# STUDIES

## THERAPEUTIC SPOT AND REGIONAL DERMABRASION IN STABLE VITILIGO

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Therapeutic spot or regional dermabrasion was carried out at 64 sites located over cosmetically unimportant hairy areas (52) and non-hairy areas (12) in 15 cases of stable vitiligo. Lesions were individually dermabraded first with either electric or manual dermabraders till pinpoint bleeding occurred. They were further deep demabraded to an appropriate depth manually. On healing, all the 64 sites were further treated with PUVA or PUVASOL. Out of 52 hairy sites 46 sites (88.5%) showed total pigmentation, 2 showed partial pigmentation and in 4 sites there was no pigmentation. Out of 12 non-hairy sites, 10 sites (88.3%) showed only perilesional hyperpigmentation along the borders with no change in the remaining 2 sites. Side effects were superficial scarring (5) and hypopigmentation (4) which improved over next 6 months. Complications were deep scarring (3) and secondary infection (1). Therapeutic spot or regional dermabrasion is useful alone or in combination with PUVA/PUVASOL for treating stable vitiligo (hairy areas).

**Key Words : Stable vitiligo, Dermabrasion, Hairy areas**

## Introduction

There are many cases of vitiligo who either fail to respond or only partially respond to the medical line of treatment. Stable vitiligo is a term coined for such cases where in addition, the disease is inactive and no new patch has develop in past 2 years. These cases of stable vitiligo may be treated by surgical methods like tattooing,[1] organ cultured foetal skin allografting,[2] epidermal culture grafting,[3] melanocyte culture grafting,[4] epidermal grafting by suction blister technique,[5] thin thiersch's split skin grafting[6] or miniature punch grafting.[7] Miniature puch grafting has also been combined with medical modality like PUVA or PUVASOL for faster results.[7] Tsuji and Hamada[8] have reported successful repigmentation in stable vitiligo with topical 5% fluorouracil cream under occlusion after dermabrading vitiligo areas.

Dermabrasion is an extensively used surgical modality for treating many cutaneous problems from acne scars to removal of tattoo or tumours to the scar revision.[9-14] Therapeutic spot or regional dermabrasion[10] is a dermabrasion carried out only of the areas where the specific lesions are located. It has been successfully used for the treatment of actinic keratosis,[10] lichen simplex chronicus,[11] papular lichen amyloidosis,[12] scar revision,[13] tatto removal[14] etc. Apart from routine facial dermabrasion it is possible to dermabrade practically every portion of the body[11] by altering the methodology according to the anatomical feature of the region.

When spot dermabrasion was used for removing mismatched tatto done in a stable vitiligo case, on healing an interesting finding was noted. Not only the tattoo pigment was removed but the wound created by dermabrasion while healing induced perifollicular pigmentation in the hairy areas and perilesional pigmentation at the border in

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the non-hairy areas (personal observation). This phenomenon of re-koebnerization was also in contrast to that of koebnerization observed during medical treatment of active vitiligo. Hence a few cases of stable vitiligo were subjected to spot and regional dermabrasion. To the best of author's knowledge, the use of dermbrasion alone or along with PUVA, PUVASOL in stable vitiligo has not previously been reported.

## Materials and Methods

Fifteen patients (9 women and 6 men, age range 19-42 years) all with partially arrested generalised vitiligo participated in this study. Total 64 different sites were treated as follows : lower extremities 33, upper extremities 17, trunk 8, scalp 4 and nape of the neck 2. The lesions varied in size from ½" to 18" with either round, oval, linear or any geographical pattern.

Out of 64 sites, 12 sites were located in the non hairy areas like ankle, wrist, etc and 52 sites were located in the hairy areas as mentioned earlier. Only those hairy sites were selected where the majorty (70% or more) of the hair in the individual lesion were black. All the cases had received PUVA/PUVASOL, local or oral steroids etc in the past 2-7 years and were refractory to the medical line of treatment.

