Swimming Pool Worker Dermatoses 196

Nina R. Blank and David E. Cohen

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Abstract

On a daily basis, pool cleaners handle a variety of entities that are potentially hazardous to the skin. The aquatic environment of the swimming pool exposes workers to potential contact allergens and irritants and aids in the transmission of infectious agents. In addition, ultraviolet radiation and genotoxic substances generated as disinfection by-products may increase the risk of cancer in these individuals.

Sanitizing and pH-stabilizing agents commonly used in swimming pools include chlorine, hypochlorite salts, trichloro-s-triazinetrione (8), muriatic acid, and copper. Several of these chemicals are known to cause irritant dermatitides, and some, such as muriatic acid, may yield severe chemical burns. When mixed improperly with hypochlorite salts, TST can result in explosions, also leading to burns and even fatalities in those who work with the substance. Cases of allergic contact dermatitides following exposure to various pool water chemicals – chlorine included – have also been reported.

Many common viruses, dermatophytes, and bacteria are waterborne and capable of causing cutaneous infection in humans. Tinea pedis, verruca vulgaris, molluscum contagiosum, *Pseudomonas* folliculitis, and atypical mycobacterial

N. R. Blank (✉)
Weill Cornell Medical College, Weill Cornell Medicine, New York, NY, USA
e-mail: nina.blank@gmail.com; nib2025@med.cornell.edu

D. E. Cohen
The Ronald O. Perelman Department of Dermatology, New York University School of Medicine, NYU Langone Medical Center, New York, NY, USA
e-mail: david.cohen@nyumc.org

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infections are all skin diseases that may be transmitted by contact with infected water and thus have potential to affect pool workers.

Ultraviolet light and water disinfection by-products such as trihalomethanes (THMs) and haloacetic acids can potentially increase cancer risk in swimming pool workers. THMs and chloroform are activated to mutagens by the enzyme glutathione-S-transferase-theta (GSTT1-1). In particular, bladder cancer risk from THMs seems to be higher in subjects with GSTT1-1 gene.

There is a paucity of literature specific to swimming pool workers; therefore, much of this chapter will extrapolate from relevant skin reactions reported in populations with similar exposures. Whenever possible, studies specific to swimming pool workers will be discussed and cited.

Keywords
Pool cleaners · Pool sanitizing agents · Ph stabilizers · Alkalization agents · Algaecides · Irritant contact dermatitis · Allergic contact dermatitis

1 Core Messages

- Pool cleaners may come into contact with several entities that are potentially hazardous to the skin, including infectious agents, contact allergens and irritants, ultraviolet radiation, and genotoxic byproducts.
- Chlorine, sodium hypochlorite, muriatic acid, and trichloro-s-triazinetrione (TST) are sanitizing agents that may cause irritant and/or contact dermatitides. Muriatic acid and TST have been reported to cause severe chemical burns when handled improperly.
- Cases of allergic contact dermatitis to brominated pool water manifest as pruritic rashes and/or widespread eczema after bathing in whirlpools treated with a bromine disinfectant. Allergy to "chlorinated water" is less frequently observed.
- Many common viruses, dermatophytes, and bacteria are waterborne and capable of causing cutaneous infection. Tinea pedis, verrucae vulgaris, molluscum contagiosum, *Pseudomonas* folliculitis, and atypical mycobacterial infections are all diseases that may be acquired through contact with infected water.
- Ultraviolet light and water disinfection by-products such as trihalomethanes (THMs) and halo acetic acids may increase cancer risk in swimming pool workers. THMs and chloroform are activated to mutagens by the enzyme glutathione-S-transferase-theta (GSTT1-1). Subjects with a GSTT1-1 gene may incur greater risk of bladder cancer from THMs.

2 Introduction

Swimming pool cleaners are a population largely absent from the dermatologic and occupational medicine literature, although there are several ways in which these workers are at risk of occupational skin disease. On a daily basis, pool cleaners handle a variety of entities that are potentially hazardous to the skin. The aquatic environment of the swimming pool exposes the worker to potential contact allergens and irritants and aids in the transmission of infectious agents. When workers clean outdoor pools, exposure to ultraviolet (UV) radiation may contribute to skin cancer. Despite these risks, few original investigations of skin problems that pool cleaners experience have been performed. Good safety practice on the job may prevent these conditions in the first place; alternatively, a lack of awareness of this population's risks may account for its absence in the dermatology literature. Whenever possible, studies specific to swimming pool workers will be discussed and cited in this chapter. Pool worker dermatoses will otherwise largely be hypothesized, based upon reactions in populations with similar exposures.

