Sexually explicit material (SEM) is now routinely and publically displayed throughout numerous public venues. SEM is easily accessed through a variety of electronic devices.
This sexualized environment is man-made. Internet-based access has dramatically increased in recent years (Buzzell, 2005). Such a dramatic change in the everyday environment of most youth raises questions regarding the impact of this material on development.

Physiological response to SEM

When an adolescent is exposed to SEM, the amygdala of the limbic system is activated (Redoute et al., 2000; Karama et al., 2002; Ferretti et al., 2005; Walter et al., 2008). Activation of the amygdala (Figure 1) concomitantly initiates physiological processes that flood the brain and body with neurotransmitters and hormones. These include cortisol (Mirolli, Mannella, & Baldassarre, 2010), testosterone (Viau, 2002), epinephrine (Mirolli et al., 2010), norepinephrine as both a hormone and a neurotransmitter (Mirolli et al., 2010; Arnsten, 2009), and dopamine (Mirolli et al., 2010; Arnsten, 2009). A critical function of the amygdala, therefore, is to enhance the perception of stimuli that has emotional significance (Anderson & Phelps, 2001). Afferent neuronal connections to the amygdala include visual, auditory, olfactory, and gustatory (Mirolli et al., 2010). In light of the extensive innervation to the amygdala, SEM could be considered visual images, reading or hearing sexual material, sexting, or listening to music with sexual themes.

Activation of the amygdala concomitantly initiates the following: 1) the hypothalamus activates neurons in the brain stem and spinal cord initiating the sympathetic division of the autonomic nervous system resulting in systemic release of epinephrine and norepinephrine; 2) the hypothalamus stimulates the pituitary gland, resulting in activation of the hypothalamic–pituitary–adrenal (HPA) axis resulting in cortisol release and the hypothalamic–pituitary–gonadal (HPG) axis resulting in testosterone release (Viau, 2002), 3) the nucleus accumbens is

activated via dopamine. For a comprehensive review of the amygdala and its innervations and regulation of somatic processes see Mirolli et al. (2010).

Additionally, the function of the prefrontal cortex is decreased and the function of the basal ganglia is increased due to the release of neurotransmitters, in particular dopamine and norepinephrine (Arnsten, 2009; Radley, 2005; Hanson et al, 2012). In other words, the brain's prefrontal cortex (center of careful decision-making) is impaired, while the basal ganglia (prone to impulsivity) is strengthened.

With the basic physiological response delineated, the following five sections will highlight adolescent physiological paradigms that distinguish them from adults. These differences will then be looked at in the context of SEM. These will be followed by a working model summary and a discussion.

Overactive dopamine system exacerbates pleasure seeking

Dopamine is considered the "pleasure neurotransmitter" (Arias-Carrion et al., 2010). Dopamine is unique in that it has the ability to cause craving or wanting (Volkow, 2006; Berridge, 2006). SEM activates the dopaminergic reward system of the brain (Negash et al., 2016). The nucleus accumbens is considered the pleasure center of the brain and located in the basal ganglia (Chambers, 2003; Jentsch, Roth, & Taylor, 2000; Jones, 2010). Dopamine-containing neurons are not only extremely adaptable (Chen, Hopf, & Bonci, 2010), they are also found to be more abundant in early adolescence, and they undergo neuronal pruning with advanced maturity (Somerville & Jones, 2010; Andersen et al., 1997; Wahlstrom, White, & Luciana, 2010). The nucleus accumbens has the highest density of dopamine receptors in early adolescence (Tarazi, Tomasini, & Baldessarini, 1998).

Adolescents are more susceptible to the pleasurable effects of dopamine because they have increased receptors for it (Wahlstrom et al., 2010; Teicher, Andersen, & Hostetter Jr., 1995). Wahlstrom et al. (2010) stated, "Increased receptors support the notion that limbic and striatal dopamine systems may be in a state of overdrive during adolescence." The dopamine system develops more quickly than the inhibitory serotonin system (Chambers, 2003).

