The official newsletter of the Pharmacy Association of Nova Scotia

PHARMACY LIFE

Winter 2021

PANS
PHARMACY ASSOCIATION OF NOVA SCOTIA

In this issue:

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Have an interesting story idea or know of a pharmacist or pharmacy technician we should profile, we want to hear about it.

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Message from the Chair and CEO:

By: Diane Harpell, Chair of the Board and Allison Bodnar, CEO

There is no doubt about it. It is been a hard 18+ months. There has been a lot asked of pharmacy - and pharmacy team members have risen to the challenge. And it is taking its toll.

We hear you.

We know that some pharmacy team members feel like they are at a breaking point. We know that some of you are thinking about throwing in the towel and leaving the profession.

It has been hard. Really hard.

We, at PANS have been trying to understand what we can do to support our members during this difficult and tumultuous time.

We have learned that those who work in pharmacies are not unique in their experience - although we also know this will unlikely make any of you feel better.

In what is being called the "Great Resignation of 2021" or the "Big Quit," more than 20 million Americans have quit their jobs since April of this year. In recent research conducted by McKinsey & Company , they found that 40 percent of respondents were likely to leave their employment in the next three to six months. Of those respondents who had quit their job in the past six months, 36 percent did so without having another job to go to. That number rises to 42 percent if the person worked in healthcare or social-assistance.

Some Canadian economists are quick to say that they are not seeing the same wave of resignations in Canada as they are in the United States, but then qualify it with a "not yet." The percentage of adult Canadians who voluntarily left jobs within the past couple of months and didn't immediately resume working at another place of employment is on the rise.

As McKinsey points out, you cannot fix what you don't understand.

At PANS, we are working to understand the root causes of what appears to be growing fatigue and discontent in our profession. Is it as straight-forward as COVID-19 burnout or is there something more systemic within the profession that needs to be addressed? Is there a shift occurring with our society that goes beyond profession?

These are the questions we are asking as we begin our strategic planning for the next five years.

We know a lot has been asked of you, but we need your help as we move forward. Things can only improve if we all work together to address the challenges we face.

Over the past year and half, the spotlight has been on pharmacy. We have impressed upon people what we can do. We also know, we cannot keep working in ways that could potentially impact the health of our pharmacy teams to the point in which they want to leave the profession.

As we continue to work on this issue, PANS

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Message from the Chair and CEO

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staff has compiled some resources to help our members through these difficult times.

There are two webinars available on our website - free of charge for PANS members - that deal with burnout and resiliency. We also have a list of resources on the PANS COVID-19 page (pans.ns.ca/covid-19).

Links to the webinars and online resources can also be found on **page 27** of this issue of Pharmacy Life.

Demands on pharmacy will remain as we continue through this pandemic and beyond. This is because you are a valuable and vital part of Nova Scotia's healthcare system. You can do so much more today than you could ten years ago. This means we cannot work the same as we did ten years ago.

Through our demonstration projects, such as Prescription to Thrive, we are learning how to work smarter instead of harder. There is still room for pharmacies to sign up for the program, and we encourage anyone who would like to know how pharmacy facilitator Glenn Rodrigues can help pharmacy teams implement initiatives to both grow the business and build stronger, healthier and happier teams. We encourage you to reach out to Glenn.

Finally, we want to end with this: THANK YOU.

Pharmacy team members do not hear thank you enough.

The work you are doing is extraordinary and historic. You are doing something that has never been done before. What you are doing is not just providing a vaccine, you are providing hope all while filing in the growing gaps in healthcare in this province.

We know that there are some members of the public who may not see it that way. We know that pharmacy teams members have been on the receiving end of verbal abuse because there is a small minority that do not like decisions that have been made by public health experts to keep us all safe. You don't deserve that. We hear you. We see you. And we continue to work with government on this issue.

We want to share with you that there is a much larger population in this province that is eternally grateful for your efforts to keep us healthy and safe.

At PANS, we are incredibly fortunate to receive emails from the public letting us know just how much pharmacy is appreciated.

Recently, we have been getting a lot of emails from mothers. Such as the email from a mother whose child had a terrible fear of needles and it took three trips to three pharmacies to finally successfully get the COVID-19 vaccine. This mother said every pharmacist they went to was incredibly compassionate and patient and that each one contributed to the success of her daughter getting immunized.

We received another call recently from a mother whose child was prone to fainting and the pharmacist made an arrange with a clinic in the same building so the patient could lie down when they were vaccinated. Knowing this was a possibility, greatly reduced the child's stress and the parent's and they were extremely grateful for this pharmacist's extra effort to make sure they felt safe and supported.

We get emails saying, "I just wanted to email you to let you know how amazing my pharmacy is."

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Sometimes, the negative voices can be louder than the positive ones - but it does not mean they are in the majority. But those negative voices have a huge impact and sometimes can become internalized.

We are so lucky to have amazing pharmacy professionals in this province. We want you to feel safe and encouraged and proud of your profession.

The full PANS team is here to support you. If there is anything that PANS can do to support you, we encourage you to reach out to any member of the PANS team at 902-422-9583 or by email.

For quick reference here are the emails for PANS staff:

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Halifax Pharmacy Aims to Improve Access To Healthcare Services for the 2SLGBTQ+ Community

by: Jenna Lumsden, Communications & Marketing Assistant, PANS

Greg Richard, pharmacist and owner of Boyd's Pharmasave in Halifax, recalls the moment he recognized the need for a safe and inclusive pharmacy spaces for the 2SLGBTQ+ community. After graduating Dalhousie's College of Pharmacy in 2016, Richard worked in a number of community pharmacies in New Brunswick – from small community pharmacies to larger corporate chains.

He says that he was inspired to open a pharmacy that specifically caters to the unique needs of the 2SLGBTQ+ community after seeing first hand the need for improved access to knowledgeable and trained professionals.

"We have seen patients transfer from over 50 km away to use our services, which speaks to the need for affirming-spaces like ours," he says.

Boyd's Pharmasave differs from other pharmacies in the province in the way that it has taken a keen interest in improving access to healthcare specifically for the 2SLGBTQ+ community, and vocalizes their mission to create safer spaces for folks to receive pharmacy services.

"Many patients experience difficulty finding healthcare practitioners trained and comfortable assessing, treating and administering gender-affirming hormones," he says. "I hope to be able to act as a resource for both patients and other healthcare professionals alike."

GENDER AFFIRMATION HORMONE THERAPY

Gender-affirming hormone therapy, such as testosterone and estrogen, is the primary medical intervention sought by transgender and gender-fluid people. It is used to alter hormone levels to help a person match their outward characteristics with their gender identity.

Boyd's Pharmasave is currently the only pharmacy that offers gender-affirming hormone administration in Nova Scotia. It also offers assistance with beginning the transition process, linking patients to other healthcare providers, and strives to familiarize the greater community with gender-neutral terminology and inclusive language.

Boyd's Pharmasave, Agricola Street, Halifax

Although a small community pharmacy with limited staff, Boyd's Pharmasave takes special consideration to reflect the diversity of its community – understanding the importance of creating a space for patients to ask questions that they may feel uncomfortable asking elsewhere.

Richard explains how lack of inclusiveness in healthcare services can create negative impacts on individuals' health outcomes.

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"It's easy to forget how it can be extremely intimidating for some patients to self-identify their gender and/or sexual orientation to their healthcare practitioner," he says. "Not knowing with certainty how accepting their healthcare practitioner will be can create fear and hesitant for the patient, which often prevents them from accessing critical healthcare services."

INCLUSIVE PHARMACY SERVICES IN NOVA SCOTIA

Overall, Richard feels that Nova Scotia pharmacists have done a good job in providing inclusive healthcare services across the province, although he feels more can still be done.

"In my opinion, pharmacists and pharmacy team members should try to identify gaps in the system for their patients, and work to fill these gaps in their own pharmacies," he says. "This may mean taking time to learn about ways to create affirming-spaces, revisiting clinical resources on trans-therapies, or reaching out to other healthcare professionals to collaborate."

According to Richard, every pharmacist he knows would hope that their 2SLGBTQ+ patients would feel comfortable approaching them for any of their unique healthcare-related needs.

"We are all armed with the knowledge, or at minimum, the resources, and the ability to provide safer spaces," he says regarding pharmacists in Nova Scotia. "But we need to work harder to show the community that we are there to provide them with access to equitable healthcare services."

Regardless of the steps taken, Richard insists that the most important thing is to show your patients that you are in their corner no matter what, and that you will approach their care without judgement or bias.

He reiterates the importance of simple changes, such as implementing intake forms that include the option to indicate pronouns and gender identity, and including pronouns on business cards and name tags.

Greg Richard, pharmacist and pharmacy owner

"The most rewarding aspect of all of this is hearing how we have been able to take way a lot of stress surrounding our patients' pharmacy experience and the administration of their hormones," he says.

THE FUTURE OF INCLUSIVE HEALTHCARE FOR 2SLGBTQ+ INDIVIDUALS

Richard says he hopes to continue providing unique services that disproportionately affect the 2SLGBTQ+ community, such as PrEP prescribing and HIV point-of-care testing. Although Nova Scotia has made great strides in improving equal healthcare access to all individuals, there is still a long way to go.

"If I could relay one message to the 2SLGBTQ+ community, it would be that we recognize the challenges you may have experienced getting to this moment in your life, and we are committed to ensuring that accessing healthcare services, and more specifically pharmacy services, is as challenge-free as possible going forward."

ONTARIO PHARMACISTS ASSOCIATION
Advocating Excellence in Practice and Care

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Therapeutic Options

FOCUS ON: ADHD IN CHILDREN AND ADOLESCENTS

By Tiffany Barker, BSc, BScPhm, RPh

INTRODUCTION AND EPIDEMIOLOGY

Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by symptoms of inattention, hyperactivity, and/or impulsivity.[1,2] It has worldwide prevalence and is the most common pediatric neuropsychiatric condition, affecting 4-12% of school-aged children in North America.[1,2] Boys are more commonly affected by ADHD than girls, with male to female ratios being 4:1 and 2:1 for the predominantly hyperactive and inattentive subtypes, respectively.[3] Although it may be perceived that ADHD is over-diagnosed, the prevalence rates of ADHD have remained stable over the last 30 years.[1]

ETIOLOGY AND PATHOPHYSIOLOGY

The etiology of ADHD remains uncertain but may include genetic, neurological, and environmental factors.[1,4] The genetic etiologies of ADHD are complex; several genes have been associated with this disorder and it has been found to be highly heritable (76% heritability in twin studies).[1] Neuroimaging studies in children with ADHD have shown structural and functional abnormalities in various areas of the brain.[3] Other potential etiological influences include prenatal exposure to tobacco smoke or alcohol, prematurity, and low birth weight; however, the significance of these factors is uncertain.[1,3] Although the pathogenesis of ADHD has not been conclusively determined, an imbalance of catecholamine (dopamine and norepinephrine) metabolism in the cerebral cortex appears to play a key role.[3]

SIGNS AND SYMPTOMS

Three core symptoms characterize ADHD – inattention, hyperactivity, and impulsivity.[2] Symptoms of inattention may include difficulty maintaining focus in school, home, or play activities; seeming not to listen (even when spoken to directly); not following instructions; being easily distracted by extraneous stimuli; forgetfulness in daily activities such as homework and chores; difficulty organizing tasks; and losing items necessary for activities.[1,5] Hyperactivity and impulsivity symptoms may include excessive fidgeting; difficulty remaining seated when necessary; excessive talking; interrupting others; difficulty awaiting turns; and appearing to always be "on the go".[1,5] In adolescents, these symptoms may also include feelings of restlessness, substance use, risky sexual behaviour, or impaired driving.[5] Core symptoms appear to a greater extent than would be expected for the child's age/developmental stage; often interfere with normal development; and adversely impact academic, emotional, or social functioning.[2,3] An ADHD diagnosis is based on the presence of six* or more symptoms of the ADHD symptom criteria (as defined by the *Diagnostic and Statistical Manual of Mental Disorders*, Fifth Edition [DSM-5]).[1,2] Symptoms must be present for at least six months, occur in more than one setting (e.g., home and school), and lack alternate causes.[1,2,5] Diagnostic subtypes include inattentive, hyperactive-impulsive, and combined categories, with the latter presentation being the most common.[1,2,5] It has been suggested that ADHD may be diagnosed in children as young as four years of age.[1,5] ADHD is often a lifelong condition; symptoms usually appear before 12 years of age and continue to have a significant impact into adulthood in greater than 50% of diagnosed children and adolescents.[1,2] Over time, the impact and presentation of ADHD can change, necessitating regular re-evaluation of ADHD symptoms and their effect on functioning.[1]

