1

The Nature of Tics and Habits

Overview of the Nature of Tics and Habits

History

The first references to tics go back to medieval times. In the fifteenth century, two Dominican monks reported the case of a priest who could not help but grimace and emit vocalizations, whenever he was praying (Kramer and Sprenger, 1948). Later in 1825, Jean-Marc Gaspard Itard described tics in a systematic way for the first time (Itard, 1825). The latter reports the case of a 26-year-old French noblewoman, the Marquise de Dampierre, who presented involuntary convulsive spasms and contortions at the level of the shoulders, neck, and face. Shortly afterwards, he also reported the presence of “spasms affecting the organs of voice and speech,” and notes the presence of strange screams and senseless words in the absence of a circumscribed mental disorder.

The Gilles de la Tourette syndrome is named after the French neurologist Georges Gilles de la Tourette, who, in 1885, described again the condition of the Marquise de Dampierre, now aged 86 years old, who continued to make abrupt movements and sounds also known as tics. The same year, Tourette described eight other patients with motor and vocal tics, some of whom had echo phenomena (a tendency to repeat things said to them) and coprolalia (utterances of obscene phrases) (Gilles de la Tourette, 1885) which was consistent with similar observations from American clinicians 1 year later (see Dana & Wilkin, 1886). In a doctoral dissertation published under the supervision of Tourette and Charcot, Jacques Catrou, documented 26 other cases (Catrou, 1890) with more details. The merit of Gilles de la Tourette’s report, consisted not only in gathering remarkable clinical descriptions of the symptoms that were little documented, if ever, until then, but also in describing the fluctuating evolution of what became known as the Gilles de la Tourette Syndrome (Gilles de la Tourette, 1885).

Subsequently, there were few systematic investigations, clinical observations, or particular etiological developments during the first half of the twentieth century. Rather, during this period, a psychoanalytic explanation prevailed, with little or no notable empirical support (Ascher, 1948; Ferenczi, 1921; Mahler, 1944; Mahler, Luke, & Daltroff, 1945). In the 1960s an experimental drug treatment...
(i.e., haloperidol) surfaced for tics (Seignot, 1961). These results encouraged clinical trials in the United States, which further supported the beneficial effects of neuroleptics (Corbett, Mathews, Connell, & Shapiro, 1969; Shapiro 1970; Shapiro & Shapiro, 1968). These seminal investigations instigated the race to find an effective pharmacological treatment and, therefore, the search for a neurobiological etiology, relegating to the background, the psychoanalytic, and the behavioral approach as well (Shapiro & Shapiro, 1971 Shapiro, 1970; 1976).

**Idea of a Tourette or Tic and Habit Spectrum**

A majority of patients with Tourette’s also face various concomitant problems (Freeman et al., 2000), which include obsessive-compulsive disorder (OCD) or at least some obsessive-compulsive symptoms, attention deficit hyperactivity disorder (ADHD), depression, and anxiety disorders.

**Current Diagnostic Criteria of Tics and Habits**

**Nosology of the Gilles de la Tourette syndrome and tic disorders**

Tic disorder and Tourette’s syndrome are currently classified in the Diagnostic and Statistical Manual of Mental Disorders Version 5 (DSM-5) (APA, 2013) with motor disorders listed in the neurodevelopmental disorder category. A tic is defined as a sudden, rapid, recurrent, non-rhythmic motor movement or vocalization. Tics can be present in the form of simple or complex multiple motor or vocal tics. The complex tics are contractions of a group of skeletal muscles, resulting in complex and repetitive movements, such as hopping, contact with certain objects or people, grimacing, abdominal spasms, tapping, movements or extension of the arms or legs, shoulder movements in sequence, copropraxia (unintentionally performing sexual gestures), or echokinesia (imitation of a gesture). Simple tics are defined as non-voluntary repetitive contractions of functionally related groups of skeletal muscles in one or more parts of the body including blinking, cheek twitches, and head jerks among others. Vocal tics can also take the form of simple (e.g., coughing, sniffing, clearing throat) or complex tics, such as coprolalia (using profanity and obscene words) or palilalia (involuntary repetition of syllables, words, or phrases).

