

Sensory stimulation via the visual, auditory, olfactory and gustatory systems can modulate mood and depression

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Abstract

Depression is one of the most common mental disorders, predicted to be the leading cause of disease burden by the next decade. There is great deal of emphasis on the central origin and potential therapeutics of depression whereby the symptomatology of depression has been interpreted and treated as brain generated dysfunctions filtering down to the periphery. This top-down approach has found strong support from clinical work and basic neuroscientific research. Nevertheless, despite great advances in our knowledge of the aetiology and therapeutics of depression, success in treatment is still by no means assured. As a consequence, a wide net has been cast by both clinicians and researchers in search of more efficient therapies for mood disorders. As a complementary view, the present integrative review advocates approaching mood and depression from the opposite perspective: a bottom-up view that starts from the periphery. Specifically, evidence is provided to show that sensory stimulation via the visual, auditory, olfactory, and gustatory systems can modulate depression. The review shows how—depending on several parameters—unisensory stimulation via these modalities can ameliorate or aggravate depressive symptoms. Moreover, the review emphasises the bidirectional relationship between sensory stimulation and depression. Just as peripheral stimulation can modulate depression, depression in turn affects—and in most cases impairs—sensory reception. Furthermore, the review suggests that combined use of multisensory stimulation may have synergistic ameliorative effects on depressive symptoms over and above what has so far been documented for unisensory stimulation.

KEYWORDS

affect, bottom-up model, mood, multisensory stimulation, top-down model

1 | INTRODUCTION

Depression is one of the most common mental disorders and is predicted to be the leading cause of disease burden by 2030 (Kessler, 2012; Malhi, 2018; World Health

Organization, 2012, 2017). While clinicians and researchers in the field have greatly advanced our knowledge of the aetiology of depression and provided new therapeutic methods, success in treatment is still by no means assured (Akil et al., 2018; Blackburn, 2019;

Johnston et al., 2019; Trivedi et al., 2006). As a consequence, a wide net has been cast by both clinicians and researchers in search of more efficient therapies for mood disorders. To this end, clinical work and basic research have targeted the central mechanisms that underline the aetiology of depression and provided the basis for medical treatment. Thus, there has been a great deal of emphasis on the central origin and potential therapeutics of depression. The symptomatology of depression ranging from psychomotor retardation and anhedonia to cognitive impairment consequent to the emergence of the psychopathology have been interpreted as centrally generated dysfunctions filtering down to the periphery. This top-down approach has found overwhelming support from clinical work benefitting among others from pharmacotherapy (Krishnan & Nestler, 2010; Matveychuk et al., 2020; Murrough & Charney, 2012; Sanacora et al., 2012), various invasive and noninvasive centrally applied electrical/magnetic stimulation techniques (Andrade et al., 2010; Askalsky & Iosifescu, 2019; O'Reardon et al. 2007; Qi et al., 2020; Sackeim, 2000; Singh et al., 2020) as well as deep brain (Dandekar et al., 2018; Kisely et al., 2018) and vagal stimulation (Carreno & Frazer, 2017; Conway & Xiong, 2018; George et al., 2002).

Thus, the field of depression therapy and research has been solidly based for several decades on a central approach. Alongside this central approach, there is a complementary view that approaches mood and depression from the opposite perspective: a peripheral, bottom-up view that starts from the periphery and moves up centrally to integrate with the major central structures and functions that have been implicated in the aetiology of depression. Two review articles in the last decade (Canbeyli, 2010, 2013) advanced the complementary view that stimulation via the unisensory modalities such as vision or audition as well as physical exercise has the power to alleviate or aggravate depressive symptoms depending on several parameters such intensity, frequency, duration and quality of stimulation. The papers marshalled ample evidence to suggest that a bottom-up approach to depression not only should figure in the therapeutics of depression but may also provide clues to its pathogenesis. Approximately a decade after the original review, there is a plethora of new supportive evidence that expands the scope of the bottom-up view to emphasise the relevance of the bottom-up approach for a comprehensive evaluation of depression.

Before considering the potential merits of the bottom-up approach proposed earlier in the two review papers and espoused in the present paper as essential part and parcel of depression research, the reasons for the overwhelming current emphasis on the central view of

the pathology must be addressed. First and foremost, depression is mainly if not exclusively a human affliction that requires urgent action since it is now considered to be a worldwide health threat. Second, even though research on animal models of depression has provided valuable insights into depression (Krishnan & Nestler, 2011; Czéh et al., 2016; Ménard et al., 2016; Hao et al., 2019; Unal & Canbeyli, 2019), depression is basically a major human health burden at the individual and societal levels (WHO, 2016, 2018; GBD 2018; Malhi, 2018). Lastly, vast progress over several decades in the neurochemistry, neurophysiology and neuroanatomy of depression involving humans as well as animal models seems to justify an exclusively central, top-down approach. Thus, depression is overwhelmingly considered as a dysfunction in the neurophysiology and neurochemistry of a fronto-limbic circuitry components of which are not only more numerous now than just a few decades ago but also considerably better understood thanks to advances in neuroimaging, as well as therapeutic superficial and deep brain stimulation techniques (Duman et al., 2019; Jones & Nemeroff, 2021; Kisely et al., 2018; Nugent et al., 2019).