COMORBID DISORDERS

Comorbid psychiatric disorders, including anxiety, depression, oppositional defiant disorder, tic disorders, autism spectrum disorder, and learning disabilities are common, with one major study citing occurrence in 70% of school-aged children with ADHD.[1,3] For example, anxiety affects 11-30%, and learning disabilities affect more than 31% of children and adolescents with ADHD.[1] Comorbid disorders may require concomitant or prioritized treatment, depending on severity (information regarding treatment of comorbidities is beyond the scope of this article).[1,3]

TREATMENT

A multimodal treatment strategy, using both nonpharmacologic and pharmacologic interventions, is the recommended approach for the management of ADHD in children six years of age and older and adolescents.[1,2,6] Therapies should be individualized and will generally be required throughout a patient's life.[1]

Goals of therapy include[1,2]:

- Significantly decreasing core ADHD symptoms
- Increasing academic, emotional, behavioural, and social functioning
- Enhancing self-esteem
- Improving overall quality of life

Nonpharmacologic Therapy

Integrating psychosocial interventions into the ADHD treatment approach is recommended.[2] A variety of options exist and include behavioural interventions, parent training, social skills training, and cognitive behavioural therapy.[1,2,6] Non-pharmacologic treatment (e.g.,

* Six or more symptoms are required for diagnosis in children and adolescents <17 years of age; five or more symptoms are required for older adolescents and adults aged ≥17 years.[1,2]

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parent-administered behavioural therapy) alone is recommended as first-line therapy in children under six years of age; behavioural therapy is more effective and has more lasting benefits than psychostimulants in this age range.[1,6,7] A large RCT in older children (7-9 years old), however, found that the use of behavioural therapy alone was inferior to pharmacologic therapy alone in reducing core symptoms of ADHD and that multimodal treatment was more effective than either therapy alone for other outcomes (e.g., decreasing anxiety, improving self-esteem and social interactions).[2]

Pharmacologic Therapy

Medication is recommended as part of the multimodal treatment approach in children and adolescents who have impairments in learning or academic performance or in behavioural and social functioning due to core ADHD symptoms.[2,8] Several factors should be considered when selecting a medication to treat ADHD, including the patient's age and ability to adhere to medication; the duration of medication effect required by the patient's symptoms; mode of delivery of the various pharmaceutical formulations; concomitant psychiatric or medical comorbidities; drug interactions; and affordability/reimbursement.[1,9] A patient's symptom profile and/or family history cannot predict which medication will be most effective; ADHD medications can reduce both inattention and impulsive/hyperactive symptoms and response tends to be individual.[1] Pharmacologic treatment may be used in children four and five years of age who do not adequately respond to behavioural therapy, following an assessment of the risks and benefits, and should be done under the care of a specialist (discussion of pharmacotherapy in children under six years of age is beyond the scope of this article).[1,2,6]

Comprehensive information regarding starting doses, titration schedules, and maximum daily doses of ADHD medications can be found in the Canadian ADHD Resource Alliance (CADDRA) Practice Guidelines[1], product monographs, and other references below. Generally, the dose is slowly increased until 1) treatment goals have been achieved; or 2) adverse effects prevent further dose increases; or 3) the maximum recommended dose is reached.[1] The aim is to reach a dose at which treatment goals are achieved with minimal adverse effects.[1,9]

First-Line Therapy

Long-acting psychostimulants (methylphenidate and amphetamines) are first-line medications in the treatment of ADHD.[1] Their exact mechanism of action is unknown, but they are thought to increase the levels of norepinephrine and dopamine at the central nervous system synaptic clefts by blocking reuptake and/or increasing release of these catecholamines.[1,10] These first-line medications have the most favourable risk-benefit profile and greatest degree of effectiveness (as measured by effect size).[1] The efficacy of both classes of long-acting psychostimulants is generally similar and the choice of class is determined by physician and patient preferences.[1,2] Psychostimulants have a quick onset of action with a reduction in core symptoms typically seen within the first week of therapy (their effects are usually stable at a given dose within 1 to 3 weeks).[1,2,11] However, individual patients may have a better response to one class versus the other. Before moving to second-line agents, an adequate trial of therapy of each drug class is recommended.[1]

Long-acting formulations of psychostimulants are advantageous since they reduce the need for multiple dosages thereby improving medication adherence and avoiding the need for medication administration at school.[1,2] The longer duration of effect enhances symptom coverage, decreases the risk of rebound hyperactivity, and may improve tolerability.[1,2] Psychostimulants are controlled drugs and have the potential for misuse and diversion; long-acting preparations may reduce the risk of diversion compared to immediate-release products.[1,2]

Long-acting psychostimulants are available as different formulations with varying delivery systems, medication release properties, durations of effect, and administration options (Table 1). Considerations for selecting a long-acting psychostimulant product should include the duration of medication effect necessary to treat the patient's symptoms; the patient's ability to swallow pills; affordability/reimbursement; and the potential for abuse, misuse, and diversion.[1]

Second-Line Therapy

Second-line agents for the treatment of ADHD comprise short- and intermediate-acting psychostimulants and the non-stimulant medications atomoxetine (a selective norepinephrine reuptake inhibitor) and guanfacine XR (a selective alpha$_{2A}$-adrenergic receptor agonist).[1,12,13] The therapeutic effect of atomoxetine is believed to be associated with its potent inhibition of the presynaptic norepinephrine transporter.[12] Guanfacine stimulates alpha$_{2A}$-adrenergic receptors in the brain, although its exact mechanism of action in the treatment of ADHD is not well understood.[13,14] Non-stimulant agents are considered second-line due to lower response rates observed clinically and in studies when compared to psychostimulants.[1] Both atomoxetine and guanfacine are not controlled drugs and have no known abuse potential.[1,2]

Second-line medications may be used when a patient experiences suboptimal response or significant adverse effects with first-line options or when a patient lacks access to long-acting psychostimulants.[1] Alternatively, use of short- and intermediate-acting psychostimulants can provide flexible dosing when medication coverage is required for only minimal hours of the day; these medications may also be used as add-on therapy to extend the effects of long-acting psychostimulants.[1,2]

Atomoxetine and guanfacine XR have slower initial onset of action than psychostimulants (1 to 4 weeks and 1 to 2 weeks, respectively), and maximum response may not be achieved until 6 to 12 weeks and 4 weeks, respectively.[1,10,11] These non-stimulants may be used as monotherapy or as adjunctive therapy to long-acting psychostimulants for patients who have suboptimal response to first-line medications.[1] However, the use of atomoxetine as adjunctive therapy is off-label and should be reserved for complex resistant cases.[1] Atomoxetine or guanfacine XR may also be prescribed when a contraindication to psychostimulant use exists (e.g., high risk of stimulant misuse).[1]

Third-Line Therapy

Bupropion, clonidine, and tricyclic antidepressants (desipramine, imipramine, nortriptyline) are examples of third-line (and off-label) medications for the treatment of ADHD in children and adolescents.[1,2,10,15,16] Medications in this category are less effective and/or carry greater risk of adverse effects than first- and second-line agents.[1,2] These medications are generally reserved for cases of treatment resistance, unacceptable side effects, or significant comorbidities and referral to an ADHD specialist is recommended when the use of third-line options is considered.[1,2,10]

GENERIC FORMULATIONS

Health Canada deems a generic product

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to be bioequivalent to a brand name product when it produces a similar maximum concentration (Cmax) and area-under-the-curve (AUC), however the time to maximum concentration (Tmax) may be better related to the duration of effect than Cmax or AUC. The Canadian ADHD Practice Guidelines' committee notes that some patients report reduced effect from medications when switched from a brand name to generic product and recommends that the decision to switch be made individually, involving the patient/family. Follow-up should occur, monitoring for changes in effectiveness or adverse effects. Therefore, this committee considers generic long-acting psychostimulant products to be second-line agents.[1]

ADVERSE EFFECTS AND MONITORING

In general, psychostimulants are well-tolerated and considered safe in treating children and adolescents with ADHD.[1] Both classes of long-acting psychostimulants have similar tolerability, however, a switch from one class to the other may improve tolerability in some patients.[1] Many psychostimulant adverse effects are mild, short-term, and reversible with dose adjustments.[1,10]

Adverse effects of psychostimulants may include appetite suppression, weight loss, and poor growth.[2,10] Three-year follow-up in a large RCT showed that children receiving psychostimulants were an average of 2.7 kg less in weight and 2 cm shorter in height when compared with children who did not receive medication during the same time.[2] Other studies have found final adult height to be minimally affected in most children and youth receiving psychostimulant therapy for ADHD.[8,10] Height and weight should be measured prior to initiating pharmacotherapy and continually monitored.[1,2] Strategies for managing appetite suppression include providing maximal nutrition when appetite is least suppressed (e.g., mornings prior to stimulant dose, evenings); reducing portions and increasing snack times; considering nutritional supplements; or considering a dose reduction, alternate agent, or drug holiday.[1,2] Patients taking atomoxetine should also be monitored for appetite suppression and weight loss.[10]

Psychostimulants and atomoxetine may cause increased heart rate and blood pressure whereas guanfacine XR may decrease heart rate and blood pressure; these vital signs should be measured prior to initiating therapy and regularly monitored.[1,2] In young patients with normal physical examination and no personal or family history of heart disease, routine electrocardiogram (ECG) monitoring at baseline or follow-up is not recommended.[1,8] Cardiology consultation should be considered in patients with confirmed or suspected heart disease or in the event of significant changes in heart rate, blood pressure, or ECG.[1,2] Adherence to alpha$_2$-adrenergic agonists (guanfacine, clonidine) is essential since abrupt discontinuation creates risk of hypertensive crisis (discontinuation of these medications requires tapering).[1,2]

Suicidal thinking and psychosis have been reported rarely with psychostimulant and atomoxetine therapy.[2,10] Suicide-related events have been reported at various times throughout therapy, however, they are of greatest concern at the beginning or end of treatment or upon dose adjustment.[2] Psychostimulants are contraindicated in patients with a history of mania or psychosis.[1] Patients on any type of ADHD medication should be monitored for changes in mood or thought patterns, anxiety, irritability, and changes in behaviour or sleep.[1]

Psychostimulants have been associated with new onset of tics.[10] ADHD often coexists in patients with tic disorders and although psychostimulants may exacerbate tics for some of these patients, these medications can be safely used in most.[1,2] Recent studies suggest that worsening of tics with initiation of psychostimulants is often coincidental and related to the waxing and waning pattern of tics.[1] Atomoxetine rarely exacerbates tics and may be considered as an option in patients who experience worsening of tics with psychostimulants.[1,10]

Concerns may exist regarding substance abuse potential in patients receiving psychostimulant therapy.[2] ADHD itself is a risk factor for substance use disorder.[2] Findings of research regarding the effects of pharmacologic treatment for ADHD on substance use risk in adolescence or adulthood vary. Some studies have found that treatment with psychostimulants in childhood may be associated with decreased risk of future substance-related problems whereas other studies have shown that psychostimulant therapy neither increases nor decreases this risk.[1,2,6] Psychostimulant medications can be diverted or misused; short-acting formulations have greater risk of this due to their pharmacokinetic profile and easy crushability.[1] Patients should be counselled on the harms of psychostimulant misuse and diversion.[2]

For more comprehensive information regarding contraindications/precautions for ADHD medications and their adverse effects, refer to the CADDRA Practice Guidelines[1] and other references below.