Tic disorders are grouped into three main classifications in the DSM-5: Tourette’s disorder (307.23), persistent chronic motor or vocal tic disorder (307.22), and provisional tic disorder (307.21).

The criteria for Tourette’s disorder are (a) the presence of both multiple motor and one or more vocal tics at some time during the illness, although not necessarily concurrently; (b) tics that may wax and wane in frequency, but have persisted for more than 1 year since first onset; (c) onset is before age 18 years; and (d) a disturbance that is not attributable to the physiological effects of a substance or another medical condition (e.g., Huntington’s disease, post-viral encephalitis). For the persistent chronic motor or vocal tic disorder, single or
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Multiple motor or vocal tics have been present during the illness, but not both motor and vocal. The other criteria are similar to those for Tourette’s disorder. In provisional tic disorder single or multiple motor and/or vocal tics are present, and tics have been present for less than 1 year since first tic onset. Criteria have never been met for Tourette’s disorder or persistent (chronic) motor or vocal tic disorder (see examples in Table 1.1).

Habit disorders and body focused repetitive behavior (BFRB)

Another concomitant clinical problem often associated with tic disorder is body focused repetitive behavior (BFRB), also known as habit disorder. BFRB represents a clinical term that includes various diagnoses, such as trichotillomania,
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Skin picking, and onychophagia, and may also include certain types of teeth grinding. More precisely, habits are impulsive and destructive body focused behaviors that are repetitive, automatic, and serve no obvious function. These include hair pulling (trichotillomania; TTM), skin picking (BS), nail biting (NB), teeth grinding, and knuckle cracking, among others. They are variably described as impulse control disorder, stereotypic movement disorder, and obsessive-compulsive spectrum disorders (see definitions of each habit disorder in Table 1.2).

Despite the heterogeneity of symptoms in the BFRB category, their main clinical signs are directed toward the body, in reaction to feelings of discomfort, which is often present in tic disorder. In the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, text revision (DSM-IV-TR), trichotillomania was categorized as an impulse control disorder, not elsewhere classified, and was associated with skin picking and onychophagia (APA, 2000). In the DSM-5, trichotillomania and skin picking are now classified within the obsessive-compulsive and related disorders category, while onychophagia and dermatophagia are mentioned as “other specified obsessive-compulsive and related disorders.” According to the current diagnostic criteria, typically patients must have BFRB when they repeatedly engage in body focused activities (other than hair pulling or skin picking), make repeated attempts to reduce or stop the behaviors, and experience significant distress or impairment caused by the behaviors. Despite the fact that these disorders have been relocated to the obsessive-compulsive category, impulse control and feeling of sensory discomfort remain an important communality of their profile. In addition, this incapacity to resist a specific impulse or urge is a characteristic shared by tic disorder and BFRB patients.

Tic disorder and BFRB or habit disorders are considered similar, and the relationship between these two entities is sometimes clinically unclear: the complex movements in BFRB can often be confused with complex tics. Another good

| Table 1.2 Examples of body focused repetitive disorder of hair pulling, skin picking, nail biting, neck cracking, body symmetry, and idiosyncratic |
|-----------------------------------|-----------------------------------|-----------------------------------|
| **Hair pulling**                  | **Skin picking**                  | **Nail biting**                   |
| ● Recurrent pulling out of own hair, resulting in noticeable hair loss | ● Repetitive picking of blemishes or healthy skin causing tissue damage using fingers, tweezers, pins, or other instruments | ● Insertion of fingers into mouth, with contact between nails and teeth |
| ● Hair may also be eaten or rolled into balls and swallowed | ● Repetitive picking of blemishes or healthy skin causing tissue damage using fingers, tweezers, pins, or other instruments | ● Teeth replace scissors or nail clippers |
|                                   | ● Repetitive picking of blemishes or healthy skin causing tissue damage using fingers, tweezers, pins, or other instruments | ● Nail often bitten beyond nail bed and cuticles, drawing blood |
| **Neck cracking**                 | **Body symmetry**                 | **Idiosyncratic**                 |
| ● Twitching neck or flexing knuckles until joints crack and crack is felt or heard | ● Complex body movements to even up feeling of symmetry on either side | ● Any self-destructive repetitive movements to give feeling of relief and emotional regulation |
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reason to characterize tic disorder and BFRB together is mainly related to their response to treatment. Our experience allows us to see habits as part of the Tourette spectrum. Table 1.3 gives a list of all problem habits that we consider to fall under the Tourette/tic/habit spectrum and why. We do not include bruxism (teeth grinding), although it responds well to the CoPs treatment program.

