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Full Length Research Paper

# The Pharmacological Modulation of Human Photosynthesis: A Real Hope for Bhopal, India

Arturo Solís-Herrera\*, María del Carmen Arias Esparza C.\*\*, Martha P. Solís-Arias\*\*.

Human Photosynthesis Study Center, \*, \*\*. López Velarde 108 y 109, centro; Aguascalientes, Aguascalientes, México, CP. 20000. Phone: 0524499150042, fax: 0524499160048,

\*Corresponding Author Email: comagua2000@yahoo.com

Abstract

Massive cases of accidental acute pesticide poisoning account for significant morbidity and mortality worldwide, especially in developing countries. There are no reliable estimates as to how many people per year suffer from pesticide-related health effects. This is due to several reasons including a lack of standardized case definition. The case definition includes of all circumstances of poisoning including suicide, homicide, and non-intentional (accidental exposure) and occupational. In developing countries, where there is insufficient regulation, lack of surveillance systems, less enforcement, lack of training, inadequate access to information systems, poorly maintained or non- existent personal protective equipment, and larger agriculturally-based populations, the incidence are expected to be higher. The use of pesticides banned in industrialized countries, in particular highly toxic pesticides, obsolete stockpiles and improper storage techniques may provide unique risks in the developing world. One of the most dramatic examples was with methyl isocyanate, in Bhopal, India, in 1984, and whose effects continue to date.

Keywords: Human Photosynthesis, Melanin, water splitting, energy, Alzheimer, Bhopal.

## INTRODUCTION

On the night of 2/3 December 1984, a major accident occurred in Bhopal, India, there was an apparently accidental leakage of methyl isocyanate storage tank at a pesticide plant owned by the Union Carbide Corporation (Bogard 1989). This accident triggered a long-term industrial crisis for the entire population of Bhopal, and the consequences continue to date, and also for government agencies in India, and for the Union Carbide Corporation. Industrial crises are processes of severe disruption and harm that originate in industrial activities and technological systems. They affect people, property, and the natural environment. Communities, corporations, and government agencies are sometimes restructured in the wake of these crises.

Bhopal gas tragedy was a methyl isocyanate leak incident in India, considered the world's worst industrial disaster. Methyl isocyanate is and intermediate to produce the pesticide carbaryl (Sevin<sup>TM</sup>). Briefly the chemical process employed was methylamine + phosgene = methyl isocyanate + 1-naphtol = carbaryl. Bhopal gas tragedy was a methyl isocyanate leak incident in India, considered the world's worst industrial disaster. Methyl isocyanate is and intermediate to produce the pesticide carbaryl (Sevin<sup>TM</sup>). Briefly the chemical process employed was methylamine + phosgene = methyl isocyanate + 1-naphtol = carbaryl.

The Bhopal gas tragedy was preceded by several accidents of exposed plant's workers with some deaths. The methyl isocyanate leak included problems such as: storing the methyl isocyanate in large tanks and filling beyond recommended levels, failure of several safety systems due to poor maintenance and safety systems being switched off to save money. Beside workers were forced to use English manuals.

## Acute effects

The initial effects of exposure were coughing, vomiting, severe eye irritation, and a feeling of suffocation, at the light of human photosynthesis there were an abrupt diminishing of turnover rate of the water dissociation and its consequent reformation of the water molecule, thereby the low levels of free chemical energy into the cell, producing a generalized failure of the systems that form the human body. The severe coughing reflects damage to the lungs, for instance.

During Bhopal gas disaster. Lung was one of the most commonly affected organs, Sajal, 2012). The exact nature of the toxic responsible for this disaster is disputed and multiple chemical substances are attributed (Rao et al., 1991). Bhopal. The MIC aerosol was also super-added with its pyrolytic products i.e. hydrogen cyanide, nitrogen oxides and carbon monoxide besides contaminants such as phosgene and mono-methylamine (Sriramachari, 2004). Most of the immediate deaths after the disaster were due to involvement of lung. Various studies have documented the high incidence of respiratory morbidity among the surviving population due to single high level exposure of MIC (Weill, 1987).

