**Journal of Business and Economics, ISSN 2155-7950, USA**
**April 2021, Volume 12, No. 4, pp. 400-408**
**DOI: 10.15341/jbe(2155-7950)/04.12.2021/004**

**http://www.academicstar.us**

# Potentials of the Medicinal Synthetic Aluminum-Magnesiumsilicate {MSAMS: $Al_4(SiO_4)_3 + 3Mg_2SiO_4 \rightarrow 2Al_2Mg_3(SiO_4)_3$} for the Economy

*Maduike C. O. Ezeibe, Favor I. O Onyeachonam*
*(College of Veterinary Medicine, Michael Okpara University of Agriculture, Umudike, Nigeria)*

**Abstract:** Molecules of Aluminum-magnesium silicate (AMS), WHO-approved medicine/pharmaceutical stabilizing agent consist of Nanoparticles which have negative electrical charges on their surfaces and positive charges on their edges. RNA viruses, including HIV and Covid-19 virus have positive electrical charges. DNA viruses and abnormal cells are negatively charged. Therefore, AMS prevents attachment of viruses to cells, by opposite charges-electrostatic attraction. Thus, viral replication is inhibited and extra-cellular viruses, mopped. The Nanoparticles also adsorb onto abnormal cells so that tumor cells are mopped and infected cells, destroyed. As Nanoparticles, they are able to reach viruses and abnormal cells in all organs/tissues. When 100% of each viral infection is mopped, patients suffering the disease recover. Nigeria does not have deposits of AMS but there are abundant deposits of Aluminum silicate and Magnesium silicate in the country. These minerals which are also WHO-approved medicines, were used for a reaction to get the Medicinal synthetic AMS (MSAMS, Antivirt®). Dextrose monohydrate (Glucose®) was formulated with the MSAMS to convey the charged Nanoparticles (by active transport) across mucous membranes into blood, for circulation to organs/tissues. The MSAMS has proved effective against all nine viruses so far studied including HIV. As adjuvant, it has potentiated all five antimicrobial drugs so far studied and made them achieve ≥ 95% reduction of infection-loads (preventing Antimicrobial Resistant infections: AMR). Also, at 75% of their doses, antimicrobials formulated with MSAMS and used with antioxidants, regain efficacy against AMR. So, the MSAMS prevents AMR and makes drugs recover effects against AMR. Use of these solid minerals which are abundant in the country (Aluminum silicate and Magnesium silicate) to synthesize MSAMS which provides solutions to the three major health challenges of the world (Viral diseases, Abnormal-cell diseases including Cancers/other tumors and AMR) would diversify and enhance Nigerian economy.

**Key words:** Medicinal synthetic Aluminum-magnesium silicate; Antiviral; Anti-tumor; Cure/prevention of drug-resistance

**JEL code:** I1

## 1. Introduction

Maduike C. O. Ezeibe, Ph.D., Professor, College of Veterinary Medicine, Michael Okpara University of Agriculture Umudike; research areas: veterinary medicine, virology, drug development. E-mail: maduikeezeibe@yahoo.com.
Favour Onyeachonam, Dr., MSc., College of Veterinary Medicine, Michael Okpara University of Agriculture Umudike; research areas: veterinary medicine, pharmacology. E-mail: pleasfavo@gmail.com.

Developing antiviral medicines is difficult such that most viral diseases are taken to be incurable. Two other major medical challenges are tumors/cancers (abnormal cell diseases) and Antimicrobial resistant infections (AMR).