Current Multidimensional Etiology of Tics and Habits

Prevalence, comorbidity, and behavioral problems

Once considered an uncommon syndrome, the Gilles de la Tourette Syndrome is no longer considered as a rare condition and is now regarded as being almost as common as schizophrenia. The DSM-IV-TR set the current prevalence rate of 5 to 30 children out of 10 000 and 1 to 2 adults out of 10 000 (APA, 2000). The Tourette International Consortium reported that the male to female ratio is 4.4 to 1 with a mean age of onset of 6.4 years (Freeman, 2007). A family history is present in 52% of patients, while attention deficit hyperactivity disorder (ADHD) is observed in 56%, and obsessive compulsive disorder (OCD) is observed in 55%. In terms of clinical course, tics tend to disappear in adult life in approximately half of patients and remain in only 10% of patients (Robertson et al., 2012). However, some authors claim that this proportion is inaccurate due to the variance characterizing the target population (Lanzi et al., 2004; Leclerc, Forget, & O’Connor, 2008). Current epidemiological studies indicate a prevalence of 1 individual in 200 would be more realistic, especially due to the presence of comorbid disorders (Freeman et al., 2000; Hornsey, Banerjee, Zeitlin, & Robertson, 2001; Kadesjö & Gillberg, 2000; Mason, Banerjee, Eapen, Zeitlin, & Robertson, 1998; Wang & Kuo, 2003). Furthermore, as noted by O’Connor (2005), the presence of tics or habits may be an important source of distress. This can take the form of phobias, depression, social anxiety, concerns about self-image, a decrease in self-esteem, relationship problems, and so on. The possible unsystematic presence of obscene gestures (copropraxia) or language (coprolalia) may also cause significant distress. In a study on the interference of tic disorder with daily activities, several socio-economic difficulties were reported: marital conflict, unemployment, mobility restrictions, and so on. This type of difficulty is perceived as being due to the presence of tics or

<table>
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<th>Physical</th>
<th>Psychological</th>
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<tr>
<td>● Hair loss, follicle damage, scalp irritation, repetitive strain injuries</td>
<td>● Shame, guilt, and embarrassment</td>
</tr>
<tr>
<td>● Skin scarring, sores, and infections</td>
<td>● Avoidance of swimming, windy weather, visiting hairdresser, doctor, and dentist</td>
</tr>
<tr>
<td>● Damage to fingers, gums, and teeth</td>
<td>● Significant social and occupational impairment</td>
</tr>
<tr>
<td>● Joint dislocation</td>
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habits, but, to date, little epidemiological investigation has examined this field (O’Connor et al., 2001). Simple tics develop before complex tics and phonic tics (see Figure 1.1).

