EXPERIMENTAL SUPPORT FOR PRINCIPLE OF DYNAMIZATION, LAW OF SIMILARS AND FOR NEW SCIENCE OF ULTRAMICROXENOPATHY

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INTRODUCTION

Homoeopathy has stood the test of time as an *art* of healing but has not yet established as a science. One of us (RRS) has been searching[1-6] for the scientific bases of Homoeopathy for the past more than a decade. He has published new theories to explain the effects and mechanisms of the processes of dynamization[5], for the law of similars[6], and to propose the new science of Ultramicroxenopathy.[4, 6] The present paper reports the results of experimental investigations, on the treatment of alloxan induced diabetes in rats, which were designed by him to test important aspects of these theories.

MATERIALS AND METHODS

Animals used in these experiments were male albino wistar rats with 180 to 340g body weight. Alloxan ($C_4H_2N_2O_4$. $2H_2O$, M.W. 160.09, purity 98.5%) was obtained from Loba Chemie.

Urine sugar was estimated with qualitative Benedict solution. Quantitative estimations of blood sugar were made with Folin Wu method[7] by, taking 0.05ml blood sample from tail vein and 0.2ml standard solution and by measuring O.D. at 620nm wave length in Rockman Model 35 Spectrophotometer. The accuracy of our technique was about $\pm$ 10% in the region around 100 mg/dl.

Good quality lactose, double glass distilled water and dehydrated ethyl alcohol were used (by AA) to prepare potentized 30c alloxan in alcohol by Hahnemann's method[8]. The processes of dynamization were omitted for preparing dilutions of alloxan upto 100^{30} fold in alcohol.

Only those rats were included in these studies for whom the urine sugar was nil and blood sugar within normal range (80 to 120mg/dl). For inducing diabetes, intraperitonial injections, of 100 to 150mg per kg body weight alloxan dissolved in distilled water, were given after overnight fast. To minimize mortality due to hypoglycemic shock glucose was added to drinking

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water for two days after the alloxan injections. Significant levels of glycosuria and hyperglycemia were taken to indicate the establishment of diabetes mellitus. Diabetic rats were divided into groups of (a) untreated diabetes, (b) diabetes treated with potentized 30c alloxan, and (c) diabetes treated with 100^{30} fold diluted alloxan or with potentized 30c streptozotocin so that the body weight and levels of blood sugar were comparable or matching in different groups. Rats with higher levels of blood sugar were preferably included in the treatment group than in the untreated group so that the effect of treatment could be shown more strikingly and the mortality could be reduced.

To avoid the 'alcohol effect', if any, the alcoholic 30c alloxan and 30c streptozotocin were diluted 10fold in distilled water and succussed before administration to rats of treated group as intraperitonial injections in 0.1ml volume. Similarly 100^{30} fold diluted alloxan in alcohol was also diluted 10fold in distilled water before administration. The untreated group was given 'sham treatment' with 0.1ml injections (I.P.) of 10 fold diluted alcohol in water.

RESULTS AND DISCUSSION

Preliminary studies during March through May 1981 were undertaken with the limited objective to see whether alloxan induced diabetes could be treated with potentized alloxan at a dilution where the probability of finding a molecule in the dose was less than one. The results of these investigations are shown in Table 1. In the 'mild to moderate diabetes' sub-groups B, the starting blood sugar after alloxan injection ranges between 145 and 198 mg/dl for both the treated and untreated rats. After 18 days' treatment with potentized 30c alloxan the blood sugar returns to values within the normal range of 80 to 120 mg/dl whereas in the untreated subgroup B, it remains higher (156-175) than even the starting (154-163) values for 4 rats and for the 5th falls from 198 to only 178 mg/dl. In the subgroups A for the severe diabetes', the starting range of 304 to 509 mg/dl falls to values within 83 to 124 mg/dl after 30 days' treatment. In the untreated subgroup A, the blood sugar continues to be significantly high (286 to 380 mg/dl). Curative effect of treatment with 30c alloxan is therefore clear and significant.