3 Irritant Dermatitis

Like other workers whose jobs dictate time spent with wet hands, pool workers are at baseline increased risk of skin symptoms, especially irritant contact dermatitis of the hands (Nielsen 1994;

Behroozy and Keegel 2014). The cumulative effect of handling multiple chemicals then places this population at added risk of occupational dermatitides. Commonly used pool chemicals are listed in Table 1.

Sanitizing agents commonly used in swimming pools include chlorine, bromine, iodine, hypochlorite salts, cyanuric acid, and trichloro-s-triazinetrione (TST).

Chlorine is the most commonly used pool disinfectant and is occasionally listed in texts as a chemical irritant (Fisher 1987). The CDC's Morbidity and Mortality Weekly Report reported chemical-induced dermatitis in 8 of 12 individuals within several hours of swimming in a pool with excessive free chlorine levels (10–17 ppm, state limit 5.0 ppm) and an inappropriately high pH of 9.0 (state pH maximum 8.0). Comorbid symptoms included headache, cough, sore throat, vomiting, and difficulty urinating (Hlavsa et al. 2014). In a study of pool hydrotherapists, the prevalence ratio of presumed irritant dermatitis was higher in pools chlorinated by gaseous chlorine than in pools disinfected with liquid- and solid-dissolved chlorine compounds (PR = 1.49, CI 1.17–1.89, $p = 0.017$) (Pardo et al. 2007). Pool workers handling various chlorine products should thus be aware of these risks.

Bromine is a disinfectant commonly used in whirlpools because it kills *Pseudomonas* at high temperatures more effectively than chlorine (Kim and Seo 1999; Fitzgerald et al. 1995). Irritant reactions appear to be more common from brominated swimming pools than from chlorinated water. Bromo-chloro-dimethylhydantoin (BCDMH) is one brominated product implicated in an outbreak of presumed irritant contact dermatitis among pool swimmers in the UK in 1983, although few patch tests were administered to rule out allergic sensitization (Rycroft and Penny 1983). BCDMH dermatitides have been described to manifest as widespread eczema, intense pruritus, and urticaria (Rycroft and Penny 1983; Loughney and Harrison 1998). Chemical burns secondary to bromine exposure have also been reported in workers despite the use of protection garments and washing of the skin after work. There is a characteristic delay in symptom manifestation, with blisters appearing 1–5 days after bromine contact. Bromine-injured areas should be washed with copious water to avoid extension of damage, and workers should be instructed to throw away all clothing with suspected bromine contact (Kim and Seo 1999).

Table 1 Chemical names

Sanitizing agents
Chlorine
Bromine
Iodine
Hypochlorite salt
Cyanuric acid
Trichloro-s-triazinetrione
Alkalinization agents and pH stabilizers
Sodium carbonate
Sodium bicarbonate
Sodium bisulfate
Hydrochloric (muriatic) acid
Algaecide components
Elemental copper
Copper sulfate
Quaternary ammonium ions

Iodine may also be used as a pool disinfectant (Nielsen 1994) and is often conjugated to polyvinylpyrrolidone to create povidone-iodine (PVP-I), commonly known as Betadine. PVP-I in pools is less irritating to swimmers than the chloramides that accumulate in chlorinated pools (Nielsen 1994) and in general is considered to have a low irritant potential when handled properly. PVP-I has, however, been reported to cause irritant contact dermatitis in exposed surgical patients, with presentations ranging from patchy erythema to vesicles, bullae, and burns (Borrego et al. 2016; Murthy and Krishnamurthy 2009). In Borrego's series of PVP-I-induced dermatitis, 13/13 patients patch tested were negative for PVP-I sensitivity (Borrego et al. 2016). Pool workers who mishandle PVP-I may therefore be at risk for acute irritant contact dermatitis.

Hypochlorite salts are bleaching and sanitizing agents that come in granular or tablet form. Sodium hypochlorite (bleach) is a known skin irritant (Hostynek et al. 1989; Piggott et al. 2007), although other unusual dermatologic reactions to bleach have been reported as well. In

1974, Coskey described onycholysis in a lifeguard, who for two consecutive summers was responsible for adding 16% sodium hypochlorite to her pool (Coskey 1974). Pellicano et al. described a young woman who developed linear IgA bullous dermatosis after an irritant contact dermatitis caused by sodium hypochlorite (Pellicano et al. 1997).

