



IJRASET

International Journal For Research in
Applied Science and Engineering Technology



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Volume: 10 Issue: II Month of publication: February 2022

DOI: <https://doi.org/10.22214/ijraset.2022.40193>

www.ijraset.com

Call:  08813907089

E-mail ID: ijraset@gmail.com

The Effects of Malnutrition on the Immune System of Those Living in Food Deserts and Food Swamps

Gabriel Vizgan
SUNY Downstate, USA

Abstract: *Malnutrition is a leading cause of immunodeficiency worldwide; in particular, vitamin A and protein malnutrition seem to wreak devastating effects on various types of white blood cells. This research paper undertakes a primarily qualitative approach, curating and summarizing the most cited published findings on the effects of vitamin A and protein deprivation on leukocytes, neutrophils, eosinophils, lymphocytes, monocytes, macrophages, B cells, and T cells. All were found to be decreased in serum levels and/or have diminished efficacy in those that were malnourished, statistically significant to a $p \leq 0.05$. This research is relevant as it may provide another compounding variable when regarding why those of lower socioeconomic class – especially those living in food deserts and food swamps – seem to experience disease at a greater incidence and burden than their more affluent age and sex matched counterparts.*

I. INTRODUCTION

The efficacy of the human body's ability to defend against, and defeat, invaders is dependent upon the responses of both the innate and adaptive branches of the immune system. The innate arm of the immune system is nonspecific and utilizes physical and chemical barriers in order to make it difficult for foreign pathogens to invade, and subsequently colonize, the host (Nicholson, 2016). Should these barriers prove insufficient, the innate immune system is capable of launching a more targeted attack, albeit still not specific to the particular invader. This branch of the immune system is composed of: neutrophils, which are recruited to the site of infection to amplify an inflammation reaction through the release of cytokines; complement, which is able to create transmembrane pores in invading cells leading to osmotic lysis; eosinophils, which protect the host from parasitic infection through the encouragement of IgE production; mast and basophil cells, which increase blood vessel permeability for the ease of passage of other defense mechanisms; and natural killer cells, which directly lyse their target (Nicholson, 2016).

Often enough, though, pathogens manage to overwhelm the capability of the innate immune system or escape its notice altogether. In such cases, the responsibility of ridding the body of these invaders falls on the adaptive division of the immune system. To achieve this, the body employs two classes of cells – the B and T cells – in order to promote a response that is both specific and targeted at the particular invader (Nicholson, 2016). This is accomplished by a B or T cell being presented a unique antigen from the invading pathogen by an antigen presenting cell, resulting in their differentiation and propagation creating a colony of cells hosting an antigen surface marker that directly complements the invading organism. These newly differentiated cells will then either release antibodies into circulation or hone directly to the site of infection for a targeted attack, depending on whether it is a B cell or T cell respectively (Nicholson, 2016).

Of course, the innate and adaptive divisions of the immune system do not work independently of each other and there is a host of intermediate hormones, enzymes, and cell subtypes that interconnect the two (Nicholson, 2016). Furthermore, there are often cases in which the immune system does not operate altogether as a result of one or more of these factors, in either the innate or adaptive immune system, not functioning properly. Treatment protocols for such immunodeficiencies are often approached through the framework of identifying the defective element and then attempting to either substitute or circumvent it. However, recent studies have implied that a predominant cause of immunodeficiency worldwide can, in fact, be linked to malnutrition – particularly in developing countries – which can be treated simply with appropriate diet (Bourke, Berkley, & Prendergast, 2016).

Malnutrition, which can be classified as either over or under nutrition, has been labeled as one of the greatest impairments of cell mediated immunity around the world (Bourke, Berkley, & Prendergast, 2016). True, it is difficult to separate the effects of malnutrition from other risk factors plaguing those of living in poverty, but data does seem to suggest that, when infected with the same pathogen, a malnourished patient is more likely to succumb to the infection than their age and sex matched well-fed counterpart. This effect seems to be particularly evident in young children and the elderly (Bourke, Berkley, & Prendergast, 2016).

Malnutrition is defined by the world health organization as “the cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions (Grover & Ee, 2009).”

