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Global Warming Favors Pathogenicity of the Brain-Eating Amoebae

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With the global warming and extremely hot summers being witnessed throughout the globe in recent years, there are reports of increasing numbers of heat strokes, as well as an upsurge in the incidence of fatal encephalitis related with a group of Free-living Amoebae (FLA) so-called as the "brain eating amoebae".

There is an established relationship between the recent trend of mercury rising with the incidences of patients affected by encephalitis caused by *Naegleria fowleri* in the US and south east Asia. This parasitic amoeba is known to assume a trophozoite form (infective state) in heated environment [1] (Fig. **1A**) when the mercury touches between 4245°C. Accidental entry of water deep into the nose initiates a

cascade in the trophozoites that reach the brain by traversing the cribriform plate (Fig. **1B**) located at the root of the nose. The affected patients are known to suffer Primary amoebic meningoencephalitis (PAM), the mortality rate of which has remained around 98%, with less than 5 survivors in the past 50 years [2]. Two other related FLA, *Acanthamoeba* and *Balamuthia mandrillaris* are also heat-loving parasites that acquire a pathogenic trophozoite state in heated environment [3] (Fig. **1**). Though, a bit less flared-up than PAM, both these pathogens can also cause a central nervous system disease called Granulomatous Amoebic Encephalitis (GAE) [1, 3]. The mortality rate of the later infection has also remained around 95% [3]. Additionally, the *Acanthamoeba* spp. behaves as a host to many environmental bacteria in its trophozoite form [3] (Fig. **1D**), a state being favored by high temperatures [1]. Here we highlight the overt and covert roles played by the FLA that are favored by the high temperatures and global warming. Of the FLA, the *Naegleria fowleri* thrives in fresh waters like ponds, lakes, rivers and pools [1, 2]. This microbe acquires a cystic form (Fig. **1A-A2**) in winters, to acquire a pathogenic form in heated summers with above-mentioned temperatures [1]. Mostly, the patients with PAM have a history of exposure to fresh water in activities like swimming (Fig. **1B**), water sports, nasal irrigation (Fig. **1B**) and ablution [1, 2]. Public awareness regarding the mode of infection in PAM by altered environmental influences remains the only hope in reducing the incidence of this fatal disease [2]. As entry of contaminated water higher up in the nose is the solitary mode of infection by *Naegleria fowleri* to cause PAM [2] (Fig. **1B1**), avoidance of introducing water high up in the nose during swimming, ablution and nasal cleansing could also be important in the cases of PAM [1, 2]. Adequate chlorination of the swimming pools in parts of the globe where high temperatures are recoded annually [1] could also reduce the occurrences of PAM. The high environmental temperatures also favor the existence of active feeding trophozoite forms of (Fig. **1A- A1**) *Acanthamoeba* and *Balamuthia*, which is the state in which they infect humans (3). The *Acanthamoeba* spp. causes a vision compromising *Acanthamoeba* keratitis (Fig. **1C-C1**) (AK) and skin infections (Fig. **1C-C2**) in addition to GAE [2, 3]. If polluted water contaminated with *Acanthamoeba* is used for cleaning the contact lenses, the chances of AK (Fig. **1C-C1**) become high [3].

The decreased humidity with high temperatures is an additional factor that influences the chances of corneal trauma and predisposes it to AK in the contact lens wearers. These factors should be taken into consideration while wearing and prescribing contact lenses for visual acuity or cosmetic purposes. The stored water in reservoirs that gets heated up in summers favors the trophozoite form of *Acanthamoeba* and if the tap-water connected to these reservoirs is used to clean the contact lenses, it may predispose to AK [3]. Skin infection by this pathogen results in chronic non-healing wounds (Fig. **1C-C2**), from where the *Acanthamoeba* makes its way to the brain *via* hematogenous route (Fig. **1C**). Unlike *Acanthamoeba* spp., *Balamuthia mandrillaris* does not cause ocular keratitis, but can cause a skin or air-sinus infection, to later spread to the brain via blood and cause GAE [1, 3].

Acanthamoeba spp. imposes an additional danger by acting as a reservoir for bacterial pathogens. This hosting of bacteria (Fig. **1D**) has been associated with the acquisition of a multi-drug resistant attribute by the pathogen [3]. Once these microbes exit the *Acanthamoeba*, they are believed to cause fulminating infections in human against which the antibiotics are non-functional (Fig. **1D**). High temperatures, therefore, impose a dual threat by favoring the trophozoite stage of *Acanthamoeba*, that not only increases the chances of infection by this protist pathogen itself but favors the production of MDR strains of the hosted bacteria [3] (Fig. **1D**). The fact that *Acanthamoeba* has been found in tap waters of the hospitals where nosocomial infections are routine, it is not difficult to understand why the incidences of sepsis and nosocomial infections increase in summers [4]. Additionally, FLA are constantly detected in treated drinking water systems around the globe and present a yet undetermined emerging health risk [5].