### 1.1 What Makes Designing Antiviral Medicines Difficult

Most viruses cause immune deficiency and viral small sizes enable them infect cells which are inaccessible to medicines of large molecules. So, medicines that act by inhibiting physical features of pathogens need immunity to complement their effects against viral infections while side effect of medicines that act against viral biochemistry become intolerable to patients when treatments continue for a long time (because of similarity of viral-biochemistry and animal cell-biochemistry). Under state of immune deficiency, infections in cells that are inaccessible to large molecules cannot be cleared by existing antiviral medicines. Those inaccessible cells are the ones called "viral reservoirs" or "sanctuary cells". That is reason some viral diseases, such as HIV/AIDS are "incurable" while patients of viral diseases that do not cause severe immune deficiency, such as Covid-19, recover in millions. So, antiviral medicines should be designed to inhibit physical features/activities of viruses instead of their biochemistry (to minimize side effects) and their molecules should be smaller than viruses (≥ 5 nm) so that they can reach all infected cells.

### 1.2 Literature-Review (for Solution)

That electrostatic attraction would serve as a mechanism of inhibiting electrically charged pathogens by medicines that possess opposite electrical charges is an old scientific knowledge and that viruses and abnormal (infected/tumor) cells are electrically charged (Brooks G. F., 1998; Chen B. D., Le W. J., Wang Y. L. et al., 2016) has been discovered. That most epidemics/epizootics, including HIV/AIDS, Ebola, Lassa fever, Covid–19, viral hepatitis and Avian Influenza are viral diseases, is known. Molecules of Aluminum-magnesium silicate (AMS), WHO-approved medicine/pharmaceutical stabilizing agent, consist of *Nanoparticles* (Cristina E., Ivan P., Kevin R., (2007) that are only 0.96 nm thick (Vanderbilt R. T., 2012). That means, AMS-*Nanoparticles* are smaller than any known virus (≥ 5 nm). They have negative and positive electrically charged ends (Vanderbilt R. T., 2012) but unlike abnormal cells, healthy cells are neutral (bio-medical marker). The charges enable AMS mop/destroy viruses/abnormal cells, by opposite charges-electrostatic attraction. As a silicate, AMS also normalizes immunity (Suni L., Hiroaki H., Megumi M. et al., 2014) and as a stabilizing agent (Brent W., Gunderson Gigi H., Ross K. H. I. and John C. R., 2001) it enhances efficacy of antimicrobial agents for effective treatment of secondary infections.

### 1.2 The Innovations

The solid mineral, AMS ($Al_2Mg_3(SiO_4)_3$) is not found in Nigeria but the country has large deposits of Aluminum silicate ($Al_4(SiO_4)_3$) and Magnesium silicate ($Mg_2SiO_4$) which are also WHO-approved medicines. These locally abundant solid minerals were used for a reaction (Ezeibe M. C. O., 2012) to get the *Medicinal synthetic AMS*{MSAMS: $Al_4(SiO_4)_3+3Mg_2SiO_4 \rightarrow 2Al_2Mg_3(SiO_4)_3$}. Dextrose monohydrate (simple sugar) was formulated with the MSAMS, to convey the electrically charged particles across mucous membranes (Murray K. R., 2000), into blood, by active-transport.

### 1.3 The Antiviral/Antitumor Hypothesis

Mopping viruses; Destroying abnormal cells; Normalizing immunity; Effective treatment of secondary infections, would cure any viral/abnormal-cell disease including, Cancers, HIV/AIDS and Covid-19. The MSAMS mops electrically charged pathogens (viruses and abnormal cells) by opposite charges electrostatic

attraction. So, we are introducing opposite charges electrostatic attraction between medicines and pathogens as a mechanism of action for terminating viral infections and metastasis of tumor cells. We are also applying the principle of active transport to convey AMS (which is un-absorbable) into blood-circulation so that it functions as a systemic medicine.

### 1.4 Prevention and Cure of Antimicrobial Resistant Infections

Ability of AMS to enhance efficacy of antimicrobial agents (Ezeibe M. C. O. & Ogbonna I. J., 2016) so that secondary infections are effectively treated also helps in treating viral diseases. With enhanced efficacy, lower doses achieve desired effects. Use of lower doses for treatments minimizes side effects of medicines. When side effects are minimized, immune responses of patients improve. Enhancing efficacy of antimicrobial agents and improving immune responses of patients lead to cure of even already resistant infections which also increases chances of patients of viral diseases to recover.

