model of how diverse insular functions and computations may interact exemplified in the context of a social encounter. (1) The insula cortex receives multisensory information. In this example, it reacts to hearing or seeing people. (2) It processes the salience of incoming stimuli, that is, it responds more to the known (red silhouette) than to the unknown persons (grey silhouettes). (3) The insula assesses the valence of the stimulus, for example, is this a loved or dreaded person, by (4) integrating information from multiple brain systems, including emotional feelings for the person and cognitive information, for example what is known about the person. The insula also perceives (5) bodily feelings and (6) contributes directly to physical reactions caused by the social encounter, e.g. an increase in heartbeat or the feeling of butterflies in the stomach. (7) Through its interactions with other large-scale brain networks, the insula (8) assesses the risk of an interaction by (9) predicting the possible outcomes, i.e. acceptance or rejection. (10) Bodily feelings as well as cognitive processes, e.g. imagination of the outcome, may cause emotions like the pleasure or fear to interact. (11) The uncertainty of the other person's reaction engages the insula in deciding what to do next. Upon a decision, the insula anticipates the outcome (12) and (13) influences the behavior of seeking or avoiding the contact.

autistic symptom severity. Together, these findings indicate that both functional changes within the insula, as well as in long-range connectivity between the insula and related brain regions, contribute to the behavioral and cognitive symptoms of ASD.

Conclusion

The insula has thus been implicated in a plethora of different functions, which may at first present a somewhat confusing picture. However, a few general schemes emerge from the diverse studies that have been carried out in different species (see Figure 3 for a summary). First, despite its name, the insula is not an isolated 'island' but rather an integral brain hub connecting different functional systems underlying sensory, emotional, motivational and cognitive processing. Through its connectivity, the insula is ideally suited to monitor the current environment, as well as the present emotional and bodily states, and, based on experience, to predict how future actions may influence survival and wellbeing. It is thus crucial in determining the valence of internal and external stimuli. Together, these features explain the important roles that the insula serves in several forms of reinforcement learning, emotion control, and decision-making. It has further been suggested that the insula acts as a salience detector that marks the most relevant stimuli for further processing in other large-scale brain networks. In addition to these general roles, the insula contains multiple subregions, each characterized by different patterns of connectivity to the rest of the brain and at first sight distinct functional roles. How these different insular regions interact, whether their functions, albeit seemingly quite different, are subserved by common neuronal computations, or whether they operate as separate modules are open questions key to advance our understanding of insula function.

Homologies exist for all of the functional implications described above between human and animal models. This provides an entry point to use the animal model to unravel basic neural mechanisms of multimodal and multisystem integration, salience and valence coding, outcome anticipation, and prediction error coding. In this endeavor, the advent of recent techniques in neuronal circuit mapping and manipulation especially in rodent models will allow us to go beyond evaluating the functional implications of entire insular regions, and zoom in to deconstruct regions into functional

microcircuits and specific neuronal subpopulations.

In conclusion, the study of the neuronal mechanisms of insular function promises to deepen our understanding of the neuronal underpinnings of complex aspects of brain functioning, like the impact of feelings and emotions on flexible behaviors, such as decision-making, that occur in everyday life.

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Seeing lightness in the dark

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From intense sunlight in bright snow down to a moonless night in a dark forest, we can use light to recognize objects and guide our actions. This remarkable range mainly rests on having two different types of photoreceptors, the rods and the cones. The cones are active under daylight conditions, allowing high acuity and color vision. Rods are mainly active under very dim illumination conditions and have an exquisite sensitivity to light [1]. There are obvious detriments to visual perception in near darkness, such as a central scotoma, reduced motion perception [2], and most of all a lack of color [3]. There is only one type of rod, and thus intensity and wavelength differences cannot be disentangled when only the rods are active. This is captured well by the old saying “at night all cats are gray”, meaning that different colors inevitably get mapped onto different shades of gray. Here we show that the perception of lightness is also different for night vision: our results indicate that surfaces that appear to be white under daylight conditions, at best, appear medium gray under night vision, suggesting that activation of the cones is necessary for the perception of white.

We tested whether observers report surfaces as appearing ‘white’ under dark adaptation viewing conditions. Observers were shown ten pieces of paper, either A4 sized sheets or square cut-outs with a side length of 5 cm, that were printed such that they were equally distributed along a well-established lightness scale: the L* scale of the CIELUV color space [4]. As photopic and scotopic stimulation cannot be achieved at the same time, observers were first shown our darkest and whitest papers under bright photopic conditions and were asked to memorize these as 0% white (black) and 100% white, respectively. Later on, observers were shown each paper, one at a time, and they were asked to state their white rating for each paper, in steps of 10% along their memorized photopic white scale. We tested four illumination conditions that were all metameric to natural daylight (D65) in a counter-balanced design. The bright photopic (277 cd/m^2) and dim photopic (28 cd/m^2) conditions would mainly activate cone photoreceptors, the scotopic (1.2 x 10^{-4} cd/m^2) condition would only activate rods, and the mesopic (1.8 cd/m^2) condition would produce activity of rods and cones. For the smaller cut-outs, we also compared reports for foveal and peripheral viewing, since the fovea is dominated by cones.

Figure 1 contains the white ratings that observers reported for the papers. As expected, we find that observers properly recognize the brighter papers as lighter and the dimmer papers as darker across all illumination conditions. But while the most reflective paper is judged as white under photopic conditions, it is reported as gray under the mesopic and scotopic conditions, eliciting a report of 64% white under the scotopic conditions and of 79% white under the mesopic conditions (Figure 1A). For the smaller papers, the reported scotopic percept of white decreased to 47% white, with no significant difference between foveal or peripheral fixation (Figure 1B; one-sample t-test, darkest chip: $t(5) = 0.98$, $p = 0.37$, lightest chip: $t(5) = 0.70$, $p = 0.51$). For the scotopic conditions, we found that the darkest papers were consistently perceived as black. Under the brightest photopic condition, the A4 sized black paper appeared as a dark gray (11% white). Essentially, the lightness scale is compressed as either the stimulus becomes smaller or the illumination becomes darker, with the whiter end of the scale being more heavily compressed and the blacker end of the scale remaining relatively untouched.

These results present a stunning failure of lightness constancy, the ability of human observers to consistently judge surface reflectance as unchanging, despite changes in illumination. Modern lightness perception theories typically propose that the visual system separates surface reflectance (lightness) from direct and reflected illumination