**Neurological and physiological etiology**

Understanding the cerebral causes of tic disorder remains important because it has the potential to help cognitive-behavioral therapy (CBT) practitioners to refine the modality according to motor symptoms as well as the corporeal distribution of tics or habits. This distribution, illustrated to the patient with a body diagram, helps to locate tics or habits during a structured interview. Tourette’s syndrome has been considered initially to be of “nervous origin” even in the absence of an explanatory model of underlying symptoms (Gilles de la Tourette, 1885). Although important progress has been made in the past few decades due to, among other things, the series of seminal articles by the Shapiro team (Corbett, Matthews, Connell, & Shapiro, 1969; Shapiro, 1970, 1976; Shapiro & Shapiro, 1968), the cerebral origin of tics or habits remain, nonetheless, equivocal. Recent studies suggest that the mechanisms responsible for this syndrome could be attributed to a dopaminergic dysfunction provoking a higher level of cerebral activation than normal, so provoking these motor symptoms (Muller-Vahl et al., 2000). Dopamine is a neurotransmitter primarily associated with movement control as well as with rewards and pleasure. It is thus plausible to hypothesize that the networks associated with motor skills are involved in Tourette’s syndrome. Neuroimaging studies have underlined the contribution of the frontal motor cortex as well as deeper subcortical regions such as the basal ganglia, which have been structurally affected among children and adults afflicted with Tourette’s syndrome (Peterson et al., 2003). These deep nuclei project themselves via a structure called the thalamus and then to different

![Diagram showing the development of tics](image-url)
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regions of the cortex also affected in Tourette’s syndrome, such as the prefrontal and somatosensory cortex (Thomalla et al., 2009). These circuit loops are involved in multiple functions such as emotions, motivation, personality, disinhibition, executive functions, motor planning, and the control of different muscles. To date, this network is probably the best candidate for explaining the expression of tic symptoms (see Figure 1.2).

Somehow, the neurobiological hypothesis seems insufficient in order to truly appreciate the complexity of this syndrome, because we know little of the functional implication of affected structures in Tourette’s syndrome. Is the entire network affected? Or is it a part of the network that affects other structures one after the other? Could it be an overactivation or an underactivation of certain networks?

Etiology of developmental and behavioral neuroplasticity in tics and habits

Tourette’s syndrome is characterized by its fluctuating nature over time, and, as we have seen in the previous section, its developmental trajectory needs to be
considered. Through longitudinal studies, certain hypotheses have underlined cerebral anomalies associated with the persistency of the symptoms in adulthood. Peterson and collaborators proposed that because it is present in every age group, certain brain alterations could constitute a global feature of Tourette’s syndrome. However, the volume decrease of deep subcortical cerebral regions, as well as the increase in volume of cortical motor regions are uniquely present among adults, which suggests that they are associated with the development of symptom maintenance among subgroups with significant and persistent symptoms of Tourette’s syndrome during adulthood. Among these individuals, there seems to be a neuroplasticity anomaly that allows counteracting for the presence of tics or habits via an overactivation of motor inhibition processes. Neuroplasticity is defined as the aptitude of the brain to alter its own structure and function following changes in the external environment, such as following a new learning concept or after psychotherapy, for instance. The brain could be able to adapt and realize such modifications following therapy, as demonstrated with neuroimaging (Deckersbach et al., 2014) and electrophysiological techniques (Lavoie, Imbriglio, Stip, & O’Connor, 2011; Morand-Beaulieu et al., 2016). According to that model, unlike adults, children with Tourette’s syndrome have a relatively larger orbitofrontal volume (Peterson, 2001; Peterson et al., 2001; Spessot, Plessen, & Peterson, 2004), which would constitute an adaptive plasticity in response to the expression of tics or habits, which, in turn, would help to inhibit them more easily. With the maturation of the frontal cortex during adolescence, this mechanism could gain strength, which explains the decrease in symptoms during adolescence and early adulthood. Among adults with persisting symptoms, this neuroplasticity could occur, but more research is needed.

These neurodevelopmental observations are compatible with CBT models (O’Connor, 2002). If the evolution and fluctuation of symptoms are related to a form of neuroplasticity, thus we propose that CBT will, in turn, improve symptoms as well as favoring neurophysiological changes corresponding to a normalization of cerebral function, a phenomenon that has recently been observed by our team (Branet, Hosatte-Ducassy, O’Connor, & Lavoie, 2010; Lavoie, Imbriglio, Stip, & O’Connor, 2011; Morand-Beaulieu et al., 2016; Morand-Beaulieu, Sauve, Blanchet, & Lavoie, 2015; O’Connor, 2005; O’Connor et al., 2001; O’Connor, Lavoie, Stip, Borgeat, & Laverdure, 2008).