The present review does not by any means advocate a dichotomic provenance, namely, central versus peripheral source of depression but will emphasise the essential relevance of the peripheral inputs to the central mechanisms involved in the pathogenesis of and the potential therapeutic tools for depression. To appreciate the importance of peripheral stimulation in modulating depression, the paradoxical status of the brain as a privileged organ of the body must be taken into account; thanks to the skull, the meninges, cerebrospinal fluid and the brain-blood barrier, the brain is the most protected organ against internal and environmental threats and physicochemical fluctuations, while at the same time it is also the most receptive to physical and sensory input from the periphery. Contrasting with these elaborate protective systems is the reality that the brain unlike most internal organs provides access to physicochemical inputs, particularly via the sensory systems of vision, audition, olfaction and gustation with the last two systems directly allowing bodily access to physicochemical stimuli in the form of smells and ingested foods and drinks. Importantly, the review not only documents the mood modulatory effects of sensory stimulation via the visual, auditory, olfactory and gustatory systems but also emphasises the growing evidence that such unisensory stimulation has the potential to alter—and often impair—the central substrates involved in mood and depression. This in turn results in modified central sensory reception further complicating the interaction between the peripheral and central processing of sensory stimulation.

The subsequent sections of the review will deal with new insights into the potential contribution of peripheral stimulation to mood modulation considering two important points in sensory reception. First, it will be shown that individual differences in sensory reception may contribute to variability in the mood modulatory effects of peripheral stimulation. Second, the review will also argue that instead of unisensory stimulation via one of the sensory systems, multisensory stimulation involving combinations of these modalities may lead to an enhanced ameliorative effect on depression. Due to limitations of space, the review will deal mainly with evidence from human studies.

2 | VISION

In addition to seeing, the visual system starting at the retinal level has also non-image forming functions that are still under investigation. One major aspect of these functions is the modulation of emotion and mood. Studies indicate that the intensity, duration, timing and spectral quality of light significantly affect the impact of the visual system on emotion and depression (Jean-Louis et al., 2005; Vandewalle et al., 2010).

2.1 | Light improves mood and alleviates depressive symptoms

Modulation of mood by light is a globally experienced phenomenon as shown by the contrast in how people generally feel during the short and long days of winter and summer, respectively. Compared with the other sensory systems to be discussed below, the ameliorative effects of light stimulation have been studied more thoroughly and utilised more effectively for clinical purposes in the form of phototherapy. Starting in early 1980s, exposure to bright light for approximately 1 h early in the morning or evening has been shown to alleviate depressive symptoms in patients suffering from seasonal (Lewy et al., 1982; Avery, 1998; Oren & Terman, 1988; Pjrek et al., 2020) and non-seasonal depression (Even et al., 2008; Kripke, 1998; Mackert et al., 1991; Pail et al., 2011).

In addition to its use in controlled clinical practice, bright light stimulation has beneficial effects on depressive symptoms in uncontrolled or non-clinical environments. Thus, inadvertent exposure to bright light in the form of staying in sunny hospital rooms has been reported to significantly shorten duration of hospitalisation for depressed patients compared with those in 'dull' rooms (Beauchemin & Hays, 1996).

Exposure to artificial bright light can also have ameliorative effect on mood in nonclinical populations. Repeated exposure in the winter to at least 1 h of light reduced depressive symptoms in healthy office workers with or without winter-related mood symptoms (Partonen & Lönnqvist, 2000). Similarly, combining bright light exposure with exercise over an 8-week period significantly reduced depressive symptoms in healthy adults compared with exercise in normal indoor illumination (Leppämäki et al., 2002).

In addition to being used as monotherapy, bright light stimulation is also utilised as an augmentation of pharmacotherapy (Al-Karawi & Jubair, 2016; Guzel Ozdemir et al., 2015; Niederhofer & Von Klitzing, 2012). A review of studies using bright light treatment along with various pharmacotherapies indicated that phototherapy improved treatment of major depression compared with pharmacotherapy alone (Penders et al., 2016).

2.2 | Inadequate light photoreception or visual dysfunction can aggravate mood and depressive symptoms

Just as bright light stimulation is conducive to good mood and can ameliorate depressive symptoms, inadequate photoreception due to low or shortened periods of illumination as in the winter months is a globally experienced source of lowered mood and exacerbated depressive symptoms. This is not just valid for those who suffer from seasonal affective disorder (SAD), a subtype of depression characterised by increased depressive symptoms in the fall and winter with remission in the summer, but also for people who live in areas receiving little sunlight throughout the year (Booker et al., 1991). Since studies attest to the depressogenic effects of low or inadequate photoreception (Espiritu et al., 1994; Jean-Louis et al., 2005; Park et al., 2007), depressive symptoms should be expected to be frequently observed in people with visual impairments that limit the level of experienced illumination. This is indeed the case as confirmed by clinical reports of significantly increased incidents of depression in people with ophthalmic dysfunctions involving lowered photoreception and/or reduced visual acuity (Rovner et al., 2002; Rovner & Shmuel-Dulitzki, 1997; Shmuel-Dulitzki & Rovner, 1997).