An adolescent's nucleus accumbens is more easily aroused than an adult's when exposed to the same stimulant (Ernst et al., 2005). Galvan et al. (2006) stated, "evidence from our study supports the notion that relative reward preference is exaggerated during adolescence: adolescents showed an enhanced accumbens response." The more robust nucleus accumbens of an adolescent makes that age group particularly vulnerable to addictive substances (Hardin & Ernst, 2009).

Steinberg (2011) stated, "We now know there's a rapid increase in dopamine activity in early adolescence—in fact there's more dopamine activity in the brain's reward center in early adolescence than at any other time of life. Because things feel especially pleasurable during early adolescence, adolescents go out of their way to seek rewarding experiences." The prefrontal cortex has enhanced dopamine drive during adolescence (Spear, 2000). Heightened DA activity may result in an apparent over-activation of incentive motivation in the absence of reliable levels of behavioral control (Wahlstrom et al., 2010).

The convergence of a zealous dopamine system with the inherent neuroplasticity of dopamine neurons results in a condition particularly conducive to dopamine exploitation during adolescence.

When viewed through the lens of SEM, the effect of dopamine, namely pleasure and craving, would be heightened in an adolescent due to their robust dopamine system.

Release of cortisol in response to SEM exposure indicates increased stress

Cortisol is a hormone that helps the body respond to stress. Cortisol also facilitates sexual arousal (Goldey & van Anders, 2012). Stress is often conceptualized as perturbation of homeostasis (Sapolsky, 2004). With this definition in mind, Goldey and van Anders (2012) hypothesized that cortisol may be just as necessary to mobilize energy to respond to positive events (including sexual ones) as negative ones. In this context SEM can be considered a stressor because of the activation of the HPA axis and the corresponding release of cortisol. Van der Maij, Buunk, & Salvador (2010) reported cortisol release in men with a brief social interaction with women, and interestingly, the more attractive the woman was rated the higher the release of cortisol as compared to controls.

Cortisol has a diminishing effect on prefrontal cortex function, the executive center of the brain (Grundemann, Schechinger, Rappold, & Schomig, 1998; Wellman, 2001). Cortisol can directly reduce the functioning of the prefrontal cortex by blocking transporters that clear dopamine and norepinephrine, thereby indirectly increasing these neurotransmitters (Grundemann et el., 1998). The prefrontal cortex is very sensitive to its environment of neurotransmitters and peak performance demands a specific neurotransmitter climate (Arnsten, 2009).

During adolescence, the HPA pathway is more pronounced than in adulthood or childhood (Walker, Sabuwalla, & Huot, 2004). Adolescence is characterized by a prolonged activation in response to stressors as compared to adulthood, which may render ongoing development of the brain vulnerable (McCormick & Matthews, 2007; Dahl & Gunnar, 2009). During the juvenile period, the stress response lasts considerably longer than adults, possibly due to immature control regions of the brain (Romeo et al., 2004). Adolescents have decreased cognitive flexibility under stressful conditions when compared to adults (Rahdar & Galvan, 2014).

Rahdar and Galvan (2014) stated, “The adolescent brain is particularly vulnerable to the effects of daily stress relative to an adult comparison group, an effect that has significant consequence for behavior.” Among adolescents, the onset and persistence of depression is linked with augmented cortisol secretion (Goodyer, Herbert, Moor, & Altham, 1991; Goodyer, Tamplin, Herbert, & Altham, 2000; Goodyer, Park, & Herbert, 2001).

Hatch (2011) stated, “The stress of viewing sexually explicit images at an early age could be seen as a neurobiological event mediating the reactions exhibited by many teens in the form of emotional and addictive symptoms.” The adolescent HPA axis is more potent physiologically compared to an adult, therefore SEM would cause a more commanding HPA response and the resulting cortisol secretion and executive function impairment would be more pronounced. In the short-term, cortisol diminishes functioning of the prefrontal cortex (Grundemann et al., 1998; Wellman, 2001). In the long-term, cortisol has a direct role in brain development and neural plasticity (Sisk & Zehr, 2005; McCormick & Matthews, 2007; Joels, 2011).