ALTERNATIVE OPTIONS

Interest in a variety of alternative therapies for treatment of ADHD may be expressed by patients or families. Omega-3 fatty acids have been researched as an alternative to pharmacological therapy but are not recommended as a replacement for standard treatment in patients with significant symptoms of ADHD; however, literature suggests that adjunctive omega-3 supplementation may be potentially useful. Dietary interventions that have been studied as alternatives to standard ADHD treatment include elimination diets (e.g., eliminating artificial food colourants, eliminating sugar) or dietary supplementation (e.g., with amino acids, essential fatty acids, vitamins, minerals); insufficient evidence exists to recommend either of these as alternatives to usual therapy.[1]

DRUG HOLIDAYS

ADHD is often a chronic condition and temporarily discontinuing ADHD medication ("drug holiday") is not routinely recommended as symptoms and functional impairment can return.[1,2,9] Decisions to use drug holidays should be made on an individual basis and involve careful monitoring of core ADHD symptoms and functioning.[2,9] Drug holidays may be beneficial when trying to assess the continued effectiveness of ADHD medication or in the event of adverse effects such as growth suppression or weight loss.[2] The period of discontinuation should be timed to avoid interference with school or work (e.g., during summer vacation).[1,2,9] Psychostimulant medications may require tapering as abrupt discontinuation can cause withdrawal symptoms in some patients (e.g., those who have received prolonged psychostimulant therapy and/or high doses).[1,2] To be of clinical benefit, non-stimulant medications must be taken continuously.[1] Drug holidays may not be feasible for atomoxetine and guanfacine XR due to their prolonged effects and longer onset of action.[9] Withdrawal is less likely with atomoxetine, which may be discontinued without tapering.[1,2,9,12] When discontinuing alpha$_2$-adrenergic agonists (guanfacine, clonidine), these medications must be gradually tapered due to a significant risk of hypertensive crisis with abrupt discontinuation.[1,2]

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Table 1. ADHD Medications for Children (6–12 years) and Adolescents (13–17 years)[1,12,13,17-25]

	Brand Name (Dosage Form)	Active Ingredient	Delivery System	Immediate / Delayed Release Ratio*	Duration of Effect^ (hours)
FIRST-LINE	**Long-acting psychostimulants**				
	Adderall XR®# (Extended release capsuleα)	amphetamine mixed salts (predominantly dextro-amphetamine)	Two types of medication-containing beadsΔ	50/50	12
	Biphentin® (Controlled release capsuleα)	methylphenidate	Multilayer beads+	40/60	10-12
	Concerta®# (Extended release tabletβ)	methylphenidate	Oros® technologyΩ	22/78	~12
	Foquest® (Controlled release capsuleα)	methylphenidate	Multilayer beads+	20/80	16
	Vyvanse® (Capsule and chewable tablet‡)	lisdexamfetamine (pro-drug of dextro-amphetamine)	Prodrugμ	Gradual delivery (prodrug)	13
SECOND-LINE / ADJUNCTIVE	**Short-acting & intermediate-acting psychostimulants¢**				
	Dexedrine®# (Tablet)	dextro-amphetamine	Immediate	100/0	4
	Dexedrine® Spansule®# (Sustained release capsuleβ)	dextro-amphetamine	Intermediate; beaded formulation	50/50	6-8
	Ritalin®# (Tablet)	methylphenidate	Immediate	100/0	3-4
	Ritalin® SR# (Extended release tabletβ)	methylphenidate	Intermediate; wax matrix	100/0; may be inconsistent	5-6
	Selective norepinephrine reuptake inhibitor¢				
	Strattera®# (CapsuleβY)	atomoxetine	n/a	n/a	Up to 24 hours§
	Selective alpha$_{2A}$-adrenergic receptor agonist¢				
	Intuniv XR® (Extended release tabletβ)	guanfacine	n/a	n/a	Up to 24 hours§

n/a = not applicable
* Certain psychostimulant products are formulated to deliver a proportion of medication immediately with the remainder being delivered using a delayed release delivery system.
^ Can vary from individual to individual.
# Generic available.
α Should be swallowed whole but contents may be sprinkled on soft food and should not be chewed or crushed. See product monograph for full administration instructions (e.g., type of soft food).
Δ Both immediate- and delayed-release beads.
\+ Both immediate- and delayed-release medication-containing layers.
β Must be swallowed whole.
Ω Outer layer releases a portion of the medication immediately and the remaining medication is gradually released from the osmotically-active trilayer tablet core (generic delivery system differs).
‡ Capsule may be swallowed whole or contents may be mixed with yogurt, water, or orange juice until completely dispersed. Chewable tablet must be chewed thoroughly before swallowing. See product monograph for full administration instructions.
μ Lisdexamfetamine (inactive) requires enzymatic transformation in the gut and blood system to release dextro-amphetamine (active).
¢ Short- and intermediate-acting dextro-amphetamine products may be used to augment Adderall XR® or Vyvanse®; short-acting methylphenidate products may be used to augment Biphentin® or Concerta®. Intuniv XR® and atomoxetine may also be used as adjunctive therapy to long-acting psychostimulants (this use of atomoxetine is off-label). Second-line medications may also be used as monotherapy.
¥ Capsule contents can cause nausea/stomach upset and are an ocular irritant.
§ Continuous coverage may be provided, including late evening and early morning periods.

REFERENCES

1. CADDRA - Canadian ADHD Resource Alliance: Canadian ADHD Practice Guidelines, 4.1 Edition, Toronto ON; CADDRA, 2020 [Accessed 2020 July 2]. https://www.caddra.ca/download-guidelines/
2. Virani A. Attention-Deficient Hyperactivity Disorder. In: Compendium of Therapeutic Choices. Ottawa, ON: CPhA. Date of Revision: 2019 July 25 [Accessed 2020 July 2]. https://myrxtx.ca
3. Krull K. Attention deficit hyperactivity disorder in children and adolescents: Epidemiology and pathogenesis. Augustyn M, Torchia M (eds). UpToDate. Waltham, MA: 2019 [Accessed 2020 July 5]. www.uptodate.com
4. Bélanger SA et al; Canadian Paediatric Society, Mental Health and Developmental Disabilities Committee. ADHD in children and youth: Part 1–Etiology, diagnosis, and comorbidity. Paediatr Child Health 2018, 23(7):447–453.
5. Krull K. Attention deficit hyperactivity disorder in children and adolescents: Clinical features and diagnosis. Augustyn M, Torchia M (eds). UpToDate. Waltham, MA: 2019 [Accessed 2020 July 2]. www.uptodate.com
6. Krull K. Attention deficit hyperactivity disorder in children and adolescents: Overview of treatment and prognosis. Augustyn M, Torchia M (eds). UpToDate. Waltham, MA: 2019 [Accessed 2020 July 2]. www.uptodate.com
7. Canadian Paediatric Society. Five Things Physicians and Patients Should Question. Choosing Wisely Canada: Paediatrics. Last Updated: 2019 July [Accessed 2020 August 23]. https://choosingwiselycanada.org/paediatrics/
8. Feldman ME et al; Canadian Paediatric Society, Mental Health and Developmental Disabilities Committee. ADHD in children and youth: Part 2–Treatment. Paediatr Child Health 2018, 23(7):462–472.
9. Krull K. Attention deficit hyperactivity disorder in children and adolescents: Treatment with medications. Augustyn M, Torchia M (eds). UpToDate. Waltham, MA: 2020 [Accessed 2020 July 2]. www.uptodate.com
10. Krull K. Pharmacology of drugs used to treat attention deficit hyperactivity disorder in children and adolescents. Augustyn M, Torchia M (eds). UpToDate. Waltham, MA: 2020 [Accessed 2020 July 2]. www.uptodate.com
11. Clinical Resource #340530. Comparison of ADHD Medications. Pharmacist's Letter/Prescriber's Letter. 2018 May [Accessed 2020 August 3]. https://ca-pharmacist.therapeuticresearch.com/
12. Eli Lilly Canada Inc. Strattera® Product Monograph. Toronto, ON. Date of Revision: 2015 October 1. Submission Control No: 184870.
13. Shire Pharma Canada ULC. Intuniv XR® Product Monograph. Toronto, ON. Date of Revision: 2019 January 23. Submission Control No: 221634.
14. PL Detail-Document #310409, Management of ADHD: When a Stimulant is Not Enough. Pharmacist's Letter/Prescriber's Letter. 2015 April [Accessed 2020 August 3]. https://ca-pharmacist.therapeuticresearch.com/
15. AA Pharma Inc. Desipramine Product Monograph. Vaughan, ON. Date of Preparation: 2012 September 19. Control No: 158145.
16. AA Pharma Inc. Aventyl® Product Monograph. Vaughan, ON. Date of Preparation: 2014 April 29. Control No: 173243.
17. CADDRA - Canadian ADHD Resource Alliance: CADDRA Guide to ADHD Pharmacological Treatments in Canada; CADDRA, 2020 February [Accessed 2020 July 8]. https://www.caddra.ca/resources/medication-chart/
18. Shire Pharma Canada ULC. Adderall XR® Product Monograph. Toronto, ON. Date of Revision: 2017 June 30. Submission Control No: 185297.
19. Purdue Pharma. Biphentin® Product Monograph. Pickering, ON. Date of Revision: 2020 January 14. Submission Control No: 224342.
20. Janssen Inc. Concerta® Product Monograph. Toronto, ON. Date of Revision: 2019 April 17. Submission Control No: 224296.
21. Purdue Pharma. Foquest® Product Monograph. Pickering, ON. Date of Revision: 2019 March 1. Submission Control No: 214860.
22. Shire Pharma Canada ULC. Vyvanse® Product Monograph. Toronto, ON. Date of Revision: 2019 July 5. Submission Control No: 220806.
23. Paladin Labs Inc. Dexedrine® / Dexedrine® Spansule® Product Monograph. St-Laurent, QC. Date of Revision: 2016 March 21. Submission Control No: 183401.
24. Novartis Pharmaceuticals Canada Inc. Ritalin® / Ritalin® SR Product Monograph. Dorval, QC. Date of Revision: 2020 June 19. Control No: 237335.
25. Health Canada. Drug Product Database. [Internet database; cited 2020 August 24]. Available from: https://health-products.canada.ca/dpd-bdpp/index-eng.jsp

Reviewed by Joanne Deshpande, BScPhm, RPh and Manal Rostom, PharmD, RPh, ACPR

Ask a Drug Information Pharmacist

Pregnancy and Breastfeeding During the COVID-19 Pandemic

JOANNE DESHPANDE, BSC PHM R.PH

The COVID-19 pandemic has created an environment that is difficult for any patient to navigate, but more so for those with chronic illnesses or medical concerns including pregnancy and breastfeeding. What is the impact of the COVID-19 virus on pregnancy? Do these patients need to take additional precautions to protect themselves and their babies? Is it safe for women who have tested positive for COVID-19 to breastfeed? These are valid concerns that may be addressed by pharmacists.

Does pregnancy put women at higher risk of acquiring the COVID-19 virus?

The transmission, signs and symptoms and, most importantly, prevention of COVID-19 infection, are no different in a healthy pregnant woman than in the general population. The same steps of prevention (e.g., frequent hand washing, avoidance of touching the face, physical distancing and use of personal protective equipment (PPE) where appropriate) should be followed both at home and in the workplace. Pregnant women who are healthcare workers or employed in essential services can continue to work as long as the appropriate protective measures are followed; no additional protection is needed because of pregnancy. In situations where work-related exposure to COVID-19 is considered to be substantial, accommodations or absence from work may be a consideration for pregnant employees. Pregnant women with concomitant medical conditions such as cardiac disease, hypertension, or pulmonary disease may need to take extra precautions due to their comorbidities, rather than the pregnancy.[1,2]

If a pregnant woman acquires the COVID-19 virus, will she develop more serious complications?

Due to physiological changes that take place in pregnancy, lower respiratory tract infections, such as pneumonia, have typically been associated with higher rates of hospitalization and admission into intensive care units in this population. During the previous global outbreaks of other highly pathogenic coronaviruses, SARS and MERS, a small number of cases in pregnancy did involve severe morbidity, as well as maternal mortality. Although case reports of spontaneous abortion, stillbirth, intrauterine growth restriction and preterm birth were reported during these outbreaks, there were also a number of pregnancies with positive outcomes despite maternal infection. The Society of Obstetricians and Gynaecologists of Canada (SOGC) felt that the adverse pregnancy outcomes were most likely attributed to the severity of maternal respiratory compromise.[3] Over 500 cases of COVID-19 infection in pregnancy have been confirmed, with the majority of cases being mild to moderate in severity and the rate of critical illness comparable to non-pregnant women of similar ages. Although limited data exists, these case reports reinforce that pregnant women are not at higher risk of infection nor of severe morbidity, compared to non-pregnant women of the same age.[1]

The majority of babies born to women infected with COVID-19 have been healthy with near-term prematurity being the most common adverse outcome.[1,3] Early data suggested that preterm births occurred in as many as 30% of pregnant women infected with COVID-19; however, recent reports indicate lower figures of 6-15%.[1] There has been little evidence to suggest a vertical (mother-to-infant) transmission of the virus; however, in a cohort study done in Wuhan, China, 3 of 33 neonates born to COVID-19 positive mothers presented with early-onset COVID-19 infection. Samples of amniotic fluid, cord blood and breast milk were all negative for the virus; however, a vertical transmission could not be ruled out. The 3 neonates with COVID-19 infection had mild symptoms and all recovered.[4] To date, there has also been no evidence of COVID-19 causing birth defects; however, due to the limited number of reported cases of infection during the first trimester when embryogenesis occurs, risk of congenital anomaly due to COVID-19 cannot be fully excluded.[3,5]

How will a COVID-19 infection affect pregnancy?