2.3 | Depression can modulate visual processing and photoreception

The centrifugal/top-down relation between depression and vision involves modification—in most cases

impairment (Bubl et al., 2010; but see Golomb et al., 2009)—by depression of visual processes starting at the retina reaching up to subcortical and cortical levels. Depressed patients are challenged in visuospatial tasks (Asthana et al., 1998), and the visual system is sensitive to manipulations of mood and to depressive symptoms as shown by alterations in visual perception of faces according to different emotional states induced by music (Lolij & Meurs, 2011). High trait anxiety interacting with presentation of fearful faces exacerbates visual contrast sensitivity (Ferneyhough et al., 2013). In fact, impaired perception of emotion in depression may be a stable deficit (Kohler et al., 2011; Leppänen et al., 2004; Muller et al., 2014).

In addition to the modulation of visual attention involving specific features, particularly facial ones, affect can modulate the scope of visual processing as shown by the report that induced sad or happy moods promote local versus global visual features in a drawing task from memory (Gasper & Clore, 2002). Furthermore, affective states can bias gaze and visual attention to salient visual features (Vuilleumier, 2015).

Several studies emphasise the fact that perceptual deficits in visual processing in depression are not limited to emotional and affective states but point to a more profound impairment in the functioning of the visual system starting at the retinal level. Depression is reported to result in dysregulation of visual motion inhibition as measured by performance on motion detection with a center-surround suppression task (Norton et al., 2016). Similarly, Desseilles et al. (2009) found in an fMRI study that patients with major depressive disorder had difficulty detecting nonemotional stimuli (presented at a fixed central visual location) because of abnormal filtering of irrelevant stimuli presented in the periphery.

Modulation of visual processing by affective states or depression starts at the retina and continues at subcortical and cortical structures. (Cosker et al., 2020). Using electroretinogram (ERG) to measure retinal function, Lavoie et al. (2009) found significantly lower cone ERG in patients suffering from SAD. Compared with controls sensitivity to blue light as measured with pupillary light reflex is increased in depressed patients and is reduced after antidepressant treatment (Seggie et al., 1989).

Neuroimaging studies reveal that emotions and depression can modulate central processing of visual input (Adolphs, 2004; Bar & Neta, 2007; 195 Meier et al., 2007). For example, an fMRI study with depressed patients showed differential asymmetric activation of several limbic structures that were correlated with severity of depression (Lee et al., 2007). Another study using color and slides with emotional images found that compared with healthy controls depressed patients displayed

reduced responses in late stages of visual processing as measured with event-related potentials (ERPs) which recovered after pharmacotherapy (Pause et al., 2003).

3 | AUDITION

For humans, auditory stimulation, particularly music, is not only an effective but also very likely the oldest means of modulating emotions and mood as it has been practiced universally in the last several thousand years (Heise et al., 2013; Nizamie & Tikka, 2014; Rollin, 1998). However, both the methodical applications and neuroscientific investigations of the mood modulatory aspects of different auditory stimulation, particularly musical treatment, are relatively recent (Aalbers et al., 2017; Abbott, 2002; Croom, 2012; Kemper & Danhauer, 2005; Sachs et al., 2015). There is now strong evidence for the bidirectional-reciprocal relationship between hearing and mood and depressive disorders; a variety of auditory stimulation can modulate mood and depressive symptoms, while depression has the capability to alter auditory perception.

3.1 | Auditory stimulation can modulate mood and alleviate depression

A large range of auditory exposure varying from musical compositions to a variety of non-musical stimulation can modulate mood (Albersnagel, 1988; Clark, 1983; Gagner-Tjellesen et al., 2001). While auditory stimulation has been shown to alleviate depressive or anxious mood (Bouhuys et al., 1995; Hsu & Lai, 2004; Pignatiello et al., 1986; Smith & Noon, 1998; Thompson et al., 2001; Tornek et al., 2003), acute or chronic exposure to noise has negative psychological consequences (Gomez & Danuser, 2004; Stansfield, 1992). There is a paucity of systematic studies on the effects of noise on depression in humans. Nevertheless, a large-scale study in Amsterdam assessed depressed mood by means of the nine-scale Patient Health Questionnaire over approximately 5 years with a large number of subjects living in various locations with different levels of ambient traffic noise. Results indicated a significant positive association with depressed mood for those exposed to noise levels greater than 70 dB compared with the reference group who experienced 45- to 54-dB exposure (Leijssen et al., 2019).

Mood modulatory nature of auditory stimulation is not limited to musical compositions. For instance, stimulation with musically enhanced birdsong early in the morning induces positive mood and decreases depression as measured by Beck Depression Inventory (BDI) and

Profile of Mood States (Goel & Etwaroo, 2006; Goel & Grasso, 2004). Similarly, inducing binaural auditory beats by presenting tones of slightly different frequency to the two ears so as to create EEG activity in the delta/theta (1.5 to 4 Hz) range has been shown to result in negative mood compared with induction of activity in the beta wave range of 16 to 24 Hz (Lane et al., 1998).