Haemogram, blood sugar and routine urine were done of all patients. Bleeding tendencies were ruled out. Their BCG scars or old scars were examined for keloidal tendency and informed consent was obtained.

Main instruments used were either locally manufactured non expensive Dr Manekshaw's manual metallic dermabraders of various sizes or more convenient motor driven wire brushers or coarse diamond fraise mounted on electric hand machine and revolving at 5000 to 30,000 rpm (average 15,000 rpm).

After surgical preparation and isolation, local anaesthesia was given with 1% xylocaine with adrenaline intradermally and subcutaneously so as to balloon out the lesions in 13 out of 15 cases. In 2 cases where both the lower extremities were involved extensively, they were subjected one by one to regional dermabrasion under general anaesthasia of IV Ketlar - ketamine hydrochloride combined with local tumescent anaesthesia. Tumescent anaesthesia fluid consisted of (1) 1 litre of normal saline, (2) 50 ml of 1% xylocaine with 1:1000 adrenaline and (3) 12.5 ml of soda bicarbonate 1 mEq. The lesions were dermabraded individually first with either electrical or manual metallic dermabraders upto the junction of mid and deep papillary dermis. They were further deep dermabraded manually for better depth control upto the junction of upper and mid reticular dermis. Perimeter of the lesions were then feathered gently into the surrouding normal skin. Important landmarks were standardised while dermabrading. Various landmarks and the corresponding level of depth in the skin are as follow :

1. Loss of colour - Epidermis

2. Multiple punctate bleeding - superficial papillary dermis.

3. Change in bleeding pattern - junction of mid and deep papillary dermis. Bleeding vessels are fewer, larger and bleed more rapidly.

4. Faint whitish pink lines - Junction of papillary and reticular dermis.

5. Breaks in these lines - Junction of upper and mild reticular dermis. This indicates that dermabrasion has reached optimum depth and it should be terminated at this

level or else deep permanent scarring will occur.

Two broad types of dermabrasions were carried out in this study. SUPERFICIAL being upto the junction of mid and deep papillary dermis and DEEP being upto the junction of upper and mid reticular dermis. Standardized landmarks as per the depth were followed accordingly. Either type of demabrasions were carried out as per the condition of the skin overlying the vitiligo lesion. They were as follows :

1. In majority of sites (58) where overlying skin was not lichenified superficial dermabrasion was carried out.

2. In 8 sites where the overlying skin surface was lichenified and rough due to previous PUVA/PUVASOL therepy deep dermabrasion was carried out.

All the wounds were covered with double layer of framycetin tulle followed by gauze pieces, roller bandage and elastocrepe bandage. Patients were asked to raise the dermabraded part, rest for 24 hours and return for change of dressing after 48 hours and then subsequently every 3rd to 4th day for a period of 2 weeks. Usually the oozing and draining was observed upto first two dressings. All patients were covered with broad spectrum antibiotic, antinflammatory and analgesics for first few days. Small oral dose of steroid 5-10 mg of prednisolone for first 2-3 days resulted in lesser oozing and soaking of bandage.

Post surgically PUVA/PUVASOL was given in all the cases for 1 to 3 months. Fifteen patients were followed up for 6 months, 6 for 1 year and 2 for 1½ years.

## Results

There was considerable soaking of dressing in all 64 sites due to serous discharge in the first 24-48 hours necessitating change of dressing. Superficially dermabraded sites (58), stopped oozing after first change of dressing and they healed normally in one to two weeks with no scarring. In other 8 sites which were deep dermabraded soaking gradually reduced over next 3-4 days and disappeared after one week and the sites healed with erythema (8), hypopigmentation (4) and superficial (8) or deep (3) scarring over next 2-3 weeks or more.

Out of 52 hairy sites, 46 sites (88.5%) showed total repigmentation, 2 showed partial pigmenatation and in 4 sites there was no pigmenation. The sites healed with erythematous shiny skin and perifollicular pigmenation in 1-2 weeks. All the patients were put on PUVA or PUVASOL for the next 1-3 months. During this time, the pigmented island spots coalesced together to cover the whole area. The pigmenation was intense and dark in colour. It gradually reduced in intensity and matched with surrouding skin colour over next 3-6 months.

