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Full Length Research Paper

Histological signs of oligodendroglioma in the brain of rats fed with diet containing yaji: The complex Nigerian suya meat sauce

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***Yaji* is a complex mixture of groundnut cake powder, additives, spices and salt. The production and consumption of *Yaji* is not regulated despite the excitotoxic, apoptotic and tumourigenic potentials of some of its active principles. This has been the basis for several scientific investigations aimed at determining the effect of *Yaji* on different body organs. The present study on the brain is intended to determine its tumourigenic potentials. Eighteen weeks old white albino rats of an average weight of 170 g were used for this study. They were divided into eight groups (A - H) of three subgroups each. Subgroup 1, 2 and 3 represents experimental periods of 2, 4 and 6 weeks respectively. Group A rats served as control and were fed with normal feed (growers mash) only, while groups B - H served as the test groups and were fed with normal feed plus graded levels of *Yaji* (B, 10%; C, 20%; D, 30%; E, 40%; F, 50%; G, 60%; and H, 70%). At the end of the respective experimental periods, test group rats were sacrificed in order to harvest the brain tissues for tissue processing. We observed that the stained brain tissue micrographs from test group F3 (6 weeks; 50%) presented features that were histologically similar to those of oligodendroglioma and the incidence appears to be high dosage/duration dependent. This result implicates the active principles in *Yaji* and suggests that at high doses, *Yaji* is capable of inducing brain tissue damage as well as tumour formation.**

Key words: *Suya*, *Yaji*, additives, spices, oligodendroglioma.

INTRODUCTION

Yaji is the meat sauce for a Nigerian meat delicacy called *Suya.* It is a complex mixture of groundnut cake powder, additives, spices and salt (Okonkwo, 1987). According to Igene and Mohammed (1983), "*Suya* is a popular, traditionally processed, ready to eat Nigerian meat product, which may be served or sold along streets, in club houses, at picnics, parties, restaurants and within institu-tions". Omojola et al. (2008) described it as "one of such intermediate moisture products that is easy to prepare and highly relished", while Uzeh et al. (2006) identified it as "a mass consumer fast food whose preparation and sales along the streets, are usually not done under strict hygienic condition".

Historically, *Yaji* was named after a 14th century Hausa ruler called "Yaji (meaning the 'hot one')" (Betumiblog, 2006). The spices in it are ginger, cloves, red pepper, and black pepper (Nwaopara et al., 2004). These spices contain gingerol (Witchtl, 2004), eugenol (Krishnaswamy and Raghuramulu, 1998), capsaicin (Collier et al., 1965), and piperine (McGee, 2004) as active principle respectively. The other three constituents-white maggi (or Ajinomoto), salt and groundnut cake powder, contain monosodium glutamate (Omojola, 2008), sodium chloride (Carson et al., 1998) and oil (Fageria et al., 1997) as active principle respectively. This indicates that *Yaji* is a complex combination of ingredients with active principles that are potentially harmful when consumed in excess (Southgate, 1993).

Unfortunately, the production and consumption of *Yaji* is yet to be regulated and this has been the basis for several scientific investigations aimed at determining the effect of *Yaji* on body organs (Nwaopara et al., 2004; 2007a; 2007b; 2008a; 2008b; 2009). Some of the histolo-

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gical findings on the Pancreas (Nwaopara et al., 2004), Liver (Nwaopara et al., 2007b), and Kidney (Nwaopara et al., 2008), suggest that an excessive consumption *Yaji* can induce pancreatic, liver and kidney damage. The present study therefore, is intended to determine the effect of *Yaji* on the brain as there are reports that some the active principles in *Yaji* like capsaicin, piperine and monosodium glutamate, have excitotoxic, apoptotic and tumourigenic potentials (Choi, 1988; Blaylock, 1997; Lipton and Rosenberg, 1994; Whetsell and Shapira, 1993; Olney, 1989; Olney et al., 1997; Sugimoto et al., 1998; Ankarcrona et al., 1998; Martin et al., 2000; Bellamy, 2008; Rothstein and Brem, 2001).