Recent years have seen an increased interest in and use of music therapy for depression either as a low-cost monotherapy (Kemper & Danhauer, 2005; Lin et al., 2011) or as augmentation or enhancement to established standard psychotherapies (Erkkilä et al., 2011; Gold et al., 2009; Zhao et al., 2016). Minimal negative side effects, practical applicability tailored not just to individuals but to groups, combined with cost efficiency make it very likely that music therapies will figure more prominently in treatment of depression in the future, particularly with more systematic studies of the optimal parameters (Choi et al., 2008; Hillecke et al., 2005; Leubner & Hinterberger, 2017).

3.2 | Impaired audition can aggravate mood and induce depression

Some types of auditory stimulation and music can lower mood and induce depressive symptoms (Abbott, 2002; Kemper & Danhauer, 2005; Pignatiello et al., 1986; Smith & Noon, 1998; Thompson et al., 2001). On the other hand, compromised hearing resulting in limited auditory reception can also result in negative mood and increased depressive symptoms. For instance, not only total deafness but also compromised audioreception due to hearing impairment result in increased rates of depressive symptoms (Blazer & Tucci, 2019; Leigh et al., 1989; Leigh & Anthony-Tolbert, 2001; Magilvy, 1985; Steinberg et al., 1998; Watt & Davis, 1991; Zazove et al., 2006). Moreover, tinnitus, a hearing disorder involving perception of sound without an external source, is significantly correlated with depression (Stephens & Halam, 1985; Sullivan et al., 1988; Budd & Pugh, 1995; Langguth et al., 2011). Interestingly, a large-scale Korean study based on self-reported hearing impairment showed that individuals with hearing loss had significantly more depressive symptoms than those who had good hearing (Han et al., 2019).

3.3 | Low mood or depression can affect auditory function

Emotion and affective states can influence auditory perception at various levels of processing (Erhan et al., 1998;

Brandao et al., 2001). Perception of auditory loudness occurs early in the auditory processing hierarchy; Wang et al. (2009), for instance, reported that negative affect can modulate the central auditory response to speech within 20 ms. Pollock et al. (2006) demonstrated that subjects with high anxiety sensitivity had lower thresholds for reception of heartbeat. Compared with controls, auditory tones are rated as louder by subjects with negative affect (Siegel & Stefanucci, 2011) and thresholds for pure tones are decreased in depressed subjects with traumatic disorder (Aubert-Khalifa et al., 2010).

Several lines of research indicate that depression alters auditory perception at various stages of processing (Doose-Grünefeld et al., 2015). Depression impairs the evaluation of affective prosody (Schlipf et al., 2013; Uekermann et al., 2008) and music (Naranjo et al., 2011). Another study reported that compared with controls, depression did not alter the pleasure derived from a pleasant pure tone, but significantly reduced the pleasure from the repetition of the tone (Collet & Duclaux, 1986).

In addition to findings that locate the impairment of auditory reception in depression at early stages of processing, there are studies that indicate cortical involvement in the negative effect of depression on auditory function. An fMRI (Michael et al., 2004) and a magnetoencephalography (Tollkötter et al., 2006) study showed that depression can alter habituation in the auditory cortex to repeated presentation of sine tones. Hearing loss and auditory perceptual asymmetry are also reported in depression (Bruder et al., 1975; Malone & Hemsley, 1977). Several studies with depressed patients report a reduction of the left ear (right hemisphere) advantage for nonverbal tasks compared with non-depressed subjects (Bruder et al., 1981; Johnson & Crockett, 1982; Overby et al., 1989). Similarly, assessment of auditory sensitivity by means of pure tone and click audiometry in depressed patients indicated bilateral hearing loss and auditory asymmetry in the form of poorer hearing in the left ear (Yovell et al., 1995).

4 | OLFACTION

Olfaction is evolutionarily the oldest sensory system providing vital cues to humans and animals as they navigate in their natural environment. That odour that can modulate emotions is a universal phenomenon and an idea not lost to producers of expensive perfumes. More importantly, several decades of research have shown that depending on several parameters including quality, intensity and duration odours can modulate mood and depression (Kadohisa, 2013; Komori et al., 1995; Kontaris et al., 2020; Rochet et al., 2018; Schiffman et al., 2000).

4.1 | Olfactory stimulation improves mood and alleviates depressive symptoms

Several studies with nonclinical subjects indicate modulation of mood and depressive symptoms by pleasant and unpleasant odourants. For understandable reasons there are only a few studies that have assessed the deleterious effects of malodorous smells on mood and depression. Nevertheless, two studies using unpleasant odours akin to smell of rotten eggs, sodium sulfide (Weber & Heuberger, 2008) and dimethyl sulfide (Knasko, 1992) reported negative effects on mood while vanillin, a pleasant odour had the opposite effect. Conversely, several studies report elevated mood as a result of exposure not only to natural pleasant odourants such as lemon oil (Kiecolt-Glaser et al., 2008), lavender fragrance (Field et al., 2005; Goel & Grasso, 2004), Japanese cedar wood odour (Matsubara & Ohira, 2018) and citrus fragrance (Komori et al., 1995) but also to a synthesised odour derived from glycine/glucose Maillard reaction (Zhou et al., 2016). While most studies that have demonstrated ameliorative effects of odours were conducted in experimental environments, others indicate that mood can be improved by ambient odour stimulation in a natural outdoor setting (Glass & Heuberger, 2016) or in a dental office (Lehrner et al., 2000, 2005).