It is estimated that in 2010 more than 925 million people in the world were malnourished, which contributed to about a 33% increase in global disease burden (Black, et al., 2008). Interestingly, it was found that even children with less severe forms of malnutrition were succumbing to infection at greater rates than their well-fed equals, when adjusted for other confounding factors (Black, et al., 2008). Furthermore, though studies have primarily focused on underdeveloped countries thus far, it is quite possible that populations in developed countries, such as the United States, could actively be experiencing similar epidemics of malnutrition-based immunodeficiency if located in food deserts – areas with little access to fresh produce – and/or food swamps – areas saturated with fast-food restaurants – due to the lack of necessary nutrients in their diets.

According to Bourke, Berkley, & Prendergast (Bourke, Berkley, & Prendergast, 2016) malnutrition is a leading cause of immunodeficiency worldwide and may prove to be a confounding factor in the explanation of why those living in lower-income neighborhoods may present with greater health disparities. This research paper examines the following research questions: (1) What are some of the systemic effects of malnutrition on the immune system? and (2) Are those living in food deserts and/or food swamps at greater risk for acquiring pathogenic infections based on their dietary intakes? This research is important as it may provide a further confounding variable in the understanding of why those living in lower-income neighborhoods may face a greater health burden than a more affluent citizen of the same city. Furthermore, it may prove to be an adequate measure of compliance with human rights regulations; if a certain population is succumbing to infection more often than the general population it could potentially indicate systemic malnutrition.

II. LITERATURE REVIEW

The link between malnutrition and altered immunocompetence has been hypothesized about since at least the 1970's. However, there have been few human longitudinal studies documenting the specific effects of malnutrition on immunocompetence due to the particularly immoral nature such research would constitute. Therefore, most of the current research available on this topic exists in the forms of retrospective case studies of children in resource-poor settings, animal models, and systemic reviews (Ibrahim, Zambruni, Melby, & Melby, 2017). What follows is a summary of the relevant results from the most cited peer reviewed articles on this topic, broken up based on the specific cell-types of the immune system.

Monocytes make up the largest portion of all circulating white blood cells and play a key role in modulating the immune system as well as actively attacking invaders. However, through experimentation with mouse models, two independent research teams were able confirm their hypotheses that protein malnourishment significantly impaired the number and function of circulating monocytes as well as their migratory form, macrophages. Though the mechanism was not fully understood, the team behind Cunha, et al. (2013) noticed that when mice were kept on a protein deficient diet their bone marrow experienced significant hypoplasia. In their experiment, two-month-old Balb/c mice were subjected to diets with only a 2% protein content while the control group of the same species of mice was given a standard 12% protein diet. The malnourished animals were found to have decreased expression of CD45 and CD117 in their bone marrow, implying low hematopoiesis. That being said, while the authors Morris, et al. (2011) attained similar results when creating their own mouse models with female Balb/c mice, they took their experiment a step further and discovered that upon feeding starved mice a protein-adequate diet, bone marrow cellularity, mucosal protein levels, and mucosal DNA all recovered and were near control levels after eight continuous days of sufficient nutrition.

Monocytes are not the only immune cell that require adequate protein levels to function, though. Nayak et al. (1989) discovered decreased neutrophil ability in 25 children with protein calorie malnutrition who were admitted to the department of pediatrics in S.P. Medical College in Bikaner. The children were all aged up to twelve years old and, when compared to a control group of 25 sex and age matched children with healthy diets, were found to have significantly impaired ability to kill bacteria in vitro ($P < 0.001$); a linear relationship between the amount of serum protein found in a child and the number of bacteria killed during incubation was present (Nayak, Sethi, Aggarwal, Chadda, & Kumar, 1989). However, this may have been a compounding and nonspecific effect of general malnutrition, as Twining, et al. (1996) was able to attribute decreased neutrophil activity to Vitamin A deficiency. Using WAG/Rij/MCW rats as an experimental and control group, they found that the neutrophils of vitamin A deficient rats were generally more hyper-segmented and contained lower levels of cathepsin G than those in the control group. Building upon these results, Jimenez, et al. (2010) published that her team was able to find a statistically significant increase in the number of active neutrophils after vitamin A supplementation in 68 malnourished preschool children.

Similar to neutrophils, a link has been made between natural killer (NK) cell function and vitamin A levels. Bowman, et al. (1990) presented Lewis rats of various ages diets that were either restricted or absent of vitamin A and compared their natural killer cell activity to that of age and sex matched control groups being fed normal diets.