Chronic inflammation of the respiratory tract is attributed for the long-term morbidity and respiratory function abnormality. Study conducted within a few months after the disaster had demonstrate the presence of bronchiolitis obliterans and a subsequent study reported the development of pulmonary fibrosis and interstitial pneumonia confirmed both histo-pathologically (Vijayan et al., 1995). In 1985, one year after the disaster, spirometry of gas victims shown that the mixed pattern was the commonest lung function abnormality (Rastogi et al., 1988).

Obstructive pattern was the commonest abnormality observed (50.8 %), restrictive patter in 13.3%; and normal spirometry in 35.8 %. Irreversible obstruction was seen in 24 % and reversible obstruction in 26.8 % of cases.

Our histo-pathological observations in lung of Wistar rat As exposed groups, are congruous with the restrictive pattern in 49.1 % reported by Naik et al., 1986, fifteen weeks after the disaster. The histological changes produced by As in our laboratory animals were decreased significantly in the experimental group in which, while the animal was exposed to As at the same period time photosynthesis process was intensified pharmacologically, as can be seen in the following photomicrograph:

The persistent small airways obstruction among survivors of the 1984 disaster may be attributed to gas exposure (Beckett, 1998).

Thousands of people succumbed by the morning hours. Within few days leaves on tree yellowed and fell off. The dissociation of the water molecule in plants and humans has impairment common, a previously observed fact; i.e. cold turn down the photosynthesis process in humans and plants, and also pesticides, herbicides, etc. Sadly Union Carbide was more interested in getting information from Indian government than in helping the relief work. The causes of death were choking, circulatory collapse, cerebral and pulmonary edema, tubular necrosis of the kidneys, fatty degeneration of liver and necrotizing enteritis. These data of generalized failure are characteristic of low levels of free chemical energy into the cell medium. Recall without a stable source of energy, cell will die in seconds.

Clinical studies have shown chronic illnesses such as pulmonary fibrosis, bronchial asthma, chronic obstructive pulmonary disease (COPD), emphysema, recurrent chest infections, keratophaty and corneal illnesses in exposed cohorts.

The water is under intense and constant movement through the cell, which is a fundamental process for life, when is significantly damaged, it's manifested by swelling of the affected tissues. At microscopic examination data cells show necrosis or accidental death, as the histological findings show no order as we can find in apoptosis.

Without an adequate level of free chemical energy, the cell is unable to do what she can do and that has made millions of years. Therefore, try to correct fault fails, is a formidable challenge, given the complexity of an organism that has four billion years of evolution is far beyond our ability to abstraction. Suffice it to recall that we are not able to make one hair.

And the large number of deaths and the terrible consequences that disaster survivors crawl leaves no doubt. Survivors continue to experience higher incidence of reported health problems including febrile illnesses, respiratory, neurologic, psychiatric and ophthalmic symptoms Figure1a and 1b.

The persistent toxic effects of MIC in the survivors of the tragedy have been studied, and the results displayed a significant increase in the levels of al circulating inflammatory biomarkers in the MIC exposed group in comparison to non-exposed cohorts (Bhargava et al., 2010), being the mechanism poorly understood. However at the light of human photosynthesis the golden standard returns to work, if the failure is widespread, we must first think about energy, and in the case of the eukaryotic cell we mean the free chemical energy levels, which, according to the persistent symptomatology, they are impoverished chronically.

### Toxicity of Methyl Isocyanate; McConnell et al., 1887).

Methyl isocyanate (MIC) is a highly toxic chemical that was responsible for the deaths of over 2000 people, hospitalization of over 50 000, and significant exposure of over 320 000 people in Bhopal India.

MIC is used for manufacture of various pesticides, the most important of which are aldicarb, carbaryl and methomyl. MIC, made by reacting methylamine with phosgene, has a boiling point of 39.1 °C at 1 at m, a



**Figure 1a.** Cornea, Guinea pig, developing corneal angiogenesis at 15 days to reduce tissue photosynthesis intentionally with a single drop of aromatic compounds that decrease the turnover rate of water dissociation and re-formation.

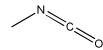


**Figure 1b.** On the cornea of Wistar rats, the angiogenic response is also clear and remarkably pure, with sparse fibrous tissue. The human photosynthesis in this case, of mammals, seems to antagonize the number of blood vessels. The greater the intensity of photosynthesis means smaller number of blood vessels and vice versa.

density of 0.960 g/cm<sup>3</sup>, and a vapor pressure of 348 Torr at 20  $^{\circ}$ C. It is flammable and highly reactive with water.