4.2 | Depression can modulate olfactory processing

Just as odours modulate mood and depressive symptoms, depression in turn can alter olfactory processing and sensitivity to odours (Flohr et al., 2017; Hoenen et al., 2017; Hur et al., 2018; Pollatos et al., 2007; Qazi et al., 2020). For instance, a study showed that depressed patients who were comparable with controls in the olfactory task before antidepressant treatment significantly increased their sensitivity to iso-amyl acetate 6 weeks after treatment onset (Gross-Isseroff et al., 1994). Pause et al. (2001) reported a reduction in olfactory sensitivity in depression proportional to the severity of the symptoms which was subsequently alleviated by antidepressant treatment. Similarly, a negative correlation was observed between sensitivity to odourants and the level of depression in healthy subjects as measured by BDI (Pollatos et al., 2007). One important aspect of this top-down influence on olfaction is the fact that the emotional and olfactory systems are intricately related with shared neuroanatomical structures (Laudien et al., 2006; Savic, 2001; Zald & Pardo, 1997, 2000). This is dramatically evident in reduced volume of olfactory bulb in major depressive disorders resulting in impaired olfactory function

(Negoiias et al., 2010). In fact, changes in the olfactory bulb volume and olfactory dysfunction have been proposed as potential biomarkers for depression (Atanasova et al., 2008; Brand & Schaal, 2017; Croy et al., 2014; Croy & Hummel, 2017; Rottstaedt et al., 2018). A psychophysiological study (Pause et al., 2003) assessed central processing of olfactory cues by means of chemosensory ERPs (CSERPs) in patients with major depressive disorder to a pleasant and an unpleasant odour. Results indicated impairment in the early level of olfactory stimulus processing compared with healthy controls. Similarly, CSERP recordings in healthy subjects revealed impairment in early aspect of olfactory processing when transient learned helplessness was induced by means of an unsolvable social discrimination test (Laudien et al., 2006).

Despite strong evidence for modified central olfactory system, there is no consensus as to the impact of depression on the peripheral effects of odourants in depressed patients with considerable inconsistencies in the reporting. For example, olfactory sensitivity is reported as unaltered in non-seasonal depression (Amsterdam et al., 1987; McCaffrey et al., 2000) and either unaltered (Oren et al., 1995) or increased (Postolache et al., 2002) in seasonal depression. Nevertheless, in addition to odour identification impairment (Khil et al., 2016), several studies have found a reduced sensitivity to pure olfactory (rather than trigeminal) cues in depressed patients with relatively severe symptoms (Lombion-Pouthier et al., 2006; Serby et al., 1990). Pause et al. (2001) reported a reduction in olfactory sensitivity in depression proportional to the severity of the symptoms which was subsequently alleviated by antidepressant treatment.

Factors that may contribute to the disparity in these reports arise from the fact that olfactory dysfunction may present itself as altered thresholds for and changes in sensitivity or emotional responses to odourants (Kohli et al., 2016). Differences in the medication status as well as duration of depression may also contribute to the inconsistent findings (Pabel et al., 2018). Nevertheless, according to two recent systematic reviews (Kohli et al., 2016; Taalman et al., 2017), a majority of studies reveal olfactory dysfunction in depression.

5 | GUSTATION

Unlike the other sensory systems taken up above which are usually subject throughout the day to varying degrees of ambient stimulation not all of which are voluntarily sought and willingly accepted, gustatory stimulation is usually individually initiated and punctate in nature in

the form of meals and snacks. Moreover, more so than the other sensory systems taken up earlier, gustatory cues are often bimodal in character, olfactory cues accompanying and modulating the impact of foods. Additionally, food items and eating habits are more closely determined by cultural norms and availability of foodstuffs dependent at least partially on geographical location and seasonal variability. Despite all these complications, there is a great deal of evidence for a bidirectional interaction between food intake and affect in that what we eat can modulate mood and depression, which in turn can impact our eating habits. What makes this bidirectional interaction difficult to delineate precisely at this stage but a worthy scientific challenge is the fact that the process of gustation from chewing to digestion directly involves more bodily systems including the gastrointestinal tract and the brain than the other sensory modalities (Breit et al., 2018; Ekstrand et al., 2017; Lach et al., 2018; Morkl et al., 2018; Vincis & Fontanini, 2019; Winter et al., 2018; Yang et al., 2020; Zheng et al., 2016).

5.1 | Foods can modulate mood and depression

Studies indicate that eating a chocolate bar or an apple can elevate mood compared with eating nothing in healthy female subjects (Macht & Dettmer, 2006). Similarly, palatable but not unpalatable chocolate briefly improves negative mood induced by a film clip (Macht & Mueller, 2007). The mood modulatory effect of chocolate goes beyond its transient mood ameliorative effect after a single episode of limited consumption (Meier et al., 2017) to protracted antidepressant effect when chocolate is consumed regularly over a prolonged period of time (Jackson et al., 2019). On the other hand, chocolate consumption as a result of emotional eating in stressful conditions may prolong dysphoric mood (Parker et al., 2006).