### 1.5 What Made HIV/AIDS "Incurable"

Reason existing ARVs do not achieve permanent cure for HIV/AIDS patients may be that their molecules are too large to cross physiological barriers. For that limitation, they do not reach HIV infections "hidden" in some cells. So, even when viral loads in blood of patients they are used to treat become undetectable, the infection may still remain "hidden". Since the MSAMS is made of ultra-*Nanoparticles*, it crosses physiological barriers and reaches every virus and every virus-infected cell in every organ/tissue. And since it acts by a physical effect, it is safe for any treatment-duration needed to terminate any viral-infection (including HIVinfections).

## 2. Efficacy-trials of MSAMS-formulations

### 2.1 Antivirt® (MSAMS-formulation, for Viral and Abnormal Cell Diseases in Man) Antivirt® on HIV/AIDS with MSAMS Acting as Antiviral Medicine

Experiment: Nigerian Institute of Medical Research (NIMR), certified the Antivirt® toxicologically safe by testing it on mice before recruiting three HIV/AIDS volunteers (all adults), for phase one clinical trial of the medicine, patented in Nigeria, as broad spectrum antiviral medicine and antiretroviral medicine (Ezeibe M. C. O., (2017). A formulation of the MSAMS and Ampicillin trihydrate (Antivirt A®) and a formulation of the MSAMS alone (Antivirt B®) were submitted by the inventor for treatment of the patients which started in December 2019 and lasted till May 2020. The patients were placed on oral medication with Antivirt®A for 30 days, at dose rates of 50 mg of the MSAMS/kg body weight and 7.5 mg of MSAMS-stabilized Ampicillin trihydrate/kg body weight, daily. After the first 30 days, they were placed on Antivirt® B at dose of 50 mg/kg. Each patient also took Immunace extra protection®(Vitabiotics, England), as source of antioxidants, at the rate of one tablet everyday. The Antivirt® was taken at night, at least two hours after dinner (empty stomach) and the patients were asked to eat no other thing (except water) once they took the Antivirt® till the following morning. The Immunace extra-protection® was taken in the morning, immediately after breakfast (full stomach). If a patient needed to take any other oral medicines for any reason, such other medicine was taken at least two hours before the Antivirt® or two hours after. Viral loads of the patients were tested for every month. Their recovery rates were also assessed by their doctors, every month.

**Results:** Means of their viral loads (ranked) increased ($P \leq 0.05$) from 10.00±7. 21 to 11.30±5.51 in the first month (unmasking "hidden infection) before decreasing ($P \geq 0.05$) to 10.67±6.81 (in the second month),

8.67±5.68 ($P \leq 0.05$: in the third month), 9.00±5.57 ($P \leq 0.05$: in the fourth month) and 7.33±6.03 ($P \leq 0.05$: in the fifth month): Table 1.

This amounted to increase in viral load (instead of decreasing) by a mean of 41.03% in the first month (unmasking of "hidden HIV-infections") before the viral loads started decreasing to 22.62% — increase (in the second month), 54.18% — decrease (in the third month) 55.27% — decrease (in the fourth month) and 76.69% — decrease (in the fifth month). The viral load-reduction rates are as on Table 2 while the laboratory results as reported by government agency (NIMR) are on Table 3.

WHO, reported that there is inverse relationship between viral loads and CD4-lymphocyte counts (immunity) in HIV/AIDS patients (WHO, 2007). Reduction of viral loads by as much as 76.69% suggests immunity of the patients may have normalized (CD4 ≥ 500). Synergy between normalized immunity and viral-mopping mechanism of the medicine would lead to recovery from HIV/AIDS.

Since the Antivirt® reduced infection of HIV, an RNA virus (positively charged), by as much as 76.69% in five months, despite severe immune-deficiency associated with HIV, the medicine will terminate infections of *Covid-19 virus* (another RNA virus which is not associated with severe immune-deficiency) in a much shorter treatment-duration (few days).