Out of 12 non-hairy sites, 7 sites (58%) showed perilesional hyperpigmenation uniformally all along the border. Three sites (25%) showed perilesional hyperpigmentation but in patchy manner along the border. Thus ten sites (83.3%) out of 12 showed perilesional hyperpigmentation. In the remaining two sites no such change was noticed. In four sites, there were isolated 2-3 pigmented spots scattered in the centre of the lesion. The pigmentation of the border and of the central isolated pigmented spots became intense and dark in colour when patients were put on PUVA/PUVASOL. There was marginal migration of pigment from these areas into the centre or the surrouding vitiligenous areas in the first 1-3 months. It remained static without further advancement after that.

All the deep dermabraded 8 sites healed with erythema, hypopigmenation and superficial scarring. Erythema gradually diminished over next one month and disappeared by 3 months. Superficial scarring in the form of smooth shiny skin improved cosmetically over next 6 months disappearing completely in 3 sites, however, in 5 sites it remained permanently and this thus remains a major side effect of this procedure. Hypopigmentation gradually improved over 3-6 months disappearing in 4 sites, but remained present in varying degree as another side effect at 4 sites.

Complications occurred at 3 sites. Out of these in 2 sites where the dermabrasion was carried out unevenly to the depth of reticular dermis, the wounds healed with atrophic parchment like superficial and deep scars with persistant erythema, hypopigmentation and at places depigmentation. This persisted even at the end of 1 year. In remaining one site, there was secondary bacterial infection due to improper local wound care or premature discontinuation of oral antibiotics on the part of the patient. However with improved local wound caring and with long term oral cephalosporins there was delayed wound healing (5-6 weeks) with superficial and deep scarring.

## Discussion

Spot or regional dermabrasion was found to be usuful in stable vitiligo when combined with occlusion of the wounds with 5-fluorouracil cream.[8] In this study it was found to be useful in hairy areas of stable vitiligo. The pigmentation was enhanced when combined with PUVA/PUVASOL. The procedure is carried out under local anaesthesia on OPD basis. One can treat areas of any size with this method by carrying out single or multiple sessions of spot dermabrasions. Where the lesions cover a very large area such as full extremity, regional dermabrasion can be carried out under general anaesthesia combined with local tumescent anaesthesia. The procedure is quicker and simpler as compaed to the other surgical modalities. There is immediate near total perifollicular pigmenatation on healing. The perifollicular pigment islands coalesce together, become intense and this process of pigmentation is enhanced in the next 1-3 months on further treatment with PUVA or PUVASOL. As it is used therapeutically in cosmetically non important areas, no special expertised training is required.

In this study, there was total repigmentation in 88.5% of hairy sites and there was perilesional hyperpigmentation at the borders in 83.3% of non-hairy sites. There was partial pigmenation in two hairy sites and three non-hairy sites, however, at two sites of each totalling 4 sites, no pigmentaiton was seen. In all the sites which were deep dermabraded, the most common major side effect seen was superficial scarring at 5 sites which improved cosmetically but did not disappear over next 6 months to 1 year. In dermabrasion re-epithelization takes place from remnants of dermal appendages - sebaceous glands, hair follicles and sweat glands.[11,15] Skin overlying upper or lower extremities or trunk is not as rich as facial skin in dermal appendages and hence the epithelization is slow and wound heals with superficial scarring at places. Also during the deep dermabrasion some of the deeper appendageal structures either must have got partially planed away or traumatised. Hypopigmentation observed at 4 sites like the hypopigmentation observed after routine facial dermabrasion[10,11,15,16] persisted upto 6 months to 1 year. In routine facial

dermabrasion this post surgical hypopigmentation usually lasts upto 3-6 months. Here it was delayed and persisted. Again the reason being the facial skin has rich reservoir of melanocytes due to presence of many hair follicles than the upper or lower extremities or trunk and also face is more exposed to normal UV radiations of sunlight. Also deep dermabrasion must have traumatised the deeper structures as mentioned earlier. Both these side effects were not observed after superficial dermabrasion (58 sites). They were only observed where the overlying skin surface was rough and lichenified and hence were deep dermabraded (8 sites). These side effects may be avoided if the overlying rough and lichenified skin surfaces are treated with emollients prior to dermabrasion.