They discovered a statistically significant decreases in NK activity that directly correlated with the degree of vitamin A restriction, and a return to normal NK activity as vitamin A was reintroduced to the rats' diet. The most famous research done on the effects of malnutrition on NK cell activity was conducted by the groups Rikimaru, et al. (1998) and Salimonu, et al. (1982); Rikimaru, et al., found that the serum levels of IgA1, IgA2 and C4 were greater in 170 Ghanaian kids, aged 8-36 months, that were severely malnourished compared to age and sex matched healthy children (Rikimaru, Taniguchi, Yartey, Kennedy, & Nkrumah, 1998), and Salimonu, et al. found statistically significant decreased NK activity and undetectable IFN levels in 19 Nigerian Children (Salimonu, et al., 1982). However, neither of the researchers presented any findings that pointed to a specific dietary deficiency as the cause of the immunocompetence.

The first time a direct connection between dendritic cell (DC) activity and nutrition was made by Hughes, et al. (2009) as they discovered that 81 severely malnourished children presented to the university teaching hospital, Lusaka, Zambia had inadequate numbers of circulating DCs that improved upon feeding. However, like Rikimaru, et al. (1998) and Salimonu, et al. (1982), Hughes, et al. (2009) failed to attribute this to a specific dietary deficiency.

Thus far only elements of the innate immune system have been discussed, but that does not mean that the adaptive immune system is exempt from the effects of dietary insufficiencies. In fact, T cells and B cells have been shown to have the same reliance on protein and vitamin A for their proper function. Regarding T cells, Paiva, et al. (2010) conducted a prospective cohort study on 52 malnourished Teresian children, ranging in age from three to seven years old, in order to ascertain the effect vitamin A supplements would have on their T cell counts. The researchers found that there was a statistically significant increase in CD4 CD8 positive T cells after supplementation, implying a proliferation of functional T cells prepared to fight off infections (Paiva, et al., 2010). Furthermore, the research team behind Sakai, et al. (2006) found that mice that were kept on a strict protein free diet had significantly reduced levels of circulating CD8 T cells and responded poorly to DNA vaccines, when compared to a control group. B cells, too, seem to be dependent on vitamin A in order to properly function, according to Molrine, et al. (1995) In their experiment, they deprived C.B.-17 mice of all sources of vitamin A for seven days and then exposed them to the tetanus toxoid in order to determine the effect the deprivation would have on the mice' immune response. It was discovered that the vitamin A deficient mice had a statistically significant stunted response to the toxoid vaccine, producing very little IgG compared to the control group, implying impaired B cell action (Molrine, Polk, Ciamarra, Phillips, & Ambrosino, 1995).

III. METHODS

This research paper primarily undertook a qualitative approach, curating and summarizing the most cited published findings and presenting them in a matter that allows for logical conclusions to be drawn; only information found in peer-reviewed published articles was presented. To find these articles, PubMed was searched using the search terms "malnutrition + immunodeficiency," "malnutrition + disease," "malnutrition + food desert," and "malnutrition + food swamp." Though each research team sought to verify different hypotheses, some of them did employ similar methodology. All researchers used mean \pm standard deviation when presenting their data as well as the Student t test to determine the statistical significance between groups except for Twining, et al. (1996) who used either a one way or two-way ANOVA, as they had four experimental groups, and Bowman, et al. (1990) and Morris, et al. (2011) who both used a Tukey A post hoc test, and Jimenez, et al. who used a Wilcoxon – Mann – Whitney test; all research groups used a P value of ≤ 0.05 or less to define statistically significant. All researchers using mice or rats for their experiments had different starvation/deprivation protocols that varied by age of mice/rats, length of starvation/deprivation, and re-nourishing procedures depending on the goal of the experiment and breed of mouse/rat. White blood cell count was determined by blood smear.

IV. RESULTS

Circulating within a control group of 12 mice, there was found to be an average leukocyte count of 2790 (/mm³), neutrophil count of 189 (/mm³), eosinophil count of 9.9(/mm³), lymphocyte count of 2425 (/mm³), and monocyte count of 59 (/mm³). The protein-malnourished experimental group, on the other hand had a leukocyte count of 1008 (/mm³), neutrophil count of 33.7 (/mm³), eosinophil count of 1.4 (/mm³), lymphocyte count of 974 (/mm³), and monocyte count of 18.1 (/mm³) – all were lower when compared to control, and all were found to be statistically significant to $P \leq 0.05$ using a Student t test (Cunha, et al., 2013). Furthermore, mean bone marrow cellularity ($\times 106$ /femur) was found to have decreased to 7.14 in protein starved mice from 12.5 in the control but increased to 8.42 after reintroducing protein into the mice' diet – the discrepancy of results between the three group were found to be statistically significant to $P \leq 0.001$ using a Tukey's test (Morris, et al., 2011).