**Table 1 Monthly Ranked Viral Loads of HIV/AIDS Patients Being Treated with the Antivirt®**

| Patients Ranked | Viral | | Loads | | | |
|---|---|---|---|---|---|---|
| Treatment-Months | 0 | 1 | 2 | 3 | 4 | 5 |
| 1 | 16 | 17 | 18 | 15 | 14 | 13 |
| 2 | 12 | 11 | 9 | 7 | 10 | 8 |
| 3 | 2 | 6 | 5 | 4 | 3 | 1 |
| Mean | $10\pm7.21^{c}$ | $11.3\pm5.51^{d}$ | $10.67\pm6.81^{c}$ | $8.67\pm5.68^{b}$ | $9\pm5.57^{b}$ | $7.33\pm6.03^{a}$ |

**Table 2 Monthly Viral Load Reduction-rates (%) in HIV/AIDS Patients Treated with the Antivirt®**

| Months: | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| | -135.36 | -137.02 | 60.33 | 73.66 | 78.65 |
| | 59.82 | 67.58 | 76.48 | 64.38 | 69.18 |
| | -47.54 | 1.57 | 25.74 | 27.70 | 82.24 |
| Mean | -41.03 | -22.62 | 54.18 | 55.27 | 76.69 |

**Table 3 Monthly Viral Loads of HIV/AIDS Patients Being Treated with the Nigerian Antivirt®**

| 0 | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|
| 1810000 | 4260000 | 4290000 | 1720000 | 1130000 | 916000 |
| 438000 | 176000 | 142000 | 103000 | 156000 | 135000 |
| 3450 | 5090 | 5010 | 3780 | 3680 | 904 |

## 2. Admacine®(MSAMS-Ampicinllin for Animals)

### 2.1 MSAMS Acting as Adjuvant to Ampicillin Against *Salmonella gallinarum*.

**Experiment:** Four groups, each of 10 randomly selected chicks, infected with *Salmonella gallinarum* were treated with Ampicillin trihydrate (AT) for 5 days. Two groups were treated at dose rates of 10 mg and 7.5 mg of AT per Kg body weight respectively, with 100% Ampicillin. Two other groups were similarly treated with the

Ampicillin-MSAMS drug. The fifth group served as control. Bile of 5 chicks from each group was harvested. Then 0.1 ml of bile from each chick was added to 0.9 ml of normal saline to get a 1:10 dilution. Again 0.1 ml of the 1:10 bile-dilution was added to 0.9 ml of normal saline to make a 1:100 dilution. Finally, 0.05 ml of each diluted bile was placed on Mc-Conkey agar and incubated at 37°C for 24 hours. The *S. gallinarium* colonies (X) were counted and expressed as colony forming units per ml (CFU/ml) by the formula: CFU/ml = x/5×10,000. Means of the CFU/ml of the five groups were compared for statistical differences, by ANOVA.

**Results:** Normal dose of Ampicillin (10 mg/kg) led to only 80.68% reduction ($P < 0.05$) of CFU/ml of bile of *S. gallinarum*-infected chicks. When the drug was stabilized with the MSAMS the reduction improved ($P < 0.05$) to 86.36%. Reducing the dose to 75% of recommended dose of Ampicillin (7.5 mg/kg) and stabilizing it with the MSAMS improved rate of reduction of the infection load ($P < 0.05$) to 97.84%.

**2.2 MSAMS acting as adjuvant to Ampicillin against resistant *Escherichia coli***

**Experiment:** Five groups, each of 5 randomly selected chicks, infected with Ampicillin-resistant-*E. coli* were used. Two days before infecting the chicks, 2 groups were placed on poultry feed, fortified with additional 375 mg of Vitamin A, 10 mg of Vitamin C, 75 mg of Vitamin E and 12.5 mg of Selenium for each 25 kg bag. Three groups were left on ordinary poultry feed. The 2 groups on the fortified feed were treated with Ampicillin and with the Ampicillin-MSAMS drug formulation respectively, at dose of 7.5 mg/kg for 7 days. Two of the groups on ordinary feed were treated at dose of 10 mg/kg with 100% Ampicillin and with the Ampicillin-MSAMS drug formulation respectively, for seven days while the third group on ordinary feed served as control. Means of *E. coli* CFU/*m*l of bile of the groups of chicks were compared for statistical differences, by ANOVA.