If a pregnant woman becomes infected with the COVID-19 virus, she should follow the same self-isolation precautions as a non-pregnant woman would. More frequent prenatal follow-up may be suggested depending on the symptoms of the woman and the stage of pregnancy, however, a diagnosis of COVID-19 is not an indication for delivery. Since maternal infection with COVID-19 may necessitate greater monitoring of both mother and baby, a hospital delivery is recommended.[5]

Can a COVID-19 infected woman still breastfeed?

There has been no evidence of virus transmission into breast milk. A mother with confirmed or suspected COVID-19 infection can continue to breastfeed her baby but she should follow precautions of washing her hands and wearing a face mask before breastfeeding. If using a breast pump, hand washing before use and thorough pump cleaning after use is recommended. If possible, it is recommended that someone who is not COVID-19-infected feed the expressed milk to the baby.[6]

For up-to-date information on COVID-19 and pregnancy or breastfeeding:

SOGC: https://www.sogc.org/en/-COVID-19/en/content/COVID-19/COVID-19.aspx?hkey=4e808c0d-555f-4714-8a4a-348b547dc268

CDC: https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/pregnancy-breastfeeding.html

References

1. The Society of Obstetricians and Gynecologists of Canada [SOGC]. SOGC Statement on Pregnant Workers during the COVID-19 Pandemic. 2020 May 21 [Internet]. Ottawa, ON [cited 2020 July 8]. Available from: https://sogc.org/en/content/featured-news/SOGC-Statement-on-Pregnant-Workers-during-the-COVID-19-Pandemic.aspx
2. The Society of Obstetricians and Gynecologists of Canada [SOGC]. Revised SOGC Infectious Disease Committee Statement on Health Care Workers during the COVID-19 Pandemic. 2020 March 27 [Internet]. Ottawa, ON [cited 2020 July 8]. Available from: https://www.sogc.org/en/content/featured-news/SOGC-Infectious-Disease-Committee-Statement-on-Health-Care-Workers-during-COVID19Pandemic.aspx
3. The Society of Obstetricians and Gynecologists of Canada [SOGC]. Updated SOGC Committee Opinion - COVID-19 in Pregnancy. 2020 March 13 [Internet]. Ottawa, ON [cited 2020 July 8]. Available from: https://www.sogc.org/en/content/featured-news/Updated-SOGC-Committee-Opinion__COVID-19-in-Pregnancy.aspx
4. Lingkong A, Xia S, Wenhao Y et al. Neonatal early-onset infection with SARS-CoV-2 in 33 neonates born to mothers with COVID-19 in Wuhan, China. JAMA-Pedatrics. (2020 March 26). Available from: http://doi:10.1001/jamapediatrics.2020.0878
5. Aggarwal V, Essalah A, Arwini B et al. COVID-19 in Pregnancy. OBGYN Academy. Revised 2020 May 4 [Internet, cited 2020 July 8]. Available from: https://obgynacademy.com/covid-19
6. Centers for Disease Control and Prevention. Coronavirus Disease 2019 (COVID-19): Pregnancy & Breastfeeding. Last reviewed 2020 June 25 [Internet, cited 2020 July 8]. Available from: https://www.cdc.gov/coronavirus/2019-ncov/need-extra-precautions/pregnancy-breastfeeding.html

Ask a Drug Information Pharmacist

Question: How can statin-associated muscle symptoms be identified and managed?

BY MANAL ROSTOM, PharmD, ACPR, RPh

Statins [3-hydroxy-3-methylglutaryl coenzyme A reductase (HMG-CoA) inhibitors] are considered the mainstay of treatment for hypercholesterolemia, and play a key role in preventing and treating cardiovascular disease (CVD) by reducing morbidity and mortality.[1,2] Statin therapy is associated with many benefits, including lowering of low-density lipoprotein cholesterol, stabilizing atherosclerotic plaques as well as exhibiting antioxidative properties and anti-inflammatory effects.[2]

Muscle-related adverse events (AEs) are the most commonly observed side effects from statin therapy.[2] The term statin-associated muscle symptoms (SAMS) has been used to describe these side effects and includes the following syndromes:*[1,3,4]

- **Myopathy:** general term for muscle disease; often used to describe unexplained muscle pain or weakness
- **Myalgia:** muscle ache/weakness with no elevation in creatine kinase (CK) [CK ≤ the upper limit of normal (ULN)]
- **Myositis:** muscle inflammation causing pain/weakness with elevated CK [CK > ULN]
- **Rhabdomyolysis:** muscle ache/weakness accompanied with renal dysfunction and/or myoglobulinuria with elevated CK [CK > 10 times ULN or CK > 10,000 U/L]

* The definitions provided are as outlined by the Canadian Consensus Working Group and may differ from other international guidelines.[3]

SAMS is characterized by bilateral and symmetrical myopathy that is always confined to skeletal muscle.[5] SAMS typically impacts large muscle groups, including the arms, shoulders, back, legs and pelvic girdle. Patients may describe their symptoms as cramps, muscle tenderness or weakness, stiffness, fatigue or heaviness while exercising.[1,4] Mild to moderate muscle pain or weakness may occur without a significant increase in CK levels, however if muscle pain is unbearable, patients should be advised to have their serum CK levels checked immediately.[1]

SAMS can occur with the use of any statin and the risk is highest within the first year of therapy, after a dose increase, or with the addition of a potentially interacting drug. In clinical trials, the reported incidence of SAMS is typically 1-5%, while observational studies have reported higher levels of up to 29%. This difference in prevalence is likely due to the exclusion of patients from trials who have underlying risk factors for experiencing these AEs.[1] Risk factors for SAMS can be divided into two categories:[2,3,4,6]

- **Non-modifiable risk factors:** age >75 years, female gender, low BMI, frailty, Asian ethnicity, family history of SAMS, specific genetic polymorphisms, severe renal disease, acute/decompensated liver disease, diabetes mellitus, underlying muscle disease, previous CK elevation.
- **Modifiable risk factors:** high statin dose, interactions with CYP3A4 inhibitors/substrates, interactions with CYP2C9 inhibitors/substrates, interactions with OATPIB1 (organic anion transporting peptide 1B1) inhibitors (e.g. gemfibrozil, cyclosporine, letermovir), untreated hypothyroidism, vigorous physical activity, alcohol/drug abuse, vitamin D deficiency, high grapefruit or cranberry intake

It is recommended that prior to initiation of statin therapy, prescribers and pharmacists screen for potential drug interactions, and select a statin accordingly to minimize the risk of SAMS. Screening for any underlying muscle disease or muscle pain/weakness, and clearly documenting this prior to initiation of the statin can aid in differentiating between true SAMS or myopathy unrelated to statin use. Finally, checking the patient's renal function and ensuring appropriate dosing of the statin can also prevent the occurrence of SAMS.[4]

According to a systematic review, statin discontinuation in high-risk patients was associated with a 67% increased risk of acute myocardial infarction.[1] Therefore, safely restarting and maintaining statin therapy should be prioritized, particularly in high-risk patients with CVD, as it reduces the risk of major vascular events and mortality.[5,6] Recent evidence demonstrates that the majority of patients who experience SAMS can safely tolerate therapy with a different statin or with the same statin at a lower dose.[2,4,6] For patients who continue to experience SAMS, preliminary studies suggest that taking rosuvastatin once or twice weekly may be efficacious and safe as a last resort.[6]

References

1. Alonso R, Cuevas A, Cafferata A. Diagnosis and Management of Statin Intolerance. J Atheroscler Thromb, 2019; 26: 207-215.
2. Algharably EA, Filler I, Rosenfeld S,et al. Statin Intolerance – a question of definition. Expert Opin Drug Saf. 2017 Jan;16(1):55-63.
3. Mancini GB, Baker S, Bergeron J, et al. Diagnosis, Prevention, and Management of Statin Adverse Effects and Intolerance: Canadian Consensus Working Group Update (2016). Can J Cardiol. 2016 Jul;32(7 Suppl):S35-65.
4. Cupp, M. Statin Muscle Symptoms: Managing Statin Intolerance. Pharmacist's Letter/Prescriber's Letter. June 2020.
5. Newman CB, Preiss D, Tobert JA, et al. Statin Safety and Associated Adverse Events - A Scientific Statement from the American Heart Association. Arterioscler Thromb Vasc Biol. 2019;39:e38-e81.
6. Li JJ, Liu HH, Wu NQ, et al. Statin intolerance: an updated, narrative review mainly focusing on muscle adverse effects. Expert Opin Drug Metab Toxicol. 2020. PMID: 32729743 Review.

How Home Insurance Premiums Are Calculated

by: TD Insurance

Here's what you need to know about how home insurance is calculated in Canada

Insurance companies consider many factors to estimate the likelihood that you will make a claim, and what that claim will cost. Rating factors can vary from company to company, but here are some of the main things that will affect the cost of your property insurance.

How does where you live affect your home insurance premium?

Location, location, location. Your address can make a big difference. Using your postal code, insurance companies can track claims made in that location and use the information to determine the likelihood of a claim occurring. They can then adjust premiums based on past experience in your neighbourhood. If you live in an area with a high incidence of break-ins or vandalism, for example, your rate could be higher than what you would pay in an area where those things are rare.

Is your home near a fire station or hydrant?

How close you are to a fire station is another indicator used to adjust premiums. Fire is a major concern, so it's an advantage to live near a hydrant or station. The closer you are, the better the chances of saving your property in the event of fire. In urban areas, proximity usually isn't a problem. But in more remote or rural areas, the distance may be greater, influencing the cost of insurance.
Does the age of the structure affect home insurance premiums?

As a building ages, the risk associated with it increases and so does the premium. As the overall infrastructure wears down, there is a higher risk of a faulty/leaky pipe (potential for water damage claims increases, etc.). Newer homes generally pay lower premiums and they increase as the homes age. It's worth noting that if you make updates and renovations (like replacing a roof), the effect of the building aging decreases.

Does the heating system affect the cost of home insurance?

With oil heating, you may have to pay more than you would with a gas furnace or electric heat. The risk of leaks with oil tanks increases the potential for damage to your property as well as the potential for environmental hazards, which can be very costly to remediate. Wood stoves can increase risk of fire, and older model wood stoves, (especially if incorrectly installed or maintained) are a common source of house fires.

What type of wiring does your home have?

A variety of factors associated with your electrical system can affect the risk of fire and, with it, the cost of insuring your property. Such things include breakers posing less of a risk than fuses, and older wiring increasing the risk of fire.

What type of basement does your home have?

Basements are no longer used primarily for storage and laundry. Many basements are finished (as homeowners look for more living space) and are used for recreational

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purposes often with expensive furnishings and equipment (which make for more expensive claims). As such, having a finished basement could lead to an increase in your premium. Although it's true that having a finished basement could increase the premium, it's important you provide accurate information to ensure you have adequate coverage in the event a claim occurs. Customers do not always think to notify their insurance company when they finish their basement.

How much coverage and what types of coverage do you need?

Typically, the more coverage you purchase, the higher your premium will be - however, this could save you money in the long run. Our Home Coverage includes our Million Dollar Solution and covers All Risks, Personal Liability and much more. We also offer Enhanced Home Coverage that includes extras like Family Coverage and Claims Forgiveness. There is also other optional coverage such as Identity Theft or Personal Umbrella that can be added based on your needs.

What if you live in a condo, is the insurance different?