Tics, Tourette’s, and habits are not anyway caused exclusively by brain dysfunctions. There is no compelling evidence that people with Tourette’s have neuropsychological alteration, except when it comes to organizing action and motor planning. Brain and behavior are a two-way traffic lane and it is conceivable that some brain anomaly is due to tic or habit suppression. Likewise, results from brain imaging and electrophysiology often fluctuate and it’s likely that a subsample of people with persistent tics is susceptible to higher sensorimotor activation. In any case, our approach here is to put the emphasis on processes leading to tics, which can be considered common. An alternative model considers tics at least maintained by contingencies, where negative reinforcement could be an important cycle. This heightened vulnerability to sensorimotor activation may explain the lower threshold, but also heightened attention to sensory phenomena.
**Cognitive and behavioral etiology**

The presence of tics generates significant distress among certain individuals. These signs are distinguishable in terms of phobias, depression, social anxiety, self-image concerns, a decrease in self-esteem, relationship problems (Marcks, Berlin, Woods, & Davies, 2007; Woods & Marcks, 2005), the negative perception of peers, and social rejection (Anderson, Vu, Derby, Goris, & McLaughlin, 2002; Roane, Piazza, Cercone, & Grados, 2002). Also noticeable is a particular style of behavioral planning on a cognitive level. Thus, preliminary results from the STOP questionnaire (O'Connor, 2002) suggest that Tourette’s syndrome would be defined by chronic overactivation, difficulty in staying focused, and the tendency to undertake many things at the same time (overactivity), as well as an increased investment in efforts related to motor function (overpreparation). These two components logically constitute the basic focus for therapy. In return, the treatment will sensibly lead to changes that impact on neuroplasticity.

**Environmental and psychosocial etiology**

Beyond the neurocognitive and behavioral origin of Tourette’s syndrome, psychosocial observations are critical to embrace the full picture of symptom evolution. First, environmental factors such as the presence of an academic support and the quality of social interactions can influence symptom severity (Leclerc, Forget, & O’Connor, 2008). Moreover, behavioral approaches conceptualize tic or habit manifestations as being associated with principles of learning and the management of environmental contingencies (Azrin & Nunn, 1973). Within that perspective, tics are considered as an exaggerated response involving social operant conditioning. Consequently, the tic or habit manifestation would be more frequent when the individual receives attention, or when it allows him/her to avoid an unpleasant situation (Roane et al., 2002; Woods & Miltenberger, 2001). This theory can explain the simple fact that talking about vocal tics can cause an important increase in these manifestations. These hypotheses are partly based on observations that tics fluctuate in time, but they seem incomplete and today's research states that other factors may be involved.

In light of previous statements, a sound hypothesis of tic or habit onset requires linking the role of many interrelated causes. An interactive and multidimensional model appears to be more plausible than a model based on a unilateral and linear causality (APA, 2000; O’Connor, 2005; Woods & Miltenberger, 2001).

**Social Impact and Consequences**

**Sensitivity to judgment**

There have been several studies on the social consequences of ticking (Cavanna, David, Orth, & Robertson, 2012; Cavanna et al., 2013; Crossley & Eugenio Cavanna, 2013). Tic disorder clients tend to be sensitive to the judgment of others. The comorbidities present in tic disorder can interact with quality of life, and sometimes miscomprehension of tics and cognitive, behavioral, and social
functions can interact with mood and relationship and influence quality of life and well-being. (Cavanna et al., 2012, 2013; Crossley & Eugenio Cavanna, 2013). They are often over-concerned with self-image and fear the judgment of others, including, but not limited to, how their tic or habit looks to others (Thibert, Day, & Sandor, 1995), which leads them to feel dissatisfied with themselves. Tic disorder clients also scored higher than controls on items such as feeling ill at ease with others, and self-image dissatisfaction (O'Connor et al., 2001). Although there are no data to date on whether such sensitivity and self-focus could be the result of ticking, these concerns may easily feed into the ticking loop, and feed heightened physiological tension and dissatisfaction, as suggested by the fact that even persons with low awareness of their ticking and its visibility to others still display this heightened negative self-focus.