The effect of vitamin A on neutrophil activity can be seen in the experimental results attained by Jimenez, et al. (2010) in which they found the mean number of beads a vitamin A deficient child's neutrophils could engulf increased from 3.1 to 6.3 after 30 days of vitamin A supplementation – statistically significant to $P < 0.05$ using a Wilcoxon – Mann – Whitney test.

Regarding T cells, Paiva, et al. found that amongst 631 preschool children, whom were found to be vitamin A deficient, average CD4 levels rose from 1181.38 (cells/mm³) to 1434.38 (cells/mm³) while mean CD8 positive cells rose from 773.38 (cells/mm³) to 1022.13 (cells/mm³) after supplementation, both of which were found to be statistically significant increases to $P < 0.05$ through a paired t test (Paiva, et al., 2010). Furthermore, Sakai, et al. (2006) found that protein deficient mice only had a mean 0.7% increase in CD4 positive cells, when exposed to a DNA vaccine, compared to a control group of well-fed mice who experienced a 2.8% increase on average – statistically significant to $P < 0.05$ using a Student's t test.

Furthermore, in the experiments conducted by Molrine, et al. (1995), it was discovered that when vitamin A deficient mice were exposed to the anti-tetanus toxin on average only 3.75 (µg/dl) of tetanus toxin complementary IgG was found to be in their blood serum compared to 148 (µg/dl) of IgG that was found amongst the control – statistically significant to $P = 0.005$ by two tailed t test. It was also discovered that the average total serum IgG was significantly lower in the group of vitamin A deficient mice, revealing 1,660 (µg/dl) IgG compared to the control which presented with an average of 3,240 (µg/dl) IgG (Molrine, Polk, Ciamarra, Phillips, & Ambrosino, 1995).

V. DISCUSSION

As per the literature, it is evident that nutrition plays a major role in determining the efficacy of one's immune system. The prevailing dietary factors seem to be protein and vitamin A, however, there may be other dietetics that play a more prominent role that have yet to be researched. While the direct mechanism behind vitamin A's involvement in the immune system has yet to be determined, many believe that it plays a role in thymic tissue proliferation and regulation, bone marrow cell population regulation, and white blood cell propagation (Huang, Qi, Brand, & Zheng, 2018). Similarly, the specific mechanism behind protein's involvement in the immune system has not yet been defined, but it is suspected that the during periods of protein deficiency the body simply does not allocate as many resources to the proliferation of the immune system as would be needed to maintain adequate protection from intruders (Cunha, et al., 2013). The effect of malnutrition on the immune system is still an area of active research and it is now suspected that vitamins A, D, C, E, B6, and B12 in addition to micronutrients folate, zinc, iron, copper, and selenium are all needed to support health white blood cells (Gombart, Pierre, & Maggini, 2020). More research is certainly needed, though, in order to determine the precise effect specific nutrients have on different parts of the immune system as well as their mechanisms of action.

The reason why this topic may not be receiving as much attention as it deserves could lie in the fact that world hunger has been on a decline since the year 2000, according to Global Hunger Index. However, while it may be true that most of the world is not necessarily hungry nowadays, it is still very possible that entire populations are not receiving all the dietary nutrients needed to lead a healthy life. Specifically, people living in food swamps and food deserts who have abundant access to fast-food but little to no means of acquiring healthy alternatives. According to Goodman, et al. (2020) those living in food deserts and food swaps are primarily below the poverty line, usually African American, and eat little to no fruit, vegetables, or seafood while at the same time consume large quantities of fats, sugars, and grains. This points to an inequality that only compounds the disparity already plaguing those in the lower socioeconomic status.

In fact, a study conducted by Hanson, et al. (2018) in order to determine the serum vitamin A levels in pregnant woman living in food deserts found that living in a food desert meant one had statistically significant ($P = 0.02$) greater odds of being vitamin A deficient compared to someone who lived within walking distance of an affordable grocery store. Furthermore, they discovered that those that classified themselves as non-white were 2.17 times more likely to experience the deficiency compared to their white counterparts, after accounting for confounding variables (Hanson, et al., 2018).