U.S. production is estimated to be between 30 and 35 million pounds per year.

Methyl Isocyanate



Chemical Formula: C<sub>2</sub>H<sub>3</sub>NO Exact Mass: 57.02 Molecular Weight: 57.05 m/z: 57.02 (100.0%), 58.02 (2.5%)

Elemental Analysis: C, 42.10; H, 5.30; N, 24.55; O, 28.04

MIC is highly irritating and causes severe bronchial spasms, asthmatic breathing, and chemical pneumonia when toxic levels are inhaled. Contact with intact skin, mucous membranes, and other moist surfaces such as the eyes causes irritation and burns. The nature of short and long term sequel arising from a single exposure to MIC is not well known; presently the knowledge of the MIC toxicology is scant.

The histo-pathological findings reported in lung's rats exposed to MIC were not very different from the findings of rats exposed to As. Necrosis was accompanied by transudation, exudation, and variable amount of blood. Animals that die after four days showed plugging of airways with necrotic epithelial cells, proteinaceous debris, and an influx of inflammatory cells. Compromises of pulmonary function paralleled the morphologic changes observed in the respiratory tract.

MIC is a highly toxic gas in rodents as well as humans, and As behaves similarly. Pulmonary function studies demonstrated significant pulmonary compromise and suggested the presence of persistence obstructive airway disease. In our opinion, the persistence of histopathological changes is given by the MIC or its metabolites that remain inside the body, as the data are compatible with a chronically low photosynthesis, whose typical histo-pathological characteristics are fibrosis and mitosis.

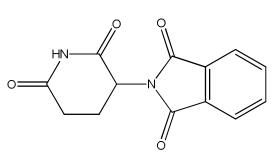
Although the literature is said that the possibilities of significant extra-pulmonary damage are low, histologic sections show liver damage, kidney and bone marrow.

#### Toxicity study, A formidable task

Animal studies are poor predictors of human reactions to exposure. The concept that animal research, particularly that relating to pharmaceuticals and environmental agents, may be a poor predictor of human experience is not new. A thousand years ago, Ibn Sina commented on the need to study humans rather than animals, and Alexander Pope's dictum: *"The proper study of mankind is man"* is well known and has been widely cited. Pharmacologists, in particular, have long recognized the difficulties inherent in extrapolating drug data from animals to man (Bracken, 2008).

Although it has been possible to detect arsenic in an autopsy since the early 1800's, occasionally slips by the coroner. Surprisingly, it is difficult to detect arsenic in a living person (David, 1993).

Thalidomide

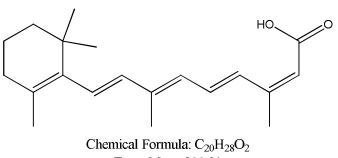


Chemical Formula: C<sub>13</sub>H<sub>10</sub>N<sub>2</sub>O<sub>4</sub> Exact Mass: 258.06 Molecular Weight: 258.23 m/z: 258.06 (100.0%), 259.07 (14.3%), 260.07 (1.8%)

Elemental Analysis: C, 60.47; H, 3.90; N, 10.85; O, 24.78

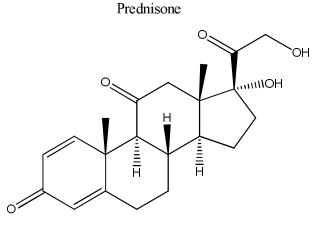
Despite its controversial past, thalidomide is currently under investigation for the treatment of several disease types, ranging from inflammatory conditions to cancer. The mechanism of action of thalidomide is complex and not vet fully understood, but there is some evidence to suggest that metabolism may play a role. Consequently, there has been a considerable effort to characterize the metabolism of thalidomide in recent years. Thalidomide undergoes biotransformation by non-enzymatic hydrolysis and enzyme-mediated hydroxylation to form a multitude of metabolites, around 1200 different molecules (Lepper et al., 2006). Thalidomide is not a teratogen in many animal species but it is in humans.

Isotretinoin



Exact Mass: 300.21 Molecular Weight: 300.44 m/z: 300.21 (100.0%), 301.21 (21.7%), 302.22 (2.3%) Elemental Analysis: C, 79.96; H, 9.39; O, 10.65

Isotretinoin causes birth defects in rabbits and monkeys as well as in humans, although not in mice or rats.