Adolescents have a unique sensitivity to the steroid hormone cortisol when compared to adults or children (Brown & Spencer, 2013). The effects of steroid hormones on the brain during this adolescent window are organizational, with effects that are permanent, compared to activational effects which are transitory (Vigil et al., 2011; Peper, Hulshoff Pol, Crone, & Van Honk, 2011). Steroid-dependent organization of neural circuits is a fundamental feature of adolescent brain development that results in structural changes that determine adult behavioral responses to hormones and sensory stimuli (Sisk & Zehr, 2005).

Cortisol is important for appropriate maturation of neuronal precursors into fully developed neurons (Joels, 2011). McCormick and Mathews (2007) stated, “Glucocorticoids (cortisol) are hormones that influence ongoing brain development and program future behavioral and

psychological responses.*"* Adolescence is a time of significant brain development and maturation of the HPA axis, thereby providing an opportunity for glucocorticoids to exert programming effects on neurocircuitry involved in learning and memory that can result in changes that affect adult cognitive function (McCormick & Matthews, 2007; 2010).

Adolescent rats exposed to stressors had diminished cognitive performance as adults compared to non-stressed counterparts. Those exposed earlier in adolescence showed a greater decline in cognitive abilities (McCormick & Matthews, 2007). An altered adrenocorticosteroid milieu during vulnerable developmental periods of neurodevelopment can impair an individual's resilience to stress in adulthood (McEwen, 2004). Juvenile stress induces lasting impairments in stress coping ability (Tsoory & Richter-Levin, 2006). Exposure to short-term stressors during juvenility or adolescent stress resulted in impaired coping responses when faced with stressors in adulthood, resembling both anxious and depressive symptoms (Tsoory, 2008). In contrast, exposure to enriched environments conducive to brain development during adolescence can impact adult cognition positively. Mice exposed to enriched environments during adolescence, but not later in life, improved Morris water maze performance (Williams et al., 2001).

Cortisol release, as a result of exposure to SEM, has the long-term ability to manipulate neurodevelopment in regards to cognition and the ability to cope with daily stressors, even throughout adulthood. This impact from SEM is not found in an adult because the organizational adolescent window has closed.

Increased testosterone is a primer for enhanced sexual behavior

At the onset of puberty, the body releases pulses of gonadotropin-releasing hormone (GnRH), which triggers a flood of sex hormones including testosterone (Dorn et al., 2003; Vogal, 2008).

Testosterone levels are their highest during adolescence and early adulthood (Mayo Clinic/ Mayo Medical Laboratories). It is important to note that adolescent males have significantly higher levels of testosterone than adolescent females (Dorn et al., 2003), exacerbating the characteristics of testosterone in males.

External sexual stimuli is associated with testosterone release (Amstislavskaya & Popova, 2004; Bonilla –Jaime, Vazquez-Palacios, Arteaga-Silva, & Retana-Marquez, 2006; Redoute et al., 2000; Exton et al., 1999; Stoleru et al., 1999). Testosterone levels are linked to sexual motivation and behavior (Udry et al., 1985). Testosterone enhances sexual anticipation and prepares the brain for further sexual stimuli (Halpern, Udry, & Suchindran, 1998; Anderson et al., 1999). The levels of free testosterone in the blood has a positive correlation with sexual thoughts in adolescent boys (Halpern et al., 1994; Udry et al., 1985). Free testosterone level also correlated with sexual desire and sexual thoughts in females (Alexander & Sherwin, 1993). Elevated testosterone is linked to increased aggressiveness and violence (Schulz & Sisk, 2006; Nelson, Leibenluft, McClure, & Pine, 2005; Goetz et al., 2014; Banks & Dabbs, 1996). Testosterone diminishes the regulatory control from the cortex over the amygdala (Van Wingen, 2010). Testosterone levels are coupled to amygdala responsiveness (Derntl, 2009; Van Wingen, 2010). Goetz et al. (2014) observed increased circulating testosterone levels were associated with heightened activity of the amygdala and aggression.