**Results:** Recommended dose of Ampicillin (10 mg/kg) led to reduction ($P < 0.05$) of load of Ampicillin-resistant *E. coli,* just by 50 %. When the drug was stabilized with the MSAMS, rate of the drug-resistant infection reduction decreased ($P < 0.05$) from 50% to 43.91%. Use of 75% of the recommended dose (7.5 mg/kg) stabilized with the MSAMS plus immune stimulants in feed of the chicks led to reduction ($P < 0.05$) of load of the resistant infection by 95.78%.

## 3. Bernazine®(MSAMS-piperazine citrate)

**Experiment:** Five groups of randomly selected mice, infected with *Helignosomoides bakeri* were treated with 110 mg/kg (piparazine), 110 mg/kg (Piparazine in MSAMS), 82.5 mg/kg (Piparazine) and 82.5 mg/kg (Piparazine in MSAMS), respectively. The fifth group served as control. *H. bakeri* Eggs Per Gramm (EPG) of feces of each mouse in the five groups were counted. Mean EPG of the groups were compared for statistical differences, by Analysis of variance (ANOVA).

**Results:** Recommended dose of Piperazine (110 mg/kg) led to only 82.94% reduction ($P < 0.05$) of EPG of feces of *H. bakeri*-infected mice. When the drug was stabilized with the MSAMS, the rate of reduction improved ($P < 0.05$) to 92.04%. Reducing the dose to 75% of Piparazine's recommended dose (82.5 mg/kg) and stabilizing it with the MSAMS improved rate of reduction of the EPG ($P < 0.05$) to 96.82%.

## 4. Ismerquine®(MSAMS-Chloroquine)

**Experiment:** Fifteen albino mice, infected by intra-peritoneal (IP) inoculation of 1 ml of blood of a donor mouse which contained $2\times10^8$ *Plasmodium berghei* per ml were randomly assigned into five groups of three each and treated at two Chloroquine dose levels (7 mg/kg and 5.25 mg/kg). Three groups were treated at Chloroquine

dose of 7 mg/kg with: Chloroquine alone, Chloroquine-MSAMS drug formulation and Chloroquine — MSAMS drug formulation plus B-vitamins, respectively. The fourth group was treated at 75% of Chloroquine-dose (5.25 mg/kg) with the Chloroquine-MSAMS drug formulation plus B-vitamins while the fifth group was not treated (control). To ensure safety for the mice and uniformity for the experiment, the two Chloroquine formulations were reconstituted, such that each mouse was drenched same volume (0.1 ml) to deliver different doses (7 mg/kg or 5.25 mg/kg) from different formulations (Chloroquine and MSAMS-Chloroquine):

For each of the treated groups, treatment was initiated 10 days post infection (PI) and lasted for 7 days. *Plamodium berghei* parasitaemia and total red blood cells counts were among parameters tested for, on days: 1, 7, 14 and 21 post treatment (PI) with their means tested for statistical differences.

**Results:**

**Mean Parasitaemia**, 42.00±15.74 of the group treated with 7 mg/kg (Chloroquine phosphate alone) did not vary ($P \geq 0.05$) from 52.50±11.99, 37.22±11.88 and 33.57±12.62 of the untreated group, the group treated with 7 mg/kg (Chloroquine-MSAMS) and the group treated with 7 mg/kg (Chloroquine-MSAMS plus B-vitamins) respectively but mean parasitaemia, 00.00±00.00 of the group treated with 75% of recommended dose of Chloroquine (5.25 mg/kg) stabilized in MSAMS plus B-vitamins was significantly ($P \leq 0.01$) lower than parasitaemia of both the untreated group and of the other treated groups.