The reason research in this area is particularly important is because it may point to another factor by which to determine the inequality faced by those living in lower socioeconomic neighborhoods. It is no secret that those residing in poorer districts have a lower life expectancy and experience a greater number of morbidities per person, on average (Chetty, et al., 2016). Furthermore, during the recent Covid-19 pandemic, it was found that those living below the poverty line, and African Americans in particular, died at a greater rate than their age and sex matched more affluent counterparts (Abedi, et al., 2020). It is not a coincidence that Abedi, et al.'s (2020) test subjects are those that live in food deserts/ swamps; while there may not be a direct correlation it certainly is something that should be considered a confounding factor.

There has been no single research study in which someone has attempted to identify each of the micronutrients missing from the diet of one living in a food desert/swamp. However, literature published up to this point does indicate that those living in such areas are not receiving all the nourishment their body needs and, at the same time, are suffering from higher infection rates, morbidities, and mortalities. Further research could prove this to be a direct correlation and potentially lead to new policy implementation that would benefit the lives of many and bring us one step closer to bridging the disparity between the different socioeconomic classes.

VI. CONCLUSION

Both vitamin A and protein, amongst other micronutrients, play a major role in regulating one's immune system. More research is needed in this area, but it appears that those living in food deserts and food swamps are malnourished in some, if not all, of these micronutrients which may be contributing to the increased incidence and burden with which these communities experience various diseases.