MATERIALS AND METHODS

The substance of study

Normally, the production of *Yaji* is not standardized as regards what the quantities in combination should be. In this study however, all the constituents were measured to determine the quantities in a given measure of *Yaji*. A weighing balance manufactured by Denver Company USA (Model 200398.1REV.CXP-3000) was used for the measurements. The constituents were purchased at Aduwawa Cattle market, Benin City, Edo State, Nigeria, and subsequently mixed together in powdery forms as directed by the dealers. The measured quantities include: Ajinomoto (150 g), black pepper (30 g), clove (39 g), ginger (78 g), and groundnut cake powder (230 g), red pepper (22 g), and salt (100 g). The total weight of these constituents summed up to 649 g.

The subjects/substance administration

Eighteen weeks old white albino rats of an average weight of 170 g were used for this study. They were divided into eight groups (A - H) of three subgroups (n = 5) each. Subgroup 1, 2 and 3 represents experimental durations of 2, 4 and 6 weeks respectively. Group A (A1, A2 and A3) served as the control, while the groups of B - H (B1 - H1; B2 - H2; and B3 - H3) served as the test groups. Group A rats were fed with normal feed (growers mash) only. The feed was purchased from Bendel Feeds and Flour Mills (BFFM), Ewu, Edo State, Nigeria. Test groups B1 - H1, B2 - H2 and B3 - H3 were fed with growers mash from the same source plus graded quantities of *Yaji* (B, 10%; C, 20%; D, 30%; E, 40%; F, 50%; G, 60%; and H, 70%) for 2, 4 and 6 weeks respectively.

The total daily feeding allowance for each experimental group was 30 g, while the feeding allowance per rat was 6 g. Test groups B (10%) received 3 g of *Yaji* daily (0.6g per rat), C (20%) received 6g of *Yaji* daily (1.2 g per rat), D (30%) received 9 g of *Yaji* daily (1.8 g per rat), E (40%) received 12 g of *Yaji* daily (2.4 g per rat), F (50%) received 15 g of *Yaji* daily (3 g per rat), G (60%) received 18g of *Yaji* daily (3.6 g per rat), and H (70%) received 21 g of *Yaji* daily (4.2 g per rat).

Feeding pellets were produced by mixing appropriate quantities of *Yaji* and feed with sprinkles of water to form a paste, which was then split into bits and allowed to dry under the sun.

Tissue processing

The animals in subgroups 1, 2 and 3 were sacrificed after two weeks, four weeks and six weeks respectively. The brain tissues harvested from the groups were immediately fixed in formaldehyde to prevent autolysis and putrefaction. Tissue processing was done according to standard procedures (fixation, dehydration, impregnation, embedding, sectioning and staining with Haematoxylin and Eosin) described by David (2004). The micrographs of the relevant stained sections were subsequently taken with the aid of a light microscope (at magnification x40).

RESULT

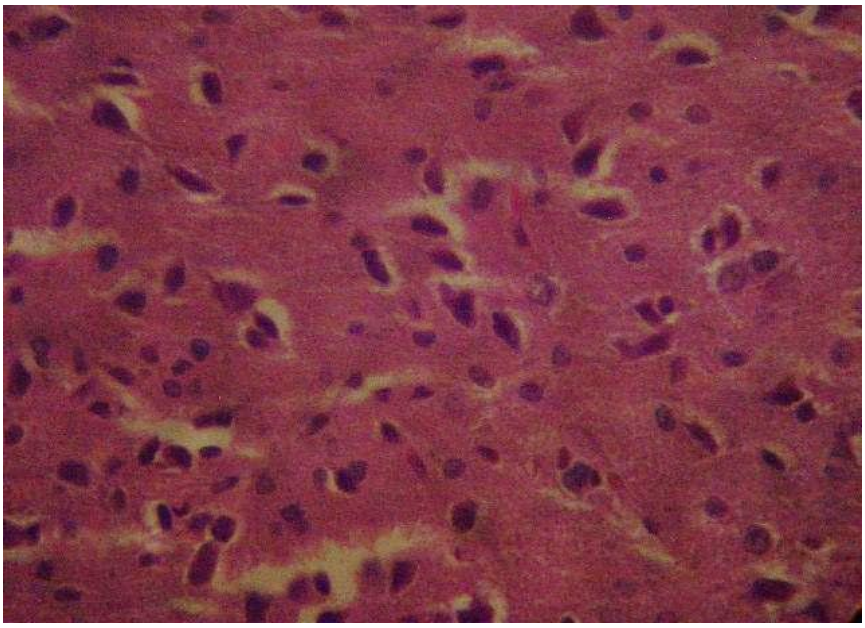
The test group micrographs presented several neurodegenerative changes. These changes include vacuoletions, eosinophilic cells, pyknotic nuclei, and gliosis. Of particular importance is the unique observation in the micrographs of test group F3 (6 weeks; 50%) as represented by Plates A and B showing proliferation of oligodendrocytes with vacuolated cytoplasm Plates A and B as well as vascular networks and haemorrhage Plate B. Both of these features are consistent with oligodendroglioma.