**Means of Red Blood Cell Count** (RBC) of groups of mice treated with Chloroquine at dose of: 7 mg/kg (46.71±3.41), 7 mg/kg with MSAMS-Chloroquine drug formulation (45.50±4.24), 5.25 mg/kg with MSAMS-Chloroquine drug formulation and vitamins (45.65±3.63) and of the untreated group (44.00±3.08) did not vary ($P \geq 0.05$) but RBC, 59.28±3.14 of the group treated with 7 mg/kg of the MSAMS-Chloroquine drug formulation and vitamins was significantly ($P \leq 0.05$) higher than mean RBC counts of the other groups.

## 5. Francoccine® (MSAMS-Sulphadimidine)

### 5.1 Against Coccidia

**Experiment:** Fifty two, day-old cockrel chicks were used in experimental studies. In the first study, at day 28 of age, all the 52 chicks were infected by oral administration of 1ml of a coccidial suspension which contained 77328 infective *Eimeria tenella* and *E. maxima* oocysts. Seven days post infection, two sick chicks were sacrificed to confirm diagnosis of coccidiosis by post mortem examination and by microscopic demonstration of coccidia. The remaining fifty chicks were assigned into five groups (A to E) of ten chicks each. Group A was treated with 5g of a drug formulation containing 20% sulphadimidin in the MSAMS, per liter of drinking water. Group B was treated with 1 g of 100% sulphadimidin per liter of drinking water. In group C, the drug formulation containing 20% sulphadimidin in MSAMS was added to their drinking water at the rate of 2 g per liter. As control for group C, 0.4 g of 100% sulphadimidin was added per liter of drinking water of chicks in group D. Group E served as untreated control.

All the treated groups received treatment for three days at first. The treatment was withdrawn for the following two days before they were treated for another three days. To assess efficacy of the treatment, clinical signs, mortality and oocysts output per gram of feces were recorded. Fecal oocysts count and mortality in the groups treated with sulphadimidin-MSAMS drug formulation were compared with those of their controls.

**Results:** Clinical signs of coccidiosis observed in the chicks, included wing drooping, inappetance, depression, ruffled feathers and bloody-diarrhea. Post mortem lesions seen at necropsy included ballooning of the

small intestines, petechial haemorrhages on serosal surfaces of the intestines. The intestinal walls were thickened, with their lumens filled with blood and tissue debris. By the end of the first round of treatment, all the clinical signs including bloody diarrhea had ceased in group C, treated with 2 g of the 20% sulphadimidin in MSAMS. The clinical signs also ceased, from the first day of second round of treatment, in group B which was treated with 1 g of the 100% sulphadimidin powder per liter of drinking water. However, the clinical signs (bloody diarrhea) persisted in group A, treated with 5 g of the sulphadimidin — MSAMS drug-formulation and in group D, treated with 0.4 g of 100% sulphadimidin per liter of drinking water. Groups A and D had mortality of 3 (30%) each. Groups B and C had 1 mortality (10%) each. Untreated group E had 9 mortalities (90%).

Parasitological assessment showed that group A, treated with 5 g of the 20% sulphadimidin in MSAMS, had the least oocyst count per gram of feces (13,000), followed by group B, treated with 1 g of 100% sulphadimidin per liter of drinking water (15,000). Group C, treated with 2 g of the 20% sulphadimidin — MSAMS drug-formulation per liter, had oocyst count of 16,000 per gram of feces while group D, treated with 0.4 g of 100% sulphadimidin per liter of drinking water, had the highest oocyst count per gram of feces (965,000). The only survivor in the untreated control group E, had oocyst count of 52,500 per gram of feces.