Eating habits and dietary patterns have impact on depression that go beyond the mostly transient effects due to a single meal or snack. The impact of food on depression is more dramatically evident when people adhere to certain types of diets and eating habits over a number of years. Surveys of large number of participants numbering in many cases in tens of thousands (Liu et al., 2016; Molendijk et al., 2018), who follow different dietary regimes (Rahe et al., 2014), over many years (Adjibade et al., 2018; Le Port et al., 2012) in different regions of the world (Akbaraly et al., 2009; Li et al., 2017; Sanchez-Villegas et al., 2018; Wolniczak et al., 2017) have provided insights into both positive and negative consequences of eating habits on depression. While these

surveys are mostly descriptive and correlational in nature without strict monitoring of adherence to the diets in question, they have provided valuable clues as to the general nature of foods that promote positive mood and prevent depression as opposed to those that have the opposite consequences. The overall picture that emerges from these surveys is that in general a Mediterranean type of diet—including fruits, vegetables and olive oil—is likelier to prevent depression and promote positive mood than a diet heavy on meats, processed foods and saturated fat. In fact, studies indicate that diets rich in fruits, vegetables and fish over number of years result in lower incidence of depression than diets with absence or shortage of these food items. Interestingly, soft drinks with added sugar or diets high in sugar content are more likely to induce depression, while coffee, tea and cocoa have the opposite effect (García-Blanco et al., 2017; Guo et al., 2014; Knuppel et al., 2017; Rothenberg & Zhang, 2019; Sanchez-Villegas et al., 2018; Vermeulen et al., 2017).

5.2 | Mood and depression modulate food intake and eating habits

The bidirectional interaction between gustation and depression is evident in a large number of studies that have discovered altered food intake and gustatory preferences in depression. Dulled or altered sensations of taste can accompany depression (Hur et al., 2018; Steiner et al., 1969, 1993), partially due to fact that most antidepressants modulate monoamine function that can distort or attenuate taste perception (Ackerman & Kasbekar, 1997; Heath et al., 2006; Schiffman, 1983; Schiffman et al., 1999). Nevertheless depressed patients report an ‘unpleasant taste’ regardless of previous antidepressant treatment (Miller & Naylor, 1989). Moreover, oral perception of fat and taste stimuli is modulated even in healthy subjects when a depressive mood is induced by video clips (Platte et al., 2013). On the other hand, dysphoric mood and depression can lead to be strong craving for food in general (Hill et al., 1991, Hill & Heaton-Brown, 1994) or more specifically for sweet tasting foods (Christensen, 1997; Kampov-Polevoy et al., 2006). Predilection for sweet foods and refined sugars is also reported in a non-clinical population with high scores on the BDI scale (Grases et al., 2019).

Depression is also often accompanied by craving for sweet foods, carbohydrates and chocolate, particularly when individuals engage in emotional eating (Camilleri et al., 2014; Ouwens et al., 2009; Rose et al., 2010). In SAD craving for sweets and carbohydrates shows a similar pattern, increasing during the winter and

decreasing in the summer (Krauchi & Wirz-Justice, 1988), while the opposite pattern is observed for sensitivity to sweet taste (Arbisi et al., 1996). Longitudinal surveys investigating the consequences of different diet patterns on depression have also discovered that depression in turn may have prolonged altered effects on gustatory preferences. Thus, a study classifying participants as depressed or not on the basis of BDI scores indicated lower consumption of legumes and fruits accompanied higher consumption of sweet foods and refined sugar in depressed than the non-depressed group (Grases et al., 2019). Adherence to unhealthy eating patterns can not only induce depression over time but also the consequent depressive state can cause people to persist in unhealthy eating regimes (Le Port et al., 2012; Wolniczak et al., 2017).

5.3 | Gut microbiota can modulate mood and depression

An important mood modulatory mechanism built into the gustatory/digestive system is the contribution of gut microbiota to maintenance of proper mental health, a phenomenon that has become a focus of research on depression relatively recently. In essence, what we eat affects the gut microbiota consisting of microbial organisms in the form of bacteria, viruses and archaea (Kelly et al., 2016; Lozupone et al., 2012; Osadchiy et al., 2020). What we eat modifies the gut microbiota, which in turn interact with the brain in a bidirectional manner neurally and hormonally (Dinan & Cryan, 2013; Mohammadi et al., 2015). The CNS modulates the composition and functioning of the microbiota neurally via autonomic regulation of the gastrointestinal system and hormonally (Cryan & Dinan, 2012; Dinan & Cryan, 2013, 2017; Osadchiy et al., 2020).

Recent studies suggest that the bidirectional nature of the gut-brain interaction is critical in modulation of mood. There is growing evidence that the gut microbiota show differences between healthy and depressed individuals. Clinical studies indicate altered microbiota in depressed patients (Jiang et al., 2015; Naseribafrouei et al., 2014), while increased incidence of depression or mood has been reported in populations exposed to microbiota altering bacterial infection (Garg et al., 2006; Lowe et al., 2014). In an interesting study, faecal microbiota from depressed and control subjects transplanted to microbiota deficient rats showed that microbiota from depressed but not control subjects induced depression-like behaviour in the recipients in the form of anhedonia and anxiety-like behaviour (Kelly et al., 2016).