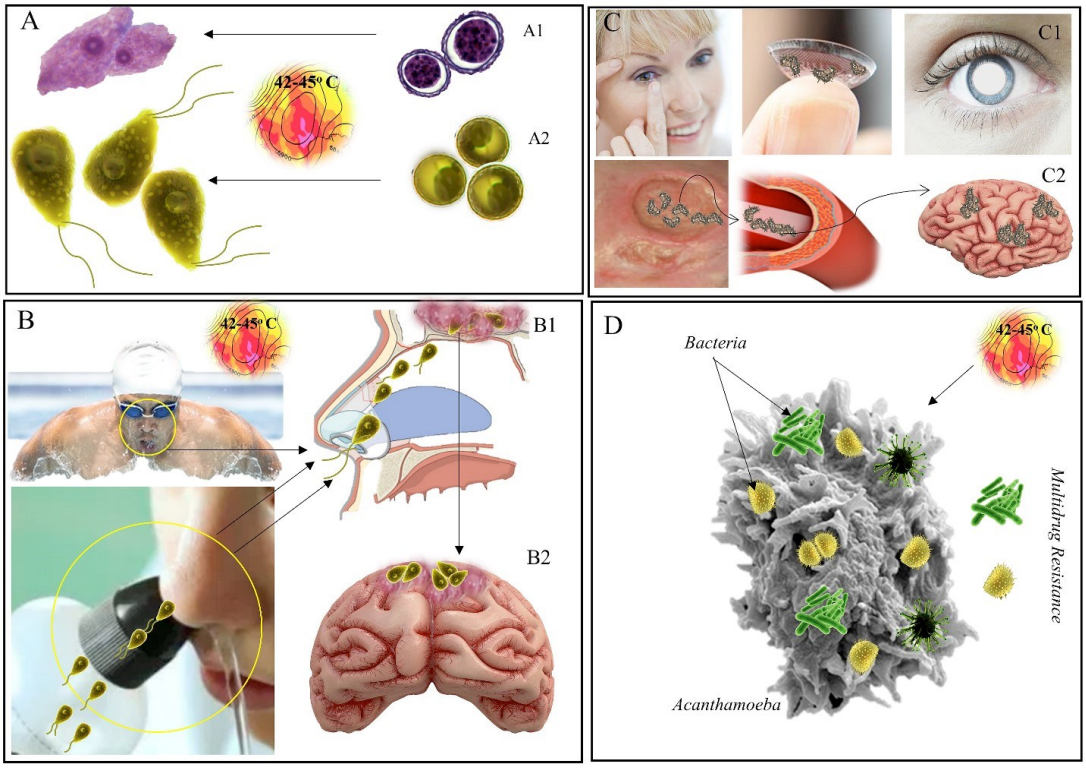

Fig. (1). (**A**) Shows high environmental temperature favoring conversation of cystic forms of free-living amoeba (FLA) into pathogenic trophozoite forms. (**B**) Mode of infection by *Naegleria fowleri.* Accidental entry of contaminated water high up in the nose enables the parasite to ascend and spread to the brain via cribriform plate (B1). Brain develops acute inflammatory lesion and causes death in PAM. (**C**) Shows mode of infection in *Acanthamoeba* keratitis resulting in corneal opacity (C1). Wound infections by *Acanthamoeba* and *Balamuthia* (C2) eventually spread to the brain via blood stream and cause GAE. (**D**) Shows bacterial hosting by *Acanthamoeba* that enables them to turn multi-drug resistant and cause nosocomial infections, particularly in high environmental temperatures.

We hint towards the enormous contribution of *Acanthamoeba* spp. in giving origins to MDR strains of bacteria and the subsequent infections that they cause, which are impossible to treat in clinics and hospitals. Cystic forms of *Acanthamoeba* (Fig.1A-A1) in particular have imposed additional burdens in eradication of the infections caused by them [3]. Cysts forms of this protist survive very well in environments like soil, water and air. *Acanthamoeba* is known to survive in cystic forms for months to years [1, 3]. Creating health-supportive environments free of contaminated soil, water and air in hospitals and clinics could reduce the occurrence of AK and GAE, in immunocompromized patients in particular, who are well known to get affected by *Acanthamoeba* and *Balamuthia* infections [1, 3]. Acquisition of FLA infections with agents like *Acanthamoeba* and *Balamuthia* via organ transplants [6] has also emerged as a new threat of infection transmission, and screening is now being done for the presence of FLA before organ transplantations.

For *Naegleria fowleri*, the health-supportive environmental deliberation that needs to be provided is adequate chlorination of swimming pool and storage waters that are used for ablution and nasal cleansing [2], especially during summers. The use of nasal clips and nasal plugs during swimming and water sports in warm fresh waters in summers can also minimize the incidences of PAM. As the mortality rates of the cerebral infections caused by these brain eating amoeba remain above 95%, until a reduction of global warming improves the summer temperatures, implementing a health-supportive environment is the only viable option to control MDR infections and the incidences of PAM and GAE.

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