Yes, condo insurance is slightly different than home insurance. A condo corporation will have a policy that covers the common areas outside of your unit. Your policy will cover damage or loss inside your unit as well as personal liability claims if someone is injured inside your home[1].

What if you rent, does it work differently?

Renter's insurance (also called tenant's insurance) is for when you rent your house or apartment from someone else. It's a good idea to have enough insurance to cover the cost of replacing your belongings in your home if they're damaged or stolen[2].

Are there ways to save on home insurance?

If you're looking to save on home insurance there are a few ways to do it. Consider adding the following to take advantage of our Home Security Savings (available to those with Home Coverage or Enhanced Home Coverage):

- A centrally monitored water alarm that sends an alert if there is a leak or flood in your house
- A centrally monitored fire or burglar alarm

Don't stop there. For more savings, here are some other helpful ways you can reduce your home insurance costs. Looking for a quote? Get a home insurance quote quickly and easily here.

If you're an existing customer and have questions about your policy, please visit the Contact Us page.

Reference Articles:

1.https://www.canada.ca/en/financial-consumer-agency/services/insurance/home.html#toc1

2.https://www.canada.ca/en/financial-consumer-agency/services/insurance/home.html#toc1

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EMOTIONAL WELLBEING

What to Do with Intense Emotions

These days, a lot of things can trigger intense emotions. Political, social, and cultural topics often bring a wide variety of perspectives and discourse. What you choose to do with your strong emotions when they show up is what matters most, particularly where your health is concerned.

Although it can be tempting to want to suppress strong emotions, ignoring them can have a negative effect on your health. A better solution is to find healthy ways to release them.

Here are some positive ways to manage your emotional energy:

Physical Activity
Moving your body is a simple way to release tension and shift your mindset. As you move, your body releases feel-good endorphins that help calm your emotions.

Therapy
Discussing your feelings with a trained therapist or trusted friend can be helpful when you need to process or work through a complicated situation.

Acupuncture
While the research[1] is still inconclusive, mostly due to the small number of subjects in studies conducted to date, there is some evidence that acupuncture can alleviate pain and anxiety by increasing endorphins.

Journaling
Because therapeutic journaling[2] requires the use of both left brain and right brain functions, it is an effective way to process challenges and reduce the intensity of emotions.

Primal Therapy
Scream therapy, or Primal Therapy[3], has been used by clinical psychologists for decades to treat anxiety, depression, and trauma. While it is an unconventional method, it has been shown to be effective for some.

Meditation
Research[4] has shown that mindfulness-based stress reduction (MBSR) techniques, such as meditation, can be an effective coping strategy when it comes to regulating emotions.

Learning how to manage stress is an important part of your wellbeing. Experiment with a few different emotional management strategies to find out which ones are most effective for you.

For additional information about Gallagher's Live Well Monthly resources, please contact:

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1. https://www.health.harvard.edu/healthbeat/relieving-pain-with-acupuncture
2. https://psychcentral.com/lib/the-health-benefits-of-journaling/
3. https://www.psychologytoday.com/us/blog/in-therapy/201002/cool-intervention-3-primal-therapy
4. https://www.sciencedirect.com/science/article/pii/S1877042814024161

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Therapeutic Options

FOCUS ON THE TREATMENT OF DRY EYES

By Joanne Deshpande, BScPhm, RPh

Dry eye disease (DED), also known as keratoconjunctivitis sicca or dysfunctional tear syndrome, is a common ailment estimated to affect over 6 million Canadians.[1,2] Previously, the incidence of DED was highest in advanced age patients but with the increased use of visual display screens, a greater number of younger people are now experiencing symptoms.[2] This article will review general aspects of DED with an emphasis on current therapies, both pharmacological and non-pharmacological.

PATHOPHYSIOLOGY AND ETIOLOGY

The Tear Film & Ocular Surface Society (TFOS) launched the Dry Eye Workshop II (DEWS II) to develop a consensus on multiple aspects of DED, using an evidence-based approach. Foremost, a contemporary definition of dry eye was adapted:[3]

> *Dry eye is a multifactorial disease of the ocular surface characterized by a loss of homeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles.*

The definition was based on the understanding that a healthy tear film depends on the combined actions of the lacrimal glands, eyelids and ocular surface.[1] The tear film, a layered structure that hydrates and prevents damage to the cornea, consists of three components: aqueous, mucous and lipid elements.[1] The aqueous phase makes up the bulk of the film, providing hydration to the eye while the lipid layer lubricates the ocular surface and forms a protective barrier to minimize evaporation.[1,4] The development of DED was previously attributed to either a decrease in tear production (aqueous deficient DED) or an increase in tear evaporation (evaporative DED).[1,3] It is now recognized that the majority (>80%) of DED cases are due to an evaporative cause or to a combination of both mechanisms.[1,4] The TFOS DEWS II consensus suggests that the core mechanism of DED is evaporation-induced tear hyperosmolarity. However, tear hyperosmolarity is also seen as a trigger for a cascade cyclical effect, inducing both direct ocular epithelial surface damage and indirect damage by initiating inflammatory mediators, and thereby, self-perpetuating the process.[3]

Aqueous deficient DED can occur because of destruction or dysfunction of the lacrimal ducts or glands. In the Western world, the main cause of aqueous deficient DED is inflammatory infiltration of the lacrimal gland, such as in Sjögren's syndrome, an autoimmune disorder affecting lacrimal gland function. Less severe forms are commonly caused by aging, however, various medications can affect lacrimal secretion and several disease states can cause either inflammatory infiltration directly or gland obstruction via conjunctival scarring.[1,3] Aqueous deficient DED may occur due to chronic contact lens use, which is linked to reduced corneal sensitivity that results in a reduced reflex sensory tear secretion.[1] A block in reflex tearing can occur with chronic use of topical anesthetics, trigeminal nerve damage or after refractive surgery (including LASIK).[1,3] Table 1 includes a list of potential disease-associated causes of DED.

Excess water loss, or evaporation, from the eye surface causes instability of the tear film resulting in hyperosmolarity. The most common cause of tear evaporation is meibomian gland dysfunction (MGD), which involves the gland that supplies the lipid component of the tear film.[1] MGD tends to develop with advanced age, particularly after the age of 50 years, but also occurs in cases of decreased androgen levels, with the use of specific medications that can induce gland atrophy (e.g. isotretinoin) or in specific skin conditions (e.g. rosacea, atopic dermatitis) or conjunctival diseases.[3]

Tear evaporation is also dependent on exposure time of the ocular surface. If blink function is decreased (e.g. in Parkinson disease or with the increased use of digital devices) or if structural eyelid abnormalities exist, the increased exposure of the ocular surface allows for greater evaporation.[1,4,5] Evaporation and symptoms worsen in cases where ocular surface irritation may be present such as with the use of topical eye drops with preservatives, chronic contact lens use and/or ocular allergy syndromes.[1,6]

PRESENTATION, DIAGNOSIS AND RISK FACTORS

Signs and symptoms of DED tend to vary widely in severity, duration and etiology.[7] Symptoms can include sensations of dryness or of a foreign body in the eye, irritation, grittiness, burning, soreness, itching or eye fatigue. Signs can include redness, increased blinking and/or paradoxical excessive tearing.[1,4,7] Patients will often report changes in vision (blurred), photophobia, contact lens intolerance or discomfort while focused on screens or reading. Exacerbating conditions can include wind, cold temperatures, decreased humidity or long screen-time exposure.

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Table 1. Risk Factors for DED* [1,3,4,7,8]

Category	Confirmed Risk Factors	Probable Risk Factors
Individual-based	Advanced age Asian race Chronic contact lens use Female sex	Low dietary intake of omega-3 fatty acids
Environmental	Air pollution Computer / Visual screen use Low humidity Second-hand smoke Windy and/or cold environment	
Medical Conditions	Androgen deficiency Connective tissue disease (including rheumatoid arthritis) Hematopoietic stem cell transplantation MGD Parkinson disease Sjögren's syndrome	Bell palsy Diabetes mellitus Glaucoma Perennial/Seasonal allergies Psychiatric conditions Rosacea Thyroid disease Viral infection Vitamin A deficiency
Medications	Antidepressants (including TCAs, SSRIs) Antihistamines Anxiolytics HRT Isotretinoin Oral contraceptives	Amiodarone Anticholinergics Antiparkinsonian agents Antipsychotics Beta-blockers Decongestants Diuretics Nicotinic acid Ocular medications containing preservatives
Ocular surface history/disease		Allergic conjunctivitis HSV Pterygium Refractive surgery (LASIK) VZV

HRT=hormone replacement therapy; HSV=herpes simplex virus; LASIK=laser-assisted in-situ keratomileusis; MGD=meibomian gland dysfunction; SSRIs=selective serotonin reuptake inhibitors; TCAs=tricyclic antidepressants; VZV=varicella zoster virus

* List is not all-inclusive; risk factors with inconclusive or limited data have not been included.

Diurnal fluctuations of worsening symptoms later in the day are commonly reported as well.[1,7]

The varying degrees of discomfort, ocular irritation and visual disability will cause many people to visit an ophthalmologist.[7] The diagnosis of DED is based on the presence of characteristic symptoms and the exclusion of other conditions that may present similarly (e.g. viral conjunctivitis, bacterial infections).[1] A comprehensive ophthalmologic examination can be done to assess the quality, quantity and stability of the tear film and workup testing should include evaluation of orbital structures including eyelids, lashes and meibomian glands.[4,7]

In many cases, the presence of bothersome symptoms can greatly impact quality of life and potentially compromise outcomes of ophthalmic surgery for vision correction. As DED can be a chronic condition and symptoms may increase in severity over time, periodic reassessment should be encouraged. Left untreated, clinically significant DED can lead to reversible conjunctival squamous metaplasia and punctate epithelial erosions of the conjunctiva and cornea. Patients with concomitant systemic inflammatory conditions are more prone to developing serious sequelae such as ocular surface keratinization, corneal scarring, thinning/neovascularization, corneal ulceration and/or severe vision loss.[7]

Numerous risk factors have been identified that may contribute to the development of DED; however, the major risk factors are advanced age and being female. Table 1 provides a list of the most consistent and probable risk factors.[7]

MANAGEMENT

The treatment of DED is not usually curative but symptomatic improvement can be achieved by increasing tear production, slowing tear evaporation or reducing ocular surface inflammation. These changes can provide significant relief while also improving visual acuity and preventing further ocular damage.[1] Several agencies, including the TFOS DEWS II, have recommended a stepwise approach to DED management, moving from a generalized course, using low-risk, over-the-counter products aimed at mixed etiologic causes to more advanced, prescribed treatments based on individual pathophysiology. As DED is multifactorial and difficult to diagnose, the stepwise approach is not meant to be rigidly adhered to; only interventions thought to benefit an individual patient should be utilized.[5]

Step 1: Environmental Strategies and Ocular Lubricants

Initial management in all patients should involve some basic non-pharmacological strategies. Since adverse ambient conditions such as air pollution, low humidity and/or extremes of air temperatures can affect tear stability, increasing humidification at home and work and avoiding cigarette smoke are recommended.[4,5] Avoiding exposure to direct air conditioning or heating vents indoors and wearing eye protection outdoors in windy conditions can be helpful.[7,8] Long periods of focused visual tasks (e.g. reading, watching TV, driving) and increased use of video display terminals (computers, phones and other devices) have been shown to decrease blink rate. Educating patients to take more frequent breaks to rest the eyes and/or incorporate blink-stimulating exercises is useful. Ergonomic factors can be implemented to reduce ocular surface exposure and eyestrain, such as lowering the level of a digital screen which causes the user to look downward.[4,5] For contact lens wearers, lens design, care system and/or frequency of replacement may need to be reconsidered, as all can contribute to discomfort.[5] In patients for whom systemic medications may be contributing to DED (see Table 1), discussion with their physician and/or pharmacist for alternatives or strategies to help (e.g. dose reduction or change from oral to topical formulation) may be warranted.[5,7] The supplemental use of omega-3 fatty acids has been widely used in DED, but studies have shown mixed results.[5] A recent large multicenter trial comparing the use of omega-3 fatty acid 3000 mg versus placebo

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over 12 months failed to show any benefit in DED signs and symptoms.[1,7,9] Finally, practising good eyelid hygiene is recommended especially in those patients who have MGD. Daily warm compresses, hypoallergenic cleansing products and gentle massaging of the lids to express lipid oils can be helpful.[4]