Calcium hypochlorite is an easily stored powder form of hypochlorite salt most commonly used in rural and small community water supplies (Yigit et al. 2009). It is a highly alkaline oxidizing agent that causes hyperhidrosis, fissuring, and painful tingling (Martinez and Long 1995). When handled inappropriately or mixed improperly with other pool chemicals, calcium hypochlorite has been known to cause explosions. A pool worker sustained first- and second-degree facial burns after mixing the salt – improperly stored at a high temperature – with water and then gazing into the violently bubbling disinfectant mixture he had just created (Yigit et al. 2009). Mixing with cyanuric acid – a pool chlorine stabilizer – was reported in 2010 to cause a blast sufficient to amputate a pool cleaner's digit and yield cutaneous burns (Shippert 2010).

Trichloro-s-triazinetrione (TST) is a sanitizing agent present in organic pool tablets. When mixed improperly with hypochlorite salts, TST may also lead to chemical burns and even fatalities (Martinez and Long 1995).

Some alkalinization agents and pH stabilizers have also been known to cause irritant contact dermatitides. Sodium carbonate is capable of producing a severe irritant dermatitis, with higher concentrations sufficient to cause ulceration. Hydrochloric (muriatic) acid, which may also be used in pool construction and cleaning, has been reported to yield irritant dermatitis and even chemical burns. Professional cleaning workers who use hydrochloric acid reported higher increased prevalence of hand dermatitis over non-cleaners (prevalence 36%, PR 1.92, 95% CI 1.22, 3.02) than did cleaners using other products (Mirabelli et al. 2012). In 2014, a pool construction worker died after collapsing into a mixture of hydrochloric acid and chlorinated rubber pool coating, in which he had remained soaking for 3 h (O'Cleireachain et al. 2014).

Both elemental copper and copper sulfate, which are components of some algaecides, are capable of producing an irritant dermatitis. Copper sulfate solution, when concentrated enough, can cause irritation. Elemental copper can irritate the skin in the presence of salt. In addition, green discoloration of hair and of seborrheic keratoses has been reported after repeated swimming in a pool with high concentrations of copper (Hinz et al. 2009; Peterson et al. 2006; Person 1985). Treatment with penicillamine shampoo (250 mg capsule dissolved in 5 ml of water and 5 ml of shampoo) using a bottled water rinse reverses green hair discoloration (Person 1985).

Quaternary ammonium ions have disinfectant properties and are often a component of algaecides. They can act as irritants and allergens even in very dilute solutions, with cross-reactivity occurring between different quaternary ammonium ions. Benzalkonium chloride is a particularly well-recognized skin irritant widely found in ophthalmologic preparations, skin cleansers, wound treatments, cosmetics, and personal hygiene products (Uter et al. 2008; Wentworth et al. 2016).

Frequent contact with solutions containing benzalkonium chloride therefore may place the pool worker at increased risk of irritant dermatitis.

4 Allergic Effects

Several of the commonly used pool chemicals detailed above may also cause allergic dermatitides. Chlorine and its by-products produce allergic reactions, albeit rarely (Nielsen 1994; Leung 1985; Sasseville et al. 1999; Hansen 1983; Dooms-Goossens et al. 1983; Kanerva et al. 1997; Neering 1977; Ng and Goh 1989; Osmundsen 1978; Bruch 2007). One case of erythema multiforme was hypothesized to be the result of swimming in a chlorinated pool, although patch testing was not performed to verify chlorine as the causative agent (Leung 1985). Chlorine and hypochlorite interconvert when dissolved in water, making it difficult to determine

which of the two might cause an allergic response following pool submersion (Sasseville et al. 1999).

Chloramine is a disinfectant and by-product of chlorinated water that is considered an allergen. It has been reported to cause patch test-positive allergic contact dermatitis on the hands and forearms of a nurse (Lombardi et al. 1989) and in a hospital cleaner (Hansen 1983), who, like pool cleaners, are occupationally exposed to the substance. Contact urticaria and respiratory symptoms after exposure to chloramine have also been reported (Dooms-Goossens et al. 1983; Kanerva et al. 1997; Neering 1977). For example, a nurse who had previously used chloramine developed contact urticaria and respiratory distress while again using the disinfectant, to which she subsequently patch tested positive with varying dilutions (Dooms-Goossens et al. 1983).