Chemical Formula: C<sub>21</sub>H<sub>26</sub>O<sub>5</sub> Exact Mass: 358.18 Molecular Weight: 358.43 m/z: 358.18 (100.0%), 359.18 (23.2%), 360.18 (3.5%) Elemental Analysis: C, 70.37; H, 7.31; O, 22.32

Corticosteroids are widely teratogenic in animals but not in humans.

Recent experience in a phase 1 study of the monoclonal antibody TGN 1412 resulted in lifethreatening morbidity in all six healthy volunteers, reflecting inadequate prediction even in non-human primates of the human response.

### Perfluorinated substances

Clear examples of toxic compounds that enter the body and can remain in either the parent compound or metabolites thereof, for the remainder of life are the perfluorinated chemicals (PFOS) (Lin et al., 2013). Higher serum concentrations of PFOS were associated with an increase of carotid intima-media thickness (IMT) in a studied cohort of adolescents and young adults.

The widespread distribution and degradation of PFCs in the environment results in a very complex exposure pattern, which makes it difficult to define the relative contribution to human exposure from different exposure pathways.

# Progressive Fibrosis due to low level photosynthesis in humans

The negative effects of MIC on the body does not seem to stop despite the passage of time, it is possible that we have another substance more (MIC) that it or its metabolites are able to remain indefinitely in the body, causing progressive damage due to its ability to decrease the photosynthetic capacity of the human.

When human photosynthesis is decreased in chronic form, the cells tend to mitosis or to synthesize collagen constantly which is characteristic of progressive fibrosis. There is a strong association between exposure gadolinium-based contrast agents and Nephrogenic Systemic Fibrosis (NSF) in patients with kidney disease, particularly in the presence of a pro-inflammatory process such as major surgery, infection, or a vascular thromboembolic event (Sadowski et al., 2007). Gadolinium chelates are commonly used to improve tissue contrast in MRI. NSF can cause systemic fibrosis, leading to disabling contractures and even death (Penfield and Reilly, 2007).

Gadolinium is a rare earth element from the lanthanide series that is used as a contrast agent in MRI because of its powerful paramagnetic properties. Its seven unpaired electrons perturb proton relaxation in water, altering thereby the complex properties of water, a fundamental actor in human photosynthesis.

Gadolinium in its unbound state is highly toxic. It is a potent inhibitor of calcium channels and has considerable cardiovascular and neurologic toxicity. Free gadolinium is deposited in liver, bone and lymph nodes and, once there; is slowly released from the boy at a rate of les of 1 % by day. The toxicity diffuse, affecting various organs and systems, is characteristic of compounds that lower photosynthesis.

# The Human Photosynthesis enhancement, a real hope for Bhopal

After 23 years of having detected in human retina the first data that led me to the discovery that the very first reaction in both plant life and humans is the same, that is, the dissociation of the water molecule. Finally, in 2012; we found the previously unknown intrinsic property of melanin to dissociate and re-form the water molecule Figure 2.

The Solís-Herrera Cycle can be schematized as follows:

## $2H_2O \leftrightarrow 2H_2 + O_2 + 4e^{-1}$

Wherein melanin molecule, in the presence of light and water expresses its previously unknown ability to absorb the entire electromagnetic spectrum and dissipate the absorbed photonic energy through the dissociation of the water molecule.

The reaction was termed as human photosynthesis by analogy with the irreversible dissociation that chlorophyll makes in plants and can be schematized as follows:

### $2H_2O \rightarrow 2H_2 + O_2$

The unsuspected capacity of melanin to make at room temperature a biochemical reaction that in laboratory needs 2000 °C is awe-inspiring. And by