Ocular lubricants, often referred to as artificial tears or tear replacements, are considered the mainstay of pharmacotherapy and a first-line option.[5,7] Although many products fall into this category, very few, good-quality clinical trials have been done to demonstrate superiority of one agent over another.[5,10] Most lubricants contain an aqueous base, but their properties differ slightly with respect to viscosity, osmolarity and/or pH, any of which can impact efficacy, tolerability and patient preference. Viscosity-enhancing ingredients include carboxymethyl cellulose, dextran, hyaluronic acid, hydroxypropyl-guar, hydroxypropyl cellulose, hydroxypropyl methylcellulose, polyacrylic acid, polyvinyl alcohol, polyvinylpyrrolidone and polyethylene glycol. These agents enhance lubrication of the eye and extend the duration of retention, thereby, protecting against dryness and encouraging tear retention at the ocular surface. These properties are also responsible for producing transient blurring of vision and/or residue build-up on the eyelids and lashes. Newer products are using a combination of viscosity-enhancing agents which may produce greater symptom control compared to single-agent products.[5]

In an attempt to improve lipid deficiency of the tear film (e.g. in MGD), lipid-based lubricant drops are becoming more common. Oil-based ingredients attempt to mimic natural meibum and include phospholipids (e.g. phosphatidylcholine, phosphtidylethanolamine), saturated/unsaturated fatty acids and triglycerides. Examples include mineral oil, castor oil, olive oil, glycerin carbomers, coconut oil, soybean oil and lecithin. Formulated as emulsions, these products usually require inversion or shaking prior to administration.[5]

Regardless of base ingredient, preservatives are commonly included to prevent microbial growth in a multi-use vial; however, chronic exposure of the ocular surface to preservatives (especially benzalkonium chloride) is known to increase irritation and induce corneal and conjunctival changes which may cause further damage and delay healing. In patients requiring frequent administration of ocular lubricants and/or requiring concomitant topical ocular therapies, a preservative-free (PF) option should be chosen. PF eyedrops have demonstrated a greater reduction of ocular surface inflammation and increase in antioxidant components of tears in patients with DED. Newer oxidative preservatives (e.g. sodium chlorite, sodium perborate, polyquaternium-1, or SofZia™) are thought to be gentler to the ocular surface but may still have some negative effects. PF drops have typically been packaged as single-use vials that are usually more expensive than multi-use bottles and may be challenging to use by patients with limited dexterity. A recent alternative to unit dose packaging of PF drops is the use of unidirectional or filter valves on multidose bottles, preventing contamination by re-entry.[5]

Step 2: Tear Conservation, MGD Therapy and Prescription Medications

If Step 1 measures are ineffective, referral to an ophthalmologist is recommended. Referral should always be a first step in severe cases of DED, in patients with underlying medical concerns (e.g. diabetes with possible neuropathy) or in the presence of ocular structural abnormalities.[1] Step 2 options involve more intensive therapies including punctal occlusion or use of moisture chamber spectacles/goggles to conserve tears, physician-assisted expression of meibomian glands or pulsed light therapy of MGD or the use of topical antibiotics (for MGD symptoms) or anti-inflammatory medications.[4,5,7]

Due to the inflammatory cascade mechanisms involved in the development of DED, a number of immunomodulating modalities have been tried. While topical glucocorticoids have demonstrated improved efficacy of DED symptoms, their associated risks of glaucoma, cataracts and opportunistic infections limit their use to short-term periods only.[5,11] Several non-glucocorticoid immunomodulating options are currently being studied. Two topical agents that have demonstrated benefit in clinical studies are cyclosporine A (CSA) and lifitegrast.[5,7]

Topical CSA, an immunomodulator with anti-inflammatory properties, has been associated with improving tear production, reducing markers of inflammation and reducing elevated tear osmolarity while also decreasing apoptosis in epithelial cells of the conjunctiva.[5,11] A recent systematic review and meta-analysis included eleven randomized controlled trials of CSA use in DED. Results confirmed the benefit of CSA in DED both subjectively and in objective clinical parameters. Efficacy appeared to be greater in older patients, possibly due to a faster clearance rate of the medication in younger patients. Of note, this study found that the beneficial effects of CSA were attenuated when combined with artificial tears, a surprising finding which needs further investigation.[11]

Lifitegrast is a lymphocyte function-associated antigen 1 (LFA-1) antagonist and decreases T cell-mediated inflammation in DED.[5] Clinical studies have demonstrated improvement in symptoms of ocular dryness and discomfort as well as in objective corneal and conjunctival tests over test periods of 3 months. Pooled safety data of lifitegrast in DED demonstrated favourable safety and tolerability. The most common adverse effect, administration site pain and irritation, improved within minutes.[5,12]

Table 2 provides an overview of the most commonly used medications in the management of mild to moderate DED.

Steps 3 and 4: Systemic Therapy, Surgical Options

If the above options are still inadequate to manage symptoms of DED, recommended Steps 3 and 4 include therapies such as oral secretagogues, autologous serum eye drops, therapeutic contact lens options, longer use of topical corticosteroids or surgical procedures. Many of the therapies are aimed at specific causative mechanisms (e.g. use of topical steroids to treat inflammation in DED), but studies to confirm efficacy and place in therapy are still needed. Due to the limited availability of evidence, these options will not be discussed.[5]

CONCLUSIONS

DED is a complex disorder with varying causes, symptoms and risk factors. The management of each patient needs to be individualized and can vary greatly amongst patients. Though it may be frustrating for patients, a trial-and-error approach to determine which therapeutic agent(s) and/or non-pharmacological strategies improve their symptoms and overall quality of life is oftentimes necessary. As pharmacists, we can help patients understand the underlying causes of symptoms and aid in product selection, both of which may maximize the benefit achieved.

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Table 2. Pharmacological Choices for the Management of Dry Eyes[1,4,5,7,8,11-14]

Therapy	Dosing⊗	Adverse effects	Comments
Ocular Lubricantsϕ∇ Examples of aqueous-based ocular lubricants: • carboxymethyl cellulose • dextran • hyaluronic acid • hydroxypropyl-guar • hydroxypropyl cellulose • hydroxypropyl methylcellulose • polyacrylic acid • polyvinyl alcohol • polyvinylpyrrolidone • polyethylene glycol (numerous brands) Examples of lipid-based ocular lubricants: • castor oil • coconut oil • glycerin carbomers • lecithin • mineral oil • olive oil • phosphatidylcholine • phosphtidylethanolamine • soybean oil (numerous brands)	Drops: starting dose of 1-2 drops into each eye tid – qid; frequency can be increased to as often as every hour Gels/Ointments: preferably applied to lower lid once daily at bedtime	• Ocular discomfort, including foreign body sensation • Transient, blurred vision with high-viscose products • Residue build-up on lashes/eyelids with high-viscose products	**Advantages** • Considered the mainstay and first-line choice in DED management • Onset of symptom relief can occur within days • Relatively safe and well-tolerated • Available in different formulations (liquid, gel or ointment); selection can be based on timing of use (gels and ointments last longer in the eye, making them preferred options for use at bedtime) and patient comfort • PF products are available; particularly preferred in patients with a pre-existing ocular surface condition or those who require frequent administration of eye drops. PF products have shown greater efficacy in decreasing ocular surface inflammation. • PF multi-use vials are available for certain products • Ointments do not support bacterial growth; these products do not require inclusion of a preservative **Disadvantages** • Few comparative RCTs to recommend one product over another • May take up to 3-4 weeks to see a significant change in symptoms • Products containing preservatives can increase irritation and induce ocular surface damage and should be avoided if eyedrops are used frequently • PF products (e.g. unit-dose vials) are typically more expensive and difficult to open • More severe cases of DED may require frequent administration
Ophthalmic Corticosteroids • loteprednol 0.5% solution • fluorometholone solution • others	1-2 drops into each eye qid 1-2 drops into each eye bid - qid	• Minor stinging upon administration • Risk of glaucoma, cataracts and ocular infections; risk increases with longer duration of use	**Advantages** • Effective in relieving moderate to severe DED symptoms over a short-term basis • Low doses can be used **Disadvantages** • Limited to short-term use (2-4 weeks as pulsed therapy) due to risk of adverse effects • Prescription only
Cyclosporine A (CSA) 0.05% emulsion (Restasis®; generic)	One drop into each eye bid After a year of therapy, dose reduction to once daily can maintain benefit in a portion of patients	• Ocular stinging and burning • May cause transient blurred vision	**Advantages** • Indicated for moderate to moderately severe DED • Long-term efficacy/safety data available for up to 40 months • QOL studies found CSA to be cost-effective compared to ocular lubricant • Twice daily dosing • PF, available as either single-use or multi-use vial **Disadvantages** • Not as effective in DED due to surgical procedures, contact lens use or thyroid orbitopathy • Conflicting data on efficacy of CSA with concurrent use of artificial tears • Requires continued use as symptoms can recur upon discontinuation • May require ≥6 weeks of therapy for noticeable improvement • May require pre-treatment with 2 weeks of topical loteprednol 0.5% to minimize stinging with CSA administration • As an immunomodulator, patients should monitor for signs of eye infection • Prescription only • Therapy is relatively costly
Lifitegrast 5% solution (Xiidra®)	One drop into each eye bid	• Transient blurred vision upon administration • Administration site reactions, eye pain and irritation • Dysgeusia (bad taste) in ~14% patients	**Advantages** • Well tolerated with most adverse effects being mild and transient • Long-term safety data available up to 12 months • PF (as single doses) • Twice daily dosing **Disadvantages** • May take several months for response to treatment • As an immunomodulator, patients should monitor for signs of eye infection • Unit dose packaging only • Therapy is relatively costly • Prescription only

bid=twice daily; CSA=cyclosporine A; DED=dry eye disease; PF=preservative-free; qid=four times daily; QOL=quality of life; RCTs=randomized controlled trials; tid=three times daily

ϕ Lists may not be all-inclusive.

∇ Not all ingredients listed may be available in Canada.

⊗ The range of dosing provided is a recommendation for ocular lubricants in general but individual product labelling should also be consulted.

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REFERENCES

1. Shtein RM. Dry eye disease. In: UpToDate. Jacobs DS, Givens J (Eds). UpToDate. Waltham, MA. [cited 2021 January 5]. Available from: https://www.uptodate.com
2. Caffery B, Srinivasan S, Reaume CJ et al. Prevalence of dry eye disease in Ontario, Canada: A population-based survey. The Ocular Surface 17(2019):526-531.
3. Craig JP, Nelson JD, Azar DT et al. TFOS DEWS II Report Executive Summary. The Ocular Surface 15(2017):802-812.
4. Rouen PA, White ML. Dry eye disease. Prevalence, assessment and management. Home Healthcare Now. March/April 2018. 36(2):74-83.
5. Jones L, Downie LE, Korb D et al. TFOS DEWS II Management and therapy report. The Ocular Surface 15(2017):575-628.
6. Craig JP, Nichols KK, Akpek EK et al. TFOS DEWS II Definition and classification report. The Ocular Surface 15(2017):276-283.
7. Akpek EK, Amescua G, Farid M, et al. Dry eye syndrome preferred practice pattern. Ophthalmology 2019;126:286-334.
8. Friesen AM. Dry eye. In: RxTx Compendium of Therapeutics for Minor Ailments. [Internet]. Ottawa, ON. Canadian Pharmacists Association. Date of revision 2019 April 24. [cited 2021 January 5]. Available from: http://www.e-therapeutics.ca
9. The Dry Eye Assessment and Management Study Research Group. N-3 fatty acid supplementation for the treatment of dry eye disease. N Engl J Med 2018;378:1681-1690.
10. Moshirfar M, Pierson K, Hanamaikai K et al. Artificial tears potpourri: a literature review. Clin Ophthalmol 2014;8:1419-1433.
11. Tuan H-I, Chi S-C, Kang, Y-N. An updated systematic review with meta-analysis of randomized trials on topical cyclosporin A for dry-eye disease. Drug Design, Development and Therapy 2020;14:265-274.
12. Nichols KK, Donnenfeld ED, Karpecki PM et al. Safety and tolerability of lifitegrast ophthalmic solution 5.0%: pooled analysis of five randomized controlled trials in eye disease. European Journal of Ophthalmology 2019;29(4):394-401.
13. Allergan Inc. Restasis MultiDose™ product monograph. Markham, ON. 2018 June 1 [date of approval].
14. Novartis Pharmaceuticals Canada Inc. Xiidra® product monograph. Dorval, QC. 2019 November 21 [date of revision].