5.4 | Differences in sensory reception is a factor in individual variability in depression

There is a great deal of individual variability not only in vulnerability to depression under similar depressogenic conditions but also in responding to similar therapeutic interventions. Factors leading to such variability are themselves important targets of research which is at present almost exclusively based on the central top-down account of depression. Thus, variability in outcomes can be accounted for among others by individual differences in susceptibility to stress (McEwen et al., 2015; de Kloet et al., 2016; Strain, 2018), psychobiological factors (Danion et al., 1996; Disner et al., 2011; Gonda et al., 2015; Grahek et al., 2019; Haglund et al., 2007; Pulcu & Elliott, 2015), neurobiological functions (Chaudry et al., 2015; Duman et al., 2019; Han & Nestler, 2017; Pitsillou et al., 2020; Villas Boas et al., 2019) and genetic/epigenetic factors (Freeborough & Kimpton, 2011; Klengel & Binder, 2013; Penner-Goeke & Binder, 2019; Ramos et al., 2016). While all these factors have been shown to contribute to the variability of the severity and duration of depressive symptomatology, there is now evidence to also consider differences in sensory reception as possible source of variability in depression.

The premise of the bottom-up sensorimotor hypothesis of depression is that sensory stimulation can modulate mood and depression. Most evidence to support the hypothesis is based on stimulation provided artificially in experimental or clinical settings. It is also important to consider the reverse side of the coin to assess the potential consequences of individual variability in sensory perception on mood and depression. Humans and animals can differ—in some cases widely—in their receptivity to and perception of sensory stimulation emanating from the environment (Acevedo et al., 2014; Aron et al., 2012). Thus, individuals can be hyposensitive or hypersensitive to sensory stimulation that may contribute to differences in emotional reactivity (Aron & Aron, 1997). While the full implication of super sensory sensitivity is still under investigation, there is evidence that differential sensory reactivity as evinced in hypersensitive as well as hyposensitivity (Acevedo et al., 2014) modulates emotional and affective assessment of sensory input from the environment (Lionetti et al., 2018). In the light of bottom-up approach to depression espoused in the present review, it is important to investigate the potential contribution of hyposensory and hypersensory sensitivity to mood and depressive symptomatology. In fact, a study that investigated sensory sensitivity in a large number of female and male undergraduates students (with two samples totaling

more than one thousand subjects) found that the subjects could be categorised as low, middle and high sensitive groups that differed in their emotional reactivity as induced experimentally by sad and happy video clips (Lionetti et al., 2018). Given the evidence provided so far, variations in sensitivity should differentially affect emotion, mood and depression. While there is as yet a paucity of research along these lines, likely due to the recency of research interest in the topic, Serafini et al. (2017) have already shown that higher sensory sensitivity and sensory avoidance are both correlated with depression. Moreover, patients suffering from seasonal depression are more likely to score high on tests assessing super sensory sensitivity (Hjordt & Stenbaek, 2019).

Another factor that must be considered in fully assessing individual variability in the impact of sensory stimulation on mood and depression is that some individuals are particularly adverse to environmental stimulation. In extreme cases such individuals who are avoidant of sensory stimulation are considered to suffer from an as yet not clearly understood phenomenon known as sensory processing disorder (SPD). First, proposed by Ayres (Ayres, 1966, 1969), SPD at first blush seems mainly as a poorly understood developmental regulatory dysfunction afflicting children. It is characterised by sensory intolerance involving hyposensitivity or hypersensitivity to non-aversive stimuli and inability to utilise sensory information in adaptive behaviour to different situational demands. As is hypothesised by the present review, SPD is related to emotional reactivity and mood disorders (Carter et al., 2011; Engel-Yeger & Dunn, 2011; Engel-Yeger, Gonda, et al., 2016; Engel-Yeger, Muzio, et al., 2016; Liss et al., 2005; Miller et al., 2009). There is evidence that the disorder is not strictly restricted to early developmental stages as it can be exhibited by adults as well (McMahon et al., 2019). In fact, a study with a large sample of female and male English adults ($n = 6147$) showed that increased sensory impairment in hearing, vision and taste linearly increased the risk of depressive symptoms (Liljas et al., 2020). Moreover, a large literature survey indicates that major depression involves sensory alterations and may be partially due to sensory perceptual disorder (Fitzgerald, 2013).

6 | ENDING ON A POSITIVE NOTE: MULTISENSORY STIMULATION MAY BE MORE EFFECTIVE IN ALLEVIATING DEPRESSION

In real life living organisms rarely encounter pure sensory stimulation in any single modality. Natural

environment almost always presents itself in the form of combination of sense data from several sensory modalities that allow humans and animals to perceive entities and situations for appropriate assessment and action. A lion, for example, in most cases is not just a visual image but an auditory and perhaps even olfactory presence as well. It is therefore critical to consider the merits of multisensory as opposed to unisensory stimulation in treatment of depression. Recent advances in sensory physiology as noted below necessitate the inclusion of multisensory stimulation in the bottom-up approach to sensory modulation of mood and depression.