Various anatomical landmarks corresponding to the related level of depth in the skin while dermabrading were standardized in this study. These are important from Indian point of view where Fluro Ethyl refrigerant cryospray[11,16] for surface local anaesthesia in not available and one has to demabrade under local or tumescent anaesthesia of mainly xylocaine. Also the skin is not stiff and hard as with cryospray and hence one has to dermabrade soft pliable skin. These standardized landmarks not only hold true for the sites dermabraded in this study, but also for the facial dermbrasion in the treatment of acne scars. Optimum depth of demabrasion is junction of upper and mid reticular dermis.[9] [11,16] Importance of dermabrading accurately to the various minimum and maximum depths is well brought out in this study.

It is well known that those with active vitiligo remain at the risk of koebnerization from trauma, surgical injury, sunburn etc. during therapy and after it has been discontinued.[17] In this study, reverse koebnerization was seen. Here, trauma of dermabrasion induced pigmentation in stable vitiligo lesions.

It is known that sometimes the inflammation induced by topical psoralens with UVA gives rise to blister formation, resulting in post inflammatory pigmentation.[18] Melanocytes can be induced to proliferate by stimulus such as ultra violet light or during an inflammatory process.[19] In this study, both the factors - inflammatory phase of the wound healing and further stimulation with ultra violet light and systemic psoralens have stimulated pigmentation. Repigmentation in stable vitiligo requires proliferation and migration of melanocytes from the reservoir into the depigmented skin. The melanocytes will migrate only a few millimeters from the pigmented edge towards the centre. The hair follicles in the centre are the main reservoirs of the melanocytes required for repigmentation.[20]

In dermabrasion re-epithelization takes place from remnants of dermal appendages - sebaceous glands, hair follicles and sweat glands.[11,15] Observation that the dermabraded hairy areas first healed with perifollicular pigmentation in this study indicates that the follicular reservoir population of melanocytes migrated to the epidermal surface during wound healing after dermabrasion and further centrifugally propagated around the hair follicle. In the non-hairy areas, the dermabraded areas healed with perilesional hyperpigmentation at the borders with migration of this pigment for few millimeters towards the centre, thus indicating that the peripheral epidermal melanocytes got stimulated during surface epithelization. However, histopathological studies are needed to confirm this.