Table 2 contains the results of extended studies with larger objectives: (i) to repeat the experiments of preliminary studies, (ii) to see the effect of dynamizations, (iii) to see if alloxan induced diabetes could be treated with potentized streptozotocin—another diabetogenic agent, (iv) to see the effect of potentized 30c alloxan on 'chronic' diabetes. The blood sugar in the group treated with 30c alloxan returns from the starting range of 179 to 501 mg/dl to that of 86 to 94.4 mg/dl on D-51, after 44 days' treatment. Further treatment of 23 days upto D-67 does not lower the blood sugar significantly. The blood sugar remains within the normal range of 87.7 to 100 mg/dl upto D-144, i.e. for 77 days of observation after stopping the treatment on D-67.

TABLE NO. 1

LEVELS OF RANDOM BLOOD SUGAR (MG/DL) BEFORE AND AFTER INJECTION ON 8.3.81 OR DAY-ZERO (D–0) OF DIABETOGENIC ALLOXAN IN UNTREATED AND TREATED GROUPS OF DIABETIC RATS—RESULTS OF PRELIMINARY STUDIES

		Before alloxan injection	Days after alloxan injection							
			D–4 (12.3.81)	D–16 (24.3)	D–20 (28.3)	D–26 (3.4)	D–33 (10.4)	D–39 (16.4)	D–58 (5.5)	D–60 (7.5.81) (fasting)
Untreated alloxan-induced diabetes	A	101.8	301	350	353	337	330	286	231	200
		89.8	408	401	404	402	395	380	—	—
		102.9	304	308	318	320	310	315	—	—
	B	101.2	163	183	180.6	175				—
		92.3	145.4	162	158.6	NB				
		110.3	154	158.6	NB	156	—	—	—	153
		101	198	189	186	178				
Alloxan diabetes treated with 30c potentized alloxan from day—8(16/3) to day-38 (15/4/81)	A	108.9	317	133.3	144	128	NB	NB	124	—
		97	509	264.8	225	175	138.8	144.6	96	—
		90.4	457.9	290	315	356	154.4	164	—	—
		94.1	304	320	330	180	106	108	83	70.3
	B	89.8	145.8	94.4	106	113				
		107.8	163	117	NB	82				
		96.9	186	117.3	110	112				
		101.8	198	106	NB	113				

N.B. No blood sample from the tail vein could be taken out.

TABLE NO. 2

LEVELS OF RANDOM BLOOD SUGAR (MG/DL) BEFORE AND AFTER INJECTION ON 20/8/81 OR DAY-ZERO (D–0) OF DIABETOGENIC ALLOXAN IN UNTREATED AND VARIOUSLY TREATED GROUPS OF DIABETIC RATS—RESULTS OF EXTENDED STUDIES

	Before alloxan injection	Days after injection of diabetogenic alloxan										
		D–7 (27.8.81)	D–19 (8.9)	D–25 (14.9)	D–39 (28.9)	D–51 (10.10)	D–55 (14.10)	D–69 (28.10)	D–90 (18.11)	D–104 (2.12)	D–116 (14.12)	D–144 (11.1.82)
Untreated alloxan-induced diabetes	92.6	389	450	435	425	NB	416	440	NB	386	320	killed*
	85.8	300	325	335	325	315	320	302	282	267	250	dead
	75.8	189	258	278	286	283	290	300	290	277	244	234
	86.4	302	NB	330	296	294	274	268	NB	257	254	227
	90.2	202	218	NB	261	NB	247	269	dead	—	—	—
Alloxan diabetes treated with 30c potentized alloxan from day–7 (27.8) to day–67 (26.10)	85	501	475	389	180	87.1	89.7	88.6	90.5	95.6	100	dead
	87.6	360	350	302	160	86.2	88.2	86.8	93.0	87.6	90.7	dead
	77.8	207	220	202	140	88.6	99.7	98.6	91.0	88.8	93.5	98.6
	86.9	295	276	203	145	94.4	92.3	92.7	85.6	91.2	87.7	killed*
	75.8	179	178	140	116	94.4	91.8	93.4	98.3	88.4	93.35	92.8
Alloxan diabetes treated with 100^{30} times diluted alloxan 27.8 to 14.9; with 30c streptozotocin 17.9 to 14.10; with 30c alloxan 19.10 to 14.12	96.2	489	658	647	557	542	575	511	326	dead	—	—
	105.6	315	325	339	257	237	287	300	261	241	241	213
	98.6	207	NB	229	218	NB	221	251	210	196	171	killed*
	75.8	279	307	NB	239	NB	267	268	216	197	207	197
	86.0	202	210	225	221	226	224	269	225	211	197	117