Recent developments suggest that the traditional view of separate primary cortical projections for each sensory system which allows for multimodal interactions beyond these primary cortical projection areas is an oversimplification (Beer et al., 2011; Calvert et al., 1999; Meredith et al., 2009; Wallace et al., 2004). In fact, a view has been presented to suggest that there are no separate unisensory primary cortical projections (Ghazanfar & Schroeder, 2006). While this may be a somewhat extreme view at this stage, there is now formidable evidence that there are multisensory neurons in what used to be considered unisensory projection areas (Atilgan et al., 2018; Bizley et al., 2007; Driver & Noesselt, 2008; Gu et al., 2019; Kayser et al., 2008; Schroeder & Foxe, 2005; Teichert & Bolz, 2018).

The mechanism by which peripheral stimulation ameliorates depressive symptomatology has to involve and engage the central neurocircuitry underlying depression. As noted in previous sections, unisensory stimulation can have ameliorative effects on mood and depression. Multisensory stimulation involving more than one sensory system therefore not only will add to the central impact but may also result in synergistic effects since primary sensory cortical areas are also responsive to inputs from other sensory modalities as noted above. Several lines of research involving combination of visual, auditory, olfactory and gustatory stimulation indicate a possible synergistic impact over and above the effects of individual unisensory combinations. There is growing evidence for cross-modal interaction or synergism when stimulation from one modality is combined with that from another (Lalanne & Lorenceau, 2004). After the seminal work by McGurk and MacDonald (1976) that showed a cross-modal interaction between auditory and visual processing, cross-modal interaction and synergism have been reported across a number of sensory combinations as between the auditory system and other sensory modalities such as vision (Chanauria et al., 2019; Ethofer et al., 2006; Kayser et al., 2010; Lovelace et al., 2003; McDonald et al., 2000; McDonald & Ward, 2000; Noesselt et al., 2010; Satoh et al., 2015; Shams et al., 2000; Tivadar et al., 2018; Watanabe &

Shimojo, 2001), olfaction (Castiello et al., 2006; La Buissonnière-Ariza et al., 2012) and gustation (Carvalho et al., 2015, 2016; Knoferle & Spence, 2012; Lin et al., 2019; North, 2012; Yan & Dando, 2015). Similarly, several studies report cross-modal interaction or enhancement between visual stimulation and olfaction (Zellner & Kautz, 1990; Zellner & Whitten, 1999; Gottfried & Dolan, 2003; Michael et al., 2003; Koza et al., 2005; Demattè et al., 2006, Demattè et al., 2009; Seo et al., 2010; Dong & Jacob, 2016) and gustation (Delwiche, 2012; Spence, 2019; Van Beilen et al., 2011). Synergism due to combination of olfactory and gustatory cues is not only constantly experienced almost with each meal or snack but has also been documented by several studies (Djordjevic et al., 2004; Welge-Lüssen et al., 2005) including the case of ‘umami’, the fifth taste due to the conjunctive effects of monosodium glutamate (MSG) and olfactory cues from foodstuffs resulting in a synergistic enhancement of taste (McCabe & Rolls, 2007; Rolls, 2009).

7 | CONCLUSION AND FUTURE DIRECTIONS

Progress in any area of medicine is achieved with constant search for improved techniques and approaches to treat diseases. The search for new therapeutic solutions becomes urgent with increased evidence that the available cures are not fully successful and even more urgent when the disease begins to afflict large portions of the worldwide population. Depression is at this very critical stage of requiring new therapeutic approaches as it is one of the most prevalent public health problems (Malhi, 2018; Mathers & Loncar, 2006; Rehm & Shield, 2019; World Health Organization, 2017). In view of this exigency, the therapeutic approaches to depression are now considerably more varied than imaginable only a few decades ago. A major tenet and guiding principle of these new approaches is the centrifugal top-down view of both the aetiology and therapeutics of depression. The present paper is not necessarily meant as an alternative to the top-down central account of depression but as a worthy addition to the palette of theorising and clinical practice about the provenance and therapeutics of the psychopathology. The bottom-up view of depression has the added advantage of potentially providing supplementary means of therapeutic vehicles along with the more traditional pharmacotherapeutic approaches. Sensory, particularly multisensory stimulation for purposes of therapy has the added advantage of being inexpensive and, with little precaution, almost free of side effects, two factors that must weigh positively on the minds of both clinicians and patients when considering pharmacotherapies.

A practical starting point for multisensory treatment of depression may be a combination of photic and music stimulation. As noted earlier, light therapy is now a well-established monotherapy for many cases of depression. Pairing of visual and auditory stimuli conveying the same emotion is shown to amplify the affective experience of the viewer and may have therapeutic effect (Baumgartner et al., 2006; da Silva, et al., 2015; Eldar et al., 2007). Because quality, intensity and duration can be practically controlled for photic and auditory stimulation, a combination of light and music therapy is amenable to parametric assessment for achieving optimal synergism in alleviating mood and treating depression.

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CONFLICT OF INTEREST

There is no potential conflict of interest.

PEER REVIEW

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DATA AVAILABILITY STATEMENT

My article E|N15507 is a review and does not contain any empirical data that can be shared with other researchers.

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