Figure 2. Macro-photography of melanin molecule in solid state

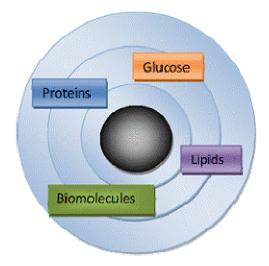


Figure 3. Formation of fibrosis or mitosis

analogy con the first reaction in plants, we termed it as photosynthesis. But in comparison, human our photosynthesis is much more efficient than that of vegetables because chlorophyll only uses between 400 and 700 nm of light wavelength (purple and red). In comparison, the human version of chlorophyll. the melanin molecule, is capable of absorbing light through the whole electromagnetic spectrum, astonishing our photosynthesis is impelled by visible and invisible light, so is able to work during night and day. Furthermore, evidence indicates that melanin can absorb any form of energy, even gravitons, and that the energy absorbed is used mainly to dissociate the water molecule. In addition, we learned that when human photosynthesis is turned down in an abrupt manner, hemorrhage and edema ensued in any part of our body, while when the down regulation is in a chronic form, then fibrosis or mitosis will develop Figure 3.

The first spark of life was the presence of free chemical energy in ways that could boost consequential reactions which were gradually forming what we call life. The carbon atom is the backbone of all biological compounds, and is created from an atom to change and joined another, and so on, thanks to the presence of free chemical energy. From the beginning until now, any biological molecule required for this free chemical energy to be created and maintained, i.e. to express the life, Biomolecules should be all the time immersed in a medium containing adequate amounts of free chemical energy unceasingly, day and night; because is the way our body imbues life to the relatively inert compounds we eat.

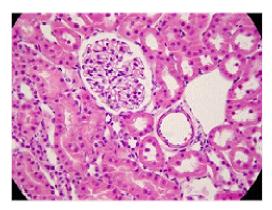
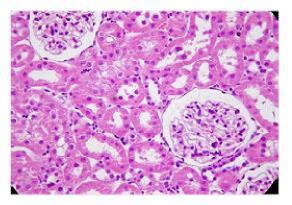
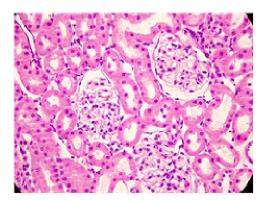


Figure 4a. Kidney, control group, H & E, 40X



**Figure 4b.** Kidney, As exposed group, note edema and hemorrhage, characteristic findings of an abrupt descent of the level of free chemical energy into the cell. H&E, 40X



**Figure 4c.** Kidney, As exposed and simultaneously treated group. H&E, 40X

I've learned over of these more than two decades, that any alteration in the functioning of the body, it starts at the molecular level and in a large number of cases, if not all, is related to the level of free chemical energy within the cell, is affected to a greater or lesser degree. Some examples of how altered lung tissue, liver and kidney in experimental animals (Wistar rats, Guinea pigs) when exposed to compounds that decrease photosynthesis.

The following histological sections were taken from an

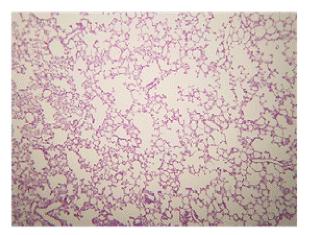


Figure 5a. Wistar rat lung stained with H & E, 5X. Control Group.

experiment whose details will be the occasion of a subsequent publication, but in general terms formed three groups of 10 animals each, male Wistar rats, 4 months old, and a group constituted the Control group, the second group was exposed to arsenic compounds that decrease photosynthesis and the third group was exposed to the same arsenic compounds of the second group, but at the same time we apply a compound we called QIAPI 1<sup>®</sup>, which has the property of enhance photosynthesis in humans and mammals. The experiment lasted four months, and at the end the animals were sacrificed by decapitation, and tissue samples were taken from various organs, including: kidney (Figure 4a-c) shown above, lung (Figure 5a-e) and liver (Figure 6a-c) as shown below.

### Comment

The human body, with four billion years of evolution, is almost perfect, is almost divine, is a self-sustaining chemical system that is much more complex than we can imagine. But yet, at the source, our body is relatively simple, because we can assume that everything comes from human photosynthesis, as any process in our body is imbued with it, is impregnated with the same, and therefore is completely ruled by it.

Or someone would argue that until the last leaf of the plant does not come and depends entirely on photosynthesis. And the same goes for us, for every one of the processes that shape us, and although we understand them, is more, not even know, and are completely dependent on the very first reaction of life (Solís-Herrera et al., 2010): the dissociation and re -formation of the water molecule in the case of humans.