Current Treatment Options

Pharmacological treatments

Pharmacological treatments are one intervention of choice to help people with Tourette’s syndrome. Various treatments have been proposed but the majority of prescription drugs, as much among adults as among children with Tourette’s syndrome, show a variable response, even sometimes on the same individual. From the beginning, let us mention that no drug can lead to the complete remission of this syndrome, and the dosage is usually adjusted, according to the presence of the dominant tic or habit or behavioral symptoms. Because of the dominant hypothesis of tics as a problem of the motor circuits and the dopaminergic system, dopamine antagonist neuroleptics are routinely the main treatment. Therefore, many researchers have observed that pharmacological agents that trigger an increase (agonist) in dopaminergic functions will exacerbate tics (Golden, 1974; Riddle, Hardin, Towbin, Leckman, & Cohen, 1987), whereas those that bring a decrease (antagonist) of the dopaminergic action tend to reduce the tic or habit frequency (Lombroso et al., 1995; Shapiro et al., 1989).

According to the Canadian guidelines for the evidence-based treatment of tic disorders (Pringsheim et al., 2012), weak recommendations were made for children for the use of typical neuroleptics (pimozide, haloperidol, fluphenazine, metoclopramide) or atypical neuroleptics (risperidone, aripiprazole, olanzapine, quetiapine, ziprasidone, topiramate, baclofen), while other treatments such as botulinum toxin injections, tetrabenazine, and cannabinoids were weakly recommended for adults only. However, strong recommendations were made for the use of guanfacine and clonidine in children, which are both antihypertensive agents and alpha 2a agonists.

Typical antipsychotics may cause extra pyramidal signs, characterized by involuntary movements, impatience, a need to constantly move, and significant trembling among other symptoms. Atypical neuroleptic or drug combinations are reserved for more complex cases as well as in the presence of associated disorders. The effectiveness of atypical neuroleptics has progressively been proven to reduce tics, despite the possibility of significant long-term side effects,
such as an increased risk of metabolic syndrome (i.e., hyperglycemia, weight gain, and diabetes). Other pharmacological agents (antidepressants or other neuroleptics) can provide positive results in reducing tics, but these results are often inconsistent and generally come from single cases and non-randomized trials (Pringsheim & Marras, 2009).

Managing tics and habits with the cognitive-behavioral approach

Azrin and Nunn (1973) were the first researchers to suggest that tics could be replaced by other behaviors. In their book *Habit Control in a Day* (1977) they suggested that the tic could be replaced by a response antagonistic to the tic tensing the opposite muscles and so impeding the tic. With practice the tic could be controlled until the tic was counter-conditioned and the urge even to tic disappeared. This same technique was applied to habits such as hair pulling, where the hands were kept occupied or in contact with a surface to impede movement to the hair or skin. The technique is termed habit reversal, and has been developed recently into a comprehensive behavioral intervention, CBIT, for tics where the main component is a competing response awareness and analysis of the stimulus and consequences of ticking in order to manage contingencies to reward non-ticking. There have been several studies affirming the validity of the CBIT approach and there are published manuals (Woods et al., 2012). Another approach particularly to managing habit disorders is the multi-modal comprehensive behavioral approach to controlling the habit, isolating and eliminating the stimulus signal for pulling whether it is sensory, cognitive, emotional, or perceptual (Falkenstein, Mouton-Odum, Mansueto, Goldfinger, & Haaga, 2015; Mansueto, Golomb, Thomas, & Stemberger, 1999). Currently, a CBT approach constitutes an effective line of treatment for adults with both tic disorders (McGuire, Piacentini et al., 2014; Wile & Pringsheim, 2013) and BFRB (Bloch et al., 2007; Gelinas & Gagnon, 2013; McGuire, Ung et al., 2014; Woods & Houghton, 2015). Another behavioral approach is exposure and response prevention, whereby the client tolerates the urge to tic whilst resisting the action of ticking (Verdellen, van de Griendt, Kriens, & van Oostrum, 2011). These approaches address the tic when it has occurred or when it is about to occur and do not look systematically at the processes leading up to and preceding the tic and tension onset as does the current CoPs approach. But the CoPs approach to management can be used in conjunction with the CBIT or comprehensive behavioral method or by itself. Another source of treatment is Keuthen and colleagues’ (2012) inspiring use of dialectical behavior therapy and the addition of mindfulness to tic and habit management, and other authors have used the addition of ACT to supplement habit reversal therapy for habits (Christenson & Crow, 1996; Flessner, Busch, Heideman & Woods, 2008a; Flessner et al., 2008b, 2008c; Keuthen et al., 2007, 2012, 2015; Woods, Wetterneck, & Flessner, 2006).