Because testosterone levels are heightened during adolescence when compared to adults, sensations of sexual motivation, anticipation and desire, as well as aggressiveness will be magnified as a result of sexually-explicit material. In addition, the inhibitory effect of testosterone on the prefrontal cortex will be more profound during adolescence as a result of an intrinsically less developed prefrontal cortex.

Testosterone, like cortisol, is a steroid hormone (Brown & Spencer, 2013). As mentioned earlier, the effects of steroid hormones on the brain during this adolescent window are organizational, with effects that are permanent, compared to activational effects which are transitory (Vigil et al., 2011; Peper et al., 2011). Steroid hormones influence neurogenesis, neurite outgrowth, synaptogenesis, receptor expression, and neuronal excitability (Sisk & Zehr, 2005; Vigil et al., 2011; Ernst, Romeo, & Andersen, 2009). The determinations made during this key window of brain organization are the basis for the adult brain (Sisk & Zehr, 2005). Vigil et al. (2011) stated, "The incidence of exogenous factors in this brain/hormone dynamic could affect the organization processes which take place in adolescence and may generate alterations in brain organization, and could manifest themselves as behavioral disorders in adult life."

Puberty is a period of dramatic synaptic plasticity in the amygdala, including changes related to sexual, aggressive, and risk-taking behaviors (Zehr et al., 2005). An intriguing relationship occurs during adolescence between testosterone and the oxytocin-secreting neurons within the amygdala. During puberty testosterone influences the proliferation of receptors for oxytocin. (Steinberg, 2008). Chibbar, Toma, Mitchell, & Miller (1990) demonstrated that testosterone regulates oxytocin gene expression during a specific development period that encompasses puberty. Puberty is a prerequisite for the steroid sensitivity of the oxytocinergic system (Chibbar et al., 1990). The amygdala is replete with oxytocin receptors (Boccia et al., 2013).

Testosterone manipulates the oxytocinergic system by binding to androgen receptors found within the amygdala (Sarkey et al., 2008). Androgen receptor activation plays a role in neuronal activity, synaptic plasticity, and behavior (Sarkey et al., 2008). Oxytocin-secreting neurons have a recognized ability to undergo synaptic plasticity as a result of stimulation (Theodosis, 2002). Additionally, Neufang et al. (2009) found that the volume of the amygdala varied as a function of testosterone and was associated with circulating testosterone levels during the adolescent period.

The amygdala, more specifically the medial amygdala, plays a key role in regulating context-appropriate social behavior (Cooke & Woolley, 2005; De Lorme, Schulz, Salas-Ramirez, & Sisk 2012; De Lorme & Sisk, 2013). De Lorme and Sisk (2013) emphasized the role pubertal testosterone exerts on suitable social information processing and adept social flexibility during adulthood. Syrian hamsters show that the variability in the increase of gonadal hormone secretion is a factor that influences expression of social behaviors in adulthood (Schulz, Molenda-Figueira, & Sisk, 2009). Pubertal testosterone organizes the regional volume and neuronal number within the medial amygdala of the adult male Syrian hamster (De Lorme et al., 2012).

The male Syrian hamster demonstrated that levels of sexual and aggressive behaviors were reduced in castrated hamsters not exposed to testosterone during puberty (Schulz & Sisk, 2006). Amygdala hyper-reactivity has been reported in people who are particularly sensitive to social threats (Lorberbaum et al., 2004). Amygdala hyperactivity predicts increased fear or anxiety and may contribute to social disorders (Amaral, 2003). Taylor et al. (2008) found a consistent link between positive stress management and reduced amygdala activity.