Two case series have reported patch test-positive cases of allergic contact dermatitis to brominated pool water disinfected with BCDMH. All 13 patients in both series developed pruritic rashes and/or widespread eczema and subsequently patch tested positive with BCDMH (Dalmau et al. 2012; Fitzgerald et al. 1995). Potassium peroxymonosulfate is used often in brominated swimming pools as an oxidizing agent to remove built-up amines and organic contaminants. It has been reported to independently cause patch test-positive contact dermatitis in swimmers (Salvaggio et al. 2013; Gilligan et al. 2010).

Allergic reactions to PVP-I have been reported in hospital patients and include cases of allergic contact dermatitis (Velazquez et al. 2009; Rahimi and Lazarou 2010), contact urticaria-angioedema (López Sáez et al. 1998), iododerma-like eruptions (Massé et al. 2008), and even anaphylactic shock (Caballero et al. 2010). In a series of 7 post-surgical patients with contact dermatitis and 30 control subjects, investigators posited that PVP-I-induced dermatitis involves both an irritant and an allergic mechanism. While PVP-I in water yielded positive patch tests in patients and controls at 48 h with diminished response at 96 h, reactions to PVP-I in petrolatum increased in intensity over time and were positive only in the study group (de la Cuadra-Oyanguren et al. 2014). There are reports of occupational allergic contact dermatitis to PVP-I in doctors, nurses, laboratory technicians, masseuses, and butchers (Sato et al. 2004), but none in swimming pool cleaners, who perhaps do not have prolonged exposure to the substance.

Allergic reactions to hypochlorite salts are also rare. One case of type I hypersensitivity to sodium hypochlorite has been reported, manifesting as a recurrent, itchy, non-tender, urticarial rash over the face, upper limb, and trunk in an operating room technician. A prick test – performed along with internal positive and negative controls – was positive (Zhe et al. 2016). Neering described a patient who developed urticaria after swimming in a pool treated with sodium hypochlorite, and the patient subsequently patch tested positive to sodium hypochlorite and calcium hypochlorite (Neering 1977). In 1989, a surgical patient was reported to develop a hypochlorite allergy when the chemical was used as a dressing disinfectant. He subsequently could not be exposed to chlorinated water without developing a generalized, week-long rash (Ng and Goh 1989). Several other reports of allergic contact dermatitis to sodium hypochlorite exist, with sensitization occurring after routine exposures to house cleaning chemicals and disinfectants (Bruch 2007).

Some algaecide components have been described to cause allergic contact dermatitides. One example is copper, which has been reported to cause patch test-positive dermatitis in exposed individuals (Fage et al. 2014). In one report, ten furniture polishers who used a solution colored with copper sulfate developed contact hand dermatitis and subsequently patch tested positive to copper sulfate (Dhir et al. 1977). This may be the report most relevant to pool cleaners, who also work with copper sulfate in solution.

Norrlind first reported cases of benzalkonium allergy in 1953 (Norrlind and Wahlberg 1962). His patients and patients reported by Wahlberg in 1962 (Wahlberg 1962) had all been treated with benzalkonium chloride for skin or wound disinfection. Since these reports, several series have investigated rates of benzalkonium chloride allergy. A Mayo Clinic review suggested an

increasing rate of patch test positivity to benzalkonium chloride between 1998 and 2010. Investigators accounted for irritant reactions in their readings (Wentworth et al. 2016). Other epidemiologic studies have revealed contact allergic sensitivities of 0–10% (Perrenoud et al. 1994; Dastychová et al. 2008).

Reports of allergy to benzalkonium chloride are numerous and of varied etiology. Many reports of allergic contact dermatitis to benzalkonium chloride come from people in the health-care professions. Nurses, who have many opportunities to be exposed to disinfecting agents such as benzalkonium chloride, are frequently affected (Suneja and Belsito 2008). There are previous reports of an allergy developing after cast material containing benzalkonium chloride was used to set broken bones (Stanford and Georgouras 1996; Staniforth 1980). Huriez found high sensitization rates in patients with a long history of using topical ointments with quaternary ammonium ions (Huriez et al. 1962). This study may have particular relevance for pool workers, whose contact with such compounds would also occur over a more protracted period of time.

5 Infections

Many common viruses, dermatophytes, and bacteria are waterborne and capable of causing cutaneous infection in humans. Fungal infections such as tinea pedis, *Mycobacterium marinum*-induced granulomas, *Pseudomonas* folliculitis, molluscum contagiosum, and verruca vulgaris are all skin diseases that may be contracted through infected water.