DISCUSSION

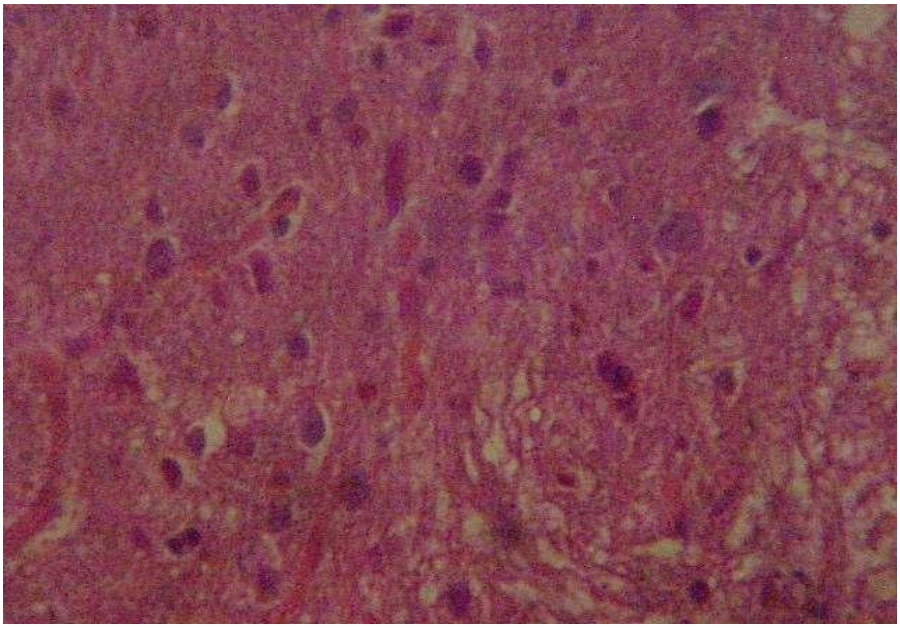
The histological features observed in test group F3 (6 weeks; 50%) are similar to those described for oligodendroglioma (tumours derived from oligodendrocytes), which, according to Stevens et al. (2007), is commonly seen in the cerebral hemispheres and composed of homogenous sheets of cells with uniform rounded nuclei, a vacuolated cytoplasm forming a 'halo' around each nucleus and a network of finely branching small blood vessels. Usually, tumours exert their harmful effects by growing into vital structures or by causing swelling of the brain around the tumour resulting in secondary compression of vital structures (Stevens et al., 2007). Like all other infiltrating gliomas, oligodendroglioma has a very high rate of recurrence and cannot be completely resected because of their diffusely infiltrating nature (Wikipedia, 2009).

Our findings seem to implicate the active principles in *Yaji*, as there is evidence that MSG is tumourigenic (Bellamy, 2004; Rothstein and Brem, 2001). There is evidence also, that excitotoxic destruction facilitates brain tumor growth. This by implication, indicate that the combined influence of all the excitotoxic elements in *Yaji* such as MSG, capsaicin and piperine is one factor that might as well account for the observed tumour formation. Of course, the excitotoxicity of MSG is well known (Espinar et al., 2000; Rothstein and Brem, 2001; Urena-Guerrero et al., 2003) and capsaicin administration causes degeneration of neurons (Jansco et al., 1977; Ritter and Dinh, 1993; Chard et al, 1995; Wood, 1993). Piperine also, is said to be cytotoxic (Unchen et al., 1998) as it promotes DNA damage (Piychatuwarat et al., 1995), which is itself, a significant trigger for apoptosis (selective cell damage).