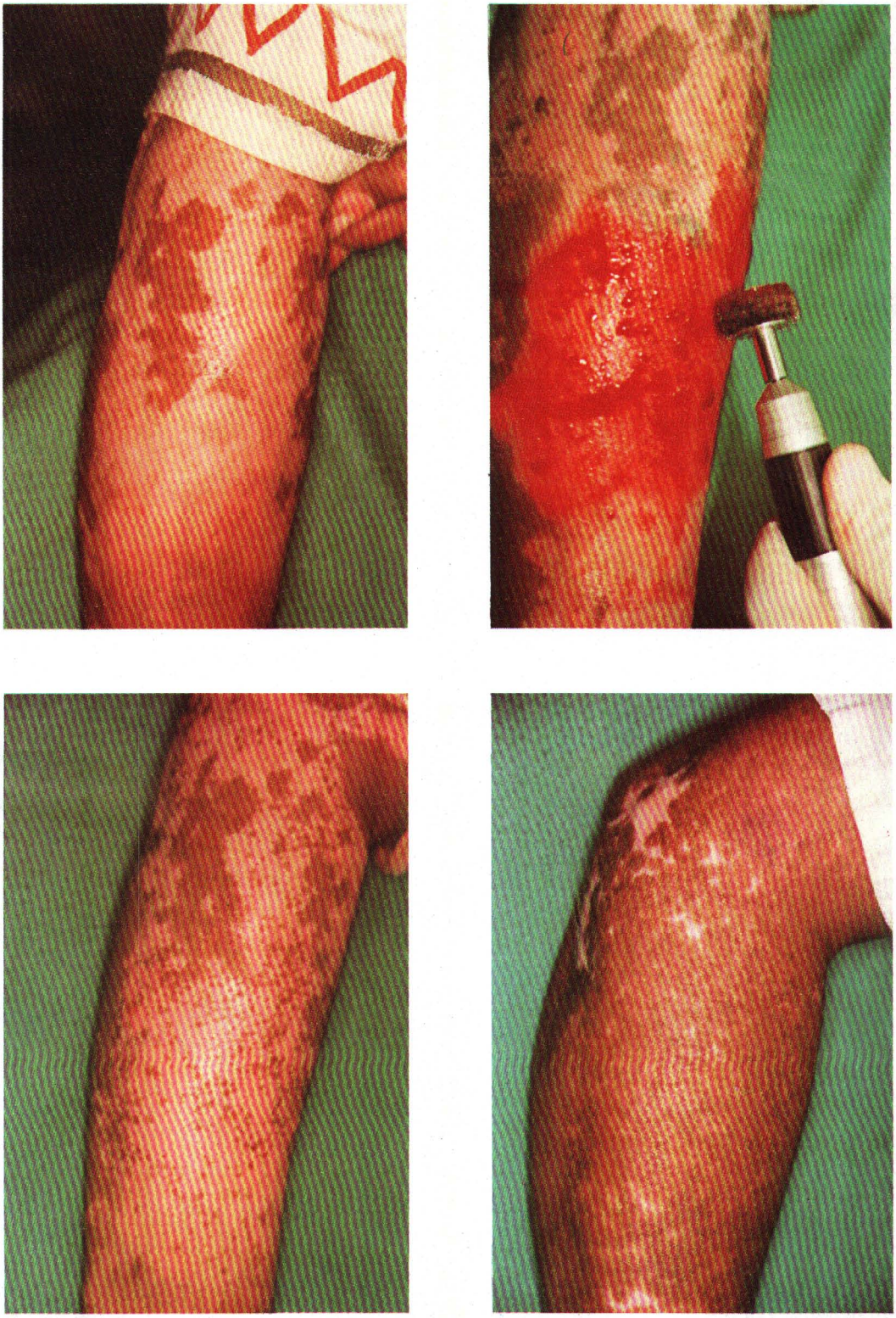
Tsuji and Hamada[8] lightly dermabraded the epidermis of vitiligo lesions applied 5% fluorouracil cream for 7 to 9 days under occlusion till the wound eroded. The authors state the fluorouracil probably induces division of follicular melanocytes which migrate to the surface after epithelization of the treated site.[8] Here, dermabrasion alone could have been responsible for repigmentation and fluorouracil may have only acted as an irritant and prolonged the healing phase of the dermabraded area. In the present study, the perifollicular pigmentation enhanced, intensified and spread further after PUVA/ PUVASOL bringing out the importance of combination of PUVA or PUVASOL with the surgical modality of spot dermabrasion in stable vitiligo of hairy areas.

Various cells of the body participate in the inflammatory and proliferative phases of wound healing by releasing many growth factors. Two such growth factors - endothelial growth factor and fibroblast growth factor are known to be mitogenic for the melanocytes.[20] Whether these factors were released and played any role in the pigmentary process by stimulating the follicular reservoir of melanocytes in this study needs to be investigated.

This method will have application in refractory stable vitiligo cases where majority of the patches have black hair. It can also be used to treat the stable vitiligo which has 25% to 50% of the hair black. Here, once the areas get partially pigmented after demabrasion, one can then use another surgical modality like miniature punch grafting in the remaining non pigmented areas to achieve the desired end results. In the non-hairy areas, one can repeatedly abrade the perilesional border to advance the migrating pigment zone. This may be used specially at the dorsi of the fingertips where other surgical method[7] though useful is tedious and more time consuming. This method can also be used in those cases of vitiligo where the lesions have been stabilized, are not spreading and are showing slow response after 3-6 months of medical line of treatment. It will not only act as an adjuvant to medical line of treatment, but will also cut down the total treatment period.