Fungal entities commonly yield skin infections in pool employees. A cross-sectional study of 133 indoor swimming pool workers showed a significantly higher prevalence of cutaneous mycosis in pool attendants (lifeguards and trainers) compared to other swimming pool workers (office, cafe, etc.) (Fantuzzi et al. 2010). Compared with a control population who did not frequent swimming pools, a cohort of 169 swimming pool employees was significantly more likely to have tinea pedis (RR = 15), onychomycosis (RR = 3), or both (RR = 20) (Shemer et al. 2016).

Mycobacterium marinum is an atypical mycobacterium and rare contagion that is free living in water. Because the organism cannot penetrate intact skin, *M. marinum* skin infection in humans is usually preceded by a cut or abrasion (Hirsh and Johnson 1984). *M. marinum* has been isolated from several aquatic sources, including swimming pools ("swimming pool granuloma"), infected fish, or tropical fish tanks ("fish tank granuloma," "fish fancier's finger").

Incubation lasts 1 week to 2 months after exposure. An erythematous, verrucous papule then appears at the site of injury, often an easily traumatized bony prominence such as the knee or elbow (Hautmann and Lotti 1994). The papule may enlarge to form a granulomatous nodule or may form a plaque with nearby papules, and either form can ulcerate and drain purulent material. Infection in immunocompetent hosts is usually painless and self-limited and rarely disseminates (Waddington 1967). Lymphangitic, "sporotrichoid"-patterned spread may occur in 20–40% of cases (Hirsh and Johnson 1984; Dolenc-Voljc and Zolnir-Dovc 2010). Also infrequently, infection involves deeper structures to cause tenosynovitis, osteomyelitis, arthritis, bursitis, or carpal tunnel syndrome (Dolenc-Voljc and Zolnir-Dovc 2010; Gluckman 1995). *M. marinum* diagnosis is frequently delayed due to unspecific histology and low clinician awareness of the rare infection. The diagnostic gold standard is culture from a biopsied lesion, which is positive in 70–80% of cases (Dolenc-Voljc and Zolnir-Dovc 2010). Prognosis is related to disease extent and host immune response. A retrospective study of 136 patients with *M. marinum* infection showed that patients with deep structure involvement have poor prognosis, especially when an operation is required (Cheung et al. 2010).

Swimming pool-related *M. marinum* granuloma outbreaks were common early in the twentieth century (Waddington 1967; Mollohan and Romer 1961; Linell and Norden 1954) but have decreased precipitously since the 1960s, likely due to improved pool disinfection and chlorination (Dolenc-Voljc and Zolnir-Dovc 2010).

Sources do differ, however, as to whether *M. marinum* is truly resistant to chlorine (Hautmann and Lotti 1994; Gluckman 1995; Kirk and Kaminski 1976; Fisher 1988). Sporadic *M. marinum* infection has been reported in pool workers and swimmers in recent decades (Dolenc-Voljc and Zolnir-Dovc 2010; Fisher 1988; Johnston and Izumi 1987), but most recent cases are unrelated to swimming pools.

Pseudomonas aeruginosa is a waterborne, folliculitis-causing gram-negative rod whose ability to form a biofilm allows it to survive in even chlorine-treated environments (Guida et al. 2016). Its O:11 serotype is most commonly implicated in causing folliculitis. The infection is more common following whirlpool bath than swimming pool use, as high whirlpool temperatures promote organism growth and water agitation causes loss of the chlorine necessary to control bacterial overgrowth.

McCausland and Cox described the first outbreak of *Pseudomonas* folliculitis in 1975 (McCausland and Cox 1975). Since then, numerous cases have been reported, including several outbreaks related to hot tubs, spas, and inflatable swimming pools (Centers for Disease Control and Prevention 2000; Tate et al. 2003; Yoder et al. 2008). The rash is papulopustular, usually pruritic, and coincides with areas of the body exposed to the whirlpool (Sausker 1987). The folliculitis has a predilection for sites with apocrine sweat glands such as the axilla and groin. It does not occur on the palms or soles. Other manifestations of infection with *Pseudomonas* include fever, malaise and fatigue, and, occasionally, mastitis and otitis externa.