One might ask if diet types have been linked to tumour or cancer. The answer is in affirmative as there are



Plates A. (Brain H&E x40**)** showing severe distortions in cellular architecture. Note the proliferated oligodendrocytes with vacuolated cytoplasm, which are consistent with oligodendroglioma.



Plates B. (Brain H&E x40) showing severe distortions in cellular architecture. Note the proliferated oligodendrocytes with vacuolated cytoplasm and the network of finely branching small blood vessels, which are consistent with oligodendroglioma.

scientific evidence showing that less than one percent of cancer deaths in industrialized nations are attributable to food additives and industrial products (Trichopoulos and Li, 1996). Dietary factor has also been estimated to account for about one third of cancer deaths in the United States (American Cancer Society, 2000; Ames et al., 1995; Doll and Peto 1981; Ries et al., 2000). Also, there is a report associating cancers of the breast, oral cavity (primarily in smokers), and liver with an excessive consumption of alcoholic beverages (International Agency for Research on Cancer, 1988; Willett, 2001).

High dosage is one other factor that might be responsi-

ble for the changes observed. In fact, a high proportion of all chemicals, whether synthetic or natural, can be “carcinogens” if administered at the maximum tolerated dose, primarily due to the effects of high doses on cell division and DNA damage (Butterworth, 1995; Ames and Gold, 1990). At 50% administration (15 g of *Yaji*), each test rat in group F3 received 3 g of *Yaji* daily signifying a daily consumption of 0.7 g of MSG, 0.14 g of black pepper and 0.1 g of red pepper. We observed that the dose levels of MSG, black pepper and red pepper in *Yaji* as administered to the test rats, far exceeds what the normal daily values should have been for a rat of an average weight of 170 g. This assertion is hinged upon a comparison with the acceptable daily doses for a man of 70 kg whose normal daily doses for MSG, black pepper and red pepper is 3 g, 359 mg and 120 mg respectively (Giacometti, 1979; Kindell, 1984; Vitamin Supplements Guide, 2006).

The import of this is that at high doses, *Yaji* is capable of inducing brain tissue damage as well as tumour formation in a manner that is likely dependent upon the concentration of those ingredients with excitotoxic and tumourigenic potentials in a given measure of *Yaji*. It is our opinion that there is a need to regulate the production and consumption of *Yaji*.

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REFERENCES

American Cancer Society (2000). Cancer Facts and Figures—2000. American Cancer Society, Atlanta, GA.

Ames BN, Gold LS, Willett WC (1995). The causes and prevention of cancer. Proc. Natl. Acad. Sci. USA 92, 5258–5265. http://socrates.berkeley.edu/ mutagen/ames.pnas3.html.

Ames BN, Gold LS (1990). Proceedings of the National Academy of Sciences USA 87, 7772-76.

Ankarcrona M, Dypbukt JM, Bonfoco E, Zhivotovsky B, Orrenius S, Lipton SA, Nicotera P (1998). Glutamate-induced neuronal death: A succession of necrosis or apoptosis depending on mitochondrial function. Neuron 15: 961-973.

Bellamy M (2008). Eliminate MSG to greatly improve your health. Retrieved 10th October, 2009 from http://nursinglink.monster.com/news/articles/6427-eliminate-msg-to-greatly-improve-your-health.

Betumiblog (2006). "Kuli-Kuli is and buy". Retrieved. Betumi 2006.

Retrieved 27th July, 2008 from www.betumi.com.

Blaylock LR (1997). Excitotoxins, the taste that kills. Santa Fe, NM., Health press pp. 248-254.

Butterworth B, Conolly R, Morgan K (1995). Cancer Letters 93: 129-46.

Carson SH, Osborn JW, Wyss JM (1998). Hepatic innervations chronically elevate arterial pressure in wistar-Kyoto rats. Hypertension 32: 46-51.