During adolescence the amygdala can be bulked up as a result of excess amounts of testosterone. SEM is a source of excess testosterone release. The long-term implications of a robust amygdala as a consequence of the inundation of SEM affects self-regulation and appropriate social interactions even as an adult.

Immature prefrontal cortex and over-responsive limbic and striatal circuits enhances environmental sensitivity and diminishes behavioral modulation

The prefrontal cortex does not finish maturing until the early twenties (Giedd, 2004). The prefrontal cortex functions as the emotional brakes of the brain; however, it is a particularly difficult time for youth to maintain cognitive control in the face of emotionally-charged distracters (Somerville & Jones, 2010; Van Leijenhorst et al., 2010). Amygdala signaling during the teenage years is disproportionately strong when compared to adults (Vigil et al., 2011; Nelson et al., 2005; Somerville & Jones, 2010; Casey et al., 2010).

Hare et al. (2008) reported an enhanced adolescent amygdala response when compared to adults and children, irrespective of symptoms of anxiety. The late maturing prefrontal cortex causes enhanced sensitivity to environmental cues without appropriate behavioral inhibition (Somerville & Jones, 2010; Casey et al., 2010). An exaggerated ventral striatal response compared to a more tepid cognitive control response implicates a bias toward appetitive cues during adolescence compared to both adults and children (Somerville, Hare, & Casey, 2011). Somerville et al. (2011) stated, "The current behavioral findings suggest that when required to suppress behavioral approach to salient appetitive cues, adolescents' performance shows impairment not observed in other age groups."

Adolescents show a heightened activation of the ventral striatum in conjugation with rewards compared to adults (Casey, Jones, & Somerville, 2011). The nucleus accumbens presents enhanced activation to reward compared to both adults and children (Ernst et al., 2005; Galvan et al., 2006).

A study done by Negash and colleagues found that pornography is linked to immediate reward and a higher rate of delay discounting (Negash et al., 2016). Delay discounting encompasses

"impatient, impulsive, short-sighted, or lacking in self-control" (Fawcett, McNamara, & Houston, 2012).

When one considers SEM in terms of adolescents and their innately deficient braking portion of the brain (prefrontal cortex), and their disproportionately developed subcortical systems, the impact of SEM and delay discounting would be compounded.

A heightened period for neuroplasticity and behavioral influence

Next to infancy, most organizational brain growth occurs during adolescence (Sisk & Zehr, 2005; Schulz & Sisk, 2006; McCormick & Matthews, 2007; Vigil et al., 2011). Profound neuronal rewiring takes place during adolescence (Sisk & Zehr, 2005). Vigil et al. (2011) stated, "The most frequently used connections are strengthened and preserved, while synapses which have shown scarce activation degenerate." Reductions are made to energy consuming connections that do not transmit information efficiently, based on experience (Chambers, 2003). The brain fabricates more synapses than it can accommodate and weeds out those connections that have been underused (Huttenlocher, 1994; Nelson et al., 2005; Somerville & Jones, 2010).

Adolescence can be understood as a unique time to facilitate brain development that will remain with an individual throughout his or her entire adult life. Such changes in the brain set the stage for behavioral adult patterns (Schulz & Sisk, 2006; Vigil et al., 2011). Schulz & Sisk (2006) stated, "A window of neural and behavioral plasticity may close at the end of the adolescent period." Adolescence can be viewed as a time of both great opportunity and great risk. The adolescent brain is more plastic than the adult brain in response to insult (Sisk & Zehr, 2005). Vulnerability to psychiatric disorders increases during adolescence (Kessler et al., 2005; Giedd, Keshavan, & Paus, 2008). Addictive disorders identified in adults generally have onset in adolescence or young adulthood (Chambers, 2003).

