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Why HDFx, a Stress Protein, May Ameliorate the Pig-Plague Effects of The African Swine Flu Virus (ASFV)

Editorial

Burton M. Altura[1-6*]

[1] Department of Physiology and Pharmacology,
[2] Department of Medicine,
[3] The School of Graduate Studies in Molecular and Cellular Science,
[4] SUNY Downstate Health Sciences University, Brooklyn, New York;
[5] Bio-Defense Systems, Inc, Rockville Centre, New York;
[6] Orient Biomedica, Estero, Florida.

Introduction

In 1921, Robert Montgomery, working in Kenya, noticed that pigs brought from England quickly developed a contagious pneumonia and rapidly succumbed, and died [1]. When antibodies against African Swine Flu Virus (ASFV) were developed (which promoted swine fever and caused feverish chills),andfailed to promote any protection, scientists concluded that another virus, that later was characterized as ASFV must be responsible for the contagion [2]. Although ASFV is harmless in humans, so far, it can be extremely dangerous in pigs [1]. Infection in pigs usually causes death with a hemorrhagic fever in one week, akin to Ebola and Marburg virus fevers. ASFV has killed hundreds of thousands of pigs in China, The Philippines, South Korea, Vietnam, Laos, Mongolia, Cambodia, Myanmar, Africa, and eastern Europe [3]. It is thought that ASFV will kill about a quarter of the world's pig population over the next few years due to lack of a vaccine. Discovery of a vaccine or a workable ameliorative would aid farmers and the civilian population immensely.

ASFV typically invades macrophages, monocytes and other blood-formed elements, thus making host-defense problematical. These invasive cellular effects induce a massive number of inflammatory responses along with hemorrhages, particularly in the lungs [1-3]. Moreover, ASFV leads to cell death of macrophages, monocytes, leukocytes and endothelial cells [3]. The major cell types that seem to be needed in defense against ASFV are the "killer T-cells", but these important cell types appear to be inactivated or destroyed early after infection by ASFV [3]. Although a considerable amount of time and effort seems to have been spent trying to develop a reliable vaccine against ASFV, this has been fruitless in many respects [3]. Manuel Boron, at The Plum Island Animal Disease Center in the USA, has developed some interesting vaccines, but safety has turned out to be a major problem with each of four vaccines [3]. Although new approaches to development of gene-deleted vaccines are thought to be a good way for successful vaccines against ASFV, so far this has not resulted in a viable vaccine [3].

For a number of years, our laboratories have been working on peptides/small proteins, lipids and other molecules which stimulate the innate and adaptive immune systems [4-33]. Along the way, we have found and isolated a 35-40 kD conserved stress protein that is in the bodies of all mammals, including rats, rabbits, mice, guinea-pigs, dogs, cats, monkeys , baboons, and pigs that we have termed host defense factor x (i.e., HDFx) [34]].

Discovery and Physiological Attributes of HDFx

About 135 years ago, Elie Metchnikoff , the father of immunology, using injury of starfish, hypothesized that the body under stressful and injurious circumstances would produce powerful immunological stimulants which could act on different parts of the immune system and serve to protect the host against major, dangerous insults, inflammatory conditions, wounding, and diverse diseases [35]]. Metchnikoff's early studies pointed to the important contribution of macrophages and phagocytic leukocytes in natural (innate) resistance against pathogenic microorganisms. Over the past 65 years much work has been done to indicate a strong support to demonstrate an important, physiological relationship between the microcirculation, macrophages, phagocytic leukocytes, alveolar macrophages, splenic macrophages, natural killer (NK) cells, the reticuloendothelial cells (particularly Kupffer cells) ,and "pit cells" in the liver to host defense [4-33, 36-39].

Using Metchnikoff's hypothesis, and thousands of animals, we

***Corresponding Author**:
Burton M. Altura,
Department of Physiology and Pharmacology, Department of Medicine, The School of Graduate Studies in Molecular and Cellular Science, SUNY Downstate Health Sciences University, Brooklyn, New York; Bio-Defense Systems, Inc, Rockville Centre, New York; Orient Biomedica, Estero, Florida.
E-mail: burton.altura@downstate.edu

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found that diverse, injurious insults, for example, sublethal hemorrhage, sublethal intestinal ischemic shock, endotoxins, live bacteria (i.e., E. coli, S. enteriditis) , sublethal centripetal forces, trauma, circulatory shock , and combined injuries, would produce a 35-40 kD conserved protein (i.e., HDFx) which when isolated, purified and administered to diverse mammals resulted in protection (or amelioration) against these diverse forms of deadly injuries [4, 34-39].

Unique attributes of HDFx , not to be minimized, is its ability to : 1. Accelerate wound healing; 2. Restore transcapillary blood flows towards normal in injured and inflamed tissues (e.g., in lungs, kidneys, liver); 3. Restore vasomotor tone; 4. Restore lowered arterial blood pressures towards normal; and 5. Restore endotoxin-induced fevers towards normal [4, 5]. Very importantly, we have found that HDFx can ameliorate/prevent "cytokine storms" in numerous rodents and farm animals, similar to those found in ASFV [4, 40, 42].

HDFx and its Potential to Ameliorate the Deadly Effects of "Superbugs" and Possibly ASFV: Importance of Activated Macrophages and NK Cells

Most hospital-induced infections in humans are caused by gram-negative bacteria [38, 39]. These "superbug -induced infections "are not only found in humans but diverse "superbug" gram-negative and gram-positive bacteria, as well as numerous funguses and viruses which are found to cause deadly infectious diseases in domestic as well as farm animals [39]. Since HDFx "supercharges" macrophages, NK cells, phagocytic leukocytes and Kupffer cells, in all mammals we have investigated, including pigs [4, 5], it is our belief that HDFx would detoxify/prevent ASFV from causing pathologies in the lung s, kidneys and liver , and prevent/ameliorate "cytokine storms", thus preventing or eradicating ASFV-induced drops in arterial blood pressure and elevated episodes of steep rises in body temperature; prevent or ameliorate alveolar tissue , liver and renal tissue damage due to its ability to improve transcapillary nutritive blood flows, and lastly,Its ability to accelerate healing and regeneration [4, 5, 40-42]. Whether HDFx will preserve "killer T-cells" remains to be investigated. To myknowledge, HDFx is the only host-defense molecule that embodies all of these unique attributes and vital therapeutic qualities against "superbugs". Moreover, since ASFV is a hemorrhagic fever,and HDFx is thought to meliorate some hemorrhagic fevers [43], we believe it would be propitious to test HDFx's effectiveness on hundreds of infected pigs.

Conclusions and Future Thoughts

It is rather obvious that the deadly ASFV could wreak havoc worldwide should this "superbug" become airborne or mutated to infect humans like has happened with Ebola, Lassa, Nipah and other viruses through mutations. Moreover, if ASFV becomes airborne and mutated, the costs to farmers and the public worldwide could bankrupt populations and interfere with critical food supplies. This potential disaster becomes "a clear and present danger" to human-kind which demands immediate action by the UN, WHO, USDA, and CDC's. Since the specific molecular mechanisms of action for infection by ASFV (and its rapid deadly effects) are not known, after more than 100 years of research , we believe HDFx should be tried against the bodily actions of ASFV.

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