Chard PS, Bleakman D, Savidge JR, Miller RJ (1995). Capsaicin induced neurotoxicity in cultured dorsal root ganglion neurons: Involvement of calcium activated proteases. Neuroscience 65(4): 1099-1188.

Choi D (1988). "Glutamate neurotoxicity and diseases of the nervous system" Neuron 1: 623-34.

Collier HO, McDonald-Gibson WJ, Saeed SA (1965). Letter: Stimulation of prostaglandin biosynthesis by capsaicin, ethanol and tyramine, J. physiol. (Lond) 179(2): 248-62.

David K (2004). Tissue Preparation. Retrieved 20th October, 2009 from http://www.siumed.edu/~dking2/intro/tissprep.htm.

Doll R, Peto R (1981). The Causes of Cancer. Oxford University Press, New York.

Espinar A, García-Oliva A, Isorna EM, Quesada A, Prada FA, Guerrero JM (2000). Neuroprotection by melatonin from glutamate-induced exocitoxicity during development of the cerebellum in the chick embryo: J. Pineal Res. 2: 81 - 88.

Fageria NK, Balgar VC, Jones C (1997). Growth and mineral nutrition of field crop 2nd Ed. Marcel Dekker, Inc, New York 1001K p. 494.

Giacometti T (1979). Free and bound glutamate in natural products. In L.J Filer, Garattini S, Kare MR et al. (ed). Glutamate Acid: Advances in Biochemistry and Physiology. New York: Raven press pp. 25-34.

Igene JO, Mohammed ID (1983). Consumers’ attitudes towards ‘suya’ meat product. Ann. Borno. 1. Retrieved 20th October, 2009 from http://www.pjbs.org/pjnonline/fin512.pdf.

International Agency for Research on Cancer (1988). Alcohol Drinking. IARC, Lyon, France.

Jancso G, Kiraly E, Jancso-Gabor A (1977). Pharmalogically induced selective degeneration of chemosensitive primary sensory neurons. Nature 270: 741-754.

Kindell SS (1984). Studies on selected genotoxic and toxic properties of piperine. Ph.D Thesis Drexel University, Philadelphia P.A.

Krishnaswamy K, Raghuramulu N (1998). Bioactive phytochemicals with emphasis on dietary practices. Indian J. Med. Res. 108:167-81.

Lipton S, Rosenberg P (1994). "Excitatory amino acids as a final common pathway for neurologic disorders" NEJM 330: 613-622.

Martin LJ, Sieber FE, Traystman RJ (2000). Apoptosis and necrosis occur in separate neuronal populations in hippocampus and cerebellum after ischemia and are associated with differential alterations in metabotropic glutamate receptor signaling pathways. J. Cereb Blood Flow Metab. 20: 153-167.

McGee H (2004). On food and Cooking: The Science and Lore of the Kitchen. New York, Scribner pp. 427-429.

Nwaopara AO, Anyanwu LC, Oyinbo CA, Anaikot IC (2004). The Histological Changes In Pancreas Of Wister Rats Fed With Diets Containing Yaji (Local meat Sauce). J. Expt. Clin. Anat. 3(2): 44 – 47.

Nwaopara AO, Odike MAC, Ikhuoriah TA, Anyanwu LC (2007a). Potential Health Hazards in Yaji: The Complex Suya Meat Sauce. Medilink J. 8(74): 34-38.

Nwaopara AO, Odike MAC, Inegbenebor U, Adoye MI (2007b). The Combined effects of excessive consumption of ginger, clove, red pepper and black pepper on the histology of the liver. Pak. J. Nutr. 6(6): 524-527.

Nwaopara AO, Odike MAC, Inegbenebor U, Nwaopara SO, Ewere GI (2008a). A comparative study on the effects of excessive consumption of ginger, clove, red pepper and black pepper on the histology of the Kidney. Pak. J. Nutr. 7(2): 287 – 291.

Nwaopara AO, Odike MAC, Inegbenebor U, Nwaopara SO, Ekhoye EI (2008b). A comparative study on the effects of excessive consumption of ginger, clove, red pepper and black pepper on the histology of the Heart. Electronic J. Biomed. 3: 61-64.