Some of the psychiatric disorders considered the most serious have their roots in adolescent neuromaturation (Walker et al., 2004). The balance between the affective and cognitive regions of the brain is likely influenced by hormonal changes during adolescence (Ernst, Romeo, & Andersen, 2009).

Neuroplasticity that results in more robust subcortical circuits are linked to certain psychiatric conditions (Nelson et al., 2005; Walker et al., 2004; Tekin & Cummings, 2002; Casey et al., 2010; Joels, 2011). Excessive release of neurotransmitters produce structural changes in the prefrontal cortex comparable to those born with ADHD (Brennan & Arnsten, 2008). Mental illnesses that are linked to robust subcortical circuits and deficient cortex circuits include: depression disorders (Jentsch et al., 2000; Bennett, 2010; Zhong et al., 2011), anxiety (Amaral, 2003; Kim & Whalen, 2009), and Attention Deficit Hyperactive Disorder (Brennan & Arnsten, 2008; Rubia et al., 1999; Sonuga-Barke, 2002). Therefore, adolescence can be considered a time when there is a marked opportunity of the brain anatomy and physiology to be shaped, as the architecture of the brain is being established and neuronal connections are vying for importance.

Tsitsika and colleagues (2009) reported that among Greek adolescents, infrequent pornographic internet site users were twice as likely to have abnormal conduct problems as non-pornographic internet site users and frequent users were significantly more likely to have abnormal conduct problems. Negash and colleagues (2016) found that pornography is linked to immediate reward and a higher rate of delay discounting. They also noted that the delay discounting can extend even beyond the temporary state of arousal highlighting the potential long-term impact of SEM through neuroplasticity.

Due to the inherent susceptibility of the adolescent brain to undergo neuroplasticity, it would appear SEM would more easily cause neuroplasticity in an adolescent compared to an adult.

A working model summary

The following is a working model summary contrasting the physiological framework of an adolescent and an adult exposed to the same sexually explicit stimulant (Table 1). It is important to understand that the prefrontal cortex is inherently less authoritative in the adolescent because it is not as fully developed as in the adult. This finds the adolescent brain function more at the mercy of the subcortical circuits that gravitate toward immediate gratification.

Upon exposure to SEM stimulation, activation of the amygdala and the HPA axis are enhanced in the adolescent, compared with the adult. This leads to a more pronounced curtailment of the prefrontal cortex and enhanced activation of the basal ganglia in the adolescent: This condition, therefore, compromises executive function, which includes inhibition and self-control, and enhances impulsivity. Because the adolescent's brain is still developing, it is more conducive to neuroplasticity. The prefrontal cortex going "off-line," so to speak, drives the subtle rewiring that favors subcortical development. If the neuroplasticity imbalance continues over time, this may result in a relatively weakened cortical circuit in favor of a more dominant subcortical circuit, which predisposes the adolescent to continued self-gratification and impulsivity.

The adolescent's nucleus accumbens, or pleasure center of the brain, will have an exaggerated stimulation compared to the adult. The increased levels of dopamine will translate into augmented emotions associated with dopamine, such as pleasure and craving. These emotions drive addictions. In addition, the pubertal surge of testosterone may lead to higher tendencies of aggression and sexual anticipation in the adolescent.

Because of the organizational window of development during adolescence, cortisol and testosterone will have a unique affect upon brain organization or the inherent viability of various neural circuits. This effect will not be found in the adult because this specific window of organization has closed. Chronic exposure to cortisol has the potential, during the adolescent organizational period, to drive neuroplasticity that results in compromised cognitive function and stress resilience even through adulthood. The robustness of the amygdala post puberty, at least in part, depends on the magnitude of testosterone exposure during the critical adolescent developmental window. A robust amygdala is linked to heightened levels of emotionality and compromised self-regulation.