N.B. No blood sample from tail vein could be taken.
* killed on 21.12.81 for histological studies.

The potentized 30c alloxan, therefore, seems to have cured, the alloxan induced 'acute' diabetes, permanently.

During 18 days' treatment from D-7 to D-25 with 100^{30} fold diluted alloxan, the blood sugar continues to rise and shows no tendency to fall whereas the potentized 30c alloxan during the same period not only arrests the rise but also shows a fall. The probability of finding a molecule of alloxan[5] in the dose of 30c potency or 100^{30} dilution is the same, namely 1 in 10^{41}. The effects of dynamizations on the diluent medium are therefore shown strikingly. The mechanism of resonant promotion of lone pair electrons in the OH groups of the diluent medium during the processes of dynamizations has been explained separately[5].

The potentized 30c streptozotocin during its 27 days' treatment from D-28 to D-55 does not lower the blood sugar. This shows that alloxan and streptozotocin, as expected and known, induce diabetes by acting on separate sites and different molecules of biological importance, as explained earlier[6].

The potentized 30c alloxan lowers the blood sugar to normal values in less than 44 days if the treatment starts on D-7 but fails to do so even in 56 days if the start of treatment is delayed up to D-60. This shows that during these 67 days from the day of diabetes induction some such near irreversible reaction(s) and/or tissue change(s) have taken place that the 'chronic' diabetes is not as readily amenable to treatment with 30c alloxan as the acute diabetes is. Thus chronic diabetes is different from the acute diabetes not only in the duration of disease but also basically at molecular levels.

THEORETICAL EXPLANATIONS

(i) *Principle of Dynamization*: These experiments have clearly demonstrated that while potentized 30c alloxan is curative for acute diabetes the 100^{30} fold dilution of alloxan has no such properties. The dynamic processes of potentization have so changed the diluent medium that it manifests the chemical and biological properties of alloxan[5] itself to effect a cure of the acute diabetes through competitive exchange[6] (see also below). This has been postulated[5] to occur through resonant promotion of lone pair electrons in the OH groups of the diluent medium.

(ii) *Law of similars*: Let the diabetogenic alloxan molecule X act on the biomolecule M to produce the complex MX:

$$M + X \rightleftharpoons MX \qquad (1)$$

The molecule of the resonantly promoted diluent medium D_x in the potentized 30c alloxan acts curatively through the process of 'competitive exchange' according to eqns. (2a) to (2d).

$$MX + D_x \rightleftharpoons MD_x + X \qquad (2a)$$

$$MD_x \rightleftharpoons M + D_x \qquad (2b)$$

$$X \longrightarrow \text{biologically eliminated} \qquad (2c)$$

$$D_x \xrightarrow{\text{de-promoted}} D \longrightarrow \text{metabolized} \qquad (2d)$$

The alloxan induced 'acute' diabetes is amenable to treatment with 30c alloxan because the diabetogenic alloxan molecule X and the resonantly promoted diluent molecule D_x both have the same number of valency electrons with same characteristic energies and hence efficient for 'competitive exchange'. In the 'chronic' diabetes, however, the biomolecule M and hence the complex MX have changed thereby lowering the curative efficiency of 30c alloxan. Similarly, the 30c streptozotocin is not that efficient as 30c alloxan in treating alloxan induced diabetes since the biological molecules involved are different.