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Therapeutic Options

MANAGEMENT OF TESTOSTERONE DEFICIENCY IN MEN: A FOCUS ON PRACTICAL IMPLEMENTATION OF THERAPY

By Joanne Deshpande, BScPhm, RPh

In 2015, the Canadian Men's Health Foundation coordinated a multidisciplinary team to develop a clinical practice guideline for the management of testosterone deficiency, which was recognized to be a "living document" and would be updated periodically to incorporate clinical and therapeutic advancements.[1,2] The focus of this article will be to update practical points of testosterone replacement, clarifying the management of specific scenarios and providing an overview of the therapeutic options currently available, based on the most recent 2021 update of the guideline.

OVERVIEW OF TESTOSTERONE DEFICIENCY

Testosterone deficiency (TD) syndrome occurs due to either a failure of the testes to produce testosterone (primary hypogonadism) or a signalling problem within the hypothalamic-pituitary-gonadal axis that regulates its production (secondary hypogonadism).[1,2,3] Both causal pathways can result in low levels of testosterone but varying levels of gonadotropins (LH, FSH) and a combination of the two routes is common. TD often occurs in men with advancing age (hence, referred to as late-onset hypogonadism), but can occur in younger men with certain medical conditions.[2] Serum total testosterone is thought to normally start to decline in men in their mid-30s and gradually continue at a rate of 1.6% per year.[4] Actual figures of TD prevalence are not well defined, due to variability in reporting measures and more specifically, in biochemical testosterone thresholds. Recent international guidelines and studies have estimated prevalence rates of TD to be in the following ranges: 4-12% in men 50-59 years of age, 9-23% in men 60-69 years of age, and 28-49% in men over 70 years of age.[2]

Signs and symptoms of TD tend to be non-specific and vary based on age, comorbidities, and duration of TD.[2,5] Table 1 provides an overview of potential signs and symptoms, but not all need to be present for a diagnosis of TD. Sexual symptoms and fatigue are the most common presentations and tend to occur the earliest.[1]

Given the ambiguity that can be associated with the clinical presentation of TD, a diagnosis often requires a correlation between signs/symptoms and the presence of low or equivocal serum testosterone levels.[2] Serum *total testosterone* (free plus protein-bound) is considered the preferred initial screening test to diagnose TD as it is considered an accurate reflection of testosterone secretion.[1,2,6] The Canadian TD guideline recommends this sample be collected between 7 AM and 11 AM, or within 3 hours of waking for shift workers.[1,2]The rationale for this is the natural diurnal fluctuation that occurs with testosterone, reaching a maximum level at approximately 8 AM. Since food or glucose can suppress testosterone, a patient should be fasting prior to this measurement. The normal range of serum total testosterone will depend on the specific assay and reference population used, but the range is usually 10.4-31.2 nmol/L.[6] The Canadian TD guideline suggests a total testosterone level <10 nmol/L is a *reasonable diagnostic threshold consistent for TD*.[2] Typically, if a level is low or borderline low, the level should be repeated once or twice to confirm the diagnosis of TD. The measure of *free testosterone* is only recommended if an abnormality in sex-hormone binding globulin (SHBG) is suspected (commonly in obesity), or if the initial total level is near the lower end of normal; however, only specific methods of analysis are reliable and should be chosen carefully.[6] Further testing of other serum entities may be useful in evaluating whether the TD is primary or secondary and for ruling out other potential causes. Although several validated screening questionnaires for TD are available, these tools are not recommended for diagnostic purposes due to a lack of specificity but may be used for initial screening purposes instead.[2]

TREATMENT OF TESTOSTERONE DEFICIENCY

Who to Treat?

The decision to treat TD is made on an individual basis, as symptoms vary in degree and intensity. The primary goal of treatment is to mitigate the negative effects of low testosterone. Diagnosis is often confirmed by serum testosterone level and the choice to treat may also be impacted by this result. A symptomatic patient with low serum testosterone is an obvious candidate for treatment but other scenarios are not as straightforward. In the case of a patient with symptoms characteristic of TD but with a normal testosterone level, other conditions with similar symptoms (e.g., depression, hypothyroidism) need to be ruled out first. Due to difficulties in the

Table 1. Signs and symptoms associated with testosterone deficiency[1,2,3,5]

Sexual	Reduced libido
	Erectile dysfunction
	Decreased frequency of morning erections
	Decreased performance
	Delayed ejaculation, reduced ejaculate volume
	Decreased intensity of orgasm
	Infertility
Cognitive / Psychological	Depression
	Mood changes
	Irritability
	Anxiety
	Poor concentration and memory
	Decreased motivation, initiative, and self-confidence
	Insomnia / Sleep disturbances
Physical / Structural	Increased body fat
	Decreased lean muscle mass/strength
	Testicular atrophy
	Fatigue / Loss of energy
	Low bone mineral density / Low-trauma fracture
	Anemia
	Loss of facial, axillary, and pubic hair
	Gynecomastia
	Hot flushes, sweats

measurement of serum testosterone (e.g., lack of consistent reference ranges between labs, variability in sensitivity of androgen receptors, testosterone neutralizing effects of SHBG), this is a situation where other markers, such as SHBG or free or bioavailable testosterone, may need to be measured to better interpret the total testosterone levels and direct treatment. Finally, in the case of a patient who has a low serum testosterone level but is otherwise asymptomatic, the recommendation is *not* to treat with testosterone replacement unless symptoms develop.[2] The benefits and safety of using testosterone replacement in asymptomatic patients remain unclear.[5]

Use of testosterone replacement in the absence of identifiable pituitary or hypothalamic disease among aging men with a decline in serum testosterone levels has been controversial, as previous studies have not demonstrated consistent benefit in this particular population.[7,8] The Testosterone Trials were a series of seven randomized placebo-controlled studies evaluating the efficacy of testosterone on several symptoms including sexual, physical, and cognitive function, specifically in older men with symptoms of hypogonadism and low testosterone levels.[8,9] The study included 790 men over the age of 65 years who received testosterone or placebo gel for a period of one year. Benefits of testosterone therapy seen in the trials included a marked increase in bone mineral density, an increase in sexual interest and activity, increased hemoglobin, a small but significant improvement in mood, and only slight improvements in walking.[2,8,9] Symptoms of energy and cognition did not improve compared to placebo.[8] Similar to the Canadian TD guideline, the findings of the Testosterone Trials suggest testosterone replacement could be used in older men who have both specific clinical symptoms and a low serum testosterone level.[2,8] A clinical practice guideline from the American College of Physicians on testosterone treatment in adult men with age-related low testosterone has recommended testosterone be used in older men with low serum testosterone levels who are experiencing sexual dysfunction.[4]

Therapeutic Choices

Testosterone is the natural consideration for treatment of TD. Choice of testosterone preparation is dependent on personal preference, safety, tolerability and cost. Table 2 provides an overview of testosterone products that are available in Canada. Transdermal products, especially gel formulations, are commonly chosen based on their convenience, ease of use and ability to produce stable serum testosterone levels.[7] Although compounded products are often a consideration, the Canadian TD guideline suggests caution as published data has indicated significant variability in the concentration of testosterone in these products, which can impact both effectiveness and safety.[2,10] With respect to herbal or "natural" forms of testosterone (known as "T-boosters"), current evidence does not demonstrate consistent efficacy. Of the limited studies done in humans, 68% of the products had no effect on testosterone levels. Also of concern was the number of case studies demonstrating severe adverse events with these products, often due to the presence of banned substances including steroids. For these reasons, the Canadian TD guideline only recommends evidence-based treatments. In addition to providing exogenous testosterone, lifestyle modifications in the form of weight loss, dietary restriction, bariatric surgery, exercise, and better sleep patterns have also demonstrated some benefit in improving testosterone levels.[2]

Response to Therapy

The response to testosterone replacement therapy can vary depending on formulation and dose, as well as baseline testosterone level and symptoms. The current recommendation is to target improvement in serum level to a mid-normal range (total testosterone, 14-17 nmol/L).[2] The majority of men (>90%) will see a normalization of serum levels with therapy, which occurs earlier than symptom improvement.[2,10] The common sexual symptoms of TD tend to improve after 3 months of treatment while the physical/structural symptoms may require 6 or more months of therapy before improvement is seen.[2] One study treating hypogonadal men with transdermal testosterone reported these same timelines for symptom improvement, however, the full effect on bone mineral density was not seen until 24 months of therapy.[7] These timelines are important for both healthcare providers and patients to be aware of as they affect monitoring times and patient follow-up.[2]

Using these timeframe parameters, patients who have symptom improvement or resolution, combined with *at-target* or *lower-than-target* serum testosterone levels, can continue at the same dose with retesting of levels every 6-12 months.[2,10] Patients who achieve symptom resolution with an increase in testosterone levels *above* the normal range should be considered for dose reduction. If the serum testosterone level increases to within the recommended range but symptoms do not improve, a dose increase is suggested, targeting a higher serum level in the upper range of normal. If symptoms continue despite improvement in serum level and

a trial of at least 3 months therapy, an alternate diagnosis and management plan should be considered.[2]

Duration of Therapy

There is no defined duration of therapy. Since initiation of testosterone therapy is based on the presence of intolerable symptoms and/or decreased quality of life, continuation can occur if the therapy is helping and is tolerated. If significant adverse effects occur and/or there is a cessation of clinical improvement, therapy should be stopped. Testosterone therapy does not require tapering of doses upon discontinuation.[2]

Safety and Tolerability

Before starting any testosterone product, safety concerns should be assessed. Besides allergy or a hypersensitivity to testosterone products, contraindications to use may include known or suspected male breast cancer, metastatic or high-risk prostate cancer requiring androgen-deprivation therapy or the presence of unstable cardiovascular disease.[2] Several areas of concern are discussed below; in each situation, potential risks and benefits should be discussed with the patient.[1]

Prostate Cancer Concerns

A perceived increased risk of developing prostate cancer secondary to testosterone use has been an ongoing concern when prescribing these products. There is now consistent evidence to suggest otherwise. In the aforementioned large randomized controlled trial (RCT; the Testosterone Trials) of 790 men treated with either testosterone or placebo gel, only one man developed prostate cancer over a period of 12 months. Similarly, a meta-analysis evaluating 2351 men found no greater risk of prostate cancer among patients treated with testosterone therapy versus those in the placebo group. Higher testosterone levels were not associated with significantly higher prostate specific antigen (PSA) levels or a greater risk of developing prostate cancer. The Canadian TD guideline makes the recommendation that men with localized prostate cancer that have been treated and have no evidence of active disease may be *considered for a medically supervised trial of testosterone therapy.*[2] However, due to the role of androgen receptor signaling in the process of prostate cancer, patients with metastatic or high-risk prostate cancer, especially cases likely to require androgen-deprivation treatment, should avoid testosterone therapy.[1,2,5] All candidates being considered for testosterone use should ideally be screened for prostate cancer prior to initiation.[5]

Benign Prostatic Hyperplasia (BPH) and Lower Urinary Tract Symptoms (LUTS)

The impact of testosterone on progression of BPH or development of LUTS has also been reviewed. In the Testosterone Trials of one-year placebo-controlled therapy in 790 men, worsening of urinary symptoms suggestive of BPH was monitored and occurred at the same rate in both treatment arms. A review of 16 RCTs of a total of 1030 men found that testosterone therapy did not affect prostate volume. Other testosterone studies have reported improvement in urinary symptoms, peak urinary flow rates, and voided volumes, potentially due to testosterone's effect on bladder detrusor function.[2]