The therapy proposed by our group is based on the CoPs model, which aims at regulating the high level of sensorimotor activation present in these populations and preventing the buildup of tension that leads to tic bursts or to the habit disorder (Lavoie, Leclerc, & O’Connor, 2013; O’Connor, 2002; O’Connor, Lavoie, Blanchet, & St-Pierre-Delorme, 2015). Although empirical studies are limited, the
CoPs approach also has shown efficacy. Its effectiveness in treating adults affected by either disorder has been replicated (O’Connor et al., 2001, 2009, 2015). Over the past 10 years, our group has conducted a number of studies exploring the cognitive-behavioral and psychophysiological manifestations of motor activation in Tourette’s syndrome/tic disorder with the aim of linking the multi-level processes evoking tic onset with behavioral management procedures (Lavoie et al., 2013; Leclerc et al., 2008; O’Connor, 2005; O’Connor et al., 2005, 2008, 2009; Thibault, 2009). These experimental and clinical findings have led to elaboration of a cognitive-behavioral/psychophysiological model of treatment (Lavoie et al., 2013; O’Connor et al., 2015), which proposes: (a) an overactive style of planning that prevents optimal preparation for action; (b) this style leads to problems regulating arousal/inhibition processes particularly under circumstances where regulation is open-looped, controlled, and has unpredictable parameters; (c) such high levels of motor activation create tension and frustration and are likely to evoke ticking; (d) hence a CBT package, which addresses the cognitive psychophysiological sources of motor activation reducing background tension and preventing tic onset. Rather than target solely the tic implicated muscle in a competing response antagonistic to the tic, an important additional component in our CBT program is a modification of excessive overall motor activation, by targeting cognitive and behavioral/physiological sources creating tension. There were also significant changes post-treatment in measures of self-esteem, anxiety, depression, and style of planning action (O’Connor et al., 2001).

More recent clinical trials have found CoPs effective for habit disorder (O’Connor et al., 2017.).

**Therapist checklist for information on tics and habits**

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<tr>
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<tbody>
<tr>
<td>The client understands what constitutes a tic or a habit</td>
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<tr>
<td>The client understands recent findings on the nature of tics and habits</td>
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<tr>
<td>The client understands how tension maintains tics and habits and at the same time how tics and habits give relief from tension</td>
<td></td>
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<tr>
<td>The client understands the model of the program addressing downstream processes, the structure, and the evolution of the program</td>
<td></td>
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<tr>
<td>The client understands that the program progresses in stages each addressing different psychophysiological processes leading up to the tic or habit</td>
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<tr>
<td>The client understands the model and how we address flexibility in tension, preparation, and thought</td>
<td></td>
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<tr>
<td>The client chose a good metaphor to mark to progress</td>
<td></td>
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<tr>
<td>The client understands that the program is cumulative and progresses stage by stage</td>
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