While *Pseudomonas* infections more common than those caused by M. *marinum*, the infection may have less relevance to swimming pool cleaners. The hands – likely a pool worker's most exposed site – have a paucity of hair follicles and are thus unlikely to develop *Pseudomonas* folliculitis. It also appears that extended immersion in water is necessary to induce infection. "Superhydration" of the skin has been shown to promote *Pseudomonas* growth (Taplin et al. 1965; Hojyo-Tomoka et al. 1973). Hydration with occlusion – as would occur under a bathing suit – has also been demonstrated to increase *P. aeruginosa* colony counts (Hojyo-Tomoka et al. 1973). Furthermore, increased time of contact with *Pseudomonas*-contaminated water has been correlated with increased infection risk (Hudson et al. 1985). It is possible, therefore, that a pool worker's exposure to *Pseudomonas*-infected water would be too incidental to cause infection.

Rarely, *Aeromonas hydrophila* folliculitis may be associated with inflatable swimming pools and presents with the same symptoms as *P. aeruginosa* folliculitis (Julià Manresa et al. 2009).

Pox viruses causing molluscum contagiosum have been reported to spread through infected pool water (Choong and Roberts 1999), and there is an established association between recent history of swimming and development of molluscum contagiosum in children (Olsen et al. 2014). Other manifestations of viral infection – such as verrucae – may also be contracted through contact with pool water (Conklin 1990; Gentles and Evans 1973). A recent report isolated human papillomavirus, including *Betapapillomavirus* species 1 and 2, from 7 of 14 samples from indoor and outdoor chlorinated swimming pools (La Rosa et al. 2015), although verrucae-causing HPVs are typically thought to spread via direct physical contact with shower and changing room floors contaminated with infected skin fragments. Fantuzzi et al. did report a higher prevalence of verrucae in pool attendants compared with other staff; however, this did not reach statistical significance (Fantuzzi et al. 2010). Ultimately, the risk to pool cleaners of contracting viral skin conditions such as molluscum contagiosum and verrucae is poorly defined. It is unlikely that pool cleaners work in their bare feet, although it is possible that sandal-wearing in the summer may predispose them to contact with infected water.

6 Cancer

Pool cleaners can certainly be categorized as outdoor workers, although their UV exposure has not been quantified. Exposure likely varies

significantly with region, season, and indoor versus outdoor pool work, making risk of UV-induced skin cancer difficult to estimate.

The toxicity of disinfection by-products (DBPs) has been studied extensively, and some components have demonstrated carcinogenic and mutagenic properties (Panyakapo et al. 2008; Chen et al. 2011; Zwiener et al. 2007). Chlorine generates by-products from its chemical reaction with pool water's organic matter, the quantity of which – along with pH, temperature, duration of exposure, and bromide ions – can affect the reaction (Panyakapo et al. 2008). More organic products and bromide ions increase formation of halogenated organic compounds such as trihalomethanes (THMs) and haloacetic acids (HAAs). The THMs chloroform, bromodichloromethane, dibromochloromethane, and bromoforme have all been reported as human carcinogens. A risk assessment study using the USEPA (United States Environmental and Protective Agency) method on swimming pool water concluded that the worst-case scenario (highest possible THM concentrations) yielded an unacceptable level of lifetime cancer risk (Panyakapo et al. 2008). The presence of other carcinogens (haloacetic acids, haloketones, and chlorophenols), along with poor ventilation in indoor pools, may all further increase cancer risk (Panyakapo et al. 2008; Chen et al. 2011).

No studies have explicitly implicated DBPs in causing skin cancer. One preliminary case-control study reported odds ratios of 2.4 (95% CI 0.9–6.7) for basal cell carcinoma and 2.1 (0.7–7.0) for squamous cell carcinoma among users of public water systems with the highest levels of THMs, as compared to reference groups with trace or no THMs. While these findings lacked statistical significance, the authors felt the hypothesis that DBP exposure could affect skin cancer pathogenesis warranted further study (Karagas et al. 2008).

THMs and chloroform are activated to mutagens by the enzyme glutathione-S-transferase-theta (GSTT1–1). GSTT1 is expressed in the skin (Karagas et al. 2008); however, bladder cancer risk from THMs seems particularly high in subjects with GSTT1–1 gene. This gene is present in 80% of Caucasians but in only 20% of Asians. Studies on alternative methods for water purification also demonstrated that bromide disinfection by-products are more cytotoxic and carcinogenic than chlorine-treated water (Zwiener et al. 2007).

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