Cardiovascular Risks

The association between testosterone levels and cardiovascular risk has been conflicting. The data indicates that untreated testosterone-deficient men are at greater risk of obesity, diabetes, dyslipidemia and metabolic syndrome, all of which can increase their risk of cardiovascular events. The majority of studies found that hypogonadal men given testosterone demonstrated cardiovascular benefit.[1,2] Conversely, more recent studies have reported increased cardiovascular risk with the use of exogenous testosterone, however, these studies were thought to contain significant methodological flaws.[2] The United States Food and Drug Administration has conducted a review of the data and found the evidence for a causal effect was weak, but still mandated precautionary labelling on testosterone products.[5] Similar action was taken due to a concern about a possible association between testosterone therapy and increased incidence of venous thromboembolic events; however, these cases were considered anecdotal and no definitive evidence of this has been found.[10] A large, long-term RCT is needed to clarify these effects.[2] Currently, a meta-analysis accruing data from 20,000 men in North America, Europe and Australia is looking to clarify the effect of exogenous testosterone on the incidence of cardiovascular events and other outcomes.[11] In the meantime, the Canadian TD guideline makes the recommendation that men with stable cardiovascular disease, who meet criteria for testosterone therapy, are candidates for use but should be monitored accordingly.[2]

Fertility Preservation

In addition to risk assessment, one of the concerns to be aware of when considering testosterone use is the patient's desire for fertility preservation as the use of exogenous testosterone can ultimately cause male infertility. Administration of testosterone results in negative feedback to the pituitary gland, which suppresses production of sperm and testosterone at the testicular level.[2] Patients who want to maintain fertility should avoid isolated use of exogenous testosterone and opt for measures that increase their endogenous serum testosterone production instead, such as human chorionic gonadotropin (hCG), selective estrogen receptor modulators (SERMs; e.g., clomiphene, tamoxifen) and/or aromatase inhibitors (e.g., anastrozole, letrozole).[2,10] Consultation with a fertility specialist is recommended, especially since many clinicians have unknowingly prescribed testosterone products in the past to help with fertility.[2]

CONCLUSIONS

Testosterone deficiency has the potential to negatively impact multiple facets of a man's well-being. By being aware of who may best benefit from therapy and what therapeutic options are currently available, pharmacists can ensure therapy is tailored to the individual patient and that monitoring of response is timely and appropriate.

REFERENCES

1. Morales A, Bebb RA, Manjoo P, et al. Diagnosis and management of testosterone deficiency syndrome in men: clinical practice guideline. CMAJ 2015; 187(18):1369-1377.
2. Grober ED, Krakowsky Y, Khera M, et al. Canadian Urological Association clinical practice guideline: testosterone deficiency in men – evidence-based Q&A. Can Urol Assoc J 2021; 15(5):E234-43.
3. Snyder PJ. Clinical features and diagnosis of male hypogonadism. In: UpToDate. Matsumoto AM, Martin KA (Eds). UpToDate. Waltham, MA. [cited 2021 May 3]. Available from: https://www.uptodate.com
4. Qaseem A, Horwitch CA, Vijan S, et al. Testosterone treatment in adult men with age-related low testosterone: a clinical guideline from the American College of Physicians. Ann Intern Med 2020; 172:126-133.
5. Bhasin S, Brito JP, Cunningham GR, et al. Testosterone therapy in men with hypogonadism: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. May 2018; 103(5):1715-1744.
6. Snyder PJ. Lab Interpretation: low testosterone in men. In: UpToDate. Matsumoto AM, Mulder JE (Eds). UpToDate. Waltham, MA. [cited 2021 May 3]. Available from: https://www.uptodate.com
7. Snyder PJ. Testosterone treatment of male hypogonadism. In: UpToDate. Matsumoto AM, Martin KA (Eds). UpToDate. Waltham, MA. [cited 2021 May 3]. Available from: https://www.uptodate.com
8. Snyder PJ. Approach to older men with low testosterone. In: UpToDate. Matsumoto AM, Schmader KE, Martin KA (Eds). UpToDate. Waltham, MA. [cited 2021 May 3]. Available from: https://www.uptodate.com
9. Snyder PJ, Bhasin S, Cunningham GR, et al. Effects of testosterone treatment in older men. N Engl J Med 2016; 374:611-24.
10. Mulhall JP, Trost LW, Brannigan RE, et al. Evaluation and management of testosterone deficiency: AUA guideline. The Journal of Urology. 2018 August; 200:423-432.
11. Yeap BB, Marriott RJ, Adams RJ, et al. Androgens In Men Study (AIMS): protocol for meta-analyses of individual participant data investigating associations of androgens with health outcomes in men. BMJ Open 2020;10:e034777.
12. Taro Pharmaceuticals Inc. Taro-Testosterone product monograph. Brampton, ON. 2018 October 25 [date of preparation].
13. Pharmascience Inc. pms-Testosterone product monograph. Montreal, QC. 2018 January 22 [date of revision].
14. Clinical Resource #360308. Comparison of Testosterone Products. Pharmacist's Letter/Prescriber's Letter. March 2020.
15. Pfizer Canada Inc. Depo-Testosterone product monograph. Kirkland, QC. 2018 July 12 [date of revision].
16. Taro Pharmaceuticals Inc. Taro-testosterone cypionate injection product monograph. Brampton, ON. 2020 January 29 [date of preparation].
17. Cytex Pharmaceuticals Inc. Testosterone cypionate injection product monograph. Halifax, NS. 2007 December 21 [date of revision].
18. Witherspoon L, Fitzpatrick R, Patel P, et al. Clinical pearls to managing men's health conditions during the COVID-19 pandemic. Can Urol Assoc J 2020;14(5):E161-6.
19. Valeant Canada LP. Delatestryl™ product monograph. Laval, QC. 2014 August 19 [date of revision].
20. BGP Pharma ULC. Androgel® product monograph. Etobicoke, ON. 2019 February 12 [date of revision].
21. Paladin Labs Inc. Testim® 1% product monograph. St Laurent, QC. 2017 February 3 [date of revision].
22. Taro Pharmaceuticals Inc. Taro-testosterone gel product monograph. Brampton, ON. 2020 October 19 [date of preparation].
23. Allergan Inc. Androderm® product monograph. Markham, ON. 2018 February 28 [date of preparation].
24. Acerus Pharmaceuticals Corp. Natesto® product monograph. Mississauga, ON. 2019 April 4 [date of revision].

Table 2. Testosterone deficiency treatment options[2,4,5,7,12,13,14,15,16,17,18,19,20,21,22,23,24]

Formulation	Drug (brand/generic) Strength / Dosage Form / Availability	Usual Dose	Comments
Oral	Testosterone undecanoate (generics only) 40 mg capsule	120–160 mg PO per day in 2 divided doses for 2–3 weeks then 40–120 mg PO per day, based on serum level* and clinical effect	**Advantages** • ease of administration • immediate discontinuation is possible **Disadvantages‡** • gastrointestinal AEs • must be taken with a meal as fat enhances absorption; from day to day, bioavailability can be variable
Injectable	Testosterone cypionate (Depo-testosterone®, generics) 100 mg/mL; 2 mL single dose ampoules or 10 mL vial	200 mg IM every 2 weeks	**Advantages** • long-lasting formulations allow for biweekly or monthly administration • injectable testosterone may be more cost-effective than transdermal and intranasal formulations **Disadvantages‡** • a longer time frame between doses can be associated with greater fluctuation in testosterone levels, producing variations in symptoms, especially in energy, mood, and libido • injection site pain/reactions • higher risk for erythrocytosis (increased hemoglobin and hematocrit) than with transdermal preparations • rarely, cough immediately after administration • IM injection usually requires clinic visit
	Testosterone enanthate (Delatestryl™) 200 mg/mL; 5 mL vial	100–400 mg IM every 4 weeks OR 100–150 mg IM every 2 weeks	
Transdermal	Testosterone gel (Androgel®, Testim®, generics) 1% w/w Available as 2.5 g (=25 mg testosterone) or 5 g (=50 mg testosterone) unit dose packets; Androgel® also available as a metered-dose pump of 60 actuations (1.25 g per actuation)	5 g applied topically once daily in the morning Apply to clean, dry, intact skin of the upper arms, shoulders, and/or abdomen, according to specific product recommendations[β] (ideally an area that can be covered by a short-sleeved shirt)	**Advantages** • less fluctuation of testosterone levels than with injectable formulations • serum testosterone levels remain steady over a 24-hour period • better skin tolerability than with patches • ease of administration **Disadvantages‡** • testosterone concentrations may vary depending on application technique • skin irritation (erythema, induration, or burning) are the most frequently reported AEs • strict adherence to application precautions is necessary to prevent transfer of testosterone to women or children via skin-to-skin contact or contact with clothing/towels/sheets that have been in direct contact with application site; virilization has been reported in children as a result of secondary exposure and is listed as a black box warning • Testim® may be associated with musk-like odour • washing or swimming after application of gel should ideally be spaced by 5–6 hours • can be slow-drying, sticky and messy to apply
	Testosterone patch (Androderm®) Available in two strengths: –2.5 mg testosterone per 24 hours (12.2 mg patch) or 5 mg testosterone per 24 hours (24.3 mg patch)	5 mg patch applied once daily at night Apply to clean, dry skin on back, abdomen, upper arms, or thighs; application site should be rotated, allowing an interval of 7 days between use of the same site	**Advantages** • can achieve relatively stable serum testosterone concentrations, allowing consistency in symptom management • convenient to use **Disadvantages‡** • skin reactions at application site (erythema, burning, allergic contact dermatitis, blister reactions) occur in up to one-third of patients; mild skin irritations may require treatment with a corticosteroid cream • use of multiple patches (2–3) may be required in men with body weight >130 kg or in those with low serum testosterone levels • adhesion of patch may be difficult on skin that is hairy, oily or sweaty
Intranasal	Testosterone nasal gel (Natesto®) 4.5% w/w Available as a metered-dose pump of 60 actuations; (5.5 mg testosterone per actuation)	11 mg (=1 actuation IEN) intranasally twice daily Based on serum testosterone level* and clinical response, dose may be increased to a maximum dose of 11 mg three times daily	**Advantages** • may be the preferred option in cases where fertility preservation is desired as it potentially results in less suppression of spermatogenesis • reduced risk of person-to-person transfer of testosterone • ease of administration **Disadvantages‡** • not recommended for use in patients with chronic nasal conditions or alterations in nasal anatomy (including sinus disease or nasal mucosal inflammatory disorders such as Sjögren's syndrome); should not be used during episodes of severe rhinitis • AEs include nasopharyngitis, rhinorrhea, sinusitis, scabbing and epistaxis, typically mild to moderate in severity • frequent dosing can be inconvenient • approved by Health Canada but currently not available due to supply issues

AE=adverse effect; IEN=into each nostril; IM=intramuscularly; PO=orally

* Preferred serum testosterone level is total (free plus protein-bound) testosterone.[1,2,6]

‡ All testosterone products share similar adverse effects and potential for drug interactions; only formulation-specific details have been listed.

β Androgel® and Taro (generic) administration instructions specify application to the upper arms, shoulders, and/or abdomen whereas Testim® application instructions specify only the upper arms and shoulders.[20,22]

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Resources: Managing Streszx Burnout

PANS Webinars (Free for PANS Members)

Fostering Resiliency in Pharmacy Practice: Implications in Covid-19 and Beyond

COVID-19 What's Normal? - Coping with anger, fear and loss within yourself and among your team

Online Resources and Tools

The 8 Steps to Resilience

Public Health Agency of Canada Wellness Together Mental Health and Substance Use Support Portal

What's Normal - Tips for the Frontline for Coping in a Crisis

Canadian Mental Health Association: Coping with COVID

Boost Your Immunity to Stress

Compassion Fatigue - Community Sector Council of Nova Scotia

Recognizing the Signs of Burnout and Compassion Fatigue - Canadian Veterinary Medical Association

How to Set Healthy Boundaries: 10 Examples + PDF Worksheets

"Psychology Works" Fact Sheet: Workplace Burnout - Canadian Psychological Association

Centre for Mental Health in the Workplace

Health Canada: Occupational Health

PANS COVID-19 Resource Page

Informative Articles

Is there a cost to protecting, caring for and saving others? Beware of Compassion Fatigue - CMHA

6 Causes of Burnout and How to Avoid Them: Harvard Business Review

McKinsey Quarterly: 'Great Attrition' or 'Great Attraction'? The choice is yours.

The 'Great Resignation'? It's not happening in Canada - Globe and Mail

Telephone Resources

Provincial Mental Health Crisis Line: 1-888-429-8167 (available 24/7)

The Kids Help Phone: 1-800-668-6868 (available 24/7)

*click item to access online

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