REFERENCES

- [1] Abedi, V., Olulana, O., Avula, V., Chaudhary, D., Khan, A., Shahjouei, S., . . . Zand, R. (2020). Racial, Economic, and Health Inequality and COVID-19 Infection in the United States. *Journal of Racial and Ethnic Health Disparities*, 1-11.
- [2] Black, R. E., Allen, L. H., Bhutta, Z. A., Caulfield, L. E., Onis, M. d., Ezzati, M., . . . Rivera, J. (2008). Maternal and Child Undernutrition. *Lancet*, 243-260.
- [3] Bourke, C. D., Berkley, J. A., & Prendergast, A. J. (2016). Immune Dysfunction as a Cause and Consequence of Malnutrition. *Cell Press*, 386-389.
- [4] Bowman, T., Goonewardene, L. M., Pasatiempo, A. M., Ross, A. C., & Taylor, C. E. (1990). Vitamin A Deficiency Decreases Natural Killer Cell Activity and Interferon Production in Rats. *Nutrition and Immunology*, 1264-1273.
- [5] Chetty, R., Steiner, M., Abraham, S., Lin, S., Scuderi, B., Turner, N., . . . Cutler, D. (2016). The Association Between Income and Life Expectancy in the United States, 2001-2014. *JAMA*, 1750-1766.
- [6] Cunha, M. C., Lima, F. d., Vinolo, M. A., Hastreiter, A., Curi, R., Borelli, P., & Fock, R. A. (2013). Protein Malnutrition Induces Bone Marrow Mesenchymal Stem Cells Commitment to Adipogenic Differentiation Leading to Hematopoietic Failure. *Plos One*, 1-12.
- [7] Gombart, A. F., Pierre, A., & Maggini, S. (2020). A Review of Micronutrients and the Immune System-Working in Harmony to Reduce the Risk of Infection. *Nutrients*, 236-242.
- [8] Goodman, M., Thomson, J., & Landry, A. (2020). Food Environment in the Lower Mississippi Delta: Food Deserts, Food Swamps and Hot Spots. *International Journal of Environmental Research and Public Health*, 1-13.
- [9] Grover, Z., & Ee, L. C. (2009). Protein Energy Malnutrition. Elsevier, 1055-1068.
- [10] Hanson, C., Schumacher, M. V., Lyden, E., Su, D., Furtado, J., Cammack, R., . . . Weishaar, K. (2018). Fat-soluble vitamins A and E and health disparities in a cohort of pregnant women at delivery. *Journal of Nutritional Science*, 1-8.
- [11] Huang, Z., Qi, Y. L., Brand, D., & Zheng, S. G. (2018). Role of Vitamin A in the Immune System. *Journal of Clinical Medicine*, 258-274.
- [12] Hughes, S. M., Amadi, B., Mwiya, M., Nkamba, H., Tomkins, A., & Goldblatt, D. (2009). Dendritic Cell Energy Results from Endotoxemia in Severe Malnutrition. *The Journal of Immunology*, 2818-2826.
- [13] Ibrahim, M. K., Zambruni, M., Melby, C. L., & Melby, P. C. (2017). Impact of Childhood Malnutrition on Host Defense and Infection. *American Society for Microbiology*, 919-971.
- [14] Jimenez, C., Leets, I., Puche, R., Anzola, E., Montilla, R., Parra, C., . . . ia-Casal, M. (2010). A single dose of vitamin A improves haemoglobin concentration, retinol status and phagocytic function of neutrophils in preschool children. *British Journal of Nutrition*, 798-802.
- [15] Molrine, D. C., Polk, D. B., Ciamarra, A., Phillips, N., & Ambrosino, D. M. (1995). Impaired Human Responses to Tetanus Toxoid in Vitamin A-Deficient SCID Mice Reconstituted with Human Peripheral Blood Lymphocytes. *INFECTION AND IMMUNITY*, 2867-2872.
- [16] Morris, H. J., Carrillo, O. V., Llauro, G., Alonso, M. E., Bermúdez, R. C., Lebeque, Y., . . . Venet, G. (2011). Effect of starvation and refeeding on biochemical and immunological status of Balb/c mice: an experimental model of malnutrition. *Informa Healthcare*, 438-446.
- [17] Nájera, O., González, C., Cortés, E., Toledo, G., & Ortiz, R. (2007). Effector T lymphocytes in well-nourished and malnourished infected children. *Clinical and Experimental Immunology*, 501-506.
- [18] Najera, O., Gonzalez, C., Toledo, G., Lopez, L., Cortes, E., Betancourt, M., & Ortiz, R. (2001). CD45RA and CD45RO isoforms in infected malnourished and infected well-nourished children. *Clinical and Experimental Immunology*, 461-465.
- [19] Nayak, K., Sethi, A., Aggarwal, T., Chadda, V., & Kumar, K. K. (1989). Bactericidal Power of Neutrophils in Protein Calorie Malnutrition. *The Indian Journal of Pediatrics*, 371-377.
- [20] Netea, M. G., Wijmenga, C., & O'Neill, L. A. (2012). Genetic variation in Toll-like receptors and disease susceptibility. *Nature Immunology*, 535-542.
- [21] Nicholson, L. B. (2016). *The immune system*. Portland Press, 275-301.
- [22] Paiva, A. d., Rondó, P. H., Vaz-de-Lima, L. R., Oliveira, C. d., Ueda, M., Gonçalves-Carvalho, C., & Reinaldo, L. G. (2010). The Impact of Vitamin A Supplementation on the Immune System of Vitamin A-deficient Children. *Int. J. Vitam. Nutr. Res.*, 188-196.
- [23] Rikimaru, T., Taniguchi, K., Yartey, J., Kennedy, D., & Nkrumah, F. (1998). Humoral and cell-mediated immunity in malnourished children in Ghana. *European Journal of Clinical Nutrition*, 344-350.
- [24] Sakai, T., Mitsuya, K., Kogiso, M., Ono, K., Komatsu, T., & Yamamoto, S. (2006). Protein Deficiency Impairs DNA Vaccine-Induced Antigen-Specific T Cell but Not B Cell Response in C57BL/6 Mice. *J Nutr Sci Vitaminol*, 376-382.
- [25] Salimonu, L., Ojo-Amaize, E., Williams, A., Johnson, A. K., Cooke, A., Adekunle, F., . . . Wigzell, H. (1982). Depressed Natural Killer Cell Activity in Children with Protein-Calorie Malnutrition. *CLINICAL IMMUNOLOGY & IMMUNOPATHOLOGY*, 1-7.
- [26] Twining, S. S., Schulte, D. P., Wilson, P. M., Fish, B. L., & Moulder, J. E. (1996). Vitamin A Deficiency Alters Rat Neutrophil Function. *American Society for Nutritional Sciences*, 558-565.



10.22214/IJRASET



45.98



IMPACT FACTOR:
7.129



IMPACT FACTOR:
7.429



INTERNATIONAL JOURNAL FOR RESEARCH

IN APPLIED SCIENCE & ENGINEERING TECHNOLOGY

Call : 08813907089  (24*7 Support on Whatsapp)