Discussion

The more robust dopamine activation during adolescence signifies pleasure seeking behavior indicative of that age group. In addition, the anatomical propensity for striatal function would indicate that SEM can have a greater emotional and developmental impact on teens. Furthermore, since dopamine plays a central physiological role in the addiction cycle, it is not surprising that teens are highly influenced by SEM, which could lead to addictive behavior. Altogether, these factors regarding dopamine activation provide insight as to why SEM has been able to have such influence and prevalence among teens.

SEM also impacts the release of cortisol, which can lead to a compromise of stress coping abilities in teenagers. Stress has interesting effects on brain function: First, it decreases frontal lobe activity and increases striatal activity, resulting in diminished executive system function and heightened emotional and impulsive function; second, stress triggers the consolidation of memories that are closely tied to emotions, and less to logic. The amygdala plays an important role in this process, and as we have established, has important physiological connectivity with

the hypothalamus. Indeed, the aberrant function of the amygdala with the hippocampus – the primary memory consolidation structure for episodic and semantic memories - has been implicated in stress disorders such as post-traumatic stress disorder and anxiety. SEM in and of itself does not appear to be a stressful stimulus, but if teenagers have an increased release of cortisol when exposed to SEM, then they must perceive such an event to be stressful, and will consolidate the event as an emotional memory. If the emotional memory also triggers the release of dopamine, then it becomes a highly-consolidated memory associated with pleasure. These together could seed the addiction cycle in teens.

Finally, testosterone drives sexual anticipation. SEM causes testosterone release, but adolescents already have inherently augmented levels of testosterone. Sexual arousal also stimulates the release of dopamine. Again, since pleasurable experiences enhance memory consolidation through the amygdala. The long-term consequences of testosterone and the impact on the amygdala affects behavior and self-regulation. We predict that an adult who had high exposure to SEM during adolescence, while the brain was still developing, would exhibit at least an increase subcortical function, and perhaps decreased frontal lobe function. Behavioral results would include diminished self-control, heightened impulsivity, and decreased stress coping abilities. Kuhn and Gallinat (2014) reported on a study of sixty-four healthy male adults with a broad range of pornography consumption. Using 3-T magnetic resonance imaging scans, striatum neural plasticity was observed. These changes reflect intense stimulation of the reward system, although there were not base lines scans for comparison before pornography consumption began.

The prefrontal cortex is innately less developed in teens compared to an adult. This dynamic results in more sensitivity to SEM due to a decreased ability to retain cognitive control in response to stimulating material. The adolescent will be more easily distracted and will have a

harder time switching from potent stimulation to quiet cognitive mode. This could have a negative impact on school performance, self-control and emotional self-regulation, all of which could affect the normal development of inter-personal relationships.

Education concerning neural and hormonal factors during adolescence can be empowering to parents, educators, youth workers, social workers, psychologists, psychiatrists, and university faculty in corresponding fields. Understanding the impact of sexually explicit material on youth can foster a greater synergism to craft public policy that creates environments that are most conducive to optimal neural development. The issue of access and exposure of adolescents to SEM is a discussion that is worthy of our time and is only going to become more pertinent as technology advances.

This review focused on well-controlled cross-sectional experimental studies, and the conclusions that were drawn are based largely on interpolation of the data showing differences between teenage and adult physiological responses to SEM exposure. Unfortunately, a longitudinal study on the effect of SEM exposure in teenagers to adult addiction behavior has not been done. Given the sensitive nature and ethical considerations of and study with experimental conditions, we instead recommend that a retrospective public health or epidemiological study combined with an cross-sectional fMRI study on the effect of SEM exposure between the years 12-18 on young adult (26-35 years old) and middle-aged adult (36-55 years old) be conducted. We predict that adults who experienced significantly more exposure to SEM as teenagers will exhibit increased striatal function, and diminished prefrontal lobe function. In addition, a retrospective study allows for the assessment of attitudes toward sexual activity and its role in relationships. We predict that more exposure to SEM during the formidable teenage years results in a significant difference in the attitudes and perceptions of the role of sexual activity in relationships.

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