Let it, however briefly, the anatomo-clinical findings of humans beings affected by the Bhopal methyl isocyanate leak. In my view, the clinical manifestations, whatever it may be, will start at the molecular level, and when damage is progressive, subclinical manifestations go to clinic level. The symptom name is not important because the human body does not care about human classifications.

Let us start from the eukaryotic cell, developed by Mother Nature over four billion years of evolution, inevitably will do their job, and i.e. is able to carry out each and every one of the biochemical reactions that form both in time and form (Solis-Herrera et al., 2013), if the conditions are right.

And being reductive, we can say that life is a delicate dynamic balance between biomass and chemical free energy available. Being the second the most fundamental: proper levels of free chemical energy.

I dare say that if the levels of intracellular free chemical energy are within ranges compatible with life, the cell showed amazing recovery capabilities, typical of a living entity that has four billion years of continuous evolution.

So, according to our clinical experience in similar cases, even of different etiology, but in the background are similar, e.g. progressive pulmonary fibrosis, acute and chronic renal failure, congenital malformations, periventricular leukomalacia, etc.., Are alterations all likely to improve very significantly with the intensification of the true source of energy of the eukaryotic cell: human photosynthesis.

We can improve the quality of life of survivors of the Bhopal disaster, and not saying everyone, but if more of those who believe. In some cases the improvement will be gradual, but in others it will be dramatic. And the explanation is simple, the methyl isocyanate causes diffuse damage in the human body, and again, on any system, when the damage is widespread, first energy, and in the case of the human body, that means free chemical energy.

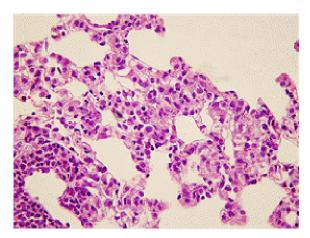
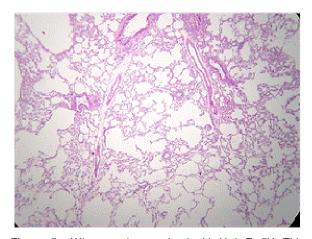


Figure 5b. Control group, lung. H & E, 40 X.



**Figure 5c.** Wistar rat lung stained with H & E, 5X. This specimen belongs to the exposed group whereas a pesticide of the pentavalent organic compounds group (arsenates) was administered through the water during three months.

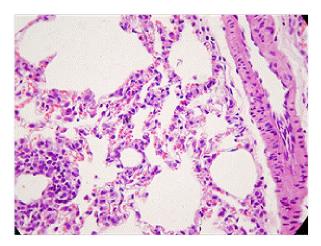
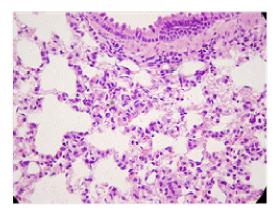


Figure 5d. At higher magnification, the lung tissue of Wistar rats exposed to arsenate show edema and hemorrhage. H & E 40X



**Figure 5e.** Lung, this group was exposed to As and at the same time were treated medically, and histological changes shows noticeable improvement of the lung tissue. H&E, 40X

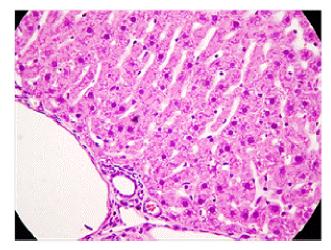


Figure 6a. Liver, control group, Wistar rat, H&E, 40X

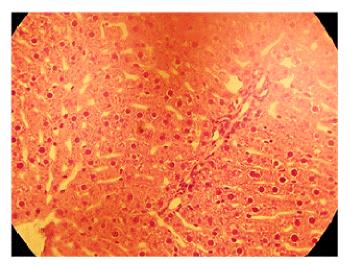


Figure 6b. Liver, As exposed group; H&E, 40X

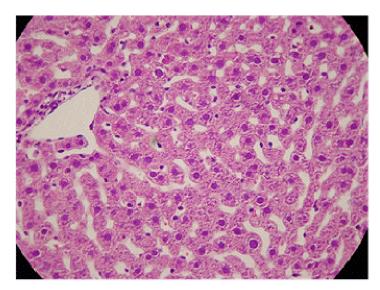


Figure 6c. Liver, exposed and treated group, simultaneously; H&E, 40X

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