## References


1. Halder RM, Pham HN, Breadon JY, et al. Micropigmentation for the treatment fo vitiligo. J Dermatol Surg Oncol 1989; 15: 1092-4.
2. Gokhale BB, Tawade YV, Bharatia PR, et al. Use of organ culture foetal skin in allografts in treatment of resistant vitiligo. Ind J Dermatol Venereol Leprol 1991; 57: 272-5.
3. Falabella R, Escobar C, Borrero I. Treatment of refractory and stable vitilgio by in-vitro cultured epidermal autografts bearing melanocytes. J Am Acad Dermatol 1992; 26: 230-6.
4. Falabella R. Grafting and transplantation of melanocytes for repigmenting vitiligo and other types of stable leukoderma. Int J Dermatol 1989; 28: 263-9.
5. Koga M. Epidermal grafting using the tops of suction blisters in the treatment of vitiligo. Arch Dermatol 1988; 124: 1656-8.
6. Behl PN, Bhatia RK. Treatment of vitiligo with autologous thin Thiersch's grafts. Int J Dermatol 1973; 12: 329-31.
7. Savant SS. Autologous miniature punch skin grafting in stable vitiligo. Ind J Dermatol Venereol Leprol 1992; 58: 310-4.
8. Tsji T, Hamada T. Topically administered fluorouracil in vitiligo. Arch Dermatol 1983; 119: 722-7.
9. Roenigk HH Jr. Dermabrasion for miscellaneous cutaneous lesions exclusive of scarring for acne. J Dermatol Surg Oncol 1977; 3: 322-8.
10. Epstein E. Dermabrasion for therapeutic purpose. In : Epstein E, Epstein E Jr , editors. Skin surgery. Philadelphia : Saunders, 1987 : 344-7.
11. Roenigh HH Jr. Dermabrasion. In : Roenigk RK, Roenigk HH Jr, editors. Dermatologic



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surgery principles and practice. New York : Marcel Dekkar 1988: 959-78.

12. Savant SS. Therapeutic regional dermabrasion in papular lichen amyloidosis of shins. Ind J Dermatol Venereol Leprol 1995; 61: 196-201.
13. Katz BE. Dermabrasion for scar revison. In: Roenigk RK, Roenigh HH Jr, editors. Surgical dermatology. UK : Martin Dunitz, 1993: 385-94.
14. Clabough W. Removal of tattoos by superficial dermabrasion. Arch Dermatol 1968; 98 : 515-21.
15. McGregor IA. Free skin grafts. In : McGregor AC, editor. Fundamental techniques of plastic surgery and their surgical applications. Edinburgh : Churchill Livingstone, 1989: 39-63.
16. Padila RS. Dermabrasion. In : Wheeland RG, editor. Cutaneous surgery. Philadelphia : Saunders, 1994: 479-90.
17. Moshe DB, Fitzpatrick TB, et al. Disorders of pigmentation. In : Fitzpatrick TB, Eisen AZ, et al, editors. Dermatology in general medicine. New York : McGraw Hill, 1987: 794-876.
18. Gokhale BB, Tawde YV, Dambre CM. Treatment of vitiligo. In : Gokhale BB, Tawade YV, Dambre CM, editors. Vitiligo a monograph India: Comprint, 1989: 122-3.
19. Boissy RE, Nordlund JJ. Biolgoy of melanocytes. In : Arndt KA, Robinson JK, Leboit PE, et al, editors. Cutaneous medicine and surgery. Philadelphia : Saunders, 1996; 1203-9.
20. Boissy RE, Nordlund JJ. Vitiligo. In : Arndt KA, Robinson JK, Leboit PE, et al, editors. Cutaneous medicine and surgery. Philadelphia : Saunders, 1996: 1210-8.