Since 5 g of 20% sulphadimidin drug-formulation contains same amount of sulphadimidin as 1 g of 100% sulphadimidin, it was expected that groups A and B would give same results. Instead, persistence of bloody diarrhea in group A and the 30% mortality were significantly different from the results in group B in which the bloody diarrhea ceased and only 10% mortality was recorded. However, the low oocyst count of 13,000 per gram of feces recorded in group A and the 15,000 per gram recorded in group B were approximately same. This suggests that sulphadimidin effectively treated coccidiosis in both groups A and B. In group C, clinical signs ceased after three days of treatment and mortality was only 10%, while group D, treated with 0.4 g of sulphadimidin, equivalent of 2 g of a 20% sulphadimidin drug formulation per liter, had a mortality of 30% and the clinical signs did not cease. Group D also had the highest oocysts count per gram of feces. These results suggest that the treatment was ineffective in group D while it was effective in group C.

It was therefore concluded that incorporating the MSAMS in sulphadimidin potentiated its anticoccidial activity. The 5 g of the 20% Sulphadimidin formulation per liter of drinking water became overdose hence the high mortality and persistence of bloody diarrhea which is clinical sign of overdose of Sulphadimidin, but with low oocyst count per gram of feces. Also, 2 g of the 20% Sulphadimidin in the MSAMS drug-formulation which is equivalent of 0.4 g of 100% Sulphadimidin per liter, which was ineffective in group D became effective with only 10% mortality, 16,000 oocysts per gram of feces and cessation of clinical signs after only three days of treatment, in group C.

The MSAMS may have potentiated action of Sulphadimidin. The relatively low oocysts count per gram of feces recorded in the only survivor of untreated control may be a result of "self cure" phenomenon.

**5.2 Against Resistant *Escherichia coli*.**

**Experiment:** Five groups of randomly selected chicks, infected with Sulphadimidine-resistant *Escherichia coli* were used for an experiment. Two groups were treated at Sulphadimidine's dose rate of 1 g/liter of drinking water with a 100 % Sulphadimidine powder and with the Sulphadimidine-MSAMS drug formulation, respectively. Two other groups were treated with the 100% Sulphadimidine and with the MSAMS-Sulphadimidine drug formulation at Sulphadimidine's dose rate of 0.75 g/liter. The fifth group served as control. After 5 days of treatment, the chicks were sacrificed and dilutions of their bile plated on Mc-Conkey agar and incubated at 37°C

for 24 hours. *E. coli* colonies in each culture were counted and expressed as CFU/ml. Means of *E. coli* CFU/ml of bile of the different treatment groups were compared for statistical differences, by ANOVA.

**Results:** Normal dose of Sulphadimidine (1 g/liter of drinking water) led to increase ($P < 0.05$) of load of Sulphadimidin-resistant *E. coli* infection by 259%. When the drug was stabilized with the MSAMS, load of the resistant infection increased further ($P < 0.05$) by 789.10%. Reducing the dose to 75% (0.75 g/liter) and stabilizing it with the MSAMS reduced load of the resistant infection significantly ($P < 0.05$) by 84.34% (Cure).

## 6. Sal-travite®(MSAMS-Cotrimoxazole)

### 6.1 Against Resistant *Salmonella pullorum*.

Experiment: Three groups (A, B and C) of chicks infected with a Cotrimoxazole-resistant *Salmonella pullorum* isolate were placed on commercial feed to which additional levels of Vitamins A, C and E were added and treated with (100 %, 75 %, and 50 %) doses of cotrimoxazole stabilized in MSAMS. Three other groups (D, E and F) were similarly infected and treated but were on the commercial feed without additional levels of the vitamins. Group G was fed with the normal feed, similarly infected but treated with 100% dose of Cotrimoxazole without the MSAMS. Group H was also fed with the normal feed and infected but was not treated.

Results: Normal dose of Cotrimoxazole could not cure Cotrimoxazole-resistant *S. pullorum* infection (77% infection-reduction ≤ 80% which leads to clinical recovery). When the normal dose was stabilized with MSAMS, it worsened the resistant infection (-212.6% and -230.96% reductions rates) but 75% of dose of Cotimoxazole stabilized with MSAMS and antioxidants achieved cure of the resistant infection (96.23% infection-reduction ≥ 95% which leads to termination of infections).