(iii) *Ultramicroxenopathy*: These experiments support the theoretical foundations of Ultramicroxenopathy[4, 6] that many a disease is caused by some pathogenic xenobiotic(s) acting on some biomolecules of vital importance and that ultramicro quantities of the same or of that xenobiotic which elicits most similar xenobiotic response in healthy subjects[3, 6] can remove the primary cause of disease through 'competitive exchange' according to the law of similars. This is in confirmation and extension of the principles of Homoeopathy —the eternal monument of Hahnemann's strength and courage of conviction!

CONCLUSIONS

(i) These controlled animal experiments have shown that potentized 30c alloxan acts curatively in alloxan induced acute diabetes although the probability of finding one molecule of alloxan in the dose[5] is about 1 in 10^{41}.

(ii) The 100^{30} fold simple dilution of alloxan exercises no curative action although the probability of finding a molecule of alloxan in the dose is the same as for 30c alloxan.

(iii) The principle of dynamization[5], the law of similars[6] and the bases of the new science of Ultramicroxenopathy[4, 6] are supported by these experiments.

(iv) The need for revising contemporary scientific theories and for proposing new theories to explain the homoeopathic laws and principles as already done[1-6] by one of us (RRS) is justified.

REFERENCES

1. Sharma, R. R. (1977): 'A Unified Theoretical Approach to Homoeopathy, Immunology and Raja Yoga and Its Consequences', *Transactions of 32nd Inter. Hom. Med. Congr.*, New Delhi, pp. 73-85 (*Ind. J. Hom.*, June-July 1980).

2. Sharma, R. R. (1979): 'Scientific Bases of Homoeopathy: The Concept of Vital Force as Molecular Mechanisms Basic to Profound Homoeostatic State', THE HAHN. GLEAN., 46 (2), 52-63 (*Ind. J. Hom.*, Aug.-Sept. 1980).

3. Sharma, R. R. (1979): 'Scientific Bases of Homoeopathy: Homoeopathic Drug Proving and Materia Medica as New Science of Xenobiology', THE HAHN. GLEAN., 46 (3), 103-107 (*Ind. J. Hom.*, Oct. 1980).

4. Sharma, R. R. (1979): 'Scientific Bases of Homoeopathy: Operational Laws of Homoeopathy as Comprising New Science of Ultramicroxenopathy', THE HAHN. GLEAN., 46 (4), 156-165 (*Ind. J. Hom.*, Nov. 1980).

5. Sharma, R. R. (1982): 'Scientific Bases of Dynamization', THE HAHN. GLEAN., 49 (1), 14-24.

6. Sharma, R. R. (1982): 'Scientific Bases of Homoeopathy, Xenobiology, Ultramicroxenopathy, Unified Therapeutics, and More'. THE HAHN. GLEAN., 49 (2), 51-61.

7. Folin, O. and Wu H. (1920): *J. Biol. Chem.*, 41, 367. (Details in H. Varley's *Practical Clinical Biochemistry*, 4th ed., Arnold-Heinemann 1969, Indian ed. 1976, New Delhi, pp. 86.

8. Hahnemann, S. (1842): *Organon of Medicine*, 6th ed., 2nd Indian ed. (1968), Calcutta, Roy Singh & Co.

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mortality. Survival was therefore considered to be unaffecld by the intake of coloring [C.I. Food Red No. 5]."

Homoeopaths have frequently complained that their results are not accepted by allopathic physicians because they contradict such implicit assumptions as the monotonicity rnle. Thus it is a consolation to know that allopathic investigators sometimes reject their own results for the same reason.

While Kon expressly limits his conclusions to long-term chronic exposure, this whole area is unexplored, and his analysis casts doubt on the monotonicity assumption in respect of other dose-response relationships.

(*To be continued*)

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