Nwaopara A, Anibeze C, Akpuaka F, Nwaopara S (2009). Antimicrobial Potentials of Yaji-Spices: The Constituents of a Complex Nigerian Suya Meat Sauce Inducing Histological Investigations. The Internet J. of Alternative Med. 6(2). Retrieved from http://www.ispub.com/journal/the_internet_journal_of_alternative_medicine/volume_6_number_2_22/article/antimicrobial_potentials_of_yaji_spices_the_constituents_of_a_complex_nigerian_suya_meat_sauce_inducing_histological_investigations.html.

Okonkwo TM (1987). "About Suya and Yaji". J. Food Agric. 1(1) 51.

Olney J (1989). "Glutamate, a neurotoxic transmitter". J. Child Neurol. 4:218-26.

Olney JW, Wozniak DF, Farber NB (1997). "Excitotoxic neurodegenera-tion in Alzheimer's disease". Arch. Neurol. 54: 1234-1240.

Omojola AB (2008). Yield and organoleptic characteristics of Suya (an

intermediate moisture meat) prepared from three different muscles of a matured bull African J. Biotech. 7(13): 2254-2257.
Piychaturawat P, Kingkaeohoi S, Toskulkao C (1995). Potentiation of carbon tetrachloride hepatotoxicity by piperine. Drug Chem. Toxicol. 18(4): 333-44.
Ries LAG, Eisner MP, Kosary CL, Hankey BF, Miller BA, Clegg L, Edwards BK eds (2000). SEER Cancer Statistics Review, 1973–1997. National Cancer Institute, Bethesda, MD.
Ritter S, Dinh TT (1993). Capsaicin induced degeneration in rat brain and retina, in capsaicin in the study of pain. Academic Press, San Diego pp. 1-26
Rothstein JD, Brem H (2001). Excitotoxic destruction facilitates brain tumor growth. Nat. Med. 7: 994 – 995.
Southgate DAT (1993). Spices and Beverages in Essential Human Nutrition and Dietitics. Edinburgh, Churchill Livingstone pp. 325 - 334.
Stevens A, Lowe SJ, Young B (2007). Wheaters Basic Histopathology (a colour atlas and Text). 4th ed. London, Churchill Livingstone pp. 1 – 34: 88 – 89.
Sugimoto T, Xiao C, Ichikawa H (1998). Neonatal primary neuronal death induced by capsaicin and axotomy involves an apoptotic mechanism. Brain Res. 807(1-2): 147-54.
Trichopoulos D, Li FP (1996). What causes cancer? Scientific American 275(3): 80-88 (1996).
Unchen S, Saito H, Nisli Yama H (1998). Death of cerebella granules neurons induced by piperine is distinct from that induced by low potassium Neurchem Res. 23(1): 97-102.
Urena-Guerrero ME, Lopez-Perez SJ, Beas Zaratel B (2003). Neonatal Monosodium glutamate treatment modified glutamic acid decarboxylase activity during rat brain postnatal development: Neurochem. Int. 42(4): 269-276.
Uzeh RE, Ohenhen RE, Adeniji OO (2006). Bacterial Contamination of Tsire-Suya, a Nigerian Meat Product. Pak. J. Nutr. 5(5): 458-460.
Vitamin Supplements Guide (2006). Cayenne pepper. Retrieved 27 October, 2009 from http://www.vitamins-supplements.org/herbal-supplements/cayenne-pepper.php.
Wikipedia (2009). Oligodendroglioma. Retrieved 27th October, 2009 from http://en.wikipedia.org/wiki/Oligodendroglioma.
Willett WC (2001). Diet and cancer: One view at the start of the millennium. Cancer Epidemiol. Biomarkers Prev. 10: 3–8.
Whetsell W, Shapira N (1993). "Biology of disease, neuroexcitation, excitotoxicity and human neurological disease." Lab Invest; 68: 372-87.
Witchtl M (2004). Herbal Drugs and Phytopharmaceuticals 3rd ed. Boca Raton FL: CRC press pp. 653-656.
Wood J (1993). Capsaicin in the study of pain. London, San Diego, Academic Press Inc (ISBN 10: 01276855X).