## 7. Conclusion

**MSAMS** is a broad spectrum antiviral medicine, antiretroviral medicine and anticancer medicine. It is also adjuvant that potentiates other medicines.

As adjuvant it improves efficacy of antimicrobials formulated with it. Improving efficacy of antimicrobial medicines reduces their doses required to achieve desired effects and their side effects. Reduction in doses, reduces costs of production and so improves profit for pharmaceutical industries while reduction of side effects enhances immune response of treated patients and leads to enough clearance of infections such that development of drug-resistance is prevented. Even already resistant infections become curable because of the enhanced immune responses. Since there is no limit to antimicrobial medicines that would require their efficacies to be improved, there would also be no limit to number of patents to register from the MSAMS. Every pharmaceutical company, everywhere in the world would require it, either as a medicine for diseases caused by electrically charged pathogens or as adjuvant (to improve other medicines) or for both purposes. Both Aluminum silicate, Magnesium silicate and Aluminum-magnesium silicate are medicines already approved by WHO. So, all that is needed is for the government to invite WHO to confirm these efficacies of the MSAMS and grant necessary approvals. That would diversify and grow the economy, post Covid-19.

**References**

Brooks G. F. (1998). *Medical Microbiology* (21st Ed.), McGRAW, San Francisco, USA.

Chen B. D., Le W. J., Wang Y. L., Li Z. Q., Wang D., Ren L., Lin L., Cui S.B., Hu J. J., Hu Y. H., Yang P. Y., Ewing R. C., Shi D. L.

and Cui Z. (2016). "Targeting negative surface charges of cancer cells by multifunctional nanoprobes", *Theranostics*, Vol. 6, pp. 1887-1898, doi: https://doi.org/10.7150/thno.16358.

Cristina E., Ivan P. and Kevin R. (2007). "Nanomaterials and nanoparticles: Sources and toxicity", *Biointerphases*, Vol. 2, pp. MR17-MR71, dol: 10.1116/12815690.PMD.20419892.

Vanderbilt R. T. (2012). *Veegum — The Versatile Ingredient for Pharmaceutical Formulations*, Inc. Technical Literature.

Suni L., Hiroaki H., Megumi M., Hidenori M., Aoko K.T., Ying C., Kozo U., Masayasu K., Yasumitsu N. and Takemi O. T. (2014). *Immunostimulation by Silica Particles and the Development of Autoimmune Dysregulation*, InTech, Open, London.

Brent W., Gunderson Gigi H., Ross K. H. I. and John C. R. (2001). "What do we really know about antibiotics pharmacodynamics?", *Pharmcotherapy*, Vol. 21, pp. 28-31, doi: https://doi.org/10.1592/phco.21.18.302S.33905.

Ezeibe M. C. O. (2012). "Medicinal synthetic Aluminum-magnesium silicate (Nanoparticles) — Antiviral agent and adjuvant to chemotherapeutics", Federal Republic of Nigeria Patents and Designs Ref No.: NG/P/2012/639.

Murray K. R. (2000). *Harpers Biochemistry*, McGraw Hill, New York.

Ezeibe M. C. O. and Ogbonna I. J. (2016). "Use of the medicinal synthetic aluminum magnesium silicate to enhance efficacy of antimicrobials, for prevention and treatment of resistant infections", *Clin. Exp. Pharmacol*, doi: 10.4172/2161-1459.C1.010.

Ezeibe M. C. O. (2017). "Antivirt®. Broad spectrum antiviral medicine and antiretroviral medicine", Federal Republic of Nigeria Patents and Designs Ref No: NG/P/2017/2418.

World Health Organization (2007). "Laboratory Guidelines for Enumerating CD4 T Lymphocytes in the Context of HIV/AIDS", World Health Organization Regional Office for